Effect of dietary particle size on gastric ulcers, assessed by endoscopic examination, and relationship between ulcer severity and growth performance of individually fed pigs

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Summary

Purpose: To assess the effects of dietary particle size on the prevalence and severity of gastric ulcers, while monitoring growth performance of individually fed pigs.

Methods: A total of 80 pigs (24.1 ± 2.9 kg, 53.1 ± 6.4 lb) were given ad libitum access to a finely ground, corn/wheat diet (578 ± 1.89 μm) for 2 weeks. All pigs were examined endoscopically, and ulcers were scored according to severity from 0–3: 0 = normal, 1 = parakeratosis, 2 = moderate ulceration, and 3 = severe ulceration. Forty-eight pigs were then selected so that each ulcer score could be represented, and the pigs were individually fed the fine diet for another 7 weeks. After 7 weeks of feeding a fine diet, ulcers were reassessed by endoscopic examination and pigs were then allotted to either the fine diet or a coarse diet (937 ± 2.19 μm) for another 3 weeks. The pigs were endoscopically examined again to observe the effects of feeding a coarse diet on the severity of ulcers. Growth performance, feed consumption, and feed efficiency were monitored throughout the experiment.

Results: Increasing ulcer severity was associated with a linear decrease in average daily gain (P < .05). Feeding a coarse diet for 3 weeks decreased the severity of ulcers (P < .05).

Implications: A change in ration from one of fine particle size to a coarser diet for at least a short period of time appears to be a practical approach to handling a clinical problem of gastric ulcers when the fineness of the feed is a probable cause.

Gastric ulceration is a common problem in the swine industry. It can be a significant cause of death in certain herds. Mortality due to bleeding from deep, erosive lesions in the pars esophageal region of the stomach can reach 1% or higher, with culling losses of 3%–5% in such herds.[1] Recent surveys indicate that the prevalence of lesions in the pars esophageal area of the stomach of slaughter hogs ranges from 32%–65%.[2,3]

There are conflicting reports regarding the association between gastric ulcers and growth performance.[4–6] It is not known whether mild-to-moderate lesions of the surface structure of the pars esophagea are associated with slow growth or reduced feed intakes.

It is well documented that a small dietary particle size increases the prevalence of ulcers compared to coarsely ground diets.[4,6–11] However, there is little information regarding whether changing the feed from a finely ground to a coarsely ground diet will promote the healing of ulcerative lesions.

The usual method of monitoring the prevalence and determining the effects of stomach lesions on growth has been to record gross lesions at slaughter. Endoscopic examination to assess stomach pathology on the live animal has had limited use.[12,13] Because gastric lesions are almost exclusively restricted to the pars esophageal region, we speculated that these lesions could be readily observed using a modern flexible endoscope.

The objective of this study was to determine whether a relationship exists between ulcer severity and growth performance using endoscopic examination to diagnose the ulcers, as well as to investigate the effect of feeding a coarsely ground diet to observe whether or not existing ulcers could be healed.

Materials and methods

Animals and diets

A total of 80 Yorkshire barrows and gilts (24.1 ± 2.9 kg, 53.1 ± 6.4 lb) were individually housed, given ad libitum access to a finely ground ($\frac{3}{32}$ inch screen, particle size = 578 ± 1.89 μm) pelleted corn/wheat-based grower diet (Table 1) for 2 weeks, and then examined endoscopically.

Forty-eight pigs were then selected so that each ulcer score would be equally represented in the experimental group. Pigs were fed the fine diet for an additional 7 weeks and then subjected to a second endoscopic examination to reassess the severity of the ulcers. Pigs were then allotted to either the same fine diet or to a diet of moderate particle size ($\frac{3}{16}$-inch screen, 937 ± 2.19 μm) so that pigs from all ulcer lesion score categories were again included in both diet groups. Pigs were fed the diets for an additional 3 weeks, and endoscoped again to observe any possible healing effects of the coarse diet. Data collected

weekly throughout the experiment were average daily gain (ADG), average daily feed intake (ADFI), and feed:gain (F:G).

