produced poliomyelitis in the same species of animal (produced by a virus from man). Most infected mice show no symptoms of Theiler's disease and are carriers, the infection being transmitted by the ingestion of material contaminated by faeces. Many competent authorities believe that the virus of the mouse disease and of poliomyelitis had a common origin, although they are now distinct, and that that common origin was the mouse. A chance mutant of the rodent virus deposited in mouse faeces on human food, may have been the origin. While this is pure speculation, it is a fact that there is no evidence that the mouse is at present, a source of human infection with poliomyelitis.

While this review includes the diseases known to be transmitted by mice, it cannot be inferred that it is complete. Far too little work has been carried out with mice to enable such a statement to be made. However, enough has been done, to justify a demand that more should be carried out.

(Reprinted from "Pest Control" September, 1949, with the kind permission of the editor).

# Inherited Facial Conformation and Susceptibility to Infectious Atrophic Rhinitis of Swine

By J. W. P. Gilman*

## Introduction

THE CONCEPT that inheritance plays a vital role in the etiology of Rhinitis in swine has been prevalent since the earliest reports of outbreaks of this disease; thus, in 1878, Schneider (17) attributes the "Sneezes" (Rhinitis) outbreak in Germany, studied by him at that time, to a congenital under-development of the nasal muscles. Jensen, (7, 8) in 1916 and later in 1933, estimated that the incidence of Rhinitis in Denmark was from 5 - 10 per cent, and, basing his opinion of the etiology of the disease mostly on negative bacteriological evidence, was inclined towards acceptance of the hereditary theory. Krage (10), in East Prussia, and Hoflund (5), in Sweden, lent further support to this view, the former claiming that the condition was most commonly encountered in good inbred herds, while the latter, in his excellent review of the literature up to that date, states, "There is apparently much in favour of the theory that atrophy of the muscles and deformity of the snout are hereditary", and goes on to suggest that such a defect may have as its primary cause some inherited glandular dysfunction. In the same year Radtke (15) published a very extensive paper dealing with swine flu and rhinitis (which conditions he attempts to relate to each other). This author stated that short nosed swine are to be especially feared when exposed

* Ontario Veterinary College, Guelph, Ont.

to rhinitis and sinusitis, as such animals are subject to extreme malformations; however, he considered the condition to be primarily an infection. He further claimed that the breed of pig infected may have a considerable influence on the course of the disease, Edelshwein and Berkshire pigs showing more pronounced symptoms than pigs of the longer nosed Landschwein breed.

Peterson (13), Thunberg and Carlstrom (19), Philips (14) and others, although unable to isolate the causative organisms, or reproduce the disease experimentally, were strongly of the conviction that Rhinitis was primarily an infectious disease. Recent work (4, 12, 15) in connection with outbreaks of rhinitis has demonstrated that the disease may be reproduced experimentally by introducing infective material from the upper respiratory tract of acutely infected animals into the respiratory tract of very young normal animals. McKay's (12) investigations indicate the possibility that *A. necrophorus* and *P. multocida,* acting in combination are the causative organisms. Attempts to implicate a filtrable virus have given consistently negative results (18).

Even although recent literature has made less specific mention of hereditary facial conformation as an etiological factor in rhinitis, belief in this association has become accepted as a matter of course and has found its way into some standard Clinical and Pathological texts (6). The situation has been further confused by papers apparently primarily concerned with the relative incidence and merits of Prognathic and Brachygnatic incisor contact, relating this purely inherited characteristic with the incidence and diagnosis of rhinitis in swine (1, 2). This hypothetical association has unfortunately been rather widely used as the basis for articles in the agricultural literature, trade journals and circular letters to breeders (11), with the result that many breeders and veterinarians practise and advise changing over from short to long-nosed swine, either by selection within the Yorkshire breed or by introducing a Tamworth sire, as a preventative measure against the further spread of rhinitis. Jones (9) mentions the general acceptance of the concept of both breed and strain (short-nose) susceptibility, but states that no data exist to support this hypothesis and quotes personal observation of a severely infected Tamworth herd. It was in an attempt to test the validity of these assumptions that the investigation reported here was undertaken.

