

INCIDENCE OF SEP-LIKE LUNG LESIONS AT SLAUGHTER BEFORE DURING AND AFTER EMPLOYING VACCINATION OF FATTENERS AGAINST *MYCOPLASMA HYOPNEUMONIA*

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Introduction

Age segregated rearing has considerably improved the health status of the pigs in Sweden. The recordings of swine enzootic pneumonia (SEP) at slaughter have been reduced from 18 % in 1994 to 2% in 2001 (1). Piglets are generally seronegative to *Mycoplasma hyopneumoniae* (*Mh*) at 25 kg bw, but seroconversion to *Mh* may take place during the fattening period (2, 3). Vaccinations of growers prior to seroconversion have been proven effective in preventing SEP in farrow-to-finish systems (2, 3). The aim of this study was to see if the load of *Mh* could be reduced in a specialised fattening herd by vaccination of pigs on arrival.

Material and Methods

Herd, Management and study groups: This study was performed in a specialised fattening herd with four closely located units. In each unit, 370 pigs were raised all in- all out. One unit was emptied, cleaned and disinfected every 4 week. Thus, the turnover time was 16 weeks and the average rearing time was 100 days (25 to 110 Kg bw). Since 1999, all pigs have been bought from one single piglet producer also employing age segregated production.

Group I comprised 13 batches of fattening pigs arriving to the herd from October 1999 to September 2000.

Group II comprised 34 batches of pigs vaccinated once against *Mh* (HYORESP[®]) on arrival to the herd from October 2000 to April 2003.

Group III comprised the first 28 batches of fatteners again not vaccinated against *Mh*. These pigs arrived to the herd from May 2003 to June 2005.

Lung lesions recorded at slaughter: The lungs of the pigs were inspected at slaughter according to rules set by the National Food Administration. To be registered as SEP, ongoing lesions of a certain magnitude had to be present in the lungs.

Blood sampling and *Mh* ELISA: From the 13 first batches in group III, blood was collected from 20 individually marked pigs on arrival and 12 weeks later. Serum antibodies to *Mh* was determined by an indirect ELISA-system previously described (4).

Results

All pigs appeared healthy on arrival, and no group medication of pigs was performed during the trial. Following vaccination, the prevalence of SEP-like lesions recorded at slaughter decreased from $13.8 \pm 7.5\%$ to $3.1 \pm 2.4\%$ ($p < 0.0001$, Figure 1). As the vaccinations ended, the incidence of SEP-like lesions registered was initially low but increased by time and was at the end of the study at the original level ($5.5 \pm 3.8\%$ during the first five batches, $9.0 \pm 2.5\%$ during batch 5-10; $8.2 \pm 5.2\%$ during batch 11-20 and; 15.9 ± 10.3 during batch 21-28). The pigs were generally seronegative to *Mh* on arrival to the herd. In the first three batches of group III all pigs remained

seronegative 12 weeks after arrival. Eighty-eight percent of the pigs in the fourth batch were seropositive at that time, and after that, the incidence of seropositive pigs ranged between 30 and 90% at that age (Figure 2).



Figure 1: The % of SEP-like lesions registered at slaughter (■ = group I; ○ = Group II; ▲ = Group III).

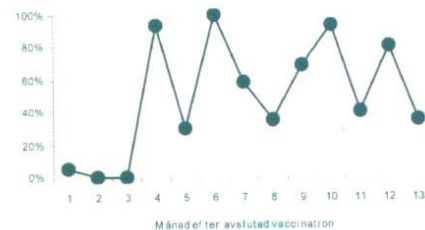


Figure 2: Prevalence (%) of pigs seropositive to *Mh* 12 weeks after arrival to the herd in batch 1-13 in group III.

Discussion and Conclusion

Vaccination of pigs on arrival was proven effective in preventing SEP and also improved growth. They were concluded economically profitable but laborious and therefore terminated. Four months later, seroreactors to *Mh* were common at the end of the rearing period and one year later the incidence of SEP-like lesions recorded at slaughter were at a similar level as prior to the vaccination period.

The obtained results suggest that *Mh* may establish during the fattening period despite employment of batch wise rearing and hygiene measures between batches. It should not be ruled out that seronegative pigs aged 10 weeks may carry low doses of the microbe which might contribute to this proliferation. Strategic vaccinations may be one way to control disease.

References

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4. Wallgren, *et al.*, (1992), Vet Microbiol 32: 363-374

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