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Genetic validation of postmixing skin injuries in pigs as an indicator of aggressiveness and the relationship with injuries under more stable social conditions


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ABSTRACT: The objective of the study was to estimate genetic correlations between skin lesions and aggressive behavior postmixing and under more stable social conditions as a potential means of selecting against pig aggressiveness. Postmixing aggression in commercial pig production is common, compromises welfare and profitability, and cannot be significantly reduced by low-cost changes to the environment. A genetic component to individual aggressiveness has been described in pigs and other species. Selective breeding against aggressiveness ought to be possible if an easily measured indicator trait can be shown to be genetically associated with aggressive behavior. Aggressive behavior was recorded continuously for 24 h after mixing, and a count of skin lesions (lesion count, LC) was recorded at 24 h and 3 wk postmixing on 1,663 pigs. Two behavioral traits were found to have a moderate to high heritability similar to that of growth traits; duration of involvement in reciprocal fighting (0.43 ± 0.04) and delivery of nonreciprocal aggression (NRA; 0.31 ± 0.04), whereas receipt of NRA had a lower heritability (0.08 ± 0.03). Genetic correlations (rg) suggested that lesions to the anterior region of the body 24 h after mixing were associated with reciprocal fighting (rg = 0.67 ± 0.04), receipt of NRA (rg = 0.70 ± 0.11), and to a lesser extent, delivery of NRA (rg = 0.31 ± 0.06). Lesions to the center and rear were primarily genetically associated with receipt of NRA (rg = 0.80 ± 0.05, 0.79 ± 0.05). Genetic correlations indicated that pigs that engaged in reciprocal fighting delivered NRA to other animals (rg = 0.84 ± 0.04) but were less likely to receive NRA themselves (rg = −0.41 ± 0.14). A genetic merit index using lesions to the anterior region as one trait and those to the center or rear or both as a second trait should allow selection against animals involved in reciprocal fighting and the delivery of NRA. Positive correlations between LC 24 h and 3 wk after mixing were found, especially for lesions to the center and rear of the body, indicating that postmixing lesions are predictive of those received under more stable group conditions. As well as reducing immediate aggression at mixing, selection on postmixing LC is expected to have a long-term impact on injuries from aggression, even after dominance relationships are established.

Key words: aggression, fighting, genetic correlations, lesion, pig

INTRODUCTION

Abrupt mixing of commercial pigs is common and results in fighting that cannot be reduced in the long term by low cost environmental changes (Luescher et al., 1990; Hayne and Gonyou, 2006). This aggression is associated with a reduction in the rate and efficiency of BW gain and poorer meat eating quality, carcass gradings, and maternal ability (Rundgren and Löfquist, 1989; Tan et al., 1991; Warriss et al., 1998; Løvendahl et al., 2005). It also increases the risk of infection, reduces immunocompetence, and increases the risk of disease spread (Morrow-Tesch et al., 1994). Involvement in postmixing aggression and the injuries caused have been found to be heritable in pigs (h² = 0.17 to 0.24, Løvendahl et al., 2005; h² = 0.22, Turner et al., 2006a). Selective breeding, therefore, has the potential to reduce aggression at little implementation cost.
An easily measured indicator trait that is genetically correlated with involvement in aggressive behavior would facilitate selection. A count of skin lesions (lesion count, LC) 24 h postmixing was shown by Turner et al. (2006b) to be phenotypically correlated with involvement in aggressive behavior. Turner et al. (2008) estimated the genetic association between LC and aggressive behavior using Bayesian methodology with a population of 1,132 pigs of which 341 were sampled for aggressive behavior. The LC was genetically correlated with involvement in reciprocal fighting ($r_g = 0.72$ to 0.76) and the delivery of nonreciprocal aggression (NRA; $r_g = 0.56$ to 0.69), but not with the receipt of NRA ($r_g = -0.06$ to 0.34). Due to the small number of animals sampled for behavior and reliance on a Bayesian analysis, a larger experiment was performed in the present study using a dam-line rather than sire-line farm with a different breed composition and in which many more animals were phenotyped for aggressive behavior ($n = 1,184$) and LC ($n = 1,660$). The present study also used a broader definition of aggressive behavior that encompassed not only injurious interactions as recorded by Turner et al. (2008), but also noninjurious pushing, which could also be perceived as stressful. By examining the genetic correlations within this data set, a more definitive assessment of the value of selection on LC as a means of reducing postmixing aggressiveness was possible.

