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Vaccination against Atrophic Rhinitis: Effect on Clinical Symptoms, Growth Rate and Turbinate Atrophy

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Baalsrud, K. J.: Vaccination against atrophic rhinitis: Effect on clinical symptoms, growth rate and turbinate atrophy. Acta vet. scand. 1987, 28, 305–311. – Two combined Pasteurella multocida/Bordetella bronchiseptica whole cell bacterins were tested in 15 swine herds in which atrophic rhinitis (AR) was a problem, though of varying significance. One half of the sows/litters in each herd was vaccinated, the other half acting as a control group. Vaccination resulted in reduced clinical symptoms of AR and an average increase in weight gain of 2.4 % in the 8 herds in which toxigenic P. multocida had been isolated, which was not statistically significant. Clinical symptoms and turbinate atrophy were also observed in vaccinated animals in most herds.
The need for improved methods to diagnose the presence of toxigenic P. multocida is discussed. Vaccination programmes in which herds are selected on the basis of the presence of toxigenic P. multocida are proposed.

pigs; toxigenic Pasteurella multocida.

Introduction

Animals suffering from atrophic rhinitis (AR) may show clinical signs such as twisted or distorted snouts, frequent sneezing and nose bleeding. Symptoms may be slight, the disease perhaps even being subclinical. The overall health situation in the herd may, nevertheless, be affected. Individuals with clinical AR, or in which turbinate atrophy is found at slaughter, have been shown to gain less weight than clinically unaffected animals in some studies (*Flesjå* 1980, *Bäckström et al.* 1985), though not in others (*Pearce & Roe* 1967, *Straw et al.* 1984).

In Norway, AR is a notificable disease. As a consequence, sale of live animals from infected herds to others is restricted. Thus, the disease is of economic significance also in herds where only weak symptoms are observed clinically. This comes in addition to the losses due to clinical or subclinical disease.

A new aspect of the pathogenesis of AR in pigs has recently been presented (*Elling & Pedersen* 1985). These authors consider that the resistance of the nasal mucosa is weakened by non-infectious agents and by Bordetella bronchiseptica-infection, and toxigenic strains of Pasteurella multocida become established on the nasal mucosa, where they produce toxin. Various experiments with new vaccines containing toxigenic P. multocida have shown that vaccination programmes may help to reduce the effect of this multifactorial disease, though complete elimination of clinical and pathological symptoms by vaccination has not yet been achi-

eved (*Schuller et al.* 1980, *Schöss* 1980, *Wittowski et al.* 1980, *Baars et al.* 1982).
In the present work 2 different B. bronchiseptica/P. multocida-bacterins were used in a field study in which the effects of the vaccines on 1) clinical signs of AR, 2) weight gain and 3) turbinate atrophy at slaughter were investigated. The objective was furthermore to compare 2 vaccines for which different vaccination regimes were employed.

Materials and methods

Herds

Fifteen combined herds (i.e. herds containing both breeding sows and slaughter pigs) located in south-eastern and south-western Norway were selected for the experiment. All had been notified as AR herds and were subjected to restrictions imposed by governmental veterinary authorities. In 10 of the 15 herds, obvious clinical AR prevailed, with a number of pigs showing deviated and deformed snouts. In the remaining 5 herds, no pigs showed obvious clinical symptoms of AR, although turbinate atrophy was regularly observed at slaughter. No medication programmes were followed and no other special efforts were made in any of the herds for prophylactic or treatment purposes.
Altogether 1271 fattening pigs from 217 Norwegian Landrace sows were included in the experiment (Table 1). The animals were marked individually at 4–8 weeks of age in order to allow identification at slaughter. Half the sows were selected at random and placed in a vaccine group, the other half being left as an unvaccinated control group. The experimental period lasted until each sow in the herd had produced 1 litter taking part in the experiment. In herds in which only some of the slaughter pigs were included in the experiment, the pigs conserved were selected at random.

Vaccination programme

Two different vaccines were used. Vaccination regimes recommended by the manufacturers were followed.
Vaccine 1[1] contained piliated and pili-enriched B. bronchiseptica and toxigenic P. multocida (serotype D) with an aluminium hydroxide adjuvant. The bacterial strains used in the vaccine were isolated from pigs with AR. The sows were vaccinated 6 and 2 weeks prior to expected farrowing and, in addition, all piglets from these sows were vaccinated twice at the age of 1 and 3 weeks. The vaccine was administered intramuscularily with a dosis of 3 ml for sows and 2 ml for piglets. This vaccine was used in 6 herds.
Vaccine 2[2] contained 1 strain of B. bronchiseptica and 2 strains of P. multocida (both serotype D), one of them toxigenic. These strains also originated from pigs with AR. The vaccine contained a mineral oil adjuvant. Using this vaccine, the sows were vaccinated 10 and 4 weeks prior to expected farrowing. Each dosis of 2 ml was administered intramuscularly. The piglets were not vaccinated. This vaccine was used in 9 herds.

