Influence of Weaning, Diet Particle Size, and Dietary Zinc Concentration on Growth and Stomach Morphology of Young Pigs

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Stomach ulceration in the pig is a serious health concern in the U.S. swine industry, and often results in pig death or poor finishing pig performance. Ulceration in the pig occurs in the upper region of the stomach near the esophageal opening. This region is not protected by the thick mucous layer which coats the remainder of the pig stomach. Fasting or major reduction in feed intake, pelleting or fine grinding of the diet, crowding, and transportation have all been implicated as factors which may contribute to ulceration in pigs. The factors which have received the most attention are pelleting and fine grinding of the diet. However, we reported in the 1996 Purdue University Swine Day proceedings that fasting appears to be the primary instigator of ulcerative tissue damage in the grow-finish pig, and that diet preparation may play a role in maintaining fasting-induced tissue damage or allowing the tissue to repair. The newly weaned pig goes through a transition from a milk based diet to dry feed, which is often in a finely ground and pelleted form. These factors may contribute to the initiation of stomach ulceration in the pig at a young age. Therefore, the objective of these experiments was to evaluate the influence of weaning, fasting, and diet composition on growth and stomach morphology of the young pig.

Materials and Methods

Diet composition and particle size. Corn-soybean meal based diets were used in all experiments, with the coarse and fine ground diets having mean particle sizes of 750 and 550 microns, respectively. A commercially prepared, pelleted starter diet was also used as a treatment in the first experiment.

Weaning, fasting, and diet particle size. A total of 52 barrows with an initial weight of 18 lb (approximately 21 to 28 days of age) were used. Ten barrows were killed and stomach morphology evaluated immediately after weaning. An additional 10 barrows were placed in separate nursery pens with 8 nonlittermate pigs. Pigs had ad libitum access to a commercially prepared pelleted diet for 72 hours. The 10 pigs were then killed and stomach morphology evaluated. An additional 32 barrows were weaned and placed 2 pigs/pen with nonlittermates and denied access to feed for 24 hours. Following a 24-hour fast, 8 pigs were killed and stomach morphology was evaluated. Sixteen of the remaining 24 pigs were individually housed and fed either a 750 or 550 μ m diet. Pigs were fed .44, .88, 1.32, and 1.76 lb/day in two equal meals. The remaining 8 pigs were assigned to a 550 μ m diet and were fasted for 24 hours every 7 days. Weekly intakes of this fasted group were adjusted to be similar to the unfasted pigs. At the end of the 28-day feeding period, the 24 barrows were weighed and killed, and stomach morphology was evaluated.

Weaning, fasting, diet particle size, and dietary zinc source. A total of 72 barrows with an initial weight of 18 lbs were used. Twelve pigs were killed immediately after weaning and stomach morphology was evaluated. Sixty barrows were weaned, housed 2 pigs/pen, and fasted for 24 hours. Following the 24-hour fast, twelve additional pigs were killed and stomach morphology was evaluated. The remaining 48 pigs were individually housed and assigned to one of six dietary treatments (8 pigs/treatment). Dietary treatments included 750 and 550 µm diets containing 100 and 3,000 ppm Zn from zinc sulfate. Pigs were fed twice daily as described previously. At the end of the 28-day feeding period all pigs were killed to evaluate the influence of diet particle size and zinc level on stomach morphology following fasting.

Results and Discussion

Stomach morphology and appearance. The esophageal region of the pig stomach is actually a continuation of the esophagus which extends down into the stomach. In contrast to the rest of the stomach which is protected by a thick mucous coat, the esophageal region's only defense is a thick layer of epithelium, which is the same type of cells from which skin is derived. Under normal circumstances, this epithelial layer is rather thick, has a pearly white consistency, and is extremely pliable. Ulceration of the esophageal epithelium initially manifests itself as a keratinization, or hardening, of the epithelial layer. As keratinization progresses, the cells begin to absorb a yellow stain from bile which has been refluxed from the small intestine into the stomach. Progression of tissue damage results in erosion, or sloughing off, of the surface epithelial layer. As erosion continues, ulceration develops resulting in bleeding from the esophageal region and swelling of the area around the ulcer. Severe swelling of the esophageal region may result in closure of the esophageal opening.