### Materials and Methods

Four litters, two Yorkshire (Y III and IV) and two Tamworth (T II and T III), containing 34 piglets in all, were exposed to infectious rhinitis, while four similar litters of control animals were raised on rhinitis-free premises. All litters except T II and III were out of sows which had previously farrowed one or two litters each, while the two Tamworth litters were from gilts. All the sows of a particular breed were acquired from a single rhinitis-free premises and were bred to the same pure-bred boar of that breed; thus all piglets within each breed were at least half sisters and brothers.

Environmental conditions such as housing, rations and management of the sows and piglets to be exposed to infection, were standardized as far as possible. All litters were born during July and August and, excepting controls, were housed in the same piggery. Runts and unthrifty individuals were destroyed within forty-eight hours of birth in the test

litters, reducing the Tamworth litter numbers to eight pigs each, and the older Yorkshire sow's litters to nine each.

All animals were x-rayed and weighed individually at weekly intervals from one to eight weeks of age, inclusive. A portable x-ray machine was used and all plates were lateral views of the head, using a standard focal distance of 30", with an exposure of 2 m.p.s. Measurements of the nasal cavity were taken from these x-ray plates, using the tip of the nasal bone as an anterior land mark and a point on the cribriform plate between second and third ethmoid meatuses as the posterior land mark. It was found from experience with two previous litters (not included in this report) that physical measurements, using such instruments as calipers, were unsatisfactory, due to difficulties in restraining the animals, and locating satisfactory land marks to even approximately delimit the extent of turbinate and ethmoid development.

### Exposure

Test animals were equally exposed to infectious atrophic rhinitis between the fourteenth and twenty-eighth day. In accordance with a rabbit tissue method followed by McKay (12), six instillations of infective material were given at two to three day intervals. This method of transmitting the disease may be summarized as being dependent upon the following steps:—

1. Obtaining the original material from the nasal cavities of acute clinical cases.
2. Injecting such material subcutaneously into rabbits.
3. Making a suspension of the necrotic material from the resulting rabbit lesions.
4. Instillating this suspension into the nasal passages of the pigs to be infected at a very early age, preferably during the first few weeks of post natal life.

The material used to produce the rabbit lesions for this investigation was taken from three acute cases of rhinitis, all from the same infected premises, and, although it was impossible to instill all the test piglets with inoculum from the same rabbit lesion, each litter shared at least three of its six instillations with another litter of the opposite breed. The infective material used for these instillations was obtained from rabbit lesions after several serial passages.

It was felt that these animals, all being housed in adjoining pens under the same roof, and receiving equal quantities of infective material from the same source, were probably subjected to a more uniform exposure than would be the case under contact conditions alone.

### Post Mortem

All test animals were killed at 56 days of age and immediately examined. For the ensuing analysis, two systems of post mortem classifications were used:

1. *P. M. Classification A*—Classification based upon presence or absence of Infectious Atrophic Rhinitis: animals were classed as Positive only if they showed macroscopic destruction of the Schniderian membrane and a definite softening (decalcification) of either turbinate or ethmoid bones. All other animals, including doubtful pigs showing a considerable degree of inflammation and congestion of the nasal membranes, were here classed Negative.

2. *P. M. Classification B*—A graduated classification based on the

severity of the pathological changes, without consideration of whether the infection was unilateral or bilateral. Such a classification must of necessity be somewhat arbitrary, thus it has not been used in the ensuing analysis to establish a correlation table against length of nasal cavities.

| Class | Gross Pathological Changes |
|---|---|
| 1 (negative) | Negative. |
| 2 | Very slight congestion of nasal m.m. |
| 3 (doubtful) | Inflammation and congestion of nasal m.m. |
| 4 | Extensive Inflammation and exudate but no pitting of m.m. or definate decalcification of turbinates. |
| 5 (Positive $x^1$ group 1) | Inflammation - exudate - pitting of nasal m.m. with slight softening (decalcification) of turbinates. |
| 6 | As above (5) with extensive decalcification of turbinates. |
| 7 (Positive $x^2$ group 2) | Extensive atrophy of turbinates and ethmoids. |
| 8 | Complete disappearance of turbinates, nasal m.m. apparently intact. |

*Note*—Bacteriological examinations of the nasal cavities of all heads of exposed animals were also made by means of direct smears, aerobic and anaerobic blood agar plate cultures. Although a variety of organisms were recovered, it is of interest to note that Pasteurella and a pleomorphic filamentous gram-negative rod, tentatively identified as Actinomyces necrophorous were the only organisms consistently found in all heads classified in groups 3 to 8 above.

