Pasteurella multocida and Bordetella bronchiseptica in Atrophic Rhinitis and Pneumonia in Swine

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ABSTRACT

A total of 163 pigs from nine farrow-to-finish herds representing various levels of atrophic rhinitis (AR) were selected for postslaughter examination of AR and pneumonia. Nasal swabs and lungs were cultured for detection of Bordetella bronchiseptica and Pasteurella multocida. Seventythree pigs were examined at eight weeks of age and 90 contemporaries at six months of age. Mean AR scores were 1.21 and 1.11 for the eight week and six month old pigs, respectively (0 = normal, 3 = severe). In individual pigs increasing AR score was related to increasing pneumonia score in eight week old pigs but not in six month old hogs. In eight week old pigs, B. bronchiseptica and P. multocida were isolated more frequently from pigs with higher AR scores. From nasal swabs of six month old hogs, Bordetella was almost never recovered while Pasteurella was frequently isolated but not found to be related to AR score. Toxigenic type D P. multocida was isolated from nasal cultures of only seven (4%) pigs and from lung cultures of only one pig. Pasteurella was never isolated from lungs of the eight week old pigs and Bordetella never from the six month old hogs. The isolation rate of P. multocida. predominantly type A, from lungs of six month old pigs increased from 11% in grossly normal lungs to 86% in lungs with severe pneumonia. Pigs

from one herd free from lesions of AR and pneumonia were also examined; type A *P. multocida* was isolated from nasal cultures of one of six eight week old pigs.

Somatic antigens of *P. multocida* were determined for 94 nasal and 20 lung isolates. Somatic serovar 3 was found in 93% of the nasal isolates and in all lung isolates. Serovars 4, 5, 7 and 12 were found in low numbers, either alone or in combination with 3. The somatic serovar showed no relationship with either capsule type or pathology.

RÉSUMÉ

Cette expérience portait sur 163 porcs, issus de neuf troupeaux où s'observaient divers degrés de rhinite atrophique, que les auteurs choisirent pour étudier les lésions de rhinite atrophique et de pneumonie. Ils utilisèrent la culture bactériologique pour rechercher Bordetella bronchiseptica et Pasteurella multocida, dans des écouvillons nasaux et des échantillons pulmonaires. Cet examen porta sur 73 sujets, âgés de huit semaines, et sur 90 de leurs congénères, âgés de six mois. La gravité movenne des lésions de rhinite atrophique, selon un barème de 0 à 3, s'établit à 1,21, chez les premiers, et à 1,11, chez les seconds. Chez les premiers, mais non chez les seconds, la gravité de la rhinite atrophique se révéla proportionnelle à

celle de la pneumonie. Chez les premiers, l'isolement de B. bronchiseptica et de P. multocida s'avéra proportionnel à la gravité de la rhinite atrophique. Chez les seconds, les écouvillons nasaux ne recelèrent presque jamais B. bronchiseptica, tandis qu'on en isola souvent P. multocida, mais sans rapport avec la gravité de la rhinite atrophique. Pasteurella multocida du type D toxinogène se retrouva dans les écouvillons nasaux de seulement sept (4%) porcs et dans les poumons d'un seul. On n'isola jamais P. multocida, des poumons des porcs âgés de huit semaines, ni B. bronchiseptica, de ceux des sujets âgés de six mois. Le taux d'isolement de P. multocida, surtout du type A, des poumons des porcs âgés de six mois, passa de 11%, pour ceux qui semblaient normaux, à 86%, pour ceux qui affichaient des lésions marquées de pneumonie.

L'examen bactériologique des cornets et des poumons de sujets d'un troupeau témoin se solda par l'isolement du type A de *P. multocida*, à partir d'écouvillons nasaux de seulement un porc âgé de huit semaines.

La recherche des antigènes somatiques des souches de *P. multocida*, isolées de 94 échantillons nasaux et 20 pulmonaires, permit d'identifier le sérovar 3, dans 93% des isolats nasaux et 100% de ceux des poumons. Elle permit également d'identifier les sérovars 4, 5, 7 et 12 moins fréquemment, seuls ou en association avec le

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sérovar 3. Le sérovar somatique n'afficha aucune relation avec le genre de capsule ou les lésions nasales et pulmonaires.

