EFFECT OF COMPETITIVE EXCLUSION ON SALMONELLA SHEDDING IN SWINE

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Despite the efforts of researchers and public health agencies, the incidence of human salmonellosis has continued to increase over the past 20 years. Salmonellosis is now the most common cause of food-borne gastroenteritis. The number of reported cases of human \textit{Salmonella} infection exceeds 40,000 per year. The Communicable Disease Center estimates that the true annual incidence of human salmonellosis in the United States may be as high as 4 million cases.

\textit{Salmonella} have been isolated from all vertebrate hosts from which they have been sought with the possible exception of healthy fish in unpolluted water (Taylor and McCoy, 1969). Swine, cattle, and poultry are known carriers of salmonellae (Bean and Griffin, 1990; Lammerding et al., 1988). \textit{Salmonella} have been associated with food-borne illness in humans (Bean and Griffin, 1990). Over two million cases of meat and poultry food-borne disease occur in humans in the United States per year at a cost of over one billion dollars (Menning, 1988; Roberts, 1989). Human infection with salmonellae typically occurs through the ingestion of contaminated food or food products, resulting in severe gastroenteritis (Fedorka-Cray et al., 1995). \textit{Salmonella} in swine are responsible for millions of dollars in lost revenue to the swine industry (Sockett 1991; Todd, 1989). Transmission of the pathogen amongst swine can occur both by the fecal-oral route and intranasally, resulting in colonization of and dissemination from the gastrointestinal tract and organs such as lung and tonsils (Fedorka-Cray et al., 1995). \textit{Salmonella} colonize and inhabit the cecum of swine (Currier et al., 1986), and the ceca of poultry has been shown to be the primary site of salmonellae colonization (Hudault et al., 1985) and serve as a reservoir for salmonellae in other food producing animals. The digestive tract of the newborn pig is usually sterile but rapidly develops a microflora characteristic of the species as it is exposed to a traditional commercial environment (Fuller, 1989). During the first few weeks of life immature gastrointestinal systems are highly susceptible to colonization by enteropathogens, however, the presence of a stable mature microflora helps the animal resist infections, particularly in the gastrointestinal tract (Fuller, 1989).

Recently, our laboratory has employed the use of continuous-flow culture technology to develop microbial competitive exclusion cultures for swine. The ability of the culture to protect the gastrointestinal tract against \textit{S. cholerasuis} colonization is currently being tested in our laboratory and initial studies have focused on the on the ability of this culture to decrease fecal shedding of

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*S. cholerasuis* in baby and newly-weened pigs.

The intact cecum from an adult pig was removed and transferred to an anaerobic chamber where portions of the cecum contents were collected, mixed with glycerol and stored at -70 Celsius. Cecal contents were later thawed and used as an inoculum for a continuous-flow culture. Culture medium was Viande Levure broth and the culture was maintained with a turnover time of 24 hr at 39 Celsius under anaerobic conditions. The culture (Pcf1) reached steady state conditions after 5 vessel turnovers as measured by a constant pH and fermentation acid profile. An additional culture designated Pcf3 was developed from Pcