RELATIONSHIP BETWEEN FRONT LEG SOUNDNESS AND MYCOPLASMA HYOSYNVOIAE INFECTION IN Duroc SWINE

L. S. LAWRISUK1, R. F. ROSS2, M. F. ROTHCHILD1, L. L. CHRISTIAN1, and D. L. MEEKER1

1 Department of Animal Science
2 Veterinary Medical Research Institute
Iowa State University, Ames, Iowa 50011 U.S.A.

SUMMARY

The relationship between degree of Mycoplasma hyosynoviae infection and front leg soundness in Duroc swine was examined in three lines of pigs (structurally sound, control and unsound) from a population divergently selected for front leg soundness. Analysis of sera collected between 6 and 26 weeks of age yielded complement-fixing antibody titers which were interpreted as being indicative of the degree of infection with M. hyosynoviae. Statistical analysis of several variables defined using titer levels failed to reveal significant differences between the three selected lines but trends indicated higher titer values for those pigs from the unsound line. Heritability estimates for the defined titer variables ranged from .28 ± .25 to .51 ± .28.

INTRODUCTION

Mycoplasma hyosynoviae causes uncomplicated, nonsuppurative polyarthritis in 3 to 6-month-old (40 to 100 kg) swine (Ross and Duncan, 1970). This organism can also cause arthritis in older and sexually mature swine, but rarely affects animals less than 3 months of age (Ross and Switzer, 1968). M. hyosynoviae arthritis is characterized by sudden lameness which may vary in severity. Lameness may become chronic or animals may completely recover (Ross and Duncan, 1970). The disease more frequently affects heavily muscled and faster growing swine. It results in economic losses due to reduced growth rates and trimming of affected limbs at slaughter.

Many factors have been shown to affect clinical presentation of the disease. These include age, virulence of the mycoplasma strain, conformation and prior joint damage (Ross 1985). Ross (1985) observed that joints infected with M. hyosynoviae also tended to show degenerative lesions of osteochondrosis and proposed that joints with prior damage were more likely to have impaired defense against the entrance and persistence of the mycoplasma. The observations that heavily muscled animals frequently have joint damage caused by osteochondrosis and that clinical evidence of M. hyosynoviae infection is often seen in heavily muscled pigs further support the proposed relationship between M. hyosynoviae infection and prior joint damage.

This investigation was primarily concerned with relating the degree of M. hyosynoviae infection to the degree of leg soundness in Duroc swine to test if pigs exhibiting structural unsoundness were more susceptible to infection with M. hyosynoviae. To examine this relationship, a population consisting of three lines divergently selected for front leg soundness was used (Rothschild et al., 1985). Previous reports indicated that the three lines were genetically divergent for front leg soundness. Degree of M. hyosynoviae infection was determined by measurement of the prevalence of complement-fixing antibody to the organism. Higher antibody titers were interpreted as being indicative of a higher degree of infection.
MATERIALS AND METHODS

Animals used in this experiment were purebred Duroc swine from the first replicate of the fourth generation of Iowa State University's front leg soundness selection study (Rothschild et al., 1985). The original animals used in the selection study were evaluated by three observers using a 1 to 9 (worst to best) scale for front structure (bone alignment, shoulder angle and toe size) and front movement (walking style, freedom and ease of movement). Pigs were then assigned to one of three lines on the basis of their front structure score. Highest scoring pigs were assigned to the high (structurally sound) line, average scores to the control line and lowest scores to the low (structurally unsound) line. Once assigned, these pigs and their descendants remained in the same line. Successive generations underwent divergent selection for front structure. The evaluation procedure was continued for successive generations without observers knowing the descent of the pigs.

Sera were collected from pigs of the first replicate of the fourth generation at 6 weeks of age and at subsequent 4 week intervals until pigs reached market weight. Antibody levels in the sera were determined by a microtiter complement fixation technique (Slavik and Switzer, 1972). Titers were recorded as the reciprocal of the highest dilution of serum resulting in 30% or less hemolysis. Titer values were transformed to log₂ for analysis.

