Cholecystokinin Octapeptide Immunization: Effect on Growth of Barrows and Gilts

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ABSTRACT: A study was conducted to validate the previously reported growth response to cholecystokinin octapeptide (CCK-8) immunization in barrows and was extended to include gilts. Group-penned barrows and gilts were used to represent conditions in the swine industry. Thirty-two animals, 19 barrows and 13 gilts, were randomly assigned by sex to four pens and two treatments. The control groups were immunized with human serum globulin (hSG). The treated groups (CCK) were immunized with the C-terminal octapeptide of cholecystokinin conjugated to human serum globulin. Specific binding of CCK-8 was confirmed at 29 d after the primary inoculation. Antisera titers were highly variable throughout. The mean titer reached a peak on d 57 and then declined. Body weight gains during the last 49 d, the period during which titers were expressed, were compared by ANOVA. The treatment effect on gain was significant \( (P = .018) \); the sex effect approached significance \( (P = .071) \); the treatment \( \times \) sex interaction effect was not significant \( (P = .82) \). Least squares mean gain of the CCK group was 8.4% greater than of the hSG group, 41.4 vs 38.2 kg, respectively. A significant linear regression coefficient for gain vs antisera titer was obtained for barrows \( (P = .03; r^2 = .44) \) but not for gilts. Several carcass variables showed trends similar to that of BW gain, but the treatment effects were less robust \( (P < .05 \text{ to } .10) \). These results generally confirm the findings of the previous study; CCK-8 immunization stimulated growth of barrows by 7.5% in the present and by 10.8% in the previous study. The CCK-8 immunization seemed to stimulate growth of gilts by 9.3% in the present study.

Key Words: Appetite Control, Feed Intake, Growth, Growth Factors, Satiety, Cholecystokinin

Introduction

Ad libitum feed intake is not sufficient to realize the full growth potential of swine; superalimentation of young swine at 30% above the ad libitum intake of littermate control pigs increased the rate of gain by 40% (Pekas, 1985). The large response to superalimentation illustrated the benefits that could be obtained from an increase of appetite and voluntary feed intake in swine. Appetite regulation in the growing pig is precise (Pekas, 1983a,b). However, the mechanisms involved in appetite regulation are not known. Exogenous cholecystokinin (CCK) suppressed feed intake in rats (Gibbs et al., 1973). The findings from numerous studies exploring the role of CCK in appetite regulation have been reviewed (Mutt, 1980; Thompson, 1980; Walsh, 1981). Studies of the physiological role of CCK in feed intake control in pigs are scarce (Kidder and Manners, 1978; Houpt, 1982). Injections of porcine CCK to fasted pigs caused a dose-dependent suppression of feed intake (Anika et al., 1981). The first attempt to neutralize circulating CCK in swine, to inhibit CCK suppression of appetite, increased feed intake by 8.2% and BW gain by 10.6% during a 35-d period in which antisera titers were highest (Pekas and Trout, 1990). The efficiency of gain was not significantly affected, however.

The purposes of the present study were 1) to confirm the growth response of barrows to cholecystokinin octapeptide (CCK-8) immunization, as observed in our first experiment, 2) to extend the observations to gilts, and 3) to examine whether such
Materials and Methods

Thirty-two crossbred pigs (Landrace, Yorkshire, Chester White, Large White), 19 barrows and 13 gilts, 73 d average age and 20.9 kg average weight, were used. This confirmation study was conducted with barrows and gilts mixed in group pens to ensure that used. This confirmation study was conducted with barrows and gilts mixed in group pens to ensure that growth response was expressed when CCK-8-immunized animals competed directly with carrier (human serum globulin; hSG)-immunized control animals in the same pen.

The pigs received a conventional 18% CP corn-soybean swine diet (Table 1). Body weight was recorded on d 1 and at 7-d intervals throughout the study to compute planned statistical comparisons of BW and gain during the period of expression of measurable titers, as in our previous study.

