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EFFECT OF ID AND IM ADMINISTRATION OF A PRRSV MLV VACCINE IN PIGLETS ON APOPTOSIS-RELATED SERUM BIOMARKERS

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Introduction

PRRSV-infection can induce cell apoptosis both *in vivo* and *in vitro*. Soluble Fas-sFas/APO-1 is an inhibitor of apoptosis, while angiotensin II (AT-II) plays an essential role in the apoptosis. This study investigated the ID and IM administration of a PRRSV MLV vaccine in piglets on apoptosis-related serum biomarkers.

Material & Methods

The study included 104 piglets (2 weeks-wks of age) from a commercial PRRSV-positive pig farm (13x4 groupsx2 replicates); group A: IM vaccination with Porcilis®PRRS at 2 wks, group B: ID vaccination with Porcilis®PRRS at 2 wks, group C: ID placebo and group D: IM placebo. Blood samples were collected from the same 3 pigs/group/replicate at 4, 7 and 10 weeks of age. Sera were examined by qRT-PCR for PRRSV (type 1, 2) and by ELISA for sFas and AT-II.

Results

The qRT-PCR results for PRRSV at 4 wks were negative in all groups, at 7 wks only group A was negative and at 10 wks all groups were positive. No differences in sFas levels (pg/ml) were observed over time in vaccinated groups, while sFas levels increased in unvaccinated groups: C (4vs7wks:199.2±32.0vs329.3±35.4, 4vs10wks:199.2±32.0vs390.0±52.3, 7vs10wks:329.3±35.4vs390.01±52.3) and D (7vs10wks:271.8±76.7vs370.6±66.0). Significant differences among groups of sFas levels were noticed only at 10 weeks (A:293.9±49.8, B:281.7±56.8, C:390.0±52.3, D:370.6 ±66.0; AvsC, AvsD, BvsC, BvsD) and of AT-II at 7 weeks (A:0.26±0.5, B:3.44±6.1, C:0.55±1.0, D:17.6±18.4; AvsB, AvsD, BvsD, CvsD).

Discussion & Conclusion

In unvaccinated piglets, increased sFas levels reveal apoptotic suppression in comparison to vaccinated piglets. In the latter, vaccine-derived immunity limit the infection and may contribute to the reduced Fas expression, suggesting a weak induction of lymphocyte-mediated cytotoxicity. Finally, higher sFas levels were observed with ageing, possibly due to persistent PRRSV infection. The results suggest that AT-II maybe involved in the pathogenesis of PRRSV and especially in the induction of apoptosis in immune cells.