Particle size determination

Particle size was determined by the method of American Society of Agricultural Engineers (ASAE).[14] A 100-g sample of complete mash feed (sampled prior to pelleting) was placed in a Ro-tap® shaker, which consists of 15 Tyler mesh brass sieves descending in screen size. All material left on each screen was weighed and entered into a logarithmic equation for particle size determination. Subsequent particle size analysis showed that the fine diet had a geometric mean particle size of 578 ± 1.89 µm and the coarse diet had a geometric mean particle size of 937 ± 2.19 µm.

Endoscopic examination

Feed was removed from the pens 12 hours prior to endoscopic examination because an empty stomach is required for visualization of the

pars esophageal area. Water was freely available until pigs were removed from pens to be anaesthetized. Anesthetic induction was performed with intravenous pentothal (Somnotol®, MTC Pharmaceutical, Montreal, Quebec) administered via a butterfly catheter into a marginal ear vein at 10 mg per kg. Further intravenous pentothal was given as required to maintain a moderate depth of anesthesia throughout the endoscopic procedure. For the endoscopic examination, pigs were placed on their sternums on a stainless steel surgical table, and a metal mouth gag was inserted. Endoscopic examination was performed using a forward viewing flexible video scope with a working length of 100 cm and an outer diameter of 10 mm (GIF type 100 EVIS Gastrointestinal Videoscope®, Olympus Corporation, Lake Success, New York). The endoscope was rapidly passed by mouth into the cardiac region of the stomach and the stomach inflated with air via the air/water nozzle of the endoscope. The distal end of the endoscope was then retroflexed and pulled gently in an oral direction to enable visualization of the pars esophagea. After examination, the stomach was completely deflated and the endoscope was removed. The pars esophageal area was scored from 0 to 3:

- 0 = normal;
- 1 = parakeratosis;
- 2 = moderate ulceration (superficial erosive lesions covering > 25% of the pars esophagus, or a single small, deep erosion); and
- 3 = severe ulceration (one deep ulcer that covered > 10% of the pars esophagus, or more than one ulcer)

based on criteria outlined by Mackin, et al., 1996.[17] The same individual performed all the scoring.

Statistical analysis

For the first 2 weeks, this experiment used a completely randomized design. From week 2 to week 9, pigs were blocked on the basis of ulcer score, and analyzed with each pig being considered an experimental unit. In the last 3 weeks of the study, pigs were blocked on the basis of ulcer score and assigned randomly to one of two diet treatments. The general linear model[18] was performed to test for ulcer score effect on: ADG, F:G, and ADFI. If the F value for ulcer score effect was significant, differences between individual treatment means were assessed by the least squares means[18] procedure and compared using protected least significant differences.[18] Correlation analysis was performed between ulcer score and ADG, F:G, and ADFI.[18]

The general linear model procedure[18] was used to test for diet effect on: ADG, F:G, ADFI, and ulcer scores for weeks 9 to 12. Weight at week 9 was used as a covariate to test for diet effect on ADG, ADFI, and F:G between week 9 and 12. The difference between endoscopic score at week 2 and week 9, and week 9 and week 12 for individual pigs was calculated and analyzed using the general linear model[18] to test for diet effect on the progression or regression of ulcer development.

Results

The distribution of particle sizes between the two diets is shown in Figure 1. Particle size was not normally distributed for either of the diets.

