# INCIDENCE OF ANTIBODIES TO HAEMOPHILUS PLEUROPNEUMONIAE IN IOWA SWINE

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Haemophilus pleuropneumoniae was first identified as causing pneumonia in swine in the early 1960's in the United States by Olander (1963). It was not until the late 1970's that significance was attached to H. pleuropneumoniae in the United States when many swine confinement units began to suffer severe economic losses from the disease (Schultz, 1980 and Henry, 1980). The increased incidence of H. pleuropneumoniae disease is evidenced by a dramatic increase in confirmed cases at Veterinary Diagnostic Laboratories in both Iowa and South Dakota from 1977 to 1981. The increased occurrence and economic losses prompted interest in determining the incidence of antibodies to H. pleuropneumoniae in Iowa swine.

Methods: Most of our current understanding of and techniques for working with H. pleuropneumoniae have come from research done in Europe (Nicolet 1971). A complement fixation test using pooled antigens, used initially by Nielsen (1979) in Denmark and Gunnarsson (1979) in Sweden, was used in the work presented here. The antigen used was a pool of serotypes 1,2,3,4 and 5. Complement fixation was performed by the procedure of the Laboratory Branch of the Communicable Disease Center (1965) adapted to microtechnique. The procedure was done as modified for swine serum by Slavik and Switzer (1972). Swine sera were selected randomly from samples submitted to the Veterinary Diagnostic Laboratory at Iowa State University during the first 3 months of 1980. The samples represented breeding stock sold during that period as well as those herds being tested for pseudorabies validation.

Results: Serum samples were from 7348 swine representing 597 herds in Iowa, a state that markets 21 million head of hogs annually for a quarter of the United States production. A total of 32% of the individuals had complement-fixing antibody titers of 1:4 or greater (Table 1).

Table 1. Number and percent of 7348 swine negative, positive, suspect and anticomplementary for CF antibodies to H. pleuropneumoniae

| Category | Number | Percent |
|---|---|---|
| Negative | 4237 | 57.63 |
| Positive* | 2362 | 32.11 |
| Suspect | 565 | 7.68 |
| Anticomplementary | 184 | 2.50 |

*Sera with 30% or less hemolysis at dilutions of 1:4 or higher were classified as positive.

The number and percent of herds with one animal or more with CF antibodies are ranked according to the highest titer detected in Table 2. We arbitrarily selected 1:16 as the lowest definitive titer. As such, the percent of herds with at least one animal with a CF titer of 1:16 or greater was 67.3%. In infected herds, 45% of the animals had a titer of 1:16 or greater. The average number of samples tested from herds was 12.3. Forty-five percent may be taken as an emperical estimate, $\hat{P} = 0.45$, of the probability an animal is infected in an infected herd. Determination of the H. pleuropneumoniae status of a given herd of any size with 95% confidence can be achieved by testing sera from 6 animals.

Table 2. Number and percent of 597 herds of swine with CF antibody titers ranked by highest titer detected[a,b]

| Highest Titer Detected | No. of Herds | Cummulative No. of Herds | Percent of Herds | Cummulative Percent of Herds |
|---|---|---|---|---|
| 0 | 180 | 180 | 30.1 | 30.1 |
| 1:4 | 6 | 186 | 1.0 | 31.2 |
| 1:8 | 9 | 195 | 1.5 | 32.7 |
| 1:16 | 29 | 224 | 4.9 | 37.5 |
| 1:32 | 78 | 302 | 13.1 | 50.6 |
| 1:64 | 130 | 432 | 21.8 | 72.4 |
| 1:128[c] | 165 | 597 | 27.6 | 100.0 |

[a]Anticomplementary and suspect reactions excluded from this tabulation

[b]Average number of samples tested per herd 12.3 (Range 2-20)

[c]1:128 highest dilution tested.

Conclusions: The finding that 67.3% of the herds in Iowa had swine with antibodies to one or more of the 5 recognized serotypes of H. pleuropneumoniae was surprising. Although the incidence of pleuropneumonia caused by H. pleuropneumoniae has increased during recent years, clinical disease appears in far less than 67.3% of the herds, the proportion we found to have serologic evidence of infection. It seems likely, however, that many of these infected herds have the potential for developing pleuropneumonia or for spreading it to other herds.

References: Gunnarsson, A. Am. J. Vet. Res. 1979. 40:1564-1567. Henry S. Proc. George A. Young Conf. and Nebr. SPF Conf. 1980:39-41. Laboratory Branch of the Communicable Disease Center: Public Health Monograph 74, 1965. Nicolet, J. Zbl. Bakt. I. Abt. Orig. 1971, 216:487-495. Olander, H. J. PhD Thesis. 1963. Univ. of Calif. Schultz, R. A. Proc. George A. Young Conf. and Nebr. SPF Conf. 1980:43-47. Slavik, M. F., Switzer, W. P. Ia. State J. Res. 1972, 47:117-128.

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