$x^1$—Includes all animals showing acute symptoms of infectious atrophic rhinitis.
$x^2$—Includes only those pigs in which the turbinate bones had completely or almost completely disappeared

### Analysis of Experimental Results

A comparison of nose length and body weight of the litters of test piglets at both one and four weeks of age, with their controls from the same breed, showed that no appreciate variation existed either in mean or standard deviation at those ages (Table I).

TABLE I. Nose lengths (mm.) and weights (lbs.) at 7 and 28 days. Control and exposed pigs grouped according to breed.

| Breed | Lot | No. Litters | No. Pigs | Length of Nasal Cavity in mm. | | | | Weight in lbs. | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | 7 days | | 28 days | | 7 days | | 28 days | |
| | | | | Mean | S.D. | Mean | S.D. | Mean | S.D. | Mean | S.D. |
| Tamworth | Exposed | 2 | 16 | 41.69 | ±1.89 | 58.19 | ±3.79 | 5.07 | ±0.26 | 12.11 | ±1.43 |
| | Control | 2 | 17 | 41.94 | ±2.40 | 58.94 | ±2.82 | 5.54 | ±0.74 | 12.86 | ±2.67 |
| Yorkshire | Exposed | 2 | 18 | 31.06 | ±3.27 | 43.39 | ±2.98 | 5.11 | ±0.91 | 13.90 | ±1.82 |
| | Control | 2 | 17 | 29.88 | ±2.14 | 42.18 | ±2.12 | 5.05 | ±0.86 | 10.79 | ±1.82 |

Clearly any attempt to correlate length (development) of nasal cavity with susceptibility to, or degree of infection with, Rhinitis on Autopsy, would be invalid if based on measurements taken at a time subsequent to that at which the infective agent had become established in the nasal mucous membranes of exposed pigs, more particularly as in this condition a retardation of the normal development of the nose and upper jaw are considered to be a constant symptom of the disease. That this defect may manifest itself, in severe cases, within a week or two of infection, was borne out by our observations. However, as there was no

significant difference between nose lengths of exposed and control animals at four weeks (Table 1), all analyses in this report have been based on measurements made at this age, and post mortem classification A and B, mentioned above; these data are summarized in Table 2. None of the 34 control animals contracted infectious atrophic rhinitis.

TABLE 2. NOSE LENGTHS AT 4 WEEKS AND PM CLASSES (CLASSIFICATIONS A AND B) AT 8 WEEKS OF FOUR LITTERS OF PIGS EXPOSED TO INFECTIOUS RHINITIS.

| | Litter T II | | | Litter T III | | | Litter Y III | | | Litter Y IV | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pig No. | Nose length in mm. at 28 days | PM Class at 56 days A | B | Nose length in mm. at 28 days | PM Class at 56 days A | B | Nose length in mm. at 28 days | PM Class at 56 days A | B | Nose length in mm. at 28 days | PM Class at 56 days A | B |
| 1 | 59 | − | 3 | 55 | + | 7 | 41 | − | 1 | 42 | − | 4 |
| 2 | 65 | − | 4 | 53 | + | 7 | 40 | − | 2 | 45 | + | 6 |
| 3 | 57 | + | 7 | 56 | + | 8 | 45 | − | 4 | 43 | + | 5 |
| 4 | 59 | − | 3 | 58 | + | 7* | 39 | + | 6 | 44 | − | 2 |
| 5 | 60 | − | 2 | 65 | − | 4 | 48 | − | 4 | 47 | − | 4 |
| 6 | 63 | + | 5 | 55 | + | 8 | 41 | − | 3 | 44 | + | 5 |
| 7 | 61 | + | 5 | 55 | + | 8 | 48 | − | 4 | 41 | − | 3 |
| 8 | 58 | − | 3 | 52 | + | 7* | 49 | + | 5 | 41 | + | 5 |
| 9 | | | | | | | 40 | + | 6 | 43 | − | 3 |

*Pigs T III—4 and 8 died at 6 and 7 weeks of age respectively The immediate cause of death in each case appeared to be a secondary pneumonia.

