INTRODUCTION

Despite gastric ulcers being common in pigs, the pathophysiology of the condition in swine is poorly understood. Among other factors diet and bacterial infection have been incriminated as causes of the condition (1). This study had the objective of investigate the effects of two wheat-based diets, previously tested in weaners and shown to have protective and deleterious effects on the stomach respectively (2), on spiral bacterial populations in the gastric mucosa.

MATERIALS AND METHODS

Sixteen pigs were fed one of two isoproteic diets based on raw finely ground wheat (RW, n=8) or the same finely ground wheat, which also had been subjected to high pressure and temperature extrusion (EW, n=8). The diets used had a similar composition to commercial weaner diets. The diets were offered as creep feed from one week of age. At three weeks of age the pigs were weaned, and placed in individual cages with water and food provided ad libitum. Daily feed intake and weekly weight gain also were recorded.

At 35 days of age the pigs were euthanised, the stomachs cut open along the greater curvature and examined for the presence and severity of macroscopic lesions. A scoring system was used to grade the severity of the lesions (0=normal, 1=hyperkeratosis, 2=erosions, 3=ulcers). Tissue samples were taken from the Pars oesophagea-cardiac junction and fundic areas and placed in individual sterile vials containing 100% ethanol. DNA was extracted (Qiagen) from these samples and PCR analysis was performed using specific primers, based on the 16S rRNA gene, designed to amplify DNA from Helicobacter spp., H. heilmannii, Campylobacter spp. or Arcobacter spp.

Stomach content pH and dry matter were measured and also the pH of the contents of the proximal and distal small intestine were recorded. Data analysis was performed using the Chi square test.

RESULTS

Ulceration was found in 7 (87.5%) pigs on the RW diet and in 1 (12.5%) of the pigs on EW groups. There was a highly significant (P<0.0051) effect of diet on the occurrence of lesions. No association was found between the presence of Helicobacter spp. (including H. heilmannii) and ulcers, and indeed few pigs were colonised with these bacteria. No positive PCR for Arcobacter spp. results were obtained. In contrast there was a statistically significant association between Campylobacter spp. and diet (P = 0.0385), with pigs fed extruded wheat being more frequently colonised by Campylobacter spp. A summary of results is shown in the table. Similarly, pigs without ulcers (predominately on the extruded wheat diet) were much more likely to be colonised by Campylobacter spp. than were pigs with ulcers (P = 0.0385).

Table 1. Results of PCR analysis for Campylobacter spp. in healthy and ulcerated stomachs.

<table>
<thead>
<tr>
<th>Diet</th>
<th>Healthy Camp.</th>
<th>Healthy Camp.</th>
<th>Ulcerated Camp.</th>
<th>Ulcerated Camp.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>RW</td>
<td>1</td>
<td>0</td>
<td>3</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>EW</td>
<td>7</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>8</td>
</tr>
</tbody>
</table>

There was no significant correlation between ulcers and pH of the stomach contents, proximal or distal small intestinal contents, however a trend for pH values of stomach contents to be higher and intestinal contents to be lower was seen in pigs without ulcers. All ulcerations in the present study were in the cardiac area.

There was no significant difference in dry matters of stomach contents between diets or between healthy and ulcerated stomachs. Feed intake and weight gain were not significantly different between pigs with healthy and ulcerated stomachs, but feed conversion was significantly (P=0.0013) better in pigs without ulcers (predominantly pigs fed EW).

DISCUSSION

A highly significant protective effect of extrusion of wheat on gastric ulcers in a weaner model of the disease was demonstrated. Similarly there was significantly more colonisation by Campylobacter spp. in pigs on the extruded diet.

The mechanism by which diet alters susceptibility to ulcers is uncertain, but it may be that diet acts through influences on Campylobacter spp. populations. An alternative explanation for the results is that the Campylobacter spp. may simply be influenced by the same dietary factors that predispose to ulceration (eg. increased production of stomach acid). Further work is required to distinguish between these possibilities.

In humans infected with Helicobacter pylori the pattern of gastritis is dictated by host factors such as acid output (3). It is possible that acid output also determines ulcer location in pigs, and if so this may explain why younger pigs develop ulcers in a different location from older pigs.

The trend for pigs without ulcers to have higher pH values in their stomachs is consistent with gastric acidity being involved in the aetiology of stomach ulcers. A recent study has suggested that microbial production of short chain fatty acid (SCFA) may be more important than HCl in the pathogenesis of porcine gastric ulcers (4). It is conceivable that differences in dietary substrate may have influenced patterns of fermentation in the stomach in this experiment, and altered the final pH values. Similarly fermentation may alter serum gastrin levels (5), in turn influencing production of HCl. Further investigations are needed on the relationships between diet and SCFA production in the stomach, and also on a possible influence of intestinal fermentation on gastrin output in pigs.

In agreement with previous studies we found that pigs with stomach ulcers had a worse feed conversion than pigs with healthy stomachs (6), although this outcome may have been confounded by the fact that these animals were predominantly fed different diets.

ACKNOWLEDGMENTS

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REFERENCES