Data were analyzed with the linear model:

\[ Y_{ijklm} = \mu + L_i + S_{ij} + D_{ijk} + M_k + (LM)_{il} + e_{ijklm} \]

where:

- \( \mu \) = population mean.
- \( L_i \) = fixed effect of the \( i^{th} \) line.
- \( S_{ij} \) = random effect of the \( j^{th} \) sire in the \( i^{th} \) line.
- \( D_{ijk} \) = random effect of the \( k^{th} \) dam with the \( j^{th} \) sire in the \( i^{th} \) line.
- \( M_k \) = fixed effect of the \( k^{th} \) sex.
- \( (LM)_{il} \) = fixed effect of animals of the \( i^{th} \) line and \( i^{th} \) sex.
- \( e_{ijklm} \) = random error with expectation of mean zero and variance \( \sigma_e^2 \) of the \( m^{th} \) animal, of the \( i^{th} \) sex, from the \( k^{th} \) dam and the \( j^{th} \) sire, of the \( i^{th} \) line.

Four major factors describing degree of infection with \( M. \) hyosynoviae were analyzed. These were defined as follows:

1. MEAN = mean titer; the arithmetic mean of the first five titer values measured for a pig.
2. PEAK = peak titer; the highest titer reached by a pig during the testing period.
3. PEAKA = age at peak titer; the age of a pig in days when the highest titer during the testing period was first reached.
4. AGE = age at onset of titer; the age of a pig in days when first registering a titer greater than zero.
Paternal half-sib estimates of heritability \( \left( \frac{\sigma_S^2}{\sigma_T^2} \right) \) were calculated using variance component estimates from Henderson's Method III (Henderson, 1953). Estimates of standard errors of heritability were calculated according to the procedure outlined by Dickerson (1969).

RESULTS AND DISCUSSION

Results of the analysis of variance for the titer variables are shown in Table 1. Line differences were the major point of interest in this analysis since the project was designed to investigate the relationship between leg weakness and level of *M. hyosynoviae* infection. No significant difference between lines was detected for any of the titer variables. Several major points need to be noted in explanation of this lack of differences between lines.

First, although osteochondrosis is thought to be the most common underlying lesion of leg weakness (Reiland et al., 1980), the presence of leg weakness does not always imply that lesions of osteochondrosis can be found (Goedegebuure et al., 1980). Therefore, it is not certain whether unsound pigs used in this experiment had lesions of osteochondrosis which could have increased their predisposition to infection with *M. hyosynoviae* or whether structurally sound pigs used in this experiment were free of lesions of osteochondrosis. Presence of lesions in the sound pigs could have predisposed them to higher degrees of infection with *M. hyosynoviae*. Occurrence of these circumstances could have produced the lack of line differences seen in this study.

Secondly, lack of line differences found in this experiment could be explained by comparing the location of lesions of osteochondrosis to the location in the leg commonly infected with *M. hyosynoviae*. Osteochondrosis commonly causes lesions in both the growth and articular cartilage of bones (Reiland, 1976) while *M. hyosynoviae* affects the joints (Ross and Duncan, 1970). Because *M. hyosynoviae* only affects the joints, growth cartilage lesions could still cause clinical evidence of leg weakness but not predispose pigs to *M. hyosynoviae* infection.

Since incidence and location of lesions were not examined in these pigs, it is difficult to determine which of these two possibilities occurred. Future work, which includes histopathological examination of bones from animals in this population, may make these relationships more clear.

Although analysis of variance revealed no significant differences between lines for any of the titer variables, least squares means by line for each titer variable suggested linear trends in the data (Table 2). For all four titer variables, means for the control line were intermediate to the means for the low and high lines. The trends suggested by the means lended support to the original experimental hypothesis that pigs exhibiting structural unsoundness may be more susceptible to *M. hyosynoviae* infection. The least squares means for the MEANT variable suggested that pigs in the high line tended to have lower overall titers than pigs in either the control or low lines. According to the linear trends suggested by the means for the other titer variables, pigs from the high line tended to display lower peak titers, earlier ages at peak titer and later ages at onset of titer than did pigs from the control or low lines. It must be reemphasized that although these linear trends exist, differences between lines for the four titer variables were not significant.