INTRODUCTION

Throughout the 1980's, annual polls conducted by the National Pork Producers Council have indicated that atrophic rhinitis (AR) is one of the most important disease problems affecting the swine industry in the United States (1). In a slaughterhouse survey in Illinois in 1980, Bäckström *et al* reported AR lesions in 50% of both finishing hogs and sows (2). Elanco in 1984 found AR lesions in more than 70% of finishing hogs and in 100% of herds investigated nationwide (3).

Even though AR has been described as a multifactorial disease with etiological components of heredity (4-6), nutrition (7) and environment (8,9), infectious agents appear to be the most important (10). Bordetella bronchiseptica has long been considered an important cause of AR (11). but Nielsen et al (12) later confirmed by Underdahl et al (13) and others (14,15), showed that B. bronchiseptica infections in young pigs were selflimiting and that other bacteria, in particular Pasteurella multocida, had the ability to enhance or even initiate more severe and persistent turbinate lesions (16-18). In Europe and North America, certain toxin-producing capsule type D P. multocida strains have been specifically related to AR (10,16,19,20), although the significance of these toxigenic strains has been questioned in other parts of the world, especially in Japan (21-23). The toxin is a thermolabile protein that is dermonecrotic in guinea pigs and lethal for mice (10,24-26).

In some studies, pneumonia in swine and AR have been found to be related (27,28); however in other work with individual herds (2,4) and test stations (29,30), no such relationship was revealed. Pijoan *et al* reported a predominance of *P. multocida* type A isolates from pneumonic swine lungs (31), whereas type D isolates have more often been associated with AR (10,16,20).

Previous studies in the United States, including an Illinois study in 1982 (32), on the role of P. multocida in AR and pneumonia utilized a conventional nonselective blood agar culture technique that might have favored identifying the relatively large mucoid type A colonies over the smaller type D colonies. deJong (16) and Pedersen and Barfod (17) showed that P. multocida type D strains are more easily detected in cultures from livers of mice inoculated with the contents of nasal swabs, and that the toxigenicity of P. multocida could be determined by guinea pig skin tests.

The objectives of the present investigation were to apply these techniques to study the occurrence of dermonecrotic toxin-producing strains of *P. multocida* within selected Illinois swine herds affected by AR and to determine their association with *P. multocida* capsular and somatic antigens, lesions of AR, pneumonia and the presence of *B.* bronchiseptica.

MATERIALS AND METHODS

Nine farrow-to-finish Illinois swine herds known to be affected with AR at varying levels were selected on the basis of clinical history and records of postslaughter snout examinations of market hogs. One herd (Herd 10), completely free of clinical or slaughter check evidence of AR, was added to the study as an AR-free control. Herd size varied between 100 and 300 sows. All pigs were kept in confinement and fed standard corn-soybean meal rations fortified with minerals and vitamins. In all herds, 250 parts per million of antibiotics (chlortetracycline-sulfamethazine-penicillin) were added to feed for nursery and grower pigs. In five herds (2,3,5,6,7)commercial Bordetella/Pasteurella bacterins had been administered to sows and piglets for several years without apparent success in reducing snout lesions. Those bacterins were not characterized for the toxinproducing ability of the P. multocida component.

A minimum of six or more eight week old pigs were selected from each herd and euthanized by electrocution. The external nares were cleaned with alcohol and dried with cotton; then a sterile cotton-tipped swab was inserted into each nostril to the level of the ethmoid turbinates. Snouts were cross-sectioned between the first and second premolar teeth and scored for gross evidence of turbinate atrophy according to the method described by Bäckström *et al* (0 = normal, 1 = mild, 2 = moderate, 3 = severe) (32).

The lungs of each pig were examined for gross evidence of pneumonia and scored from 0 to 3 based on the severity of lesions. Lungs with no evidence of consolidation were given a score of 0. If less than the volume of one of the anterior lobes was affected. a score of 1 was assigned. A score of 3 was given if 2 or more whole lobes were affected with pneumonia. Intermediate lesions were assigned a score of 2. Cultures were taken from lesions when present or from the right cranial lobe of normal lungs. The surface of each lung sample was seared with a hot spatula and flamed scissors were used to dissect into a bronchus or bronchiole; then material for culture was collected from the airway and surrounding tissue with a sterile cottontipped swab. In each herd, seven to 14 contemporaries of the eight week old pigs examined were allowed to grow to a slaughter weight of approximately 100 kg at about six months of age. Examination and culture of the slaughter pigs were similar to that performed on younger pigs. Nasal swabs were taken after killing and before scalding if the routine of the slaughterhouse permitted. Otherwise. nasal swabs were taken from the live pig immediately prior to slaughter.

Each nasal and lung swab was streaked directly onto 5% sheep blood agar and MacConkey agar plates. In addition to the direct streaking, each nasal swab was suspended in 1 mL of sterile saline and 0.2 mL of this suspension was injected intraperitoneally into a mouse as described by Pedersen and Barfod (17). Mouse livers were also cultured on blood agar and MacConkey agar. Colonies suspected of being either P. multocida or B. bronchiseptica were identified by standard bacteriological techniques (Minitek, BBL, Becton Dickinson and Co., Cockeysville, Maryland).

	N	Atrophic		0			D .						
	NO.	Rhinitis	Snouts				Pneumonia		Lungs				
Herd	Examined	Score ^a	ВЪ	PmA-	PmD-	PmD+	Score ^a	Bb	PmA-	PmD-	PmD+		
8 Week	Old Pigs												
1	6	1.83 ± 0.98	33	0	50	17	0.17 ± 0.41	0	0	0	0		
2	21	1.76 ± 0.77	38	19	5	5	1.00 ± 0.71	0	0	0	0		
3	6	1.50 ± 0.55	17	83	0	0	0.67 ± 0.82	0	0	0	0		
4	6	1.33 ± 1.03	17	50	33	0	1.00 ± 1.10	33	0	0	0		
5	6	1.17 ± 0.41	0	0	0	33	0.00 ± 0	0	0	0	0		
6	6	1.00 ± 1.10	0	0	0	0	0.17 ± 0.41	0	0	0	0		
7	6	0.83 ± 0.75	17	17	0	0	0.83 ± 0.75	0	0	0	0		
8	8	0.50 ± 0.53	0	25	0	0	0.13 ± 0.35	0	0	0	0		
9	8	0.13 ± 0.35	13	0	0	0	0.00 ± 0	0	0	0	0		
10	6	0.00 ± 0	0	17	0	0	0.17 ± 0.41	0	0	0	0		
6 Mont	h Old Pigs												
1	11	1.91 ± 0.83	0	45	9	9	1.60 ± 0.97	0	45	0	0		
2	7	1.86 ± 0.69	0	14	29	0	0.86 ± 1.21	0	14	0	0		
3	10	1.80 ± 1.03	0	40	20	0	0.50 ± 0.53	0	0	10	10		
4	11	1.18 ± 0.75	9	36	27	0	0.64 ± 0.81	0	0	18	0		
5	8	0.63 ± 0.52	0	38	0	0	1.00 ± 0.53	0	0	0	0		
6	10	0.90 ± 0.88	0	60	20	0	1.70 ± 0.95	0	30	0	0		
7	10	1.10 ± 0.74	10	50	50	10	0.60 ± 0.70	0	50	0	0		
8	9	0.67 ± 0.50	0	0	0	11	0.22 ± 0.44	0	11	0	0		
9	14	0.29 ± 0.47	0	57	36	0	0.71 ± 0.91	0	14	0	0		
10	10	0.00 ± 0	0	0	0	0	0.00 ± 0	0	0	0	0		

TABLE I. Herd Scores of Atrophic Rhinitis and Pneumonia, and Percentage of Isolations of Bordetella bronchiseptica and Pasteurella multocida From Snouts and Lungs of Eight Week and Six Month Old Pigs

^aData are expressed as mean \pm SD; scores are based on a 0-3 scale (0 = normal, 3 = severe)

Bb = B. bronchiseptica; PmA- = toxin-negative P. multocida type A; PmD- = toxin-negative P. multocida type D; PmD+ = toxigenic P. multocida type D

The capsule types of all isolates of *P.* multocida were determined by standard methods (33,34). One hundred and fourteen *P. multocida* isolates were further characterized for somatic antigens by the Heddleston gel diffusion precipitin test (35) using chicken antisera (National Animal Disease Center, Ames, Iowa). All *P.* multocida isolates were tested for production of dermonecrotic toxin in guinea pigs as described by deJong *et al* (16) and Pedersen and Barfod (19).