Table 1

Ingredients and analyzed composition of the diet

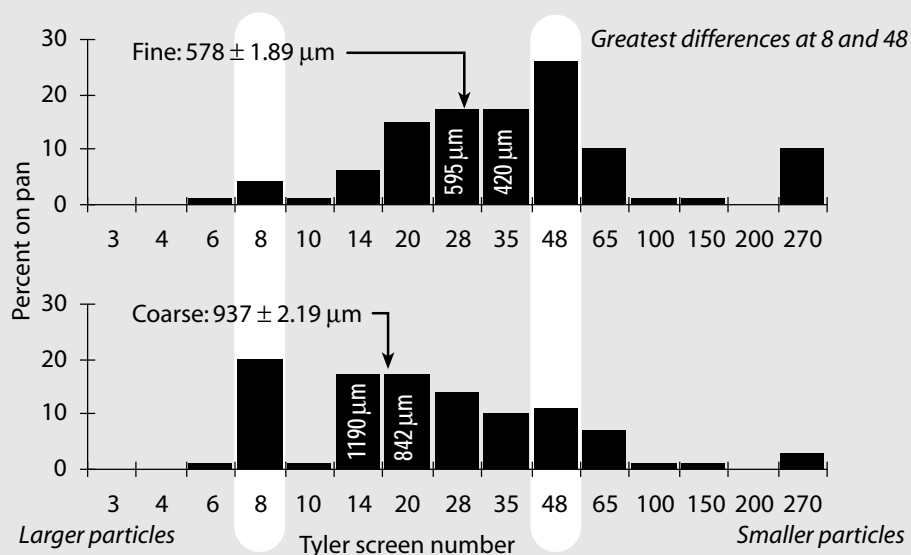| Ingredient	
Corn	40.00%
Wheat	39.65
Soybean meal, 48% CP	17.00
Limestone	1.00
Dicalcium phosphate	1.50
Cobalt salt	0.50
Mineral premix*	0.50
Vitamin premix†	0.25
Composition (calculated)	
ME	13.23 MJ/kg‡
Composition (analyzed)	
GE	14.82 MJ/kg
Dry matter	87.56% DM§
Crude protein	19.19
Fat	3.00
Ash	5.67
Crude fiber	2.96
Calcium	1.03
Phosphorus	0.88
Sodium	0.37

* supplied per kg diet: copper 15 mg, zinc 100 mg, iron 100 mg, manganese 20 mg, iodine 0.3 mg, selenium 0.3 mg.

† supplied per kg diet: 6000 IU Vitamin A , 600 IU Vitamin D_3, 24 IU Vitamin E, 1.3 mg menadione, 300 mg choline, 9 mg pantothenic acid, 3 mg riboflavin, 1.2 mg folic acid, 15 mg niacin, 0.9 mg thiamin, 0.9 mg pyridoxine, 120 ug biotin, 15 IU Vitamin B_{12}.

‡ NRC[14] book values.

§ analyzed by methods in AOCA.[15]

Figure 1

Fine: 578 ± 1.89 μm

595 μm 420 μm

Greatest differences at 8 and 48

Coarse: 937 ± 2.19 μm

1190 μm 842 μm

Percent on pan

Larger particles Tyler screen number Smaller particles

Distribution of particle sizes

Table 2

Relationship between ulcer score and growth performance over a 2-week period
for individually fed* pigs before endoscopic examination

Ulcer score[†]	0	1	2	3	r	P	SEM
n[‡]	2	29	29	13			
ADG (kg/d)	0.90[a]	0.83[ab]	0.81[b]	0.76[b]	−.15	<.1	0.02
ADFI (kg/d)	1.73[a]	1.57[b]	1.48[b]	1.44[b]	−.18	<.1	0.04
F:G (kg/kg)	1.99	1.90	1.83	1.90	−.02	NS	0.06

r sample correlation coefficient.

a,b means in a row followed by different superscripts differ P<.05.

* initial weight of pigs 24.1 ± 2.9 kg.

† ulcer scoring system after 2 weeks on fine diet; 0 = normal, 1 = parakeratosis, 2 = focal shallow ulceration, 3 = fully developed ulcer.

‡ number of pigs.

The coarse diet was negatively skewed, indicating a larger particle size, and the fine diet was positively skewed indicating a smaller particle size. Both diets were made using two different grains (corn and wheat), and therefore it is not surprising that the distribution curve has two separate peaks because different grains have different milling characteristics. The greatest difference between the diets was the percentage of particles retained on screen #8 and #48. The fine diet had approximately 5% of particles on screen #8 and 26% on screen #48, while the coarse diet had 21% on screen #8 and 11% on screen #48. Screen #8 corresponds to a particle size of approximately 2400 μm and screen #48 to an approximate particle size of 300 μm.

Three of the pigs were diagnosed as having esophageal strictures. At the initial endoscopic examination, these pigs had scarring at the junction of the esophagus and stomach that prevented the endoscope from entering the stomach. Two of the pigs with esophageal strictures subsequently died, 24 and 38 days after endoscopic examination. The cause of death in both cases was due to gastric hemorrhage from a severe and chronic ulcer of the pars esophagea.

In the first 9 weeks of the experiment, a total of five pigs died due to hemorrhage from severe gastric ulcers. At the time of the first endoscopic examination, most of the pigs had some degree of ulceration (Table 2). The initial ulcer score was related to growth performance (Table 2) (P<.1) in the first 2 weeks of growth. Pigs without ulcers had a higher ADG than pigs with severe ulcers (P<.05)(.90 versus 76 kg per day). Also, pigs that had no ulcers ate more feed than those with ulcers (P<.05). There was no difference in F:G among pigs with ulcers and those without ulcers (Table 2). Increasing ulcer severity was negatively correlated with ADG and ADFI (P<.1).

All the pigs had some degree of ulceration at the time of the second endoscopic examination (Table 3). Those pigs with a lower ulcer score had higher ADG than those pigs with more severe ulcers (P<.05) for weeks 2–9. There were no differences in ADFI and F:G for those pigs with severe ulcers compared to those with moderate ulcers,

Table 3

Relationship between ulcer score and growth performance over a 7-week period for individually fed pigs[*]

| | Ulcer score[†] | | | |
	1	2	3	SEM
n[‡]	20	17	7	
ADG (kg/d)	0.96[a]	0.95[a]	0.90[b]	0.02
ADFI (kg)	2.35[a]	2.86[b]	2.98[b]	0.21
F:G (kg/kg)	2.43[a]	2.99[b]	3.29[b]	0.04

ab   means in a row followed by different superscripts differ $P<.05$

*   initial weight of pigs 40.31 ± 5.21 kg

†   ulcer scoring system after second endoscopy at week 9; 0 = normal, 1 = parakeratosis, 2 = focal shallow ulceration, 3 = fully developed ulcer

‡   number of pigs. There were no pigs with an ulcer score of 0. Initially, 48 pigs were on test, but two pigs died and one pig could not be evaluated by endoscopy because of a severe stricture at the esophageal-gastric junction.

Table 4

Growth performance of pigs that died due to gastric ulcers and those with esophageal strictures

	Died[*]	Stricture week 2[†]	Stricture week 9[‡]	Stricture week 12[§]
n	5	3[‡]	2[¶]	6[#]
Weight(kg)	28.6	34.4	72.9	94.8
ADG (kg/d)	0.65	0.74	0.79	1.27
ADFI (kg/d)	1.38	1.60	2.19	3.03
F:G (kg/kg)	2.03	2.17	1.99	2.23

n   number of pigs

*   days lived and weight: 7 days 25.7 kg, 8 days 26.5 kg, 11 days 34.6 kg, 38 days 27 kg, 43 days 29.3 kg

†   growth performance from weeks 0–2

‡   growth performance from weeks 2–9

§   growth performance from weeks 9–12

|| two pigs died due to gastric ulcers

¶   same pig as in week 2 plus one new pig

same two pigs as in week 9 plus four new pigs

Table 5

Effect of feeding a fine (578 ± 1.89 μm) or coarse (937 ± 2.19 μm) diet on ulcer score and growth performance of individually fed pigs between weeks 9–12

Diet	Fine[*]	Coarse[†]	SEM
n	23	22	
Initial weight (kg)	80.7[a]	74.0[b]	1.08
Initial ulcer score[‡]	1.87	1.77	0.13
Final ulcer score[§]	2.10[a]	1.41[b]	0.14
ADG (kg/d)[||]	1.30	1.40	0.04
ADFI (kg/d)[||]	3.02	3.10	0.07
F:G (kg/kg)[||]	2.33	2.22	0.31

n   number of pigs.

a,b $P <.05$.