No evidence was forthcoming to support the concept that pigs of the Tamworth breed were in any way more resistant to infection with rhinitis than pigs of the Yorkshire breed. Of the 16 Tamworths exposed, 10, or 62.5% showed typical rhinitis at 56 days of age or younger, whereas, of the 18 exposed Yorkshire pigs, 7 or only 38.88% proved unquestionably susceptible. (Table 2).

It has been stated that (11, 16) even although the disease undoubtedly affects pigs of the long nosed breeds, infection in such animals follows a less severe course than in the shorter nosed types. This hypothesis was not supported under the experimental conditions of this test. (Table 3.)

TABLE 3. AVERAGE NOSE LENGTHS AND NUMBER (PARENTHESIS) OF PIGS IN EACH POST MORTEM CLASS (CLASSIFICATION B).

| Group | Post Mortem Classification B | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Negative | | Doubtful | | Positive Group 1 | | Positive Group 2 | |
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| All Animals | (1) 41 | (3) 48 | (6) 50.16 | (7) 51.45 | (6) 50.17 | (3) 41.31 | (5) 55 | (3) 55.33 |
| Yorkshires | (1) 41 | (3) 42 | (3) 41.66 | (5) 46.0 | (4) 44.25 | (3) 41.31 | — | — |
| Tamworths | — | (1) 60 | (3) 58.66 | (2) 65.0 | (2) 62.0 | — | (5) 55 | (3) 55.33 |

The "All Animals" group of Table 3, which disregards breed and considers only the factor of nose length, shows an apparently random distribution of nose lengths in regard to severity of infection, indicating little, if any, relationship between length of nose and resistance among the pigs of the sample as a whole. The "Yorkshire" portion of Table 3 likewise shows no obvious connection between nose length and resistance and, when tested on the basis of the positive-negative PM Classification A for significant difference of mean nose lengths between the infected (+) and non-infected (—) members of this group, gives the following results:—

$t = 0.061$; d.f. $= 16$; $P > 0.5$ which is not significant.

A similar analysis of the Tamworth breed data gave these somewhat different results:—

$t = 2.62$; d.f. $= 14$; $P = 0.02$

That this latter t test result suggests some positive relationship between susceptibility to the disease and a relatively short nose within this breed is further supported and more graphically illustrated when these data are examined in the light of a fourfold Contingency Table[3] (Table 4), in which all animals with a nose length greater than 58.19 mm., the breed average at 28 days, are considered as belonging to the "Long" class and vice versa for the "Short" class. The result obtained from this test for agreement between expectation and observation shows that infection with rhinitis among Tamworth pigs is significantly more frequent among short nosed individuals than among long nosed ones. Thus if the hypothesis of proportional susceptibility among Tamworths of both classes were true, an observation such as this would be improbable.

TABLE 4. FREQUENCY OF INFECTION WITH RHINITIS AMONG SHORT AND LONG NOSED TAMWORTH PIGS.

OBSERVED

| | Susceptible | Resistant | No. of Animals |
|---|---|---|---|
| Long | 2 | 5 | 7 |
| Short | 8 | 1 | 9 |
| No. of Animals | 10 | 6 | 16 |

Probability of 8 or 9 Short Nosed Animals being susceptible due to chance alone = 0.0239.

TABLE 5. FREQUENCY OF INFECTION WITH RHINITIS AMONG LITTER MATES IN TAMWORTH PIGS

OBSERVED

| | Susceptible | Resistant | Total |
|---|---|---|---|
| Litter T II | 3 | 5 | 8 |
| Litter T III | 7 | 1 | 8 |
| Total | 10 | 6 | 16 |

Probability of finding 7 or 8 susceptible individuals in Litter T III due to chance alone = 0.0574.