The hSG-immunized control group, nine barrows and seven gilts, and the cholecystokinin octapeptide (CCK)-immunized group, 10 barrows and six gilts, were inoculated according to the following immunization protocol. Primary inoculation was by subdermal-subcutaneous injection at three sites on the posterior-ventral abdominal wall on d 1 (d 1 denotes the 1st d of the experiment). Booster inoculations were on d 15, 29, and 43. Blood samples were drawn from the vena cava on d 1, 15, 29, 43, 57, 71, and 92. Serum was separated after centrifugation and stored at -20°C until antisera titer analysis.

The CCK-8-hSG and hSG antigens were prepared by glutaraldehyde conjugation as described by Pekas and Trout (1990). The final vaccines were prepared by ultrasonic emulsification of equal volumes of conjugation reaction fluid and of Freund’s complete adjuvant. Each dose of CCK-8 vaccine contained 1 mg of CCK-8-hSG conjugate in 1 mL; each dose of hSG vaccine contained only an equivalent amount of hSG. Freund’s incomplete adjuvant was substituted in the booster vaccines.

Antibody titers were calculated from the specific binding of $^{125}$ICCK-8, desulfated, measured at four dilutions in .01 M PBS, 1:10, 1:100, 1:1,000, 1:10,000. The $^{125}$ICCK-8 was prepared and the binding assay was conducted as previously described (Pekas and Trout, 1990). Antisera titer in this report is the serum dilution that would give 50% specific binding of the radioligand, and was computed using a simple linear regression. Cross-reactivity of selected samples of antisera with gastrin was examined by estimating the specific binding titer against $^{125}$Igastrin (Dupont, Boston, MA) using the same dilutions, buffer, and assay method as for $^{125}$ICCK-8.

The animals were slaughtered in an abattoir. Weights of the animals before slaughter and of the fresh and chilled carcasses were measured. Other measurements were recorded from the right side of each chilled carcass: weight, length from first rib to tip of pelvic bone, and backfat thickness over the first rib, last rib, and last lumbar vertebra. The loin was sectioned between the 10th and 11th ribs, the exposed muscle was traced, and the area was measured. Weights of the five wholesale cuts (ham, loin, Boston butt, picnic, and belly) were measured three times: raw wholesale cut, after exterior fat was removed, and after bone was removed.

Variables were compared by ANOVA in accordance with the randomized complete block design, with subsampling, in which pen was the block. The source for animal (treatment × sex × pen) was used to test the effects of treatment, sex, and pen and of their interactions. Computer software by SAS (1989) was used for statistical computations. The Proc GLM was used for the analysis as required for the unbalanced design. Linear regressions were computed to describe relationships between BW gain vs antisera titer.

Results

Table 1. Composition of the diet

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn</td>
<td>55.8</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>25.0</td>
</tr>
<tr>
<td>Oats</td>
<td>10.0</td>
</tr>
<tr>
<td>Whey</td>
<td>5.0</td>
</tr>
<tr>
<td>Dicalcium phosphate</td>
<td>2.4</td>
</tr>
<tr>
<td>Limestone</td>
<td>.8</td>
</tr>
<tr>
<td>Iodized salt</td>
<td>.4</td>
</tr>
<tr>
<td>Choline chloride</td>
<td>.2</td>
</tr>
<tr>
<td>Vitamin premix</td>
<td>.2</td>
</tr>
<tr>
<td>Trace mineral premix</td>
<td>.2</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Materials and Methods

Thirty-two crossbred pigs (Landrace, Yorkshire, Chester White, Large White), 19 barrows and 13 gilts, 73 d average age and 20.9 kg average weight, were used. This confirmation study was conducted with barrows and gilts mixed in group pens to ensure that space and management conditions were typical of those in the swine industry, in contrast to the small pens and intense laboratory atmosphere of our first study. Animals were randomly assigned by sex to four pens and then to two treatments within pen. Eight additional animals from the same animal stock were also randomly assigned to each pen to obtain the minimum space recommended for the swine industry. Each 2.1-m x 9.6-m pen had a 2.1-m x 3.1-m slatted floor over an automatic flush pit at one end and a solid concrete slab floor at the other end. One self-feeder, with four feeder spaces, was situated over the solid floor in each pen. Therefore, feed intake of individual pigs or of treatment groups could not be measured. The pigs received a conventional 18% CP corn-soybean swine diet (Table 1). Body weight was recorded on d 1 and at 7-d intervals throughout the study to compute gain after measurable antisera titers were expressed and to compute planned statistical comparisons of BW and gain during the period of expression of measurable titers, as in our previous study.