*   mean particle size 578 ± 1.89 μm.

†   mean particle size 937 ± 2.19 μm.

‡   ulcer scoring system; 0 =normal, 1=parakeratosis, 2=focal shallow ulceration, 3=fully developed ulcer , ulcer score at week 9.

§   initial ulcer score at week 9 used as covariate.

|| weight at week 9 used as covariate.

The mean difference in ulcer score between week 2 and week 9 was +0.50, indicating that ulcers tended to increase in severity during this period. The mean change in ulcer score between week 9 and week 12 was +0.23 and −0.43 for pigs fed the fine- and coarse-ground diets, respectively. These data indicate that ulcer severity continued to increase for pigs receiving the fine diet, but decreased for those receiving the coarse diet ($P<.05$) (Table 6).

Discussion

The data reported in this study reveal that as ulcer severity increases, growth performance of individually fed pigs decreases. These results agree with one previous study,[4] but are in contrast to other experiments.[5,9] Our result indicates that not only those pigs that have severe gastric ulcers suffer a depressed growth rate, but also that pigs with a moderate ulcer problem also have depressed ADG. By individually feeding the pigs, more accurate records of growth performance were obtained. Likewise with the endoscopic examination at different stages of growth, we could observe the development or change in ulcer severity throughout the experiment. This methodology has only been used to a limited extent in previous studies.[19]

Although the prevalence of gastric ulcers in young growing pigs is unknown, we speculate from this study that the

but these growth parameters were significantly different in pigs with mild stomach lesions ($P<.05$).

At week 9, new pigs were found to have developed esophageal strictures at the second endoscopic examination. Those pigs that died due to gastric ulcers and/or were diagnosed with esophageal strictures had lower growth performance than pigs with less severe ulcers (Table 4).

At the final endoscopic examination, those pigs that were fed the coarse diet for 3 weeks had lower mean ulcer scores than those fed the fine diet ($P<.05$) (Table 5). There was no difference in growth performance between pigs fed the finely ground diet (578 μm) and those fed the more coarsely ground diet (937 μm) over the 3-week period (Table 5).

Table 6

Effect of diet on change in ulcer score* between endoscopic examination at weeks 2, 9, and 12
for pigs receiving a finely (578 ± 1.89 µm) ground diet or a more coarsely (937 ± 2.19 µm) ground diet

	No change in ulcer score	Increase in ulcer score	Decrease in ulcer score	Mean ulcer change	SEM
Week 2–9 (n=46)					
fine*	20	8	18	+0.50	0.16
Week 9–12 (n=45)					
fine*	8	5	10	+0.22[a]	0.12
coarse[†]	11	1	11	–0.43[b]	0.12

*   ulcer scoring system; 0=normal, 1=parakeratosis, 2=focal shallow ulceration, 3=fully developed ulcer.
a,b   data in a row with different superscripts differ significantly, *P*<.05.
†   mean particle size 578 µm.
‡   mean particle size 937 µm.

prevalence of pars esophageal lesions in these animals may be quite high. Almost all the animals demonstrated some degree of stomach lesion at the time of the first endoscopic examination when the pigs were approximately 10–12 weeks old.

Unfortunately, only two pigs were without stomach lesions at the beginning of the study. These pigs performed better than all others, but it is difficult to make strong conclusions based on such a small sample and for only a 2-week period. However, throughout the experiment, the relationship between severity of stomach lesions and performance was consistent: as the stomach lesion score increased, growth performance decreased.