Furthermore, although aggression is most severe during the 24 h postmixing (Ewbank, 1976), a basal level of aggression persists thereafter (Stookey and Gonyou, 1994; Coutellier et al., 2007). This could affect long-term social stress and welfare and has been shown at the phenotypic level to affect growth performance (Stookey and Gonyou, 1994) and antiviral immunity (de Groot et al., 2001). The genetic correlations between LC 24 h postmixing and 3 wk later were also assessed in this study.

**MATERIALS AND METHODS**

**Ethical Approval for Animal Experimentation**

The procedures described were approved by the institutional Animal Ethics Committee. Governmental licensing was sought but not required.

**Animals and Housing**

The subjects were 1,660 pigs (898 purebred Yorkshire and 762 crossbred Yorkshire × Landrace; 419 intact males, 382 castrates, and 859 females) from a commercial dam line nucleus herd. All castrates were crossbred animals. The pigs were the progeny of 85 sires and 250 dams, and a pedigree file incorporating grandparents and great-grandparents was available.

Pigs were penned in littermate groups until 70.5 (SD 4.3) d of age and 27.6 (SD 5.6) kg of BW, when they were mixed into new groups of 15 as described below.

The pens into which the pigs were mixed were part slatted (29% slats, 71% lightly bedded solid flooring). Dry pelleted food was available ad libitum from a single space feeder, and water was also available ad libitum from a nipple drinker. The floor space allowance was 0.85 m²/pig (pen dimensions 4.0 × 3.2 m), and the mean ambient temperature was 19.4°C (SD 2.9).

**Mixing and Lesion Counting Protocol**

Single sex and single-breed groups of 15 were formed by mixing 3 pigs from each of 5 littermate groups. Mixing occurred between 1000 and 1400 h. As far as possible, pigs of a similar BW were mixed to achieve a similar within-pen variation to that of the rest of the unit. Immediately before mixing, the sex, breed, litter details, premixing LC, and ear-tag number (females and intact males) or ear notch number (castrates) were recorded for each pig. After 24 h, the animals were weighed, and a postmixing LC was recorded from which the premixing LC was subtracted. The number of fresh lesions was counted by a single observer throughout. No account was taken of the size of the lesions. Fresh lesions were those with a bright red color or with apparently recent and intact scabs. Lesions to the anterior part (head, neck, shoulders, and front legs), central part (flanks and back), or caudal part (rump, hind legs, and tail) of the body were recorded separately. Around 3 wk after mixing at 89.8 (SD 5.2) d of age, lesions were again counted on 1 occasion using the same procedure. Lesions were counted only when judged to have been received within the preceding 24 h.

**Behavior**

For 1,184 animals, involvement in 3 behavioral traits during the 24-h period after mixing was recorded: duration of engagement in reciprocal aggression and the durations of delivery or receipt of NRA. Reciprocal aggression was defined as fighting lasting ≥1 s in which both pigs were seen to be pushing, head knocking, or biting the opponent. Delivery or receipt of NRA was recorded when 1 pig received such aggression but did not retaliate. The NRA occurred in the absence of reciprocal fighting or when 1 participant was resting during an ongoing fight or withdrawing at the end of a fight. Irrespective of how it occurred, all such behavior was recorded as NRA. Three observers used time-lapse video equipment to extract the duration of each behavioral bout to the nearest second. Analysis of three 1-h samples of data showed a significant degree of interobserver agreement ($r = 0.83$, $P < 0.001$).