Data were summarized using a computerized statistical analysis system (SAS Institute, Cary, North Carolina). Frequency distributions were tested for significance by the chisquare procedure and correlations were tested using Spearman's rank correlation coefficient.

RESULTS

From the nine herds investigated a total of 73 eight week old pigs and 90 six month old market hogs were examined. Mean herd scores for atrophic rhinitis varied from 0.13 to 1.83 in the young pigs and from 0.29 to 1.91 in the market hogs (Table I). A

total of 17 (23%) of the eight week old pigs showed no gross lesions of AR, 31 (42%) had mild lesions, 18 (25%) moderate lesions and seven (10%) severe lesions (Table II). The overall mean AR score was 1.21. Corresponding figures for the older hogs were 23 (25%), 43 (48%), 15 (17%) and nine

TABLE II. Distribution of Eight Week and Six Month Old Pigs by Atrophic Rhinitis Score, and Means of Atrophic Rhinitis Scores in Relation to Nasal Isolation of *Bordetella bronchiseptica* and *Pasteurella multocida*

Bacteria	No of	Atrophic Rhinitis Score					
Isolated	Pigs	0	1	2	3	Mean \pm SD	
8 Week Old Pigs							
Bb	9	1	3	3	2	1.67 ± 1.00	
Bb and PmA-	3	1	0	2	0	1.33 ± 1.15	
Bb and PmD-	2	0	0	1	1	2.50 ± 0.71	
PmA-	12	1	6	5	0	1.33 ± 0.65	
PmD-	4	0	2	1	1	1.75 ± 0.96	
PmD+	4	0	2	I	I	1.75 ± 0.96	
No isolation							
of Bb or Pm	39	14	18	5	2	0.87 ± 0.83	
Total	73	17	31	18	7		
6 Month Old Pigs							
ВЪ	1	0	1	0	0	1.00	
Bb and PmA-D-	1	0	1	0	0	1.00	
PmA-	30	10	12	4	4	1.07 ± 1.01	
PmA- and PmD-	5	3	2	0	0	0.40 ± 0.55	
PmD-	14	1	8	4	1	1.36 ± 0.74	
PmD+	3	0	1	1	1	2.00 ± 1.00	
No isolation							
of Bb or Pm	36	9	18	6	3	1.08 ± 0.87	
Total	90	23	43	15	9		

Bb = B. bronchiseptica; PmA- = toxin-negative P. multocida type A; PmD- = toxin-negative P. multocida type D; PmD+ = toxigenic P. multocida type D

(10%), respectively, with an overall mean AR score of 1.11. None of the six eight week old pigs and ten six month old pigs in the tenth herd showed gross lesions of AR.

Among eight week old pigs, *B.* bronchiseptica and *P.* multocida were more frequently isolated from pigs with higher AR scores (p < 0.05). There was no significant association between bacterial isolation rate and AR score among the six month old hogs. The herd isolation rate of *B.* bronchiseptica varied from 0% to 38% in nasal swabs from the eight week old pigs; however, *B.* bronchiseptica was isolated from only two of the six month old hogs (Table I).

Isolation rates for P. multocida varied from 0% to 83% in the young pigs and from 11% to 90% in the older hogs. Pasteurella multocida type A was isolated from nasal swabs of one of six eight week old pigs from the ARfree herd (herd 10); neither Pasteurella nor Bordetella was isolated from the six month old hogs in this herd. The isolation rate of P. multocida from nasal swabs was markedly improved by mouse inoculation. Pasteurella multocida was isolated by direct blood agar culture from 20.9% of nasal swabs, while it was isolated after mouse inoculation from 43.5%. Using both methods, 47.9% of all pigs were identified as intranasal carriers of P. multocida.