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Results

The mean of specific binding of radiolabeled CCK-8 by 1:10 diluted serum samples on d 15 for all CCK-immunized animals was 3.7% (n = 16, SE .96), for the
barrows was 2.6% (n = 10, SE .89), and for the gilts was 5.6% (n = 6, SE 1.98). Antisera titers were estimated for most animals immunized against CCK on d 29 and 43. Titers > 5 (1:5) were rare before d 43. The mean titer reached a peak on d 57 and then declined (Figure 1). Variation of the CCK-8 antisera titers was high; the CV was 3.7%, n = 16, SE = .96. Specific binding of the radioligand was not detected in any serum samples from hSG control pigs at 1:10 dilution.

As pointed out in the first analysis without the covariate, the treatment × day interaction was significant because the rate of gain for the CCK and hSG groups shifted in the middle of the study and, therefore, the shift coincided with the period during which antisera titers exceeded 1:10 values. The planned statistical test of the effect of CCK-8 immunization on growth required that gain be compared during the period in which antisera titers were measurable. Body weight gain during the period of titer expression was adjusted for the significant difference of BW in the period before titer expression by including the covariate described above. Gain during the period from d 43 to 92, 49-d gain, was computed and analyzed by ANOVA. The model included treatment, sex, pen, and treatment × sex effects and treatment × sex × pen was the error term.
Figure 2. (A) Graph of the covariate-adjusted least squares means of BW for the four treatment × sex subgroups vs day on experiment. The covariate was BW on d 43. The ANOVA model included treatment, sex, pen, day, and treatment × sex × day effects. (B) (inset) Graph of the unadjusted least squares means of BW of the same four subgroups. Note spread of means in the period from d 1 to 43, before antisera titers were measurable.

The results are summarized in Table 2. This evidence confirms that CCK-8 immunization increased gain and that barrows and gilts responded in a parallel manner. The least squares mean gain during the 49-d evaluation period was 41.4 kg for the CCK animals and 38.2 kg for the hSG animals, which represents an 8.4% increase of gain from CCK-8 immunization. The sex effect approached significance; the treatment × sex interaction was not significant.

Wide variation of antisera titers among the CCK animals provided an opportunity to examine the relationship between antisera titer and BW gain. The relationship was examined by linear regression as in the previous study (Pekas and Trout, 1990). The area under the curve, of titer plotted against day on experiment (see Figure 1), for the 63-d period (d 29 to 92) was computed and expressed in titer-day units (TD/63 d). The regression equation of BW gain (kg/63 d) vs titer (TD/63 d) for the barrows was gain (kg/63 d) = 52.01 + .001549(TD/63 d); the regression coefficient (slope) was greater than zero (P = .03) and the variation explained by the regression gave $r^2 = .44$. The equation for gilts was gain (kg/63 d) = 52.97 − .000545(TD/63 d); the slope was not different from zero (P = .41) and the variation explained by the regression gave $r^2 = .17$.

Carcass measurements were compared using an ANOVA model with treatment, sex, pen, and treatment × sex effects, and treatment × sex × pen was the error term. Body weight on d 43 was included as a linear covariate to adjust for differences of BW during the period before antisera titers were measurable. A preliminary analysis revealed the tendency for carcass shrinkage (weight lost from the fresh carcass from chilling until dissection) to be different between treatment and sex groups (P < .07). Shrinkage was 7.9% greater for CCK than for hSG animals and 6.5% greater for gilts than for barrows. Consequently, shrinkage was included in the final analysis as a linear covariate across treatments for only the variables measured from chilled carcasses. The results are shown in Table 3. Although several carcass variables indicated that differences between treatments and sex paralleled differences in BW, the probabilities ranged from P < .05 to .10.