It was noted that 7 out of the 10 (70%) infected Tamworth pigs were litter mates (Litter T III) and furthermore, that all the susceptible members of this litter were very severe cases (Table 2). This suggested the possible influence of an hereditary litter-difference, not necessarily associated with the nose lengths of the individuals. However, a comparison of the numbers of susceptible pigs in Litters T II and T III (Table 5) gave no clear cut indication of the existence of such a hereditary litter difference, the test for independence resulting in a probability of 0.0574.

## Discussion

The limited amount of data examined in this investigation tend to refute the hypothesis that the inherited length of nasal cavity has any influence on susceptibility to infectious atrophic rhinitis in swine; neither is there any evidence to support the contention that the long nosed Tamworth breed of swine is more resistant to infection with this disease than the shorter nosed Yorkshire breed.

The results obtained support the concept that atrophic rhinitis in swine is primarily an infectious disease. However, despite equal exposure and standarized environmental conditions, only 50 per cent of the trial animals contracted the disease by 56 days of age (and these with very variable severity). This indicates that inheritance, other than that of facial conformation, may very probably exert a not inconsiderable influence as a predisposing cause in the aetiology of this disease.

Within the Tamworth breed itself, the nose lengths of susceptible animals, although longer than the longest Yorkshire, were significantly shorter than those of resistant pigs of the same breed. The picture of a clear cut positive relationship between long nose and resistance within the Tamworth breed is, however, complicated by the fact that the majority, and the most severely affected, of susceptible Tamworths were members of one litter. Although from the very limited data available, there was no positive indication of the existence of an inherent difference in susceptibility of Tamworth litters to infection with this disease, the probability of this litter difference being attributable to chance alone was less than 6%. It would seem that further work, incorporating considerably more data would be necessary to clarify this point.

It appears probable that the popular adherence to the hypothesis examined, is the result of two rather dangerous methods of reasoning:—

(1) The negative results obtained by all workers until recently in attempts to transmit the condition experimentally, or isolate the causative organism, have led to the assumption without supporting evidence, of a genetic explanation for the appearance of the disease.

(2) Observation of an apparent, but actually fortuitous, correlation between length of nose and incidence of the disease by field workers. This, in Canada at least may be due to the fact that (a) Yorkshire pigs are very common, whereas Tamworths are relatively rare and tend to form small, rather isolated groups; (b) within the Yorkshire breed itself, selection has been practised during the past several years towards a very short, dish nosed type of pig and during this period, rhinitis reached epidemic proportions. This investigator has been unable to find any evidence in the literature to support the contention that these occurrences are cause and effect.

Attempts have been made to control the disease in infected herds by breeding to a long nosed type of boar — usually a Tamworth. Piglets of the F1 generation from such a cross are almost as long nosed as their Tamworth parent (indicating that the length of nose in the Tamworth breed is probably influenced by one or more dominant genes) and although the resistance of such first generation hybrids has not been examined by this investigator, it seems highly probable that any increased resistance that does exist would here be attributable to the general effects of 'hybrid vigour'.

The contentions of Duthie (1, 2) and others, concerning the undesirability of carrying selection for dished snout within the Yorkshire breed to such extremes as to result in a brachygnathic jaw, undoubtedly merit attention *per se*. However, from the evidence of this investigation, extreme caution should be exercised in associating any such inherent differences in conformation with the incidence of an infectious disease.

**Summary**

1. Lengths of nose at 28 days and P.M. findings at 56 days were analyzed for two litters of Tamworth and two litters of Yorkshire pigs exposed to Infectious Rhinitis under experimental conditions. Related litters of control animals were maintained on Rhinitis-free premises.

2. Exposed animals were given 6 equal nasal injections of a suspension of infective rabbit lesion material between two and four weeks of age.

3. Relative measurements of length of nasal cavity were taken from x-ray plates of the head. All animals were x-rayed and weighed at weekly intervals from 1 to 8 weeks, at which age all exposed pigs were destroyed and post mortem examinations made.

4. There was NO evidence of the existence of a breed resistance on the part of Tamworth pigs.

5. NO evidence was encountered of any relationships between short nose and susceptibility within the Yorkshire breed.

6. A significant difference ($P=0.02$) between the mean nose length of susceptible and resistant Tamworth pigs was found.

