"MUCOSALIS" OF THE PIA COMPLEX - INTERACTION WITH OTHER PORCINE PATHOGENS

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The lesions of the proliferative enteropathies of the pig, intestinal adenomatosis, necrotic enteritis, regional ileitis and proliferative haemorrhagic enteropathy, are constantly associated with the presence of intracellular vibrios. From uncomplicated lesions of adenomatosis, the vibrio Campylobacter sputorum subspecies mucosalis can be recovered in large numbers from the lesions, whilst the recovery of this bacterium from the alimentary tract of non-proliferative mucosa in any numbers is rarely possible, even where the pigs have been deliberately exposed to the organism. In a sequential study of the development of the lesions of adenomatosis, mucosalis was found to multiply in association with the mucosa before the appearance of the lesions, be present in large numbers when the lesions were at their peak and to disappear from the mucosa as the changes were resolving.

Exposure of conventional pigs to C. sputorum ss mucosalis alone normally results in oral colonisation and limited recovery from the intestinal mucosa. It is not always clear whether this limited ability to recover mucosalis, especially from young piglets, is an effect of (i) the overgrowth of other bacteria on the selective media, (ii) some other factor or (iii) the genuine absence of mucosalis from the mucosa. Such a difference is clearly of some importance in evaluating the opportunity for intracellular parasitism provided by intestinal colonisation.

For these reasons a series of experiments was carried out with gnotobiotic and conventional early weaned piglets in order to establish whether intestinal colonisation of such animals took place after exposure to mucosalis and whether such colonisation resulted in intracellular parasitism. Gnotobiotic piglets exposed to mucosalis became colonised in the oral cavity and in the intestine for the duration of the first experiment (27 days) and at necropsy the organism could be recovered from the mucosa of most of the intestine of all the infected animals. Organisms were difficult to locate convincingly by any method (fluorescent antibody, silver staining or electron microscopy) but there was an absence of recognisable bacterial profiles within the cells of the infected epithelium and changes suggestive of adenomatosis were entirely absent.

Viral infections have been suggested as possible initiators of the bacterial parasitism seen in the proliferative enteropathies and porcine rotavirus must rank high in the possible candidates for such a role, both because of its ubiquitous nature and because of the type of change it induces in the epithelium. Both conventional early weaned and gnotobiotic piglets were exposed to dual infection by porcine rotavirus and to mucosalis. Infection of conventional piglets by rotavirus was aclinical and did not appear to influence the colonisation of the alimentary tract by mucosalis; infection of gnotobiotics resulted in a severe clinical rotavirus infection which once again did not provide any evidence for the intracellular parasitism of the enterocytes by C. sputorum ss mucosalis. In this experiment, the piglets were maintained until 54 days old and in neither this nor the previous infection did seroconversion take place measured by agglutinating antibody against the infecting serotype, although seroconversion to rotavirus occurred. Concurrent infection with E. coli 987 P and mucosalis was asymptomatic, whilst dual infection with cryptosporidia in some cases produced moderate enteric symptoms and lesions attributable to the cryptosporidia alone, in no case did this seem to modify the colonisation of the intestinal epithelium by mucosalis.

This search for a potentiating agent to promote intracellular parasitism by C. sputorum ss mucosalis has been without success, the work does possibly, however, support the view that the large numbers of mucosalis recovered from adenomatous epithelium are present specifically rather than non-specifically.

Selected references: Rowland, A.C. and Lawson, G.H.K. (1975). Vet. Rec. 97:178; Roberts, L., Rowland, A.C. and Lawson, G.H.K. (1977). Vet. Rec. 100:12; Roberts, L., Lawson, G.H.K. and Rowland, A.C. (1980). Res. vet. Sci. 28:145.