MUCOSAL COMPETITIVE EXCLUSION TO REDUCE *SALMONELLA* IN SWINE

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Control of *Salmonella* on the farm is currently undertaken by changes in management protocols or by use of live attenuated vaccines (Roof et al., 1992; Kramer et al., 1992). Although these vaccines are efficacious in reducing morbidity and mortality associated with salmonellosis, carrier animals are still evident within the population. While the carrier state has been defined for *S. typhimurium* and *S. choleraesuis* under experimental conditions (Wood et al., 1989; Fedorka-Cray et al., 1994; Gray et al., 1995, 1996a, 1996b), little is known about the exact modes of transmission and maintenance of the disease in swine herds.

Competitive exclusion implies the prevention of entry into a given compartment because that space is already occupied, the competing entity is better suited to establish and maintain itself in that environment or the competing entity is producing a product hostile (toxic) to its competition (Bailey, 1987). In the early 1970s, Nurmi and Rantala (1973) demonstrated that the susceptibility of broiler chicks to salmonellae colonization was probably due to the delayed establishment of intestinal microflora in chickens reared according to modern mass production methods. They also showed that salmonellae infections could be prevented by feeding the chicks anaerobic cultures of normal intestinal adult fowl flora (referred to as "competitive-exclusion" or CE; Nurmi and Rantala, 1973; Rantala and Nurmi, 1973). Since that time the efficacy of the CE concept has been demonstrated in many laboratories (Barnes et al., 1980; Bailey, 1987; Bailey et al., 1991).

Although the efficacy of CE has been demonstrated in chickens, little work has been done with CE in other species.

MATERIALS AND METHODS

We have prepared a mucosal competitive exclusion culture from the cecum of a healthy 6 week old swine (MCES) which was known to be free of *Salmonella*. This culture was propagated by methods as previously described (Barnes et al., 1980; Bailey et al., 1991) at the USDA-ARS-Russell Research Center in Athens, GA and sent frozen to NADC. The frozen culture was used to seed culture medium and repetitive passages were made every 24 h. A total of 1 to 7 passages were generated prior to inoculation of the pigs.

Sows with known farrowing dates were procured and brought to farrowing crates in isolation units at NADC. Each sow was checked every 4 h beginning 1 day before the known farrow date to insure that the first MCES culture was administrated in a timely manner. At farrowing, pigs were allowed to suckle to insure that they obtained colostrum and each pig was administered at 24 h. Pigs were challenged with $10^7$ CPU *S. choleraesuis* by intranasal given 5 ml of MCES by oral gavage between 2 and 6 hours post-farrowing. A second 5 ml instillation 48 h post-farrowing (24 h past the last MCES administration). Rectal temperatures and rectal swabs were taken daily for 7 days post-challenge from each pig and cultured for *Salmonella*. At day 7 post-challenge, all pigs were necropsied. Tissues were collected for qualitative bacteriology and included tonsil, mandibular lymph node, lung, bronchioloe lymph node, liver, spleen, middle ileum, ileocolic junction, ileocolic lymph nodes, cecum, cecal contents (CC), colon, colonic lymph nodes, and stomach wall. Quantitative bacteriology was also conducted on the cecal contents and ileocolic junction to determine the level of *Salmonella* within tissues. Bacteriologic protocols

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were followed as previously described (Gray et al., 1995, 1996a, 1996b).

In order to assess the impact the sows may have had on the pigs, sow feces was also collected and cultured prior to farrowing, within 48 h after farrowing and at day 7 post-challenge of the pigs (sows were never directly challenged with *S. choleraesuis*). Control pigs were not given MCES but were challenged at 48 h of age. Tissues were collected and processed as described above.

RESULTS AND DISCUSSION

Recovery of *Salmonella* from rectal swabs was variable. Total numbers of *Salmonella* recovered from the cecal contents and ileocolic junction are shown in Table 1.

Table 1. Total numbers of *Salmonella* recovered from the cecal contents and ileocolic junction

<table>
<thead>
<tr>
<th>Group</th>
<th>Cecal Contents</th>
<th>Ileocolic Junction</th>
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<tbody>
<tr>
<td>CE</td>
<td>3.19</td>
<td>3.13</td>
</tr>
<tr>
<td>Control</td>
<td>5.09</td>
<td>5.45</td>
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A 2 to 5 log reduction of *Salmonella* in the CC or ICJ was observed in the MCES treated pigs when compared to the controls. Total percent positive tissues from CE treated versus untreated pigs indicate a 20% difference between groups. However, examination of the gut tissue only indicates that 28% of the gut tissues were positive from the MCES treated pigs versus 79% positive tissues from the control pigs (51% difference; Table 2).

Table 2. Percent positive gut tissues from CE treated versus untreated pigs

<table>
<thead>
<tr>
<th>Group</th>
<th>Tissues (No.+/Total No.)</th>
<th>% Positive</th>
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<tr>
<td>CE</td>
<td>54/190</td>
<td>28.4</td>
</tr>
<tr>
<td>Control</td>
<td>59/75</td>
<td>78.7</td>
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While a reduction is observed in pigs already colonized with "other" *Salmonella*, the degree of protection is reduced, suggesting that in suckling pigs, an earlier administration of CE may be warranted. Salmonella reduction was imparted in CE treated versus untreated pigs. These data indicate that use of MCES may be a practical solution for control of *Salmonella*.

REFERENCES