7. Seventy per cent of the susceptible Tamworths, all advanced cases, were members of one litter. Tests for a specific difference in susceptibility between the two litters of Tamworths prove dinconclusive ($\$=0.57$).

8. Probable reasons for the widespread adherence to the apparently erroneous concept of the resistance of long nosed swine to Infectious Rhinitis are discussed.

**Bibliography**

1. DUTHIE, R. C. (1947): *Chronic Atrophic Rhinitis and Congenital Deformity of the Skull* Can. Jour. Comp. Med., 11 250-259.
2. DUTHIE, R. C. *Malformations of the Jaws in Yorkshire Swine.* (1947): Agr. Inst. Rev., 2 397-400.
3. FISHER, R. A. *Statistical Methods for Research Workers,* (1946): 92-97. (Oliver and Boyd, London. 10th Edition).
4. GWATKIN, R.; P. J. G. PLUMMER; J. L. BYRNE; and R. V. L. WALKER. (1949): *Rhinitis of Swine.* III. *Transmission to Baby Pigs.* Can. Jour. Comp. Med., 13 15-28.
5. HOFLUND, SVEN. *Report on Rhinitis Chronica Atrophicans in Swine, From a Clinical Viewpoint.* (1937): Svenska Svin. Tidscrift., 9 206-220.
6. HUTYRA, F.; J. MAREK & AND R. MANNINGER. *Chronic Purulent Haemorrhagic Rhinitis with Atrophy of the Turbinate Bones in Swine (Rhinitis Chronic Atrophicans).* Special Pathology

and Therapeutics of the Diseases of Domestic Animals. (19 46): II 459. (Alex. Eger, Chicago) Fifth Edition).
7. JENSEN, C. O. *Sneezing Disease in Swine.* Saertryk of Maanedsskrift for Dyrlaeger, (1916 28.
8. JENSEN, C. O. *Uber die Rhinitis chronica atrophicans des Schweines.* Acta Path. et Microbiol. Scand. Suppl., (1933): 16 172-179. Biol. Abst. 9 (8), (1935): 1788, 16197.
9. JONES, T. L. *Rhinitis in Swine.* Agric. Inst. Rev., (1947): 2 264-270.
10. KRAGE (1934-37) after HOFLUND, SVEN. *Report on Rhinitis Chronica Atrophicans in Swine, From a Clinical Viewpoint.* (1937): Svenska Svin. Tidscrift, 9 209.
11. Letter on Canadian Livestock Products: Industrial and Developmental Council of Meat Packers. February, 1948.
12. MCKAY, A. Personal communication and unpublished data: Ontario Veterinary College, (1948).
13. PETERSON, A., after THUNBERG, E. AND B. CARSLTROM. *Sneezing Sickness in Swine From an Epizootic Viewpoint.* Skandinavisk Veterinartidscrift, (1940): 48 711-723.
14. PHILLIPS, C. E. *Infectious Rhinitis in Swine (Bull Nose).* Can. Jour. Comp. Med., (1946): 10-33-41.
15. PHILLIPS, C. E.; H. F. LONGFIELD; AND J. E. MILTIMORE. *Porcine Infectious Rhinitis Experiments.* Can. Jour. Comp. Med., (1948); 12 268-273.
16. RADKE, G. *Investigation Concerning the Cause and Nature of Rhinitis in Swine.* Arch. Wiss. Prakt, Tierheilk, (1938): 72 371-423.
17. SCHNEIDER (1878) after HOFLUND, SVEN. *Report on Rhinitis Chronica Atrophicans in Swine, From a Clinical Viewpoint.* Svenska Svin. Tidscrift., (1937): 9 206-220.
18. SCHOFIELD, F. W. AND T. L. JONES. *The Pathology and Bacteriology of Infectious Atrophic Rhinitis in Swine:* Paper presented before the 86th Annual Conference of the A. V. M. A., Detroit, 1949.
19. THUNBERG, E. AND B. CARLSTROM. *Sneezing Sickness in Swine from an Epizootic Viewpoint.* Skandinavisk Veterinartidskrift, (1940): 48 711-723.