**Statistical Analysis**

The LC and behavioral traits showed skewed distributions (Table 1). To reduce the skewness, as well as kurtosis, and to approach normality, a log-transformation $Y = \log_e(1 + \text{observation})$ was used for all traits.
All further genetic analyses were based on the transformed data. Additionally, data from the purebred and crossbred animals had to be used in the same genetic analysis to obtain an adequate sample size.

The variance components of the genetic and environmental effects were estimated using the following multiple trait animal model:

$$
y = Xb + Za + Wc + e,
$$  

where $y$ includes the vector of observations of LC and behavioral traits, $V$ includes the fixed effects of line (purebred Yorkshire, crossbred Yorkshire × Landrace), sex (males, castrates, and females), environmental batch (14 separate mixing days reflecting the batch farrowing policy on the farm), and BW at mixing as a covariable fitted using linear regression. The vectors $a$, $c$, and $e$ represent the additive genetic effects, common environmental pen effects (into which the animals were mixed), and the environmental residual effects, respectively. The $X$, $Z$, and $W$ are incidence matrices linking the effects with $y$.

The variance-covariance structure was as follows:

$$
V = \begin{bmatrix}
a & A \otimes G & 0 & 0 \\
0 & I \otimes M & 0 \\
0 & 0 & I \otimes R
\end{bmatrix},
$$

where $G$, $M$, and $R$ represent the (co)variance matrix (each of size $9 \times 9$) due to additive genetic effects; $M$ is the (co)variance matrix due to the common environmental pen effects in which the mixing of animals took place; and $R$ is the (co)variance matrix due to residual effects. The $A$ denotes the additive genetic relationship matrix (2,419 animals), and $I$ is the identity matrix.

Genetic and environmental variance components of model [1] were estimated using REML as implemented in the program VCE-5 (Kovac et al., 2003).

### RESULTS

The strongly skewed distribution shown in the raw data (skewness = −0.8 to 27.3, Table 1) was substantially reduced by log-transformation (−1.51 to −0.06). Furthermore, the kurtosis of the distributions was substantially improved toward a normal distribution. Visual inspection of the normal probability plots also showed great improvement toward normality, although the distribution of the log-transformed traits still deviated significantly from normality using the Shapiro-Wilk test (data not shown). However, it is known that this test is powerful in rejecting the null hypothesis of normality (Shapiro and Wilk, 1965) and that REML is robust to modest deviations from normality.

Males tended to show more reciprocal aggression (0.31 SE = 0.22 log-transformed seconds per 24 h) and to deliver more (0.11 SE = 0.21), but receive less, NRA than females (−0.14 SE = 0.16), but the effect was not significantly different from zero. Moreover, purebred pigs tended to be involved in more reciprocal aggression (0.39 SE = 0.24) and delivery of NRA (0.05 SE = 0.15) than crossbreds, but again the effect was not significantly different from zero. Moreover, purebred/crossbred line were retained in the model to ensure that the genetic analysis was independent of these effects. The differences in phenotypic variances between purebred and crossbred animals were inconsistent in that crossbreds had a 13% greater variance for reciprocal aggression, but 10% less variance for delivery of NRA than purebreds. These differences in variances were nonsignificant ($P < 0.01$), and therefore, it was assumed that they were due to random sampling.

### Heritabilities

The heritabilities of the behavioral traits delivery of NRA and reciprocal aggression were moderate (0.31 and 0.43, respectively; Table 2), whereas that of receipt of NRA was substantially less (0.08). The heritabilities