The distribution among herds of different capsule types and toxigenic P. multocida strains did not show any consistent relationship to herd AR scores or type of herd therapy used on either age group of pigs (Table I). Toxin-producing type D strains were isolated from nasal swabs of only 4% of all pigs (a total of four eight week old pigs and three six month old hogs in herds 1, 2, 5, 7, 8). Toxin-negative type D strains were found in six (8%) of the eight week old pigs and in 20 (22%) of the six month old hogs; toxin-negative type A strains in 15 (21%) and 36 (40%) of the eight week and six month old pigs, respectively; and toxigenic type A P. multocida was never isolated. Of all nasal swabs, six were positive for both B. bronchiseptica and P. *multocida* and five were positive for both P. multocida capsule types A and D. Toxin-positive P. multocida

 TABLE III. Pneumonia Scores in Eight Week and Six Month Old Pigs in Relation to Atrophic

 Rhinitis Scores and Correlation Coefficients

	Eight Wee	k Old Pigs	Six Month Old Pigs		
Atrophic Rhinitis Score	No. Examined	Pneumonia Score ^a	No. Examined	Pneumonia Score ^a	
0	17	0.24 ± 0.56	23	0.70 ± 0.63	
1	31	0.42 ± 0.62	42	0.76 ± 0.88	
2	18	0.89 ± 0.83	15	0.93 ± 1.03	
3	7	0.86 ± 0.90	9	1.67 ± 1.12	

Spearman's correlation coefficients of atrophic rhinitis score and pneumonia score.

Eight '	Week Old Pig	s	Six M	S	
No. Examined	R	Р	No. Examined	R	Р
73	0.34	< 0.01	89	0.18	> 0.05

^aData are expressed as mean \pm SD; scores are based on a 0-3 scale (0 = normal, 3 = severe)

was never isolated together with a toxin-negative strain in the same pig.

For a total of 94 P. multocida nasal isolates, somatic antigens were determined. Seventy-six percent of the isolates were serovar 3; 17% gave strong precipitation lines with serovar 3 antisera and weak cross-reactions with serovars 4, 5 and 12 antisera: 5%of the isolates were serovar 12 alone or 12 with cross-reactions with serovars 4 and 7; and 2% of the isolates were serovar 4. Of 20 lung isolates of P. multocida, 80% were serovar 3 and 20% serovar 3 with cross-reactions with serovars 4 and 12. No apparent relationship was found between somatic antigen serovars or capsule types of *P. multocida* and AR score or pneumonia score in individual pigs.

Pneumonic gross lesions were more common in the six month old hogs than in the eight week old pigs (Table I). Mild lesions were found in 19 (26%), moderate lesions in ten (14%) and severe lesions in none of the young pigs and in 36 (40%), ten (11%) and seven (8%), respectively, of the older hogs. On a herd basis no significant correlation was found between scores for AR and pneumonia; however, in individual pigs AR score was correlated with pneumonia score in eight week old pigs but not in six month old hogs (Table III).

Bordetella was isolated from the lungs of only two of the eight week old pigs and never in the six month old hogs (Table I). Pasteurella was never isolated from the young pigs but was cultured from 21 of the 90 market hogs (17 type A toxin-negative, three type D toxin-negative and one type D toxin-positive). The isolation rate of P. multocida from grossly normal lungs was 5% compared to 86% from lungs with severe pneumonia (Table IV).

The capsule types of *P. multocida* isolated from the lungs did not correspond with those of *P. multocida* isolated from the snouts of the same pigs. Of 17 *P. multocida* type A lung isolates, nine corresponded to type A in nasal cultures of the same pig, five to type D and four to negative nasal cultures. Of four *P. multocida* type D lung isolates, two corresponded to type A in nasal cultures and two to negative nasal cultures.

TABLE IV. Distribution of Six Month Old Pigs by Pneumonia Score, and Means of Pneumonia Scores in Relation to Lung Isolation of *Pasteurella multocida*

No. of			Pneu	imonia So	core
Pigs	0	1	2	3	Mean \pm SD
17	3	5	3	6	1.71 ± 1.16
3	1	2	0	0	0.67 ± 0.58
1	0	1	0	0	1.00
66	32	27	6	1	0.64 ± 0.72
87	36	35	9	7	
	No. of . Pigs 17 3 1 66 87	No. of Pigs 0 17 3 3 1 1 0 66 32 87 36	No. of Pigs 0 1 17 3 5 3 1 2 1 0 1 66 32 27 87 36 35	No. of Pigs Pneu 17 3 5 3 17 3 5 3 1 1 2 0 1 0 1 0 66 32 27 6 87 36 35 9	No. of Pigs Pneumonia So 1 17 3 5 3 6 3 1 2 0 0 1 0 1 0 0 66 32 27 6 1 87 36 35 9 7

DISCUSSION

Rather than a random sample, the herds selected for this survey represented different disease levels of atrophic rhinitis independent of herd environment and therapy. Therefore, no statistical comparisons were made between herds. In a previous study, clinical signs were found to be nonspecific and poorly related to turbinate and lung lesions assessed by postslaughter examination (32). For that reason, it was decided to use only snout and lung pathology scores in individual pigs as the basis for comparison in this study.

