"MACROCELLS" OF THE PIA COMPLEX - INTERACTION WITH OTHER PORESCE PATHOGENS

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The lesions of the proliferative enteropathies of the pig, intestinal adenomatosis, necrotic enteritis, regional ileitis and proliferative hemorrhagic enteropathy, are constantly associated with the presence of intracellular parasites. From uncomplicated lesions of adenomatosis, the vibrio Campylobacter jejuni sub-species mucosalis can be recovered in large numbers from the alimentary tract of non-proliferative mucosa in any animals as 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. jejuni as mucosalis alone normally results in oral colonization and limited recovery from the intestinal mucosa. It is not always clear whether this limited ability to recover mucosalis, especially from young pigs, 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 colonization.

For these reasons a series of experiments was carried out with gnotobiotic and conventional early weaned pigs in order to establish whether intestinal colonization of such animals took place after exposure to mucosalis and whether such colonization resulted in intracellular parasitism. Gnotobiotic pigs exposed to mucosalis became colonized 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 intestines of all the infected animals. Organisms were difficult to locate consistently by any method (fluorescent antibody, silver staining or electron microscopy) but there was an absence of recognizable 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 pigs were exposed to dual infection by porcine rotavirus and to mucosalis. Infection of conventional pigs by rotavirus was limited and did not appear to influence the colonization 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. jejuni as mucosalis. In this experiment, the pigs were maintained until 94 days old and in neither this nor the previous infection did seroconversion take place as measured by agglutinating antibody against the infecting serovar, although seroconversion to rotavirus occurred. Concurrent infection with E. coli 587 P and mucosalis was asymptomatic, whilst dual infection with cryptosporidiosis in some cases produced moderate enteric symptoms and lesions attributable to the cryptosporidia alone, in no case did this seem to modify the colonization of the intestinal epithelium by mucosalis.

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