Correction page 505

5 % pentobarbital should read 9.5 % pentobarbital

- line 2 in Abstract
- line 10 in Materials and methods
- line 1 in Table 1
- line 2 in Table 2
- line 7 in Discussion
Dietary control of swine dysentery

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Introduction

In many parts of the world swine dysentery (SD) remains one of the most economically significant diseases of pigs. The condition results from infection with the aerotolerant anaerobic spirochaetal bacterium S. hyodysenteriae and may result in a severe mucosaemorrhagic colitis. In recent years, however, it has become evident that the presence of these organisms in pigs in a herd does not always result in disease (1, 2). The reasons for this are not always clear, and this led us to investigate whether the diet consumed could have a role in modulating the disease. It has previously been suggested that fermentable diets which result in acidic conditions in the large intestine may inhibit the growth of S. hyodysenteriae and prevent the development of disease (3).

Materials and methods

Groups of weaner pigs were fed either a highly fermentable unpelleted commercial diet, based on wheat and dehulled lupins (15%) or on an experimental diet based on 77% cooked rice and 21% animal protein. After four weeks adaptation to the diets, the pigs were either killed or challenged orally with cultures of S. hyodysenteriae. The latter animals were monitored for a further four weeks, and then most were killed. Pigs on the two diets were mixed daily over this period to maximise transmission of S. hyodysenteriae. At this stage two animals on the rice-based diet that had not developed SD were transferred to the commercial diet, and were rechallenged with S. hyodysenteriae.

When the pigs were killed, their large intestines and contents were weighed, the contents cultured for S. hyodysenteriae, the mucosa examined macroscopically and microscopically for lesions of SD, and the pH and concentration of volatile fatty acids (VFA) and lactate in the contents measured.

Results

The mean (±SEM) pH of caecal contents in pigs that were not challenged with S. hyodysenteriae, but which received the commercial diet (n=7) was 5.37 (±0.03), whilst it was 6.54 (±0.12) in those on the rice-based diet (n=6). Mean weights of the large intestine and its contents as a percentage of total body weight for pigs on the commercial diet was 4.53 (±0.04), compared to 1.76 (±0.10) for pigs on the rice-based diet.

Discussion

The low pH of the caecal contents, and the greater size and weight of the large intestines and contents in pigs fed the commercial diet was assumed to result from microbial fermentation of dietary residues. This low pH however did not prevent the proliferation of S. hyodysenteriae, nor the development of SD. In contrast, pigs on the rice-based diet had smaller and less well-developed large intestines, and underwent less fermentation at these sites (as judged by pH values, and VFA and lactate concentrations). We believe that these differences conferred protection from SD on the pigs fed this diet. When the diet was changed, one of two pigs developed SD. This was consistent with the rice-based diet being protective.

It is known from work in gnotobiotic pigs that S. hyodysenteriae requires the activity of other anaerobes before it will colonise the large intestine and cause lesions (4). We consider that the reduction/modification of microbial fermentation which resulted from feeding the highly-digestible rice-based diet indirectly inhibited colonisation by S. hyodysenteriae. A suitable environment was not available for the spirochaetes.

Further work is in progress to clarify the mechanisms of protection, and to develop commercially-viable protective diets.

References


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