For example, nine variables tended to be different between treatments. These variables included live BW, fresh carcass weight, chilled carcass weight, and chilled carcass length, and several variables from dissection of the chilled carcass (weight of whole, trimmed, and deboned picnic, and weight of whole and trimmed loin). For each of these nine examples, measurement values were greater for CCK than for hSG animals or carcasses. There were also indications of differences between sexes; live body, whole belly, whole Boston butt, and skin tended to be heavier from barrows than from gilts. However, longissimus muscle area seemed to be larger for gilts than for barrows.

Twelve carcass and dissected carcass variables gave probability values in the range of P < .05 to .10 for treatment × sex interactions, which may indicate that CCK-8 immunization affected barrows and gilts differently. For example, weight of trimmed and of deboned loin, weight of whole, trimmed, and deboned ham, total weight of lean dissected from the carcass, the lean proportion of the carcass weight, and the sum weight of four preferred cuts (ham, loin, Boston butt, picnic), both trimmed and deboned, were each greatest for CCK barrows, least for hSG barrows, and intermediate for CCK gilts and hSG gilts. Longissimus muscle area was largest for hSG gilts, smallest for hSG barrows, and intermediate for CCK barrows and CCK gilts. Thickness of backfat over the first rib was greatest for CCK barrows, followed by hSG gilts, then hSG barrows, and least for CCK gilts. Weight of leaf fat (fat surrounding kidneys) was greatest for hSG barrows, followed by CCK gilts, and least for CCK barrows and hSG gilts.
Table 2. Least squares means of body weight gain [kg] of hSG and CCK barrows and gilts from day 43 to 92 (body weight on day 43 was included as a linear covariate across treatments)\(^a\)

<table>
<thead>
<tr>
<th></th>
<th>Treatment</th>
<th>Residual SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>hSG</td>
<td>CCK</td>
<td></td>
</tr>
<tr>
<td>Barrows</td>
<td>39.43</td>
<td>42.37</td>
<td>4.44</td>
</tr>
<tr>
<td>Gilts</td>
<td>36.95</td>
<td>40.40</td>
<td>—</td>
</tr>
</tbody>
</table>

\(^a\)Animals were immunized against human serum globulin (hSG) or cholecystokinin octapeptide (CCK).

Discussion

All CCK-8-immunized animals in this study showed specific binding (i.e., bound radiolabeled CCK-8 in excess of nonspecific binding) after d 29. The antisera titers were highly variable in this study, as in our previous study (Pekas and Trout, 1990). The peak titer occurred approximately 14 d later, at d 57 in this study, compared with d 43 in our previous study, and the level of specific binding seemed to be lower in the

Table 3. Least squares means for weights [kg] of carcass variables listed by treatment × sex subgroups. Body weight on day 43 was included as a linear covariate across treatments for all variables\(^a\)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Treatment (sex)</th>
<th>Residual SD</th>
<th>Covariate</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CCK(B)</td>
<td>CCK(G)</td>
<td>hSG(B)</td>
<td>hSG(G)</td>
</tr>
<tr>
<td>Live weight</td>
<td>90.8</td>
<td>88.2</td>
<td>87.5</td>
<td>85.2</td>
</tr>
<tr>
<td>Fresh carcass</td>
<td>60.0</td>
<td>58.1</td>
<td>57.2</td>
<td>56.3</td>
</tr>
<tr>
<td>Chilled carcass wt</td>
<td>57.7</td>
<td>55.9</td>
<td>54.2</td>
<td>54.1</td>
</tr>
<tr>
<td>Chilled carcass length, cm</td>
<td>75.3</td>
<td>75.1</td>
<td>74.4</td>
<td>73.8</td>
</tr>
<tr>
<td>Whole ham</td>
<td>7.31</td>
<td>6.96</td>
<td>6.76</td>
<td>7.11</td>
</tr>
<tr>
<td>Trimmer ham</td>
<td>5.87</td>
<td>5.59</td>
<td>5.20</td>
<td>5.84</td>
</tr>
<tr>
<td>Deboned ham</td>
<td>5.16</td>
<td>4.92</td>
<td>4.51</td>
<td>5.16</td>
</tr>
<tr>
<td>Whole loin</td>
<td>6.93</td>
<td>6.72</td>
<td>6.23</td>
<td>6.49</td>
</tr>
<tr>
<td>Trimmer loin</td>
<td>5.17</td>
<td>5.03</td>
<td>4.51</td>
<td>5.09</td>
</tr>
<tr>
<td>Deboned loin</td>
<td>4.19</td>
<td>4.08</td>
<td>3.58</td>
<td>4.13</td>
</tr>
<tr>
<td>Whole Boston butt</td>
<td>3.60</td>
<td>3.28</td>
<td>3.58</td>
<td>3.51</td>
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<tr>
<td>Trimmer Boston butt</td>
<td>2.88</td>
<td>2.63</td>
<td>2.84</td>
<td>2.88</td>
</tr>
<tr>
<td>Deboned Boston butt</td>
<td>2.71</td>
<td>2.46</td>
<td>2.67</td>
<td>2.72</td>
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<tr>
<td>Whole picnic</td>
<td>2.89</td>
<td>2.81</td>
<td>2.64</td>
<td>2.96</td>
</tr>
<tr>
<td>Trimmer picnic</td>
<td>2.70</td>
<td>2.56</td>
<td>2.47</td>
<td>2.52</td>
</tr>
<tr>
<td>Deboned picnic</td>
<td>2.23</td>
<td>2.13</td>
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<tr>
<td>Whole belly</td>
<td>3.08</td>
<td>2.87</td>
<td>2.97</td>
<td>2.61</td>
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<tr>
<td>Trimmer belly</td>
<td>1.86</td>
<td>1.81</td>
<td>1.76</td>
<td>1.73</td>
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<tr>
<td>Kidney leaf fat</td>
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<td>.612</td>
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<td>.561</td>
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<tr>
<td>Backfat, cm</td>
<td>3.95</td>
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<tr>
<td>First rib</td>
<td>1.85</td>
<td>1.57</td>
<td>1.80</td>
<td>1.56</td>
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<tr>
<td>Last rib</td>
<td>1.87</td>
<td>1.95</td>
<td>1.60</td>
<td>1.35</td>
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<tr>
<td>Last lumbar</td>
<td>30.9</td>
<td>29.8</td>
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<tr>
<td>Longissimus muscle area, cm(^2)</td>
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<td>19.8</td>
<td>19.2</td>
<td>19.8</td>
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<td>Preferred wholesale cuts</td>
<td>16.6</td>
<td>15.8</td>
<td>15.0</td>
<td>16.3</td>
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<tr>
<td>Four, whole</td>
<td>14.3</td>
<td>13.6</td>
<td>12.8</td>
<td>14.1</td>
</tr>
<tr>
<td>Four, trimmed</td>
<td>7.13</td>
<td>7.06</td>
<td>7.08</td>
<td>6.96</td>
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<tr>
<td>Total carcass bone</td>
<td>14.0</td>
<td>13.9</td>
<td>14.3</td>
<td>11.6</td>
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<td>Total carcass fat</td>
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<td>34.9</td>
<td>32.9</td>
<td>35.5</td>
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<td>Carcass bone, %</td>
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<td>12.7</td>
<td>13.0</td>
<td>12.9</td>
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<tr>
<td>Carcass fat, %</td>
<td>24.3</td>
<td>24.5</td>
<td>26.3</td>
<td>21.5</td>
</tr>
<tr>
<td>Carcass lean, %</td>
<td>63.2</td>
<td>62.6</td>
<td>60.5</td>
<td>65.5</td>
</tr>
</tbody>
</table>

\(^a\)Abbreviations: Trt = treatment; S = sex; T x S = treatment × sex interaction; CCK = CCK-immunized treatment animals; hSG = human serum globulin-immunized control animals; B = barrows; G = gilts; Preferred wholesale cuts (ham, loin, Boston butt, picnic); Whole = whole retail cut; Trimmed = exterior fat trimmed to lean; Deboned = deboned after trimmed. Thickness and length measurements are as centimeters, area measurements as square centimeters.
present study. The average BW of CCK animals in this study was 9% lighter than that of hSG animals on d 1, a coincidental result of the random assignment of animals to treatment. Consequently, BW gain would be expected to be slower for CCK than for hSG animals in the first part of the study before antisera titers developed. Unfortunately, this complicated the planned comparisons of BW and of BW gain during the period in which antisera titers were expressed.