---

**Table 1. Means, SD, skewness, and kurtosis of the traits on the original scale and on the transformed scale**

<table>
<thead>
<tr>
<th>Trait</th>
<th>Original scale</th>
<th>Transformed scale</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Mean</td>
</tr>
<tr>
<td>LC at mixing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior lesions</td>
<td>1,660</td>
<td>19.1</td>
</tr>
<tr>
<td>Central lesions</td>
<td>1,660</td>
<td>11.3</td>
</tr>
<tr>
<td>Caudal lesions</td>
<td>1,660</td>
<td>4.1</td>
</tr>
<tr>
<td>LC 3 wk postmixing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior lesions</td>
<td>1,658</td>
<td>10.3</td>
</tr>
<tr>
<td>Central lesions</td>
<td>1,658</td>
<td>10.2</td>
</tr>
<tr>
<td>Caudal lesions</td>
<td>1,658</td>
<td>4.5</td>
</tr>
<tr>
<td>Aggressive behavior</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reciprocal aggression</td>
<td>1,184</td>
<td>563</td>
</tr>
<tr>
<td>Delivery of NRA</td>
<td>1,184</td>
<td>82</td>
</tr>
<tr>
<td>Receipt of NRA</td>
<td>1,184</td>
<td>57</td>
</tr>
</tbody>
</table>

1. Units of measurement were number of lesions/pig [lesion count (LC) traits] and seconds/pig during 24 h (behavioral traits).
2. Kurtosis is presented as 4th moment minus 3.
3. NRA = nonreciprocal aggression.
of the 3 LC traits measured 24 h postmixing were all of moderate magnitude (0.21 to 0.26). With the exception of lesions received to the caudal area of the body, theheritabilities of the LC traits were greater at 3 wk than 24 h after mixing and were comparable with those of thebehavioral traits reciprocal aggression and delivery of NRA.

**Genetic and Residual Correlations**

Genetic and residual correlations among the LC andbehavioral traits are shown in Table 2. The genetic correlation ($r_g$) between reciprocal aggression and the receipt of NRA was not significantly different from zero, whereas that between reciprocal aggression and the delivery of NRA ($r_g = 0.84$) was highly significant ($P < 0.001$), indicating that these traits share a similar genetic basis. An antagonistic genetic association was found between the delivery and receipt of NRA ($r_g = −0.41$), whereas there was a small but positive residual correlation (0.23) between these traits. Although the residual correlation was small, this indicates that random residual environmental effects played a role in encouraging animals that received NRA to be involved in its delivery, whereas there was a genetic antagonism between the behaviors.

Positive genetic correlations among the 3 LC traits 24 h after mixing were of high to very high magnitude ($r_g = 0.69$ to 0.98), particularly between central and caudal lesions. Lesions to the anterior region of the body were genetically associated with reciprocal fighting ($r_g = 0.67$), receipt of NRA ($r_g = 0.70$), and to a lesser extent, delivery of NRA ($r_g = 0.31$), whereas those to the center and caudal areas were associated primarily with receipt of NRA ($r_g = 0.80$, center; 0.79, caudal). The low residual correlations between all lesion traits and receipt of NRA suggested that the environmental component had little effect on the relationship between these traits.

Genetic correlations between the LC traits measured 3 wk after mixing were also all positive and very high ($r_g = 0.86$ to 0.97). All LC traits measured at this time point were genetically associated with aggressive behavior at mixing, being negatively associated with reciprocal aggression and the delivery of NRA and positively associated with the receipt of NRA. Residual correlations between these traits were again very small and largely not significant.

Low to moderate positive genetic relationships ($r_g = 0.28$ to 0.50) between LC recorded on the same body area 3 wk apart were found, whereas, conversely, the residual correlations were small and negative ($r_e = −0.08$ to −0.15). This implies that the random environmental effects affecting each individual pig immediately after mixing were different from those 3 wk later. In contrast, animals that were genetically predisposed to receive lesions after mixing were also genetically predisposed to receive them 3 wk later.