Weaning, fasting, and diet particle size. Pigs in this experiment were fed diets supplemented with 100 ppm Zn from zinc oxide. Growth performance was not significantly affected by diet particle size; however, pigs fed the 550 µm diets were approximately 2.0 lbs lighter than pigs fed 750 µm diets at the end of the 28 day experiment. Similarly, efficiency of conversion of feed into body weight gain was greater for pigs fed 750 µm diets (1.58 vs. 1.69 for 750 vs. 550 µm diets, respectively). The stomach morphology from pigs killed immediately after weaning was normal. Weaning followed by a 24-hour fast induced tissue keratinization. The stomach morphology from pigs which were weaned into a traditional nursery and allowed ad libitum access to a commercially prepared diet for 72 hours also showed keratinization at a level similar to that induced by the 24-hour fast following weaning. Pigs fed 750 µm diets for 28 days had normal esophageal tissue. Keratinization was present in the stomachs of the pigs fed the 550 µm diets at a level similar to that observed following weaning and the 24-hour fast. Pigs which were repeatedly fasted for 24 hours every 7 days and fed a 550 µm diet had only slightly more keratinization than the pigs fed the 550 µm diets daily. The depression in growth rate and feed efficiency observed in this experiment when a 550 µm diet was fed, while not statistically significant, suggests ulcerative tissue damage in the stomach of the young pig may adversely affect pig performance. Additionally, the 550 µm diet appeared to maintain the level of tissue damage induced by the 24-hour fast, while feeding a 750 µm diet allowed the fasting-induced tissue damage to heal. Furthermore, feeding commercially prepared starter diets may effectively maintain the tissue damage induced by fasting following weaning.

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Weaning, fasting, diet particle size, and dietary zinc source. Growth rate tended (P<.11) to be improved with 3,000 ppm Zn supplementation (.74 vs. .68 lb/day for 3000 vs. 100 ppm, respectively) and with decreasing particle size (.70 vs. .74 lb/day for 750 vs. 550 µm diets, respectively). Efficiency of conversion of feed into body weight gain was improved (P<.05) with 3,000 ppm Zn supplementation (1.54 vs. 1.40 for 100 vs. 3,000 ppm, respectively) and tended to be improved (P<.10) with decreasing particle size (1.51 vs. 1.45 for 750 vs. 550 μ m diets, respectively). As in the previous experiment, the stomach morphology of pigs weaned and immediately killed was normal. Weaning and inducing a 24-hour fast resulted in tissue keratinization. Feeding a 750 µm diet with only 100 ppm supplemental Zn following weaning and a 24-hour fast allowed for healing of fastinginduced tissue keratinization. Feeding 3,000 ppm supplemental Zn did not promote healing of fastinginduced tissue keratinization and appears to have aggravated the keratinization, resulting in severe keratinization with some erosion present in the esophageal region of pigs fed 550 µm diets. As in the previous experiment, 550 µm diets with 100 ppm supplemental Zn appeared to maintain the fastinginduced tissue damage. Although nonsignificant, pigs fed diets supplemented with 3,000 ppm Zn from zinc sulfate had a greater degree of keratinization and erosion of tissue from the esophageal region of the stomach than did pigs fed diets supplemented with Zn from zinc oxide.

Summary

Results of these experiments indicate that the esophageal region of the newly weaned pig stomach is susceptible to tissue damage induced by either a self- or management-induced fast. Furthermore, the fasting induced tissue damage appears to be maintained by a 550 μ m diet, while feeding a 750 μ m diet may allow the tissue to repair. The failure to observe consistent influences of diet particle size on growth and feed efficiency may be attributable to the relatively small number of observations per treatment for a growth assay (n = 8), or due to the confounding factors of dietary Zn supplementation in the second experiment. However, the negative impact of the 550 μ m diet on stomach morphology and on animal performance in the first experiment deserves further investigation. Supplementing nursery diets with 3,000 ppm Zn improves pig performance; however, the apparently negative impact of the supplemental Zn on stomach morphology was unexpected and also deserves further investigation.

Implications

The newly weaned pig often takes a day or two to begin consuming a finely ground and pelleted diet which is supplemented with 2-3,000 ppm supplemental Zn. Understanding the susceptibility of the young pig to these ulcerogenic factors, and the likelihood that the initial stages of ulceration are already present at the end of the nursery phase, highlight the necessity for minimizing the time feed access is restricted during later phases of production. Additionally, these results provide a model from which gastric ulceration in the young pig can be studied, and preventative measures evaluated.