The main objective of this investigation was to determine the prevalence and significance of various characteristics of P. multocida in relation to the isolation rate of B. bronchiseptica and to scores of AR and pneumonia in the herds selected. In the eight week old pigs, a positive association was found between the isolation rates of both B. bronchiseptica and P. multocida and AR lesions. No progression of AR from the age of eight weeks to six months was apparent from the data on these herds. Similar results were recently reported in another study (38). Analysis of results showed an overall low prevalence (4%) of toxigenic P. multocida and an inconclusive relationship between the isolation rates of these strains and turbinate lesions, similar to what has been reported in other studies (36-39) but different from reports indicating a very strong correlation between AR and toxigenic P. multocida (10,18-20). While the overall isolation rate of toxigenic P. multocida was low, it was isolated from five of the ten herds, representing more severe (Herds 1 and 2), as well as moderate (Herd 5) and milder forms of AR (Herds 7 and 8). Nontoxigenic P. multocida were more prevalent; these strains were isolated from 44% of the pigs and from all of the herds examined.

All toxigenic isolates of *P. multocida* were capsule type D which agrees with most other investigations (10,16,20,21,40); however, toxigenic type A *P. multocida* has also been found in association with AR (36,37,41). The ability of the toxin to produce turbinate lesions when administered intramuscularly or intraperitoneally as reported by deJong (42) and Rutter (43) raises questions concerning the sensitivity of culture techniques for recovery of toxigenic *P. multocida* from nasal swabs. If *P. multocida* colonizes the pharyngeal and/or tonsillar areas, as has been suggested (10,32), its toxin might affect the turbinates of the pig without being detected in nasal swabs. Recent development of serological techniques shows promise in assessing more exactly the role of *Pasteurella* in AR (44).

Among the six month old hogs, *P.* multocida was isolated more frequently from grossly pneumonic lungs than from grossly normal lungs. The eight week old pigs were remarkably free of *Pasteurella*, even in the presence of pneumonia.

A positive association between AR scores and lung scores is consistent with the traditional view of a link between the two diseases. One hypothesis explaining this link is that both diseases are caused by the same infectious agent; another hypothesis is that AR compromises upper airway defenses making the lungs more susceptible to disease; yet a third suggests that pneumonia makes the pig more susceptible to AR. In this study the severity of snout and lung pathology was directly related in the eight week old pigs (Table III). The numerical differences found in the six month old hogs were not significant and similar to reports from other studies (27, 30). The differences between age groups might be related to the more chronic appearance of AR lesions compared to the reversible nature of pneumonic consolidations which can develop and heal within four to five weeks. The differences between capsule types of P. multocida from snouts and lungs of the same pig, with a predominance of type A in the lungs and type D in the snouts, indicate no direct correlation between P. multocida infections in the snout and lungs.

REFERENCES

 NATIONAL PORK PRODUCERS COUNCIL. Annual surveys of swine diseases. Des Moines, Iowa, 1980-1985.

