# Diarrhea in gilts caused by excessive dietary sodium chloride

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## Summary

This case report describes how an outbreak of diarrhea in gilts was investigated and eventually diagnosed as an excess of socium chloride in the feeds.

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Porcine diarrhea has been well described in the literature. The nature of the diarrhea depends upon the pathologic or the physiologic factor(s) involved. Differential diagnoses for porcine diarrhea change as the age of affected pigs increases. For finishing to adult-aged swine, this list includes small intestinal diseases such as transmissible gastroenteritis (TGE), salmonellosis, and porcine proliferative enteropathy (PPE). Salmonellosis and PPE can also be classified as large intestinal diseases, as can swine dysentery and *Trichuris* infestations. Other less-common causes of diarrhea include mycotoxins[1] and errors in feed formulation and mixing.

The character of the diarrhea—as well as herd history, signalment, group morbidity and mortality, and postmortem lesions—may help in diagnosing the etiology of the problem. Additional diagnostics, such as bacteriologic culture, histopathology, and electron microscopy, may demonstrate agents and lesions.

Common factors such as feed, water, and environment should not be overlooked, especially when diagnostic examination for pathogens is negative.

In this case report, the cause of an outbreak of diarrhea in replacement gilts was investigated.

## Case description

### History

A 2500-sow farrow-to-wean herd in Kansas receives regular shipments of replacement gilts at selection age from a neighboring state. New replacement gilts are housed in an onsite isolation facility for a minimum of 60 days until they are moved into the sow herd. The isolation facility is located approximately 200 feet from the sow herd and has two rooms with partially slatted floors and self-feeders for two different age groups of gilts. At the time this case occurred, the 50 gilts in one room were approximately 260 days of age, and the 120 gilts in the adjacent room were 230 days of age.

Eight weeks after arrival of the second group, nearly all of the gilts began exhibiting profuse, watery, nonbloody diarrhea. Gilts also initially appeared to be off feed. There were no apparent inciting factors that occurred around the time of clinical signs, nor had there been any known breaches in biosecurity, new additions to the herd, or environmental problems. Both groups of gilts were healthy on arrival and exhibited no clinical signs prior to this incident. Overall herd health was excellent. No clinical signs were apparent in the sow herd at the time the gilts developed diarrhea.

Five tons of feed had been delivered to the isolation facility approximately 60 hours prior to the development of clinical signs. The feed was a 1.0% lysine sorghum-based meal ration that contained tylosin (Tylan®; Elanco Animal Health; Indianapolis, Indiana) at a concentration of 100 g per ton.

Since porcine proliferative enteropathy was an initial concern, treatment with neomycin was initiated via water medication immediately after clinical signs began to be observed. After 2 days of therapy, no improvement in clinical signs was seen. At this time, veterinary attention was sought.

### Examination

Upon examination, gilts did not appear to have systemic signs of disease, but they exhibited a profuse, watery diarrhea. No clinical signs had developed in the adjacent sow herd. Feed consumption had gradually increased since the outbreak of diarrhea.

Differential diagnoses included:

- PPE (ileitis),
- transmissible gastroenteritis (TGE),
- *Salmonella* enterocolitis,
- swine dysentery,
- *Trichuris suis*,
- mycotoxin, and/or
- a feed mixing error.

Five gilts were selected and sacrificed. A thorough postmortem examination was conducted and the following samples were collected from each animal:

- three fecal samples from different locations of the gastrointestinal tract (ileum, spiral colon, and cecum);
- fresh and fixed sections of the following:
  —duodenum
  —jejunum
  —ileum
  —cecum
  —spiral colon; and
- a feed sample.

Gross examination of all five carcasses was unremarkable except for watery feces present throughout the jejunum, ileum, cecum, and large bowel. Mucosal sections of the duodenum, ileum, cecum, and colon appeared normal, except for random focal areas of hyperemia throughout the jejunum. There was no evidence of parasitism. Livers and spleens were of normal size and lymphadenopathy was not seen. Visual examination of the feed revealed no gross abnormalities. It appeared fresh and did not have an abnormal odor.

Fearing the possibility of TGE, biosecurity was tightened. Foot baths were placed at the entrance of the isolation facility. The gilts in the isolation facility were to be checked by one person at the end of the day. Separate boots and coveralls were

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worn. The adjacent sow herd was carefully monitored for the onset of clinical signs. Water medication with neomycin was continued in the isolation facility pending diagnostic results.

### Postmortem diagnostic results

Selected tissue and fecal samples were immediately submitted for aerobic culture and sensitivity, histopathology, electron microscopy, immunohistochemistry, direct fluorescent antibody examination, and fecal flotation.

Tissue and fecal results for all five gilts were as follows:

- histopathology: no evidence of proliferative enteritis or swine dysentery.
- bacteriology: culturing yielded no *Salmonella* or other pathogens.
- electron microscopy: negative staining scan of pooled feces showed no virus particles.
- immunohistochemistry (IHC): negative for TGEV.
- parasitology: flotation was negative for protozoan and helminth ova in fecal material.
- direct-fluorescent antibody (IFA) test on tissue sections: negative for TGEV.

Results were obtained a few days after the herd visit. By that time, the diarrhea had subsided and no other clinical signs were apparent in the replacement gilts or the sow herd. At this time, a feed sample was submitted for analysis of crude protein, calcium, phosphorous, potassium, and sodium chloride.

## Discussion

Pathogens were ruled out as a cause of diarrhea through various diagnostics in this case. Although the initial concern was TGE, the results of IHC and IFA were negative and the adjacent sow herd did not exhibit any clinical signs several days after the onset of diarrhea in the isolation facility. One would expect that although biosecurity was tightened after the diarrhea began, TGEV would have spread to the sow herd soon after due to the relatively short incubation period of the virus and the likelihood of vector transmission.[2]

Feed analysis revealed that the ration contained 1.36% sodium chloride on a dry matter basis (Table 1). The sample on an as-fed basis contained 1.20% sodium chloride, or 10.9 kg (24 lb) per ton of feed. Problems with alterations in feed formulation are not uncommon. Dewey[3] demonstrated that the morbidity rate due to diarrhea was higher when the pigs were fed a 21% crude protein ration than when they were fed a 17% crude protein ration, and that this elevated crude protein concentration may cause diarrhea. Incorrectly formulated feed leading to an excess of flavor-enhanced vitamin premix has been implicated in an outbreak of *Streptococcus suis* type 2 disease.[4]

**Table 1:** Results of feed sample analysis in case herd with diarrhea

| Item | Dry basis | As received |
|---|---|---|
| Moisture by forced air oven | – | 11.88% |
| Dry matter | 100.00% | 88.12% |
| Crude protein | 14.19% | 12.50% |
| Calcium | 0.60% | 0.53% |
| Phosphorous | 0.39% | 0.34% |
| Potassium | 0.68% | 0.60% |
| Soluble chloride | 1.36% | 1.20% |

Improvements in growth performance have been observed when diets were supplemented with sodium and chloride.[5–7] The dietary sodium requirement of growing-finishing swine is no greater than 0.08%–0.10% of the diet and a concentration of 0.20%–0.25% added sodium chloride will meet the dietary sodium and chloride requirements of growing-finishing pigs fed a corn-soybean meal diet.[8] Based on suggested diets in the Kansas Swine Nutrition Guide[9] for a 1% lysine finishing diet, the recommended inclusion concentration of salt is 0.35% or 3.2 kg (7 lb) per ton.

Sodium ion toxicosis is a well described condition in the swine industry and is associated with water deprivation.[10,11] The major factor that influences salt toxicosis in animals is the availability of drinking water. In the presence of an ample nonsaline water supply, swine can tolerate relatively large quantities of dietary sodium chloride.[8] In this case, the dietary salt was only mildly elevated and water intake was unrestricted so no signs of toxicity were exhibited.

Maximum tolerable levels of dietary salt in animals were established as follows:

- 4% for lactating cows,
- 9% for other cattle and sheep,
- 8% for swine,
- 2% for poultry, and
- 3% for horses and rabbits.[12]

In one study, no adverse effects were reported in mature swine consuming a 3% salt diet for 11 days, even with restricted water.[11] In grazing sheep, 1.3% salt decreased body weight gains in lambs and the reproductive rate in ewes, and caused diarrhea.[13]

Sodium comprises > 90% of the cations in extracellular fluid and contributes to > 80% of the extracellular fluid osmotic load.[14] Therefore, serum sodium concentrations play a major role in the maintenance of osmotic pressure. Water moves passively along osmotic gradients created by the active transport of electrolytes (principally sodium) and it is this complex movement of ions that controls the water flux and maintains the gut-dependent secretion of the vital homeostatic mechanisms for water and electrolytes.[15] Most of the fluid transport in the small intestine is carried out by passive diffusion across the epithelium and the vascular wall through pores, following the pressure gradient of colloidosmotic pressure versus hydrostatic pressure.[16]

Based on these physiologic principles, the diarrhea observed in this case was likely of osmotic etiology and caused by excessive dietary salt. Since unlimited water was available, no signs of toxicity were seen. The elevated dietary salt may also explain why the gilts were off feed initially; this was probably due to feed refusal rather than inappetance. The previous dietary concentration of minerals to which the animals were adapted will influence the short-term response to excessive concentrations.[14] The gilts in this case were accustomed to a lower-sodium diet and thus were quickly affected by the change in dietary salt concentrations.

## Implications

- In addition to the infectious causes of diarrhea, feed mixing errors should not be overlooked.
- A feed sample should be obtained in all cases in which clinical signs are observed in the entire group over a short period of time.
- Feed analysis can be a helpful diagnostic tool when mixing errors are suspected.
- A diet containing 1.20% sodium chloride appeared to be enough to cause an osmotic diarrhea in gilts ranging from 230–260 days of age.

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