Bacteriological and toxinological investigations

Nasal swabs were taken from piglets before weaning, before any pigs in the herds were vaccinated. Between 4 and 36 swabs were collected from each herd and investigated bacteriologically for the presence of P. multocida, and isolates of the bacterium were serotyped and tested for toxin production as described previously (*Baalsrud* 1987).

[1] Experimental Pasteurella multocida/Bordetella bronchiseptica-bacterin, Schering corp., Omaha, Nebraska, USA.
[2] Nobi-Vac AR, Intervet, Boxmeer, Holland.

Clinical observations
Clinical examination of pigs in all herds participating in the investigation was carried out before the experiment started and twice during the experimental period at intervals of 6 months. As vaccinated and control animals were mixed, the overall clinical picture of AR in the herd had to be registered. The number of litters and individuals affected, and the severeness of symptoms including sneezing, dark tear staining and distorted snouts, were registered and compared.

Weight gain
The slaughter weight of dressed carcass of all pigs was recorded and divided by the age of the pig in days to obtain average daily gain.

Pathoanatomical investigations
At slaughter, snouts from all animals in both the vaccine and control groups were investigated as described previously (*Baalsrud* 1987). Nasal chonchae were graded according to the following scale: 0 – normal chonchae, 1 – some turbinate atrophy, and 2 – almost total or total turbinate atrophy.

Statistical analyses
A Students t-test was performed on the weight data within herds.

Results

Bacteriological and toxinological investigations
Toxigenic P. multocida were isolated from nasal swabs in 8 herds (Table 1), and non-toxigenic strains in another 5. In 2 herds with obvious clinical AR (herds 5 and 6), only 4 nasal swabs were collected, and the few isolates of P. multocida from these herds were found to be toxin-negative. Due to the inadequate number of samples taken, these 2 herds are listed in Table 1 as "not known" with regard to the presence of toxigenic strains. None of the P. multocida strains isolated from the herds with no definite clinical signs of AR produced toxin. Toxigenic and non-toxigenic strains were found together in quite a few of the nasal swabs, indicating that such strains may live side by side on the nasal mucosa. P. multocida was not isolated at all in herds 11 and 14. These 2 herds were characterized by good environmental conditions and no clinical signs of AR.
All the P. multocida isolates from nasal swabs, except 1 from herd 4 and 1 from herd 13, proved to be serotype D. A toxigenic serotype A strain was isolated from a nasal swab in herd 4 in which the pigs showed marked AR symptoms. In addition, 1 similar serotype A strain was isolated in this herd from a lung after necropsy of a dead 9 week old piglet. In herd 13, where no clinical symptoms of AR were seen, the only P. multocida isolate from nasal swabs was also identified as serotype A. This strain was found to be non-toxigenic.

Clinical symptoms
The change in the clinical picture of AR after introduction of vaccination in each herd is presented in Table 1. The pigs in 8 of the 10 herds with obvious clinical signs of AR before the experiment started, showed clearly less pronounced clinical signs of the disease when they were examined after the vaccination period was over. In these 8 herds, toxigenic P. multocida had been isolated from nasal swabs. In herds 5 and 6, the number of pigs affected, and the severity of the condition, seemed unchanged. In the 5 herds in which clinical AR was not observed before the experiment started, no shortened or distorted snouts were seen on clinical examination during the period of observation.

Table 1. Effect of vaccination with a B.b./P.m. bacterin on clinical picture, weight gain and turbinate atrophy in 15 herds with previous turbinate atrophy.

