Bacterial Competition as a Means of Preventing Neonatal Diarrhea in Pigs

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Baby pigs orally inoculated with a porcine strain of enterotoxigenic Escherichia coli (K88+,Ent+) showed signs of depression, severe diarrhea, and, in some instances, death. Few, if any, signs of illness occurred if baby pigs were first inoculated with a K88-possessing non-enterotoxin-producing strain of E. coli.

Neonatal diarrhea in swine has been shown to be produced subsequent to the ingestion of Escherichia coli with the capacity to produce enterotoxin (9, 12, 13, 15). Enterotoxin has been shown to cause diarrhea through activation of intestinal epithelial cell adenyl cyclase, which ultimately results in net fluid secretions into the lumen of the bowel (8).

Since only the cells of the small intestine are susceptible to the action of the toxin, E. coli, in order to be an efficient cause of disease, must remain in close proximity to the target cells of the small intestine (13). In swine, fimbrial antigens (K88) have been shown to be one mechanism by which E. coli might adhere to cells of the small intestine (1, 2, 5–7, 11, 14, 16, 17, 20).

We have recently shown that mice orally inoculated with a non-enterotoxin-producing strain of E. coli possessing K88 antigen would be protected after the oral inoculation of an enterotoxin-producing strain of E. coli possessing K88 antigen. This protection was not observed when a non-enterotoxin-producing K88-negative strain of E. coli was used (3). We concluded that this protection was afforded by competition for combining sites on the cells of the small intestine.

The purpose of the experiments reported in this paper was to determine whether the same protection could be demonstrated in newborn pigs. Specifically, experiments were designed to determine whether prior inoculation of newborn pigs with a non-enterotoxin-producing, K88-possessing E. coli (K88+,Ent−) would protect them from diarrhea after challenge with an enterotoxin-producing, K88-possessing E. coli (K88+,Ent+).

Pregnant cross-bred gilts and sows were the source of the baby pigs used in the experiments. E. coli strain P66 is K88+,Ent− of serotype O8:K40, 88ab:H9. Strain P66a is K88+,Ent+ of serotype O8:K40, 88ab:H9. Strain P66a was shown to be enterotoxigenic by the infant mouse inoculation test (4). Both strains were of porcine origin. Before inoculation of these strains, the presence of K88 antigen was confirmed by a slide agglutination test with K88-specific antiserum.

At 1 and 6 h after birth, three or four baby pigs from each litter were inoculated orally with 10⁶ cells of P66. The remaining baby pigs (usually three or four) of each litter were not inoculated at this time. Twenty-four hours after birth, all the pigs were inoculated orally with 10⁶ cells of P66a. The baby pigs were left with their mothers throughout the course of the experiment.

The baby pigs were observed every 6 h for 36 h after birth. The degree of illness was recorded using the following criteria: 0, normal appearing; 1, normal behavior, slightly loose stools; 2, mildly depressed, obviously loose stools; 3, severe depression, obviously loose stools with considerable scalding on the perineal region; 4 death.

The data (Table 1) show that P66 given before inoculation with P66a afforded a considerable degree of protection. It is our contention that the mechanism of protection is through bacterial competition. Since the K88 antigen has been shown to have a high affinity for cells of the small intestine (2), colonization of this area by the competing strain possessing the K88 antigen would be enhanced. Subsequent attachment of the K88+,Ent+ strain to the toxin-sensitive cells might be curtailed simply because of the large members of the competing strain in the small intestine or by direct blockage of the attachment site, as was observed in mice (3). In either case, the K88+,Ent+ strain
would have no place to attach and would be moved, by peristalsis, distally, away from the toxin-sensitive cells of the small intestine.

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LITERATURE CITED