- BÄCKSTRÖM L, HOEFLING D, MOR-KOC A, VINSON R, SMITH AR. Atrophic rhinitis in swine I: clinical signs, slaughter lesions, daily weight gain, disease transmission. Proc Int Pig Vet Soc Congr, Mexico City, 1982: 102.
- 3. ANON. Largest slaughter check ever finds respiratory diseases to be widespread. Report on Elanco survey. Feedstuffs 1985; 57: 8,25.
- 4. BÄCKSTRÖM L, BREMER H, DYREN-DAHL I, OLSSON H. The relationship between atrophic rhinitis, weight gain, age of the dam, and genetic disposition in a swine pedigree herd with a high incidence of the disease. Sven Vet Tidn 1976; 28: 449-455.
- 5. ELIAS B, HAMORI D. Data on the aetiology of swine atrophic rhinitis. V. The role of genetic factors. Acta Vet Hung 1976; 26: 13.
- 6. VOETS M. Genetic factors influencing atrophic rhinitis in the pig. Proc Int Pig Vet Soc Congr, Mexico City, 1982: 244.
- 7. BROWN WR, KROOK L, POND WG. Atrophic rhinitis in swine. Etiology, pathogenesis, and prophylaxis. Cornell Vet 1966; 56 (Suppl 1): 1-108.
- RUNNELS LJ. Infectious atrophic rhinitis of swine. Vet Clin North Am 1982; 4: 301-319.
- 9. HARDMAN IJHP. Control of atrophic rhinitis in the Netherlands. Proc Int Pig Vet Soc Congr, Mexico City, 1982: 109.
- RUTTER JM. Atrophic rhinitis in swine. Adv Vet Sci Comp Med 1985; 29: 239-279.
- 11. SWITZER WP. Studies on infectious atrophic rhinitis. V. Concept that several agents may cause turbinate atrophy. Am J Vet Res 1956; 17: 478-484.
- 12. NIELSEN NC, RIISING HJ, BILLE N. Experimental reproduction of atrophic rhinitis in pigs reared to slaughter weight. Proc Int Pig Vet Soc Congr, Ames, Iowa, 1976: 1.
- UNDERDAHL NR, SOCHA TE, DOS-TER AR. Bordetella bronchiseptica infection in pigs. Proc 2lst Annu Nebraska SPF Conf, 1981: 1-5.
- GILES CJ, SMITH IM, BASKERVILLE AJ, BROTHWELL E. Clinical, bacteriological and epidemiological observations on atrophic rhinitis of pigs in southern England. Vet Rec 1980; 106: 25-28.
- 15. **RUTTER JM.** Quantitative observations on *Bordetella bronchiseptica* infection in atrophic rhinitis of pigs. Vet Rec 1981; 108: 451-454.
- DEJONG MF, OEI HL, TETENBERG GJ. AR-pathogenicity tests for *Pasteurella multocida* isolates. Proc Int Pig Vet Soc Congr, Copenhagen, Denmark, 1980: 211.
- 17. PEDERSEN KB, BARFOD K. The aetiological significance of Bordetella bronchiseptica and Pasteurella multocida in atrophic rhinitis of swine. Nord Vet Med 1981; 33: 513-522.
- PEDERSEN KB. The occurrence of toxinproducing strains of *Pasteurella multocida* in SPF herds. Proc Int Pig Vet Soc Congr, Mexico City, 1982: 72.
- PEDERSEN KB, BARFOD K. Effect on the incidence of atrophic rhinitis of vaccination of sows with a vaccine containing *Pasteurella multocida* toxin. Nord Vet Med 1982; 34: 293-302.

- RHODES MB, NEW CW. Atrophic rhinitis of swine-toxigenic, type D, Pasteurella multocida. Proc 25th Annu Nebraska SPF Conf, 1985: 26-31.
- SAWATA A, NAKAI T, TUJI M, KUME K. Dermonecrotic activity of *Pasteurella multocida* strains isolated from pigs in Japanese field. Jpn J Vet Sci 1984; 46: 141-148.
- 22. OYAMADA T, YOSHIKAWA T, YOSHIKAWA H, SHIMIZU M, NAKAI T, KUME K. Lesions induced in the nasal turbinates of neonatal pigs inoculated with Pasteurella multocida and/or Bordetella bronchiseptica. Jpn J Vet Sci 1986; 48: 377-387.
- 23. NAKAI T, KUME K, YOSHIKAWA H, OYAMADA T, YOSHIKAWA T. Changes in the nasal mucosa of specificpathogen-free neonatal pigs infected with Pasteurella multocida or Bordetella bronchiseptica. Jpn J Vet Sci 1986; 48: 693-701.
- 24. NAKAI T, SAWATA A, TSUJI M, SAMEJIMA Y, KUME K. Purification of dermonecrotic toxin from a sonic extract of *Pasteurella multocida* SP-72 serotype D. Infect Immun 1984; 46: 429-434.
- CHANTER N, RUTTER JM, MacKEN-ZIE A. Partial purification of an osteolytic toxin from *Pasteurella multocida*. J Gen Microbiol 1986; 132: 1089-1097.
- 26. FOGED NT, PEDERSEN KB, ELLING F. Characterization and biological effects of the *Pasteurella multocida* toxin. FEMS Microbiol Lett 1987; 43: 45-51.
- BJÖRKLUND NE, HENRICSSON B. Studies on pneumonia and atrophic rhinitis in pigs. II. Analysis of variation in incidence. Nord Vet Med 1966; 18: 253-260.
- FLESJA KI, ULVESAETER HO. Pathological lesions in swine at slaughter. Acta Vet Scand 1980; (Suppl) 74: 1-22.