| Table 2. Heritabilities (on diagonal) and genetic (above diagonal) and residual correlations (below diagonal) between aggressive behavioral traits and lesion count (LC) traits recorded 24 h and 3 wk postmixing |
| LC at mixing | Anterior | Central | Caudal | Anterior | Central | Caudal | Anterior | Central | Caudal | Anterior | Central | Caudal | Anterior | Central | Caudal |
| Reciprocal aggression | 0.52 (0.02) | 0.29 (0.03) | 0.39 (0.03) | 0.29 (0.03) | 0.32 (0.03) | 0.23 (0.03) | 0.13 (0.02) | 0.08 (0.02) | 0.08 (0.02) |
| Delivery of NRA | 0.29 (0.03) | 0.32 (0.03) | 0.23 (0.03) | 0.13 (0.02) | 0.08 (0.02) | 0.08 (0.02) | 0.08 (0.02) | 0.08 (0.02) | 0.08 (0.02) |
| Receipt of NRA | −0.09 (0.03) | −0.08 (0.03) | −0.08 (0.03) | −0.09 (0.03) | −0.08 (0.03) | −0.08 (0.03) | −0.09 (0.03) | −0.08 (0.03) | −0.08 (0.03) |

NRA = nonreciprocal aggression. SE values are presented in parentheses.
**Pen Effects**

The proportions of the phenotypic variance due to the pen effect (the group into which mixing took place) were small and of similar magnitude for the LC and behavioral traits, ranging from 0.04 to 0.13 (Table 3). The environmental correlations between the LC traits 24 h postmixing and the behavioral traits that could be attributed to the pen effect were largely in the same direction as the genetic correlations, but generally not as large. However, although the genetic correlations indicated that the LC on all 3 parts of the body in the 3 wk postmixing were positively associated with receipt of NRA, the pen environmental correlations between these traits were small.

**DISCUSSION**

**Behavioral Traits**

The 3 behavioral traits relating to aggression during the 24 h postmixing studied here had heritabilities very similar to those previously estimated using Bayesian methodology in a different population by Turner et al. (2008). The moderate heritabilities for delivery of NRA and reciprocal aggression are within the range described for offensive aggression in pigs (postmixing fighting in sows $h^2 = 0.17$ to 0.24, Lövendahl et al., 2005) and other species (male rodents $h^2 = 0.22$ to 0.34, Miczek et al., 2001; fighting bulls, $h^2 = 0.30$, Silva et al., 2006; aggressive antisocial behavior in humans $h^2 = 0.46$, Eley et al., 2003). Conversely, a low heritability of 0.08 was found for receipt of NRA, in agreement with the lower heritability reported for receipt of aggression than delivery of aggression by Lövendahl et al. (2005). This behavior was observed during, or at the end of a reciprocal fight, or in the absence of fighting. Therefore, unlike the other 2 behaviors, receipt of NRA encompassed 3 scenarios, 2 of which involved reciprocal fighting. Although the aggression received in each of these scenarios was similar, the social context was not and may have contributed toward its lower heritability. A positive residual correlation between reciprocal aggression and receipt of NRA was found, whereas no genetic correlation was evident. This may reflect those scenarios in which the behaviors occurred together.

A strong genetic correlation (0.84) indicated that pigs involved in reciprocal aggression also delivered NRA. Turner et al. (2008) reported a similar genetic correlation (0.79) in pigs, and van Oortmerssen et al. (1985) found that reciprocal aggression in mice and the chasing of other individuals responded simultaneously to selection, supporting the view that these traits partially share a common genetic basis. In the current study, delivery and receipt of NRA showed a significant genetic antagonism with a negative correlation of $-0.41$ (SE = 0.14). This is slightly different from the findings of Turner et al. (2008), where the receipt of NRA appeared to be genetically independent from delivery of NRA or reciprocal aggression. Discrepancies between

**Table 3. Phenotypic proportions of the variance due to the effect of the pen into which the pigs were mixed together (on diagonal) and their correlations**