Heritabilities and their standard errors for MEANT and PEAKT are shown in Table 3. Heritability estimates for the other two titer variables, PEAKA and
<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Test Statistic</th>
<th>MEANT Mean Square</th>
<th>PEAKT Mean Square</th>
<th>PEAKA Mean Square</th>
<th>AGE Mean Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Line</td>
<td>2</td>
<td>Sire/Line</td>
<td>2.27</td>
<td>4.44</td>
<td>169.54</td>
<td>932.42</td>
</tr>
<tr>
<td>Sire/Line</td>
<td>16</td>
<td>Dam/Sire/Line</td>
<td>5.34*</td>
<td>12.37</td>
<td>1124.65</td>
<td>2631.00</td>
</tr>
<tr>
<td>Dam/Sire/Line</td>
<td>39</td>
<td>Error</td>
<td>2.53**</td>
<td>8.98**</td>
<td>1629.93**</td>
<td>1836.99**</td>
</tr>
<tr>
<td>Sex</td>
<td>1</td>
<td>Error</td>
<td>0.10</td>
<td>0.38</td>
<td>1478.00</td>
<td>132.10</td>
</tr>
<tr>
<td>Line x Sex</td>
<td>2</td>
<td>Error</td>
<td>0.58</td>
<td>0.49</td>
<td>11.97</td>
<td>431.74</td>
</tr>
<tr>
<td>Error</td>
<td>193a</td>
<td></td>
<td>1.42</td>
<td>4.77</td>
<td>761.52</td>
<td>782.42</td>
</tr>
</tbody>
</table>

MEANT mean=2.57, PEAKT mean=5.87, PEAKA mean=121.62, AGE mean=104.28

a Error degrees of freedom for MEANT=187.
* (P<.05).
** (P<.01).

<table>
<thead>
<tr>
<th>Line</th>
<th>MEANT Mean</th>
<th>Std. Error</th>
<th>PEAKT Mean</th>
<th>Std. Error</th>
<th>PEAKA Mean</th>
<th>Std. Error</th>
<th>AGE Mean</th>
<th>Std. Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>2.82</td>
<td>0.17</td>
<td>6.30</td>
<td>0.30</td>
<td>123.51</td>
<td>3.73</td>
<td>100.45</td>
<td>4.02</td>
</tr>
<tr>
<td>Control</td>
<td>2.53</td>
<td>0.13</td>
<td>5.76</td>
<td>0.24</td>
<td>122.66</td>
<td>2.94</td>
<td>103.26</td>
<td>3.18</td>
</tr>
<tr>
<td>High</td>
<td>2.34</td>
<td>0.15</td>
<td>5.65</td>
<td>0.27</td>
<td>118.68</td>
<td>3.36</td>
<td>108.06</td>
<td>3.63</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall Population</th>
<th>Low Line</th>
<th>High Line</th>
<th>Control Line</th>
</tr>
</thead>
<tbody>
<tr>
<td>MEANT</td>
<td>.51 ± .28</td>
<td>.68 ± .54</td>
<td>.40 ± .42</td>
<td>.58 ± .57</td>
</tr>
<tr>
<td>PEAKT</td>
<td>.28 ± .25</td>
<td>.37 ± .48</td>
<td>.31 ± .39</td>
<td>.25 ± .50</td>
</tr>
</tbody>
</table>

TABLE 2. Least squares means for titer variables.

TABLE 3. Heritabilities and standard errors for MEANT and PEAKT calculated for the overall population and within each line.
AGE, have limited meaning since they are so strongly dependent on exposure to the organism.

The large estimates of heritability indicate that a large amount of additive genetic variation may have been present, but the large standard error estimates indicate that the estimates of heritability did not differ significantly from zero. Variable environmental conditions may account for the magnitude of these values. Although the experiment was designed to try to provide equal exposure to M. hyosynoviae for all pigs, this may not have occurred under the practical production conditions of this test. Exposure may have occurred to different degrees and at different ages in the pigs. With such variability in exposure, the degree to which production of antibody is genetically governed is difficult to measure. A uniform challenge given to pigs of similar ages would help standardize environmental conditions and result in a better estimate of the genetic parameters involved. Future reports will examine results of an experiment involving a uniform challenge of pigs from this population.

CONCLUSIONS

The following conclusions can be drawn from this investigation:
1. Lack of significant differences between lines for any of the four defined titer variables suggests that degree of infection with M. hyosynoviae in Duroc swine is not significantly influenced by degree of leg soundness. A slight trend towards higher titers was seen in the low line pigs.
2. The proposed relationship between prior joint damage and increased susceptibility to M. hyosynoviae infection is neither supported nor contradicted by the results of this study since pigs were selected on the basis of visual appraisal of leg weakness with no knowledge of the location and incidence of lesions of osteochondrosis.
3. Large estimates of heritability and corresponding standard errors indicate that a large amount of additive genetic variation may be present.

REFERENCES


