THE DEVELOPMENT OF THE GASTRIC MICROFLORA OF THE PIG AND ITS RELATIONSHIP WITH DIARRHOEA AND GASTRIC ULCERATION

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

Diarrhoea (Svendsen et al. 1975; Schwartz 1971) and gastric ulceration (O'Brien 1969; Tamas & Bokori 1979; & Macarie et al. 1978) are problems of major economic importance to the pig industry. Although some of the pathogenic properties of bacteria and the mechanisms by which they disrupt the normal structure and function of the gastrointestinal tract have been identified (Moon 1978; Savage 1972), most of the work done has been in the small and large intestine. As yet the stomach mucosa has received little or no 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. Schulze (1977) characterized the anaerobic microflora of the greater curvature of the stomach but did not include the pars oesophagea. No study has been carried out to investigate the strict anaerobic microflora that colonize (adhere to) the pars oesophagea - the region most susceptible 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 pars oesophagea 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 microbes with the mucosal epithelium was determined by an analysis of the vertical distribution of isolates and by electron microscopic examination of tissue surfaces (Russell 1979; Fuller et al. 1978). By a series of washings and homogenization, microbes were subsumed into categories reflecting their vertical location on the tissue (Russell 1979). Healthy and scouring, pre- and post-weaned pigs, were anaesthetised with an overdose of pentabarbitone Na, and tissue and contents sampled before death. Facultative anaerobes were characterized by conventional means and anaerobes by the methods out-lined in the VPI Manual (Holdeman, Cato & Moore 1977).

Results and Discussion

Both the pars oesophagea and small intestine harbour large and diverse microbial populations which differ in healthy and diseased pigs. In the healthy pig there are between 10^4-10^8 microorganisms per gram of gastric contents or per gram of mucosa. The majority of microorganisms in the stomach of healthy pigs are gram positive and the dominant genera are anaerobic *Lactobacilli* and *Eubacterium*. Other anaerobes found include; *Actinomycetes, Bifidobacteria, Clostridia, Peptostreptococcus, Streptococcus* and *Veillonella*. The facultative anaerobes present include; *Aeromonas, Enterobacter, Escherichia coli, Klebsiella, Micrococcus, Serratia, Staphylococcus* 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 *Lactobacilli* 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 lumen and colonizing the mucosal epithelium. The proliferation of *E. coli* in the small intestine of scouring piglets, but not in the stomach, has been reported by Barrow et al. (1977), Schulze (1977) and McAllister et al. (1979). Barrow et al. (1980) showed that by feeding strains of *Lactobacilli* which colonize the pars oesophagea there is a reduction in the number of *E. coli* in the small intestine. Such findings, previously reviewed 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; *Klebsiella, Veillonella,* and in one case the yeast *Torulopsis blatara*. Such changes in the microflora associated with scours may cause damage to the mucosa resulting in chronic trauma to the gastric tissue. In all the scouring, weaned pigs we have observed abnormalities in the pars oesophagea ranging from parakeratotic proliferation to complete detachment of the squamous epithelial lining. All but one of the healthy pigs examined had normal pars oesophagea characterized by a normal anaerobic flora. Apart from the work done by Tannock & Smith (1970), Qureshi et al. (1978) and that discussed by Simmonson & Björklund (1978) there are few reports implicating microorganisms in the pathogenesis of gactric ulceration.

Conclusions

1) The stomach and small intestine of the pig harbour large and diverse microbial populations which differ in 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

Barrow,P.A.,R.Fuller,M.J.Newport:Infection and Immunity 1977,18(3),586;Barrow,P.A.,B.E.Brooker,R.Fuller,M.J. Newport:J.Appl.Bacteriol.1980,48,147;Fuller,R.,P.A. Barrow,B.E.Brooker:Appl.Environm.Micro.1978,35(2),582; Holdeman,L.V.,E.P.Cato,W.E.C.Moore:Anaerobe Laboratory Manual,4thEdn,1977,VPI and State University,Virginia; Macarie,I.,M.Brinzoiu,M.Bunea,L.Seiciu:Lucrari Stiintifice Institutul Agronomic N.Balcescu:,Seria C(1975/ 1976),18/19,77;McAllister,S.J.,H.J.Kurtz,E.C.Short Jr.: J.Anim.Sc.1979,49,868;Moon,H.W.:JAVMA,172,443;Qureshi, S.R.,H.J.Olander,S.M.Gaafar:Vet.Pathol.1978,15,353; O'Brien,Vet.Bull.1969,39,75;Russell,E.G.:Appl.Environ. Micro.1979,37(2) 187;Sandine,W.E.,K.S.Muralidhara,P.R. Elliker,D.C.England:J.Milk Food Technol.1972,35,691; Savage,D.C.:Soc.Gen.Microbiol.1972,22,25;Savage,D.C.: Ann.Rev.Micro.1977,31,107;Schulze,E.:Archiv.fur Experimentelle Veterinarmedizin 1977,31,299;Schwartz,W.L.: South Western Veterinarian 1971,25,11;Simmonson,A.,N. -E.Björklund:Swedish J.Agr.1978,8,97;Smith,H.W.,Bact. Rev.1977,41(2),475;Svendsen,J.,N.Bille,N.C.Nielsen,J. L.Larsen,H.-J.Riising:Nord.Vet-Med.1975,27,85;Tannock, G.W.,J.M.B.Smith:J.Comp.Pathol.1970,80,359;Tamas,J., J.Bokori:Magjar Allatorvosok Lapja 1979,30(19),677.