Herd no.	No. of contr./vacc.	Vaccine used[1]	Tox. P. m. isolated	Clinical picture of AR after vacc.	Average daily weight gain in g[3]		Average snout score[4]	
					Contr.	Vacc.	Contr.	Vacc.
1	24/24	1	yes	improved	400 ± 39	409 ± 45	0.75	0.92
2	37/29	1	yes	improved	412 ± 49	373 ± 50*	0.54	0.41
3	51/57	2	yes	strongly improved	400 ± 44	390 ± 50	0.22	0.07
4	74/10	2	not known[2]	unchanged	374 ± 47	366 ± 37	0.85	1.10
5	45/45	2	not known[2]	unchanged	404 ± 31	385 ± 48	0.86	0.67
6	54/22	2	yes	strongly improved	401 ± 83	422 ± 60	1.06	1.23
7	48/57	2	yes	improved	388 ± 47	372 ± 44	0.75	0.75
8	39/25	2	yes	improved	405 ± 94	444 ± 36*	0.64	0.80
9	42/28	2	yes	improved	340 ± 37	379 ± 56*	0.76	0.75
10	3/8	1	yes	improved	430 ± 83	458 ± 60	0.67	0.00
11	72/91	1	no	0[5]	394 ± 63	379 ± 63	0.27	0.46
12	25/20	1	no	0[5]	367 ± 35	393 ± 29*	0.00	0.00
13	24/61	1	no	0[5]	444 ± 49	439 ± 49	0.13	0.07
14	104/60	2	no	0[5]	470 ± 33	460 ± 53	0.63	0.35
15	51/41	2	no	0[5]	386 ± 38	390 ± 47	0.33	0.24

* Significantly different ($p < 0.05$)

[1] See Materials and methods for description

[2] Less than 5 samples tested

[3] Average daily weight gain = slaughter weight of dressed carcass weight/age in days

[4] 0 = no turbinate atrophy
1 = some turbinate atrophy
2 = almost total or total turbinate atrophy

[5] No clinical AR before and after vacc.

Weight gain

In 5 of the 8 herds in which toxigenic P. multocida was found, the vaccine group showed a better daily weight gain than the control group, the difference being statistically significant in 2 herds. On average, pigs in the vaccine group gained 2.4 % more than control animals in these 8 herds, which is not statistically significant. In herd 12, in which no toxigenic P. multocida was isolated, vaccinated pigs also gained significantly more weight than the control pigs. In herd 2, the vaccine group gained significantly less per day than the control group. However, 17 of the 29 pigs in the vaccine group in this herd suffered from neonatal diarrhoea whereas none of the pigs in the control group, or any of the other herds, had such problems.

Turbinate atrophy

Vaccination did not result in any marked reduction in turbinate atrophy. In 6 of the 15 herds, the vaccine group showed slightly less turbinate atrophy than the control group. In 1 herd, herd 10, no turbinate atrophy was found in the vaccine group. However, the vaccine group in this case con-

sisted of only 3 individuals. In 3 herds, the average degree of turbinate atrophy was the same in the vaccine group and the control group. In 5 herds, the vaccine group showed slightly more turbinate atrophy than the control group. This is surprising especially in herd 6, where the clinical picture of AR changed markedly after introduction of vaccination.

Complete elimination of AR was not seen in any of the herds, which was not unexpected as only one half of each herd was vaccinated. In herd 10, though, no pigs in the vaccine group showed turbinate atrophy at slaughter. In all the other herds, both vaccinated and unvaccinated animals showed moderate and, in some cases, severe turbinate atrophy.

Vaccines and vaccination regimes

No differences were seen in the effect of vaccination between herds in which the mineral oil adjuvant vaccine was used and in those in which the aluminium hydroxide adjuvant vaccine was employed.

Discussion

A positive effect of vaccination against AR was seen in about half the herds in which toxigenic P. multocida was shown to be present. The clinical picture of AR improved in more than half of the herds (8 out of 10) after vaccination of only one half of the animals in each herd over a period of half a year. This corresponds well with results from other field experiments with vaccines against AR containing toxigenic P. multocida (*Schuller et al.* 1982). None of these studies demonstrated that vaccination could eliminate AR, even though the vaccine contained the contagious agent which seems to be crucial for the development of the disease.

In the present study, vaccination reduced the severity of the disease, but did not lead to its total elimination. However, as AR is a multifactorial disease, this result was not unexpected. Furthermore, it is likely that the effectiveness of a vaccination programme will improve if all animals are vaccinated and the vaccination programme implemented for a longer period of time.

It is difficult to find any definite relationships between the observed clinical picture, weight gain and degree of turbinate atrophy at slaughter. In some herds, the clinical situation improved and the vaccinated pigs gained, on average, 35 g more per day and yet the degree of turbinate atrophy measured at slaughter was slightly more pronounced in the vaccine group than in the control group. In other herds, though the clinical situation underwent a notable improvement and less turbinate atrophy was found, weight gain was actually lower in the vaccine group.

