In a survey of a large commercial herd (J. Carr, Mayfair Farms, personal communication) it was found that some pigs that had suffered diarrhoea in early life were more prone to gastric ulceration in later life. The strong correlation between severity in infancy and ulceration in later life suggests that an early imbalance in the gastric microflora may predispose to gastric ulceration.

Diarrhoea (Swensen et al. 1975; Schwartz 1974) and gastric ulceration ("Harle 1969; Tanna & Bokor 1974; & Matha et al. 1976) are problems of major economic importance to the pig industry. Although some of the pathogenetic properties of bacteria and the mechanisms by which they disrupt the normal flora and function of the gastrointestinal tract have been identified (Chou 1978; Savage 1972), most of the work done has been in the small and large intestine. As yet the stomach mucosa has received little if any attention. Barrow et al. (1977, 1980) and Fuller et al. (1978) have recently examined the adherent microflora of the gastric epithelium of the pig, but did not use strict anaerobic methods. Schulte (1977) characterized the anaerobic microflora of the greater curvature of the stomach but did not include the pig. Our study has been carried out to investigate the strict anaerobic microflora that colonize (adhere to) the pig's stomach epithelium and are tolerant to gastric ulceration in the pig.

Materials and Methods

Strict anaerobic methods were employed to enumerate and characterize the total population of microorganisms in the stomach and small intestine of healthy and diseased pigs. Specimens were manipulated within anaerobic chambers and the isolation and culture of microorganisms was carried out both anaerobically and aerobically. The association of microorganisms with the mucosal epithelium was determined by an analysis of the vertical distribution of isolates and by electron microscopic examination of tissue sections (Russell 1979; Fullcr et al. 1978). By a series of washings and homogenization, microbes were subdivided into categories reflecting their vertical location on the tissue (Russell 1979). Healthy and diseased, pre and post-weaner pigs, were anaesthetized with an overdose of pentothal sodium and tissues and contents sampled before death. Facultative anaerobes were characterized by conventional means and anaerobes by the methods outlined in the "Textbook Manual of Bacteriology, 4th ed. (1967).

Results and Discussion

Both the pars oesophagea and small intestines of healthy and diseased pigs were cultured, and the healthy pigs show that there are between 10^2-10^5 anaerobic organisms per gram of gastric contents or per gram of contents. The majority of microorganisms in the stomach of healthy pigs are gram positive and the dominant genera are anaerobic Lactobacillus and Escherichia. Other anaerobes found include Propionibacterium, Clostridium, Peptococcus, Peptostreptococcus, Streptococcus and Veillonella. The facultative anaerobes present include Helicobacter, Butyribacterium, Porphyromonas, Catenulatus and Streptococcus. In healthy pigs strict anaerobes are the predominant microorganisms colonizing the mucosal epithelium but in scouring pigs the anaerobes are largely replaced by facultative anaerobes.

Our findings differ from those of Fuller et al. (1978) who reported that Lactobacillus and Streptococcus constitute the majority of microorganisms adhering to the pars oesophagea of healthy and scouring pigs. We have observed marked increases in the number of E. coli in the stomach and small intestine of scouring pigs both in the lesion and colonize the pig. The proliferation of E. coli in the small intestine of scouring pigs, but not in the stomach, has been reported by Barrow et al. (1977). Schulte (1977) and McAllister et al. (1979). Barrow et al. (1980) showed that by feeding strains of Lactobacillus which colonize the pars oesophagea, there is a reduction in the number of E. coli in the small intestine. Such findings, previously reported by Sandine (1972), indicate the role played by the normal flora in preventing colonization of the alimentary tract by opportunists (Savage 1977; Smith 1977).

Our results suggest that the strict anaerobic microflora may be important in maintaining the pars oesophagea in a healthy state. In the scouring pig the indigenous anaerobic microflora colonizing the mucosa appear to be displaced by microorganisms in addition to E. coli including Eubacterium, Peptostreptococcus, and in one case the yeast Torulopsis. Such changes in the microflora associated with scouring may cause damage to the mucosa resulting in chronic trauma to the gastric tissue. To all the stomach, we have observed abnormalities in the pars oesophagea ranging from parakeratotic proliferation to complete detachment of the squamous epithelial lining. But one of the healthy pigs examined had normal pars oesophagea characterized by a normal anaerobic flora. Apart from the work done by Tannatt et al. (1977) and our observations (1978) and that discussed by Bokor & Arp (1978) there are few reports implicating microorganisms in the pathogenesis of gastric ulceration.

Conclusions

1. The stomach and small intestine of the pig harbour large and diverse microbial populations which differ from healthy and diseased pigs.
2. In scouring pigs E. coli and other pathogens proliferate in the stomach, as well as the small intestine, displacing the resident anaerobic microflora.
3. Damage to the pars oesophagea which occurs in scouring pigs may be due to the activities of cytotoxic pathogens and could lead to gastric ulceration in later life.
4. Strict anaerobes may have a role in protecting the mucosa from colonization and damage by such pathogens.

References