The CCK-8 immunization stimulated growth of barrows in the present study and in previous research and seemed to stimulate growth of gilts in the present study. The growth response was delayed, and it occurred after titers were expressed in both studies; the magnitude of the growth response to CCK-8 immunization also was similar in the two studies. The growth response is considered to be a direct result of increased feed intake, as demonstrated in our first study, although it was not measured in the present study.

The significant treatment effect \((P = .018)\), but not sex or treatment \(\times\) sex interaction effects, on BW gain during the 49-d evaluation period confirms the response to CCK-8 immunization of barrows in the first study and indicates that gilts also respond to the immunization. Furthermore, linear regressions of gain vs antisera titer were significant and gave positive regression coefficients for barrows in both studies. The regression equation for the present study predicted that an average titer of 40 during a 63-d period \((TD = 2,500)\) would increase gain by approximately 4 kg per animal. The significant treatment effect and regression equation in this study are in agreement with those in our first study (Pekas and Trout, 1990), in which CCK barrows gained 8.3% more than hSG barrows \((P = .006)\), and the regression equation \((P = .045; r^2 = .34)\) of BW gain vs average binding percentage (during the antisera expression period) predicted that an average binding of 50% (at 1:180 dilution) would increase gain of the CCK-8-immunized barrows by 3.8 kg per animal. We are not able to explain why the same regression analysis for gilts in the present study was not significant. The mean and variation of computed areas under the curve \((TD/63 \, d)\) were greater for gilts than for barrows, 3,844 \((SE = 2,188)\) for gilts vs 1,665 \((SE = 660)\) for barrows. This was because one gilt was exceptional. That gilt had the largest area under the curve of all animals in the study, which was eightfold the mean value of the other five gilts. The regression analyses involved 10 barrows but only six gilts, and might be part of the reason. Moreover, we do not know which epitopes are involved in feed intake regulation, the spectrum of epitopes bound by the antisera of these animals, if differences between experiments or between barrows and gilts exist, or whether the antisera assay we used detected all epitopes involved in feed intake regulation. Perhaps there are minimal and maximal antisera titer thresholds and a linear regression may function only between these thresholds.

Although carcass and carcass dissection data in this study generally supported the statistically significant responses manifest by BW and gain, statistical probabilities for the carcass variables fell in the range of \(P < .05\) to .10 and, therefore, the carcass findings are not as convincing. Carcass responses to CCK-8 immunization must be evaluated further in future studies. The disproportionate shrinkage between carcasses from CCK-immunized and control pigs observed in this study was not encountered in a previous study (Pekas, 1991). Fresh and chilled carcasses were significantly heavier from CCK-immunized pigs than from control pigs and the composition of the carcass tissues was not altered (Pekas, 1991).

Sex and castration have effects on growth in swine that are different from those in cattle and sheep (Kay and Houseman, 1975; Ford and Klindt, 1989). There is considerable variation in growth among boars, barrows, and gilts during ad libitum feed intake. The results of numerous trials, in which at least two of the three sex-castrate groups were represented, were reviewed (Kay and Houseman, 1975) and indicated that barrows would be expected to have greater feed intake and growth than gilts in most, but not all, trials. Therefore, it seems plausible to suggest, because sex and castration can have an effect on feed intake, that the effects of CCK and thus of CCK-8 immunization on feed intake may not always be parallel in barrows and gilts, and that such nonuniform influence may explain why the regression analyses between antisera titers vs gain in the present study were not the same for barrows and gilts. Moreover, suppression of feed intake after CCK administration was more variable in female than in male rats (Wager and Levine, 1988).

Implications

These findings, combined with those of another study, establish that cholecystokinin octapeptide immunization increases growth of barrows. The antibodies produced are thought to neutralize endogenous cholecystokinin, thus inhibiting normal cholecystokinin suppression of feed intake, and in turn increasing feed intake and growth. The finding that cholecystokinin octapeptide immunization increases growth in young swine, presumably by increasing feed intake as in our previous study, represents a potential new management tool for increasing performance and economic returns for the swine industry.

Literature Cited


