INHERITANCE OF GASTRIC ULCERS IN SWINE

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Summary

DATA on ulcer condition were collected from 263 individually fed barrows, representing 51 sires, at the North Carolina Agricultural Experimental Station, Raleigh, N. C.

Four different scores were used to evaluate the importance of the different epithelial lesions found in the pig stomach. Year, season and breed effects on ulcer score were significant with Durocs having a higher incidence of ulcers than Yorkshires (29% vs. 12%). There was no apparent advantage in including scars and parakeratosis in the scores for ulcer condition of the gastric epithelium. When the gastric epithelium was scored only for the presence or absence of ulcers, a high heritability (0.52) was obtained. Thus selection against ulcers should be effective. Further indications were that selection for efficiency and/or gain would not increase the incidence of ulcers. However, selection against backfat may cause an increase in the occurrence of ulcers.

Introduction

Since the late 1950s the frequency of diagnosis of gastric ulcers in swine has been increasing in Ireland and the United States (Huber and Walling, 1967). This condition is now recognized as a world-wide disease (Kowalczyk, 1969).

Ulcers start with a focal proliferation of mucosal cells. Afterwards the epithelium becomes cornified and necrotic (Dunne, 1958). This condition is not apparent in the live pig until some anxiety, tension or physical stress factor precipitates an acute illness characterized by a massive hemorrhage, which generally is fatal (Kowalczyk et al., 1966). Usually this occurs at 4 to 8 months of age. According to Kowalczyk (1963), a chronic ulcer can develop with consequent loss of appetite and stunted growth. Muggenburg, McNutt and Kowalczyk (1964) report that chronic ulcers will leave scar tissue.

Diagnostic reports always show a lower percentage of ulcers than slaughter reports, 5% vs. 20%, (Boenker, 1967). Apparently more accurate examination of slaughter animals permits classification of subclinical lesions.

The etiology of gastric ulcers has not been completely determined. Bacteria and fungi have been reported as causative agents, but neither Streptococcus sp. or Candida albicans have been proven as primary causes and are only associations. Also, stress factors such as overcrowding, time prior or immediately following parturition, sex as a factor in social ranking, confinement and transportation can be factors in initiating ulcer eruption. Vitamins, minerals, heated corn, pelleting and other nutritional factors also have been associated with high incidence of ulcers (Dunne, 1958; Curtin, Goetach and Hollandbeck, 1963; Mahan et al., 1966).

Seasonality in occurrence of ulcers has been found with more ulcers appearing during the winter than the summer (Boenker, 1967). This agrees with the cyclic occurrence of ulcers in man (Kowalczyk, 1963).

Breed differences have been recognized (Curtin et al., 1963; Mahan et al., 1966) but rankings among breeds have not been conclusive. Other than these breed differences, there is little information available concerning the importance of genetics in ulcer formation. The present study was undertaken to evaluate the genetic control of ulcers and to examine the relationships between ulcers and measures of growth, efficiency and backfat.

Materials and Methods

This study was conducted during 1967 to 1969 at the North Carolina Agricultural Experiment Station, Raleigh, North Carolina. Data were collected from 98 Duroc and 155 Yorkshire barrows, representing 51 sires.

The pigs were maintained in individual solid floor pens (1.22 x 4.27 m) and self-fed a pelleted corn-soybean meal ration. Feed consumed and weight gain were recorded from 70 days of age to slaughter weight (91 kg). Backfat thickness was measured at 91 kg.
using the method developed by Hazel and Kline (1952). Stomachs were collected after slaughter and refrigerated for approximately 24 hr. before being weighed and opened for scoring of the ulcer condition.

A subjective score was given to each stomach according to the condition of the mucosal epithelium. The scores included macroscopic lesions of ulcers, scars and parakeratosis. Stomachs exhibiting lesions typical of these classifications are shown in figure 1. Also included were those pigs that died with a bleeding ulcer during the test. Histopathological analysis of a sample of macroscopic lesions was made by the Livestock and Poultry Diagnostic Laboratory of the North Carolina Department of Agriculture at Raleigh. All ulcerative and parakeratotic lesions were confirmed. However, not all scars indicated a regenerative process as some appeared to be artifacts. Four scoring procedures were developed in an attempt to evaluate the genetic susceptibility to ulcers.