A small particle size was fed in order to create gastric ulcers, so that the effect on growth performance could be studied. Differences in endoscopic ulcer scores from week 2 to week 9 indicated that mean ulcer score increased (Table 6), suggesting that the small particle size caused an increase in ulcer severity. The fine diet had approximately 26% of feed particles retained on screen #48, which corresponds to a particle size of 300 µm; this relatively large percentage of small particles may have been responsible for the high prevalence of gastric ulcers in this study. Likewise, the coarser diet had a relatively large percentage of feed particles retained on screen #8 (corresponding to a particle size of 2400 µm), which may have accounted for the healing effect of the coarse diet. The importance of particle size distribution with respect to ulcer development and growth performance is unknown. It is possible that removing the high proportion of fine particles is more important in ulcer reduction than the presence of the larger particles.

When pigs were fed either the coarse diet or the fine diet, there was no difference in growth performance, contrary to results in the literature. Possibly, the expected increase in performance associated with feeding a fine diet was offset by a decrease in performance due to the greater prevalence and severity of the ulcers. Pigs that received the coarse diet had a decrease in mean ulcer severity, indicating a healing effect.

Feeding the coarse diet is thought to decrease the fluidity within the stomach, and restore the pH gradient between the esophageal area and the pyloric area.[11] Decreased fluidity and mixing within the stomach is thought to prevent contact of HCl, pepsin, and bile acids with the pars esophageal area.[20] Those pigs that remained on the fine diet continued to develop more severe ulcers, reconfirming that a fine diet is more prone to cause ulcers than a coarse diet.

During the first endoscopic examination, three of the pigs had an esophageal stricture. A stricture is defined as a narrowing of the esophageal lumen that prevents the endoscope from passing or requiring a forceful passage; the stricture could be severe or mild. Esophageal strictures in humans are not uncommon; it is reported with high frequency associated with reflux esophagitis.[21] The specific etiology of strictures is unknown. Esophagitis is described by Rejeb, et al.,[22] as round or linear erosions in the esophagus caused by reflux of gastric juices consisting of HCl, pepsin, and bile acids. Reflux esophagitis and esophageal strictures may be associated with endoscopic examination, which can cause a reflux of gastric juices into the esophagus.[21] Endoscopic examination cannot be the sole cause of stricture formation, as indicated by the presence of two pigs with strictures at the first endoscopic examination. At the third endoscopic examination, six of the pigs had esophageal strictures; these may or may not have been caused by previous endoscopic examinations. The prevalence of esophageal strictures in swine is not known, but may be a much more common problem than previously thought. If this study is representative of the population, an incidence at the first endoscopic examination of two pigs with esophageal strictures out of 80 pigs suggests a prevalence of 2.5% of growing pigs. The potential for strictures to be a significant cause of variation in the growth or an explanation for "poor-doers" needs to be investigated.

Overall, this study showed that as subclinical gastric lesions became deeper and more extensive, growth performance was reduced. However, the greatest economic losses associated with gastric lesions in this study were sudden deaths due to hemorrhage and marked decreases in growth performance associated with the development of esophageal strictures. The improvement in feed efficiency associated with fine particle size[6] is such a strong economic consideration for most pork producers that it is likely to outweigh the decrease in

growth performance associated with mild to moderate stomach lesions. However, if mortality is occurring, it is apparent from this study that a change of diet to a more coarsely ground ration for a short period can quickly resolve the problem.

Implications

- Endoscopic examination proved to be a fast, effective method of assessing gastric ulcers in live pigs.
- Feeding a diet with a particle size of approximately 600 µm resulted in a high prevalence of stomach lesions.
- Increasing stomach lesion severity was associated with reduced growth performance.
- Feeding a coarse diet, after ulcers had developed, caused a decrease in the severity of ulcers.
- Particle size distribution may be important in the ulceration process, but needs to be investigated further.

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