Treponema hyodysenteriae Like Organisms from Herds Free of Swine Dysentery

R. J. Lysons*, R. M. Lemcke, J. Bew, M. R. Burrows, and T.J.L. Alexander
A.R.C. Institute for Research on Animal Diseases, Compton England RG16 ONN
and School of Veterinary Medicine, Cambridge England CB3 OES

Treponema hyodysenteriae like organisms were isolated on one occasion from each of 3 herds known to be free of swine dysentery. All three herds had a common source of breeding stock. This paper reports investigations on laboratory, pathogenicity and immunity studies using these organisms and also P18A, a strain of T. hyodysenteriae known to be pathogenic for pigs.

Materials and Methods

1) Laboratory studies. The strains of spirochaetes designated FW10, FW12 and BB310/81, were examined by tests developed to identify T. hyodysenteriae. These were the fluorescent antibody test (FAT) using absorbed sera (Hunter and Saunders 1977, Lysons unpublished). The slide agglutination (SA) test (Burrows and Lemcke, 1981); the disc-growth inhibition (GI) test (Lemcke and Burrows, 1979). The APIZYM enzyme test (Hunter and Wood, 1979) and indole production (Lemcke and Burrows, 1981). In addition, serological methods were developed to distinguish between P18A and the other strains. Hyperimmune sera was raised in rabbits to P18A and to FW10 using whole spirochaetes either as heat killed, or azide treated antigen. Agglutinin cross-absorbtion studies were carried out using antisera raised against FW10 absorbed with P18A and sera to P18A absorbed with FW10.

Pathogenicity tests. These were done in 5 to 7 week old SPF pigs (Table 1). The pigs were fed blood agar cultures of FW10 and FW12 on 3 or more occasions. Pigs were killed at intervals after the first oral dose as follows: Experiment 1, 24 to 37 days; Experiment 2, 13 or 14 days (1 or 2 pigs per group). The remaining pigs in Experiment 2 were observed for a further 23 days.

Immune Response. Serum antibody titers were determined at intervals with enzyme-linked immunosorbent assays (ELISA) using whole sonicated cells of either P18A or FW10 as antigen. Three pigs per group in Experiment 2 were dosed orally with 2×10^9 c.f.u. T. hyodysenteriae P18A on two occasions. They were killed 15 and 33 days later.

Results

Strains FW10, FW12, and BB310/81 were indistinguishable from T. hyodysenteriae by the SA and GI tests. FW10 and FW12 also gave the same enzyme pattern as T. hyodysenteriae, the same reactions with the FAT using sera absorbed with different combinations of non-pathogenic spirochaetes and were indole positive, BB310/81 was not subjected to these other tests.

When growth and haemolysis on sheep blood agar plates was compared, P18A and other T. hyodysenteriae strains of known pathogenicity were slightly more haemolytic than FW10, FW12 and BB310/81. However, the haemolysis produced by these later strains was considerably more than by Treponema innocens strain B256. Using haemolysis as a guide, FW10, FW12 and BB310/81 would be identified as T. hyodysenteriae.

Agglutination cross-absorbtion and growth inhibition studies have identified serologically different groups within the species T. hyodysenteriae (Lemcke and Bew unpublished). Thus, while growth inhibition studies suggested that FW10 was antigenically similar to P18A, agglutination cross-absorbtion indicated that they are not identical. Slide agglutination tests using hyperimmune sera to FW10 absorbed with P18A enabled cultures of FW10, FW12 and BB310/81 to be distinguished from cultures of P18A.

Pigs dosed orally with cultures of FW10 and FW12 showed no signs of diarrhoea or dysentery (Table 1) furthermore, there was no significant difference in growth rates in the 3 groups of pigs in Experiment 2 ($p<0.05$). These organisms were isolated infrequently from faeces but more often from mucosal scrapings ie: from 8 of 15 pigs examined. The serum antibody titres of pigs in Experiment 2 to P18A and FW10 rose slightly with age but there appeared to be no difference between pigs in the 3 groups.

One pig in Experiment 2 developed clinical swine dysentery after challenge with P18A. FW10 had not been isolated from 15 samples of rectal faeces taken from this pig before challenge. Another pig developed no diarrhoea or dysentery, but had colonic lesions of swine dysentery at post portem. FW10 had been isolated from this pig on two occasions before challenge with P18A.

Conclusions

Spirochaete were isolated from 3 herds which were indistinguishable from T. hyodysenteriae by a number of laboratory diagnostic tests. They were isolated from only one pig per farm. When given orally to SPF pigs they were shown to be present in the colonic mucosa of some of the pigs, but produced no clinical disease or colonic lesions.

These organisms would appear to be avirulent variants of T. hyodysenteriae, and as such they could confuse laboratory diagnosis of swine dysentery. Strain FW10 appears to be antigenically similar to but not identical with T. hyodysenteriae with strain P18A and a slide agglutination test has been developed to distinguish between them. Comparison of these two strains may reveal one or more virulence determinants of T. hyodysenteriae.

Selected references: Burrows, M.R., and Lemcke, R.M. (1981) Vet.Rec.108, 187; Hunter, D., and Wood, T. (1979) Vet.Rec.104, 383; Hunter, D., and Saunders, C.N. (1972) Vet.Rec. 101, 303; Lemcke, R.M. and Burrows, M.R. (1979) 104, 548.

Table 1. Pathogenicity and protection studies with T. hyodysenteriae FW10 and FW12

Organisms given orally	Pigs affected/pigs examined: Isolation T. hyodysenteriae from faeces	Isolation T. hyodysenteriae from colonic mucosa	Mucoid diarrhoea	Colonic lesions	S.D. following challenge with P18A
Exp. 1					
FW10	0/3	1/2	0/3	0/2	ND
FW10	0/5	3/5	0/5	0/5	ND
Exp. 2					
FW10	3/5	1/2	0/5	0/2	2/3
FW12	3/5	2/2	0/5	0/2	0/3
nil	0/4	0/2	0/4	0/1	0/3

P18A - pathogenic T. hyodysenteriae; ND - not done
S.D. - swine dysentery