According to the suggested pathogenesis of AR (*Elling & Pedersen* 1985), toxigenic strains of P. multocida are present in herds in which pigs show clinical and/or pathoanatomical signs of persistent AR. If only small numbers of toxigenic P. multocida are present they will be dominated by the rest of the nasal flora, and may thus be difficult to isolate from nasal swabs, even if these are taken from young piglets that are expected to be carriers. This could have been the situation in most of the herds in which attempts to isolate toxigenic P. multocida were unsuccessfull. Such negative findings therefore do not necessarily undermine the supposition that the presence of toxigenic P. multocida seems to be associated with clinical symptoms of AR.

Infection with B. bronchiseptica per se (i.e. in the absence of toxigenic P. multocida), can, however, cause mild turbinate atrophy and this could have been the situation in the

5 herds in which clinical symptoms were not obvious. Since the vaccines also contained the B. bronchiseptica component, it is likely that vaccination will also improve the situation in some of these herds, as indeed it did to a significant extent in 2 of them. This corresponds well with the findings of *Pedersen & Barfod* (1977), *Bercovich & Oosterwoud* (1977) and *Goodnow et al.* (1979) who all found a reduction in turbinate atrophy after vaccination with a B. bronchiseptica bacterin.

The effect of vaccination was altogether better in herds in which toxigenic P. multocida was shown to be present. Thus, a bacteriological investigation of the nasal flora prior to the introduction of a vaccination programme may allow results to be predicted to some extent. There is consequently a need for an improved test, bacteriological or serological, for the demonstration of toxigenic P. multocida in a herd.

In one of the herd in this experiment (herd 12), no clinical or pathoanatomical signs of AR were found at all, though the same herd had had a severe AR problem only 1 year earlier. This may indicate that AR had been eliminated without any special efforts being made. Such spontaneous recovery is reported once in a while, though not often. It is, however, difficult to point out any special reason as to why this phenomenon should occur.

Both the vaccines used in this experiment belong to a group of new commercially available AR vaccines, containing killed whole cells of B. bronchiseptica and toxigenic P. multocida. Improved vaccines containing a larger amount of P. multocida toxoid or modified toxoid are now being developed. Such vaccines may contribute even more to the prevention of AR, as the toxin seems to play a crucial part in development of the disease.

Two vaccines differing with regard to the adjuvant, were used, and 2 different regimes employed. The present study revealed no difference in effect between the 2. Vaccines containing aluminium hydroxide or mineral oil as adjuvant were also found to have the same effect by *Éliás & Török* (1984). Neither the present study, nor previous investigations by *Bording Jensen & Riising* (1985) clarify the question as to whether passive immunization of the piglets by vaccination of the dam or active immunization of the piglets themselves will give the best protection. When vaccination of newborn piglets is suggested, little attention seems to be paid to the possibility that maternal transferred immunity may prevent an active immune response to the same antigens. According to *Duncan et al.* (1966), infection is supposed to occur during the first days or weeks of life. Consequently, vaccination of the dam and passive transfer of immunity to the progeny would seem to be the appropriate strategy for immunoprophylaxis of AR.

Conclusion

In conclusion, vaccination against AR can be used as a means to reduce the clinical effects of the disease and perhaps to achieve a higher growth rate among pigs in affected herds in which toxigenic P. multocida is present. Herds with only mild clinical symptoms do not seem to benefit from vaccination with the types of vaccine available today.

Vaccination should not be expected to result in the total elimination of a multifactorial disease, at least, not with the vaccines against AR which are currently available.

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Sammendrag

Vaksinasjon mot atrofisk rhinitt. Effekt på kliniske symptomer, tilveksthastighet og concha-atrofi.

To tradisjonelle helcelle-vaksiner bestående av Pasteurella multocida og Bordetella bronchiseptica ble prøvet ut i 15 svinebesetninger som alle hadde nysesjuke, men varierende grad av kliniske symptomer. Halvparten av purkene/smågrisene i hver besetning ble vaksinert, resten utgjorde kontrollgrupper. Vaksinasjon førte til en reduksjon i kliniske symptomer og en gjennomsnittlig vektøkning på 2,8 % i de 8 besetningene der toksinproduserende P. multocida var blitt isolert. Men også vaksinerte dyr viste kliniske symptomer på nysesjuke og concha-atrofi.

Det trengs bedre metoder til å fastslå om toksinproducerende P. multocida finnes i svinebesetninger. Ved hjelp av dette kan en legge opp vaksinasjonsprogrammer i besetninger hvor disse bakteriene er til stede.

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