Stomach ulcers represent localized areas of the inner lining of the stomach that have become eroded and sloughed off, leaving open bleeding craters. In pigs, stomach ulcers occur at two sites: the glandular area, where the acid and enzymes are secreted to aid in the digestion of food, and the non-glandular pars oesophagea region at the top of the stomach, around the junction where the oesophagus delivers food into the stomach. This article relates to ulcers occurring around the latter region (oesophago-gastric ulcers), the most common form of stomach ulcers in pigs.

Cells lining the pars oesophagea do not secrete mucus and have limited protection against the acid pH and enzymes found in the stomach. The pig’s stomach, unlike that of many other animals, has minimal mixing abilities and is designed to be rarely empty. This is the case for animals “in nature” where vegetation forms a significant part of the diet. The most recent meal lies close to the opening to the oesophagus and lies on top of previously eaten food. Most mixing of contents occurs in the part of the stomach close to the intestine (the pyloric region which is furthest away from the oesophagus). However, intensively reared pigs are not fed continuously and are fed concentrated rations. Ingestion of these rations leads to more fluid stomach contents and more mixing of the contents. This results in a loss of the pH gradient between the oesophageal and pyloric region of the stomach. Consequently the pars oesophagea region is exposed to enzymes and acids, which damage it. The mixing of stomach contents leads to an elevated pH in the fundic region of the stomach, which in turn stimulates more acid production, further exacerbating the condition.

The cells lining the pars oesophagea respond to the insult by multiplying and thickening (hyperplasia). Hyperplasia is usually a very effective defensive mechanism, however if the hyperplasia continues insufficient nutrients are able to reach all the cells. This leads to weakening of the junctions between cells so that stomach juices can gain access to the underlying tissue structures. This initially leads to loss of the surface cells, however if the insult continues, erosions and finally ulcers develop, exposing the deeper layers of the stomach wall.

The effect of ulcers
The clinical signs observed in affected pigs predominantly reflect the

A recent study of over 15,000 pigs examined at slaughter showed that oesophago-gastric ulcers in pigs in Australia are at least as common as they are in pigs in other countries. Nearly half of all herds examined in Victoria had pigs with erosions or ulcers, but only 18% of herds in Queensland were similarly affected. There was no evidence that Helicobacter bacteria were involved in causing the condition, since they were present in both healthy and diseased pigs, nor was any influence of grain type detected. On the other hand, the use of pelleted diets and automated feeding systems were significantly associated with the condition.

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**Healthy stomach on the left and a stomach with oesophago-gastric ulceration on the right. Note the healthy white pars oesophaga in the centre bottom of the stomach on the left, surrounding the opening of the oesophagus. The pig on the right has erosions and ulceration in this area.**
degree of blood loss associated with the lesions. With significant blood losses, a pig that previously appeared healthy may be found dead with a pale carcass. In less severe cases, affected animals are anaemic and appear pale, weak, lethargic and breathe quickly. Black tarry faeces may be present and some pigs may show signs of abdominal discomfort by arching their backs and grinding their teeth. Although pigs with stomach ulcers can show dramatic clinical signs, the majority show few, if any, signs. Therefore the condition can go undetected unless post-mortem examinations are carried out or abattoir studies undertaken. Findings can also vary on post-mortem. In severe cases the stomach can be full of blood, while in less severe cases only evidence of ulceration of the pars oesophagea may be found.

**Influence on performance**

There are equivocal findings about the effect of stomach ulcers on the performance of pigs. Some authors have found that ulcers do not influence growth while others have reported a significant impact on performance. However the condition is recognised as an important cause of deaths in finishers and growers. The influence of such lesions on the overall welfare of affected pigs also should be considered.

**Causes of gastric ulcers**

Many studies have demonstrated that dietary factors, including the ingredients and physical characteristics of the diet, can influence the development of ulcers. The factors that have received the most attention are pelleting and fine grinding of the diet. Diets with a small feed-particle-size result in more fluid stomach contents which leads to increased mixing of contents, and exposure of the pars oesophagea to enzymes and acid and the onset of erosions and ulcers. In contrast, in diets with larger, more coarsely ground particles, there is less mixing and a greater gradation of pH values in the stomach.

To create a good quality pellet, grains are usually finely ground. Furthermore increasing the temperature of the feed during pelleting can lead to gelatinisation of starch. This has been shown to increase the likelihood of ulcers developing. On the other hand fine grinding and pelleting of rations may result in up to an 8% improvement in feed efficiency, and there is less feed wastage with pelleted rations. However, a 4% death loss due to gastric ulceration during the finishing phase may negate the economic benefits achieved through enhanced feed efficiency. A difficult aspect of pig management is to produce a diet that will maximise digestibility and performance but will minimise losses resulting from stomach ulcers.

Stresses associated with overcrowding, mixing and transportation also have been implicated in the development of stomach ulcers. Similarly some studies have identified fasting or major reductions in feed intake as factors contributing to the development of ulcers.

In humans there is a strong association between the presence of the bacterium *Helicobacter pylori* and stomach ulcers. However, stomach ulcers in humans predominantly occur in the pyloric region of the stomach and not in the pars oesophagea region. Although *H. pylori* is not found in pigs, similar *Helicobacter* species have been isolated from the stomachs of pigs. Reports of their role or significance in the development of ulcers in pigs have been equivocal. In the Australian study outlined below, although helicobacters were identified in the stomachs of pigs, they were present in a similar proportion in pigs with no ulcers as in pigs with ulcers. It is possible that the bacteria identified were part of the normal stomach flora of pigs.

**Results of Australian study**

Recently, in a study funded by the Australian Pig Research and Development Corporation, over 15,000 pig stomachs from 136 herds were examined at three abattoirs in Victoria, Queensland and Western Australia.
were found in the proportion of herds with erosions/ulcers/strictures between the states (47% for Victoria, 29% for Western Australia and 18% for Queensland). On an individual pig basis, the prevalences of lesions were 46% for Victoria, 35% for Western Australia and 21% for Queensland. In a detailed study conducted in Western Australia, the prevalence in culled breeding animals (77%) was significantly higher than that of finisher pigs (35.2%). This high prevalence was consistent with stomach ulcers being a common cause of sow mortality.

Within all states, pigs from some piggeries were virtually free from ulcers, whilst on others the within-herd prevalence approached 100%. Because of the variation in herd prevalence, factors that predisposed a herd to a high level of ulcers were investigated through the use of a detailed questionnaire. Specific questions were asked about the piggery including the size, breed and origin of stock; management procedures including frequency of mixing and moving pigs, transport of pigs to the abattoir; pig nutrition including number of diets fed, type of feeding (restricted, ad lib and whether the diet was wet/dry, pelleted, meal or crumble), whether an automated feeding system was used, where pigs were fed, the grain content of the diet and if antibiotics were given to pigs. Data were analysed by one-way analysis of variance or chi-square test, and significant factors then included in a logistic multiple regression model. In the final analysis, herds that fed a pelleted diet and those using automated feeding systems had significantly higher levels of ulcers than herds using meal, and using a manual feeding system. Although larger herds were more likely to have a higher prevalence of stomach ulcers, neither the number of pigs per pen (a surrogate measure of overcrowding) nor more frequent mixing of pigs were identified as precursors of ulcers. In contrast to the findings of others, in the Australian study there was no apparent influence of the grain type in the diet on the development of ulcers. This suggests that the mechanical properties of the diet are more important than the actual dietary ingredients.

Conclusions
From the Australian study and those conducted elsewhere, it is apparent that many factors, particularly dietary factors, can influence the development of ulcers in finisher pigs. Some herds have no ulcers at all, so it is clear that the condition is not inevitable. Although it is likely that husbandry and management factors can be manipulated to reduce the prevalence of ulcers, it is important that this is not done at the expense of overall pig performance. PP