<table>
<thead>
<tr>
<th>Trait</th>
<th>LC at mixing</th>
<th>LC at postmixing</th>
<th>Reciprocal aggression</th>
<th>Delivery of NRA</th>
<th>Receipt of NRA</th>
</tr>
</thead>
<tbody>
<tr>
<td>LC at mixing</td>
<td>0.00(0.01)</td>
<td>-0.09(0.06)</td>
<td>-0.11(0.05)</td>
<td>0.20(0.07)</td>
<td>-0.24(0.10)</td>
</tr>
<tr>
<td>LC at postmixing</td>
<td>0.61(0.04)</td>
<td>0.12(0.01)</td>
<td>0.32(0.07)</td>
<td>0.38(0.09)</td>
<td>0.24(0.07)</td>
</tr>
<tr>
<td>Behavioral trait</td>
<td>0.08(0.01)</td>
<td>0.11(0.01)</td>
<td>0.91(0.05)</td>
<td>0.91(0.05)</td>
<td>0.11(0.01)</td>
</tr>
</tbody>
</table>

LC = lesion count; NRA = nonreciprocal aggression. SE values are presented in parentheses.
Lesions: Genetic indicator of aggressiveness?

Turner et al. (2008) and the present findings are likely to have resulted from the use of a different population, the larger sample size, or wider definition of aggressive behavior that included noninjurious pushing in the present study. In contrast to the genetic association, there was a small but positive residual correlation between the delivery and receipt of NRA (0.23) in the present population, suggesting that animals that received NRA were influenced by random residual environmental effects to also deliver NRA to others.

Lesions 24 h Postmixing

The heritability of the LC traits 24 h after mixing were of moderate magnitude and similar to those estimated by Turner et al. (2008), although that for caudal lesions was greater than estimated previously (0.21 vs. 0.12). Lesions to the front of the body were positively genetically associated with all 3 behavioral traits and, therefore, if used in isolation, were poor at discriminating between aggressive and unaggressive pigs. Bites are typically targeted toward the head, neck, shoulders, and ears during reciprocal fighting, but are usually received on the rear of a retreating animal (McGlone, 1985; Rundgren and Lönquist, 1989; Weary and Fraser, 1999). In view of this, the strongly positive correlation between anterior lesions and receipt of NRA may seem counterintuitive. However, these anterior lesions may have accumulated during temporary breaks in ongoing reciprocal fights during which the recipient was recorded as receiving NRA. Alternatively, they may have resulted from engagement in reciprocal aggression, which was subsequently followed by retreat and the receipt of NRA. Although the space allowance per pig was generous by commercial standards, the pen dimensions may also have inhibited retreat and contributed to the accumulation of lesions on the front of withdrawing animals.

Central and caudal LC were genetically highly correlated (\(r_g = 0.98\)) and are thus essentially the same trait, suggesting that only 1 needs to be measured or that they could be combined for future use. Lesions to these areas were strongly and positively associated with the receipt of NRA, but showed only small or negative correlations with reciprocal aggression or the delivery of NRA. Central and caudal lesions are therefore good predictors of the receipt of NRA and are probably the most efficient at identifying animals that receive bouts of NRA that are independent from sequences of reciprocal aggression. A genetic merit index using anterior lesions as one trait and central or caudal lesions (or both) as a second trait should allow selection against aggression postmixing may have long-term benefits in reducing injuries from later aggression, possibly operating through the increased stability of dominance relationships formed during this early phase.

Lesion counts offer a practical and validated indicator of aggressive behavior after mixing. Counting lesions requires the use of no specialist equipment and takes under 1 min in most animals, making it considerably less time-consuming than behavioral observations made directly or from video images. The analysis has shown that skin lesions are genetically associated with the sum of injurious and noninjurious aggressive behavior, both of which may be perceived as stressful by the animal and could affect welfare and performance.
The use of a genetic merit index that applies pressure on anterior lesions as 1 trait and central or caudal lesions, or both, as a second trait ought to allow selection against pigs that become involved in reciprocal fighting and the delivery of NRA and is expected to result in a correlated reduction in lesions received under more stable social conditions. Selection may result in correlated changes in other traits of welfare or economic significance, and the genetic associations between LC traits and other traits of interest should be estimated before selection is implemented. Three further papers will describe the associations between aggression and nonsocial behaviors, productivity, and reproductive performance to allow the desirability of selection to be more widely appraised. Selection should, however, be accompanied by continued efforts to find economically viable and effective nongenetic solutions to aggression.