- 29. STRAW BE, BURGI EJ, HILLEY HD. Pneumonia and atrophic rhinitis in pigs from a test station. J Am Vet Med Assoc 1983; 182: 607-611.
- STRAW BE, LEMAN AD, ROBINSON RA. Pneumonia and atrophic rhinitis in pigs from a test station — a follow-up study. J Am Vet Med Assoc 1984; 185: 1544-1546.
- PIJOAN C, MORRISON RB, HILLEY HD. Serotyping of *Pasteurella multocida* isolated from swine lungs collected at slaughter. J Clin Microbiol 1983; 17: 1074-1076.
- 32. BÄCKSTRÖM L, HOEFLING DC, MORKOC AC, COWART RP. Effect of atrophic rhinitis on growth rate in Illinois swine herds. J Am Vet Med Assoc 1985; 187: 712-715.
- 33. CARTER GR, RUNDELL SW. Identification of type A strains of *Pasteurella multocida* using staphylococcal hyaluronidase. Vet Rec 1975; 96: 343.
- 34. CARTER GR, SUBRONTO P. Identification of type D strains of *Pasteurella multocida* with acriflavine. Am J Vet Res 1973; 34: 293-294.
- 35. HEDDLESTON KL, GALLAGHER JE, REBERS PA. Fowl cholera: gel diffusion precipitin test for serotyping *Pasteurella multocida* from avian species. Avian Dis 1972; 16: 925-936.
- 36. KIELSTEIN P, ELIAS B. Significance of Bordetella bronchiseptica and Pasteurella multocida in atrophic rhinitis of swine. Zientralbl Veterinaermed [B] 1985; 32: 694-705.
- 37. DANIEL GM, VELTKAMP G. New concepts in the etiology of atrophic rhinitis and its prevention using a *B bronchiseptica P multocida* bacterin-toxoid. Proc Am Assoc Swine Practitioners, Minneapolis, Minnesota, 1986: 77-95.

- 38. BÄCKSTRÖM LR, BRIM TA, COLLINS
- MT. Development of turbinate lesions and nasal colonization by *Bordetella bronchiseptica* and *Pasteurella multocida* during long-term exposure of healthy pigs to pigs affected by atrophic rhinitis. Can J Vet Res 1988; 52: 23-29.
- 39. BANE DP, HALL WF, WISEMAN B, COWART RP. Toxigenic Pasteurella multocida type D: an intensifier of atrophic rhinitis? (abstract no. 115). 66th Annu Meet Conf Res Workers Anim Dis, Chicago, 1985: 22.
- 40. **RIMLER RB, BROGDEN KA.** Pasteurella multocida isolated from rabbits and swine: serologic types and toxin production. Am J Vet Res 1986; 47: 730-737.
- 41. ELLING F, PEDERSEN KB. Atrophic rhinitis in pigs induced by a dermonecrotic type A strain of *Pasteurella multocida*. In: Pedersen KB, Nielsen NC, eds. Atrophic Rhinitis in Pigs. Brussels, Belgium: Commission of the European Communities, 1983: 123-135.
- 42. DEJONG MF. Atrophic rhinitis caused by intranasal or intramuscular administration of broth-culture and broth-culture filtrates containing AR toxin of *Pasteurella multocida*. In: Pedersen KB, Nielsen NC, eds. Atrophic Rhinitis in Pigs. Brussels, Belgium: Commission of the European Communities, 1983: 136-146.
- 43. RUTTER JM, MacKENZIE A. Pathogenesis of atrophic rhinitis in pigs: a new perspective. Vet Rec 1984; 114: 89-90.
- 44. BACKSTRÖM L, CHUNG WB, COL-LINS MT, CONRAD T. A comparison of different challenge methods for transmission of atrophic rhinitis to pigs from 5 weeks of age. Proc Int Pig Vet Soc Congr, Rio de Janeiro, Brazil, 1988: 41.