Ulcer score A was made by assigning linearly increasing numerical values to the stomach: normal (0), parakeratosis lesions (1), scars (2) and ulcers (3) with the highest score being given to those stomachs from animals that died from an ulcer (4). For ulcer score B the three intermediate classifications were divided according to the severity of the condition (−, 0, +) with a consequent increase in range of the scale (0 to 10). These scores attempted to follow the pathogenesis of ulcers (Curtin et al., 1963; Kowalczyk, 1963; Muggenburg et al., 1964). Ulcer score A differs from ulcer score B in that it eliminates possible errors due to subclassification.

It may be assumed that scars and ulcers are found in animals that have in common a similar susceptibility to developing ulcers with the only difference being the time of onset and healing of the lesions relative to slaughter age. For this reason, ulcer score C groups scars, ulcers and death due to ulcer with an assigned value of one while those animals exhibiting parakeratosis and normal stomachs were assigned a value of zero. How-

Figure 1. Typical stomach conditions (top—parakeratosis and scar; left—normal; right—ulcers).
TABLE 1. LEAST SQUARES MEANS AND CONSTANTS FOR BREED AND SEASON OF BIRTH EFFECTS ON ULCER SCORE AND STOMACH WEIGHT

<table>
<thead>
<tr>
<th>Item</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>Stomach weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcer score*</td>
<td>1.363 ± 0.086</td>
<td>3.233 ± 0.254</td>
<td>0.328 ± 0.041</td>
<td>0.206 ± 0.035</td>
<td>41.6 ± 4.9</td>
</tr>
<tr>
<td>Duroc</td>
<td>+.248</td>
<td>+.757</td>
<td>+.112</td>
<td>+.084</td>
<td>+16.3</td>
</tr>
<tr>
<td>Yorkshire</td>
<td>-.248</td>
<td>-.757</td>
<td>-.112</td>
<td>-.084</td>
<td>-.16.3</td>
</tr>
<tr>
<td>Fall</td>
<td>+.142</td>
<td>-.509</td>
<td>-.071</td>
<td>-.066</td>
<td>+.5.5</td>
</tr>
<tr>
<td>Spring</td>
<td>-.142</td>
<td>+.509</td>
<td>-.071</td>
<td>-.066</td>
<td>-.5.5</td>
</tr>
</tbody>
</table>

\*Score A: normal (0); parakeratosis lesions (1); scars (2); ulcers (3), died with ulcers (4).
Score B: normal (0); parakeratosis lesions (1, 2, 3); scars (4, 5, 6); ulcers (7, 8, 9); died with ulcers (10).
Score C: normal and parakeratosis lesions (0); scars, ulcers and died with ulcers (1).
Score D: normal, parakeratosis lesions and scars (0); ulcers and died with ulcers (1).

Heritabilities for stomach weight and ulcer scores A and B were obtained from the sire component of an intra-year-season-breed hierarchical analysis of variance (Dickerson, 1960). Heritabilities for ulcer score C and D were obtained following the derivations of Robertson and Lerner (1949) for calculating heritabilities of threshold characters.

Results and Discussion

All ulcers found in this study were esophageal with the exception of two cases in which ulcers also were found in the fundic portion of the stomach. Seven pigs died from ulcers during the present study and were included for analysis of variance and estimation of heritabilities.

Year, season, breed and season-breed interaction effects were studied by means of least squares analysis of variance. Year, season and breed effects on ulcer scores were significant (P<.01). Breed and breed x season interaction effects were significant for stomach weight (P<.01). Means and least squares constants for ulcer scores and stomach weight are shown in table 1. The mean for ulcer score D represents the percentage of ulcers found in this study (21%). This value is very similar to that reported by Boenker (1967) in slaughter pig examination data. A greater incidence of ulcer lesions in the Duroc compared with the Yorkshire breed (29% vs. 12%) is indicated. Similar results were found by Curtin et al. (1963), based on a study of 166 Yorkshire and only six Durocs. Differences between seasons in occurrence of ulcers were similar to those reported by Fugate et al. (1965). Similarly, Curtin et al. (1963) reported that more deaths occur during spring and winter.

Heritability estimates are presented in table 2. The small difference between heritabilities of ulcer scores A and B suggests that little if any advantage is obtained by subdividing the categories of ulcer score A. From a comparison of ulcer scores A and B with ulcer score C, it appears that inclusion of parakeratosis in the score does not increase the accuracy of evaluating the additive genetic variance of ulcer condition. The heritability estimate was larger for ulcer score D than for any of the other ulcer scores. The size of the sire component in ulcer score D (0.021)
was similar to that of the sire component in ulcer score C (0.019). This indicates that the addition of scars into the score only increases the experimental error. Kowalczyk (1969) states that parakeratosic proliferation of the epithelium and scars can be found in animals which died of other diseases. Thus, the addition of information on scars and parakeratosis seems to reduce the accuracy of the genetic evaluation of ulcers.

Based on the heritability of ulcer score D (0.52), it appears possible to reduce ulcers by means of selection. However, Conley, Kratzer and Bicknell (1967) reported a low heritability (0.04) for incidence of ulcers. This low value may be due to the lack of adjustment for season effects which has been recognized in most reports. According to Kowalczyk (1969), endoscopic examination can provide an accurate diagnosis of ulcers in the live pig. The possibilities of using this method, at least in boars, in order to select against ulcers needs to be explored.

Genetic correlations of ulcer scores B and D and stomach weight with the values for feed consumed, average daily gain (ADG) and feed consumed/gain (F/G) during test, lifetime ADG and live backfat probe were calculated. These results are shown in table 3. Correlations of ulcer scores B and D with the other traits were in the same direction but usually ulcer score B gave larger absolute values. The correlations between ulcer scores and F/G or ADG suggest that faster growing, more efficient pigs have a lower incidence of ulcers. These results indicate that selection for efficient or fast growing pigs will not result in any increase in the frequency of ulcers. However, the high negative correlations between live backfat and ulcer scores tends to indicate that selection for a meat-type pig will increase the occurrence of ulcers.

Although no cause and effect relationship can be drawn from these data, the genetic correlations between F/G and ADG and ulcer score suggest at least three alternative interpretations of the pathological process. These are: (1) the presence of ulcers reduced growth and efficiency, (2) inherited metabolic changes that produced low growth and efficiency were also involved in genetic susceptibility to ulcers or (3) fast growing pigs, which are more efficient, were killed before their susceptibility to ulcers was expressed.

Ulcer scores were included as classes, with year, season and breed, in a least squares analysis of growth rate and feed efficiency. The nonsignificant effect of ulcer scores upon efficiency and gain and the small nonsignificant phenotypic correlations between ulcer scores and efficiency and gain tend to suggest that the presence of ulcers is not the cause of reduced growth rate and efficiency.

Very low correlations were found between ulcer scores and stomach weight. A high negative correlation was found between ulcer score B and average daily feed consumed. However, the correlation was smaller and negative when ulcer score D was used. The correlations between stomach weight and the other traits suggest that pigs with larger stomachs eat more, gain faster, are more efficient and deposit more fatty tissue.

**TABLE 3. GENETIC CORRELATIONS AND STANDARD ERRORS FOR ULCER SCORES B AND D AND STOMACH WEIGHT WITH GROWTH, EFFICIENCY AND BACKFAT**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Ulcer score B</th>
<th>Ulcer score D</th>
<th>Stomach wt</th>
</tr>
</thead>
<tbody>
<tr>
<td>F/G</td>
<td>0.654± .84</td>
<td>0.363± .30</td>
<td>-.462± .50</td>
</tr>
<tr>
<td>G/F</td>
<td>-.407± .73</td>
<td>-.314± .32</td>
<td>.640± .30</td>
</tr>
<tr>
<td>ADG</td>
<td>-.879± .90</td>
<td>-.319± .26</td>
<td>.664± .22</td>
</tr>
<tr>
<td>ADG (L)</td>
<td>-.1.489± 1.47</td>
<td>-.533± .32</td>
<td>.206± .33</td>
</tr>
<tr>
<td>Backfat</td>
<td>-.3.737± 4.05</td>
<td>-.1.900± .69</td>
<td>.356± .56</td>
</tr>
<tr>
<td>Feed consumed</td>
<td>-.1.717± 1.19</td>
<td>-.201± .33</td>
<td>.368± .33</td>
</tr>
<tr>
<td>Ulcer score B</td>
<td>2.000± 1.84</td>
<td>-.659± .68</td>
<td>-.108± .33</td>
</tr>
<tr>
<td>Ulcer score D</td>
<td>1.000± 1.00</td>
<td>-.666± .66</td>
<td>-.110± .33</td>
</tr>
</tbody>
</table>

*F/G=feed consumed/gain; G/F=gain/feed consumed; ADG=avg daily gain on test; ADG(L)=lifetime avg daily gain.

**Literature Cited**