LITERATURE CITED


Grain feeding significantly (P<0.01) increased average daily gain, carrying capacity and lamb production per acre over the other three treatments. Alfalfa-bromegrass rotationally grazed over three paddocks resulted in significantly (P<0.05) greater lamb production per acre than bromegrass 3X or alfalfa-bromegrass 2X. Daily gain was not significantly affected by plant species or number of rotational paddocks. Bromegrass pasture produced more forage during the second than the first year.

Literature Cited


ERRATA


The last line of table 2, page 427, under P0, P1, and P2 should be:

\[ C_n = \frac{1}{2} + \frac{1}{2^3} \left(-\frac{1}{2}\right)^{n+1} \]


In table 1, page 632, the definition of k2 and k3 should be as follows:

\[
k_2 = \frac{1}{s-f} \left[ \frac{1}{i} \sum \frac{1}{n} \sum n^2_{ijk} - \frac{1}{i} \sum \frac{1}{n_i} \sum n^2_{ijk} \right]
\]

\[
k_3 = \frac{1}{s-f} \left[ T - \frac{1}{i} \sum n^2_{ij} \right]
\]

The results in the paper are correct as given since all results are based on the coefficients above rather than as they originally appeared.
criteria to improve prediction of weaning performance.

Literature Cited

ERRATUM
ERRATUM

Johnson, Ronald R., E. W. Klosterman and H. W. Scott. 1962. Studies on the feeding value of soybean flakes for ruminants. J. Animal Sci. 21:406. Table 5, below, was omitted from this paper. The table should be inserted on page 411.

TABLE 5. FEEDING VALUE OF SOYBRAN FLAKES IN A WINTERING RATION FOR BEEF CALVES

<table>
<thead>
<tr>
<th>Housing</th>
<th>Barn</th>
<th></th>
<th></th>
<th></th>
<th>Shed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ration</td>
<td>Hay, silage, corn</td>
<td>Hay, silage, SBF</td>
<td>Hay, silage, corn</td>
<td>Hay, silage, SBF</td>
<td></td>
</tr>
<tr>
<td>Lot no.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>No. in lot</td>
<td>16</td>
<td>16</td>
<td>24</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Av. wt. Nov. 29, lb.</td>
<td>458</td>
<td>468</td>
<td>455</td>
<td>458</td>
<td></td>
</tr>
<tr>
<td>Av. wt. Apr. 15, lb.</td>
<td>664</td>
<td>711</td>
<td>640</td>
<td>666</td>
<td></td>
</tr>
<tr>
<td>Av. daily gain 137 days, lb.</td>
<td>1.51</td>
<td>1.78*</td>
<td>1.38</td>
<td>1.52</td>
<td></td>
</tr>
<tr>
<td>Av. daily ration:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ground ear corn, lb.</td>
<td>4.0</td>
<td>4.0</td>
<td>4.0</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>Soybean flakes, lb.</td>
<td>5.0</td>
<td>5.0</td>
<td>5.0</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>Hay-crop silage, lb.</td>
<td>15.0</td>
<td>7.5</td>
<td>15.0</td>
<td>15.0</td>
<td></td>
</tr>
<tr>
<td>Mixed hay, lb.</td>
<td>5.0</td>
<td>2.5</td>
<td>5.0</td>
<td>5.0</td>
<td></td>
</tr>
<tr>
<td>Salt, oz.</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Minerals, oz.</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
<td>0.4</td>
<td></td>
</tr>
<tr>
<td>Feed per cwt. gain, lb.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ground ear corn</td>
<td>258</td>
<td>223</td>
<td>287</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soybean flakes</td>
<td>279</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hay-crop silage</td>
<td>988</td>
<td>419</td>
<td>1078</td>
<td>977</td>
<td></td>
</tr>
<tr>
<td>Mixed hay</td>
<td>329</td>
<td>140</td>
<td>359</td>
<td>326</td>
<td></td>
</tr>
<tr>
<td>Salt</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Minerals</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

* Significantly (P<.05) different from Lot 1.
ERRATUM

Christiansen, Wm. C., L. Y. Quinn and Wise Burroughs. 1962. Multiple-tube laboratory technique for studying volatile fatty acid production by rumen protozoa. J. Animal Sci. 21:707. The units in table 2 should be gm./l. not mg./l.
ERRATA

Lasley, J. F., B. N. Day and D. T. Mayer. 1963. Intra-uterine migration and embryonic death in swine. J. Animal Sci. 22:424. First sentence of next to last paragraph in second column should read: "Litters contained 1.24 more pigs where intra-uterine migration was observed and 1.73 fewer corpora lutea not represented by embryos where intra-uterine migration was observed."

Griffin, S. A. and G. M. Merriman. 1963. Thiofuradene as an anthelminthic and growth stimulant for swine. J. Animal Sci. 22:577. In table 4, the figure in the last column opposite: "No. pigs free of ascarids" should be 8 instead of 0. The figure in the next to last column opposite: "Immature-Range" should be 0–30 instead of 2–30.

Heaney, D. P., W. J. Pigden, D. J. Minson and G. I. Pritchard. 1963. Effect of pelleting on energy intake of sheep from forages cut at three stages of maturity. J. Animal Sci. 22:756. First sentence of the summary: "Pelleting decreased energy digestibility of energy intake (Kcal./WKu 0.76) by wether lambs for all forages at all stages of maturity, with the effects of pelleting becoming more marked with successive growth stages," is in error and should read: "Pelleting increased ad libitum digestible energy intake . . . growth stages."
ERRATA


In table 2 estimating equation:

\[ Y = 18.03 + 7.94X \]
\[ Y = 16.58 + 1.68X \]
\[ Y = 25.36 + 1.17X \]
\[ Y = 46.19 + 0.67X \]
\[ Y = 45.69 + 1.54X \]
\[ Y = 25.28 + 0.75X \]

should read:

\[ Y = 50.16 - 7.94X \]
\[ Y = 84.95 - 1.68X \]
\[ Y = 93.32 - 1.17X \]
\[ Y = 55.91 - 0.67X \]
\[ Y = 56.40 - 1.54X \]
\[ Y = 76.79 - 0.75X \]

In table 5 estimating equation:

\[ Y = -209.95 - 236.78X \]
\[ Y = -201.54 - 228.43X \]

should read:

\[ Y = 278.27 - 236.78X \]
\[ Y = 269.79 - 228.43X \]


Estimated initial weight \( W = W_2 - W_1 \), instead of \( W = W_2 - W_1 \).

Chalupa, William, J. L. Evans and M. C. Stillions. 1964. Influence of ethanol on rumen fermentation and nitrogen metabolism. J. Animal Sci. 23:802. The first sentence under Results and under Summary should read: Varying levels of ethanol (ml. per artificial rumen) were added \( \ldots \), instead of: Varying levels of ethanol (ml./100 gm. artificial rumen DM) were added \( \ldots \).
ERRATA


In table 3, page 349:

For phenotypic covariance of carcass grade with dressing percent; change 1.122 to —1.122.

For genetic covariance of carcass grade with area of rib eye; change —.0509 to 0.0509.

For phenotypic covariance of carcass grade with area of rib eye; change 0.4729 to —.4729.


In table 2, page 670:

Liver tissue analysis, vitamin E, mg./100 gm.; change 58.1, 51.9, 62.7, 62.0, 57.2, 65.0, 69.4, 67.1 and 66.6 to 7.3, 6.5, 7.8, 7.8, 7.2, 8.1, 8.7, 8.4 and 8.3, respectively.


On page 681, line 6 of the right hand column, change sections to selections.

On page 684, line 7 of the left hand column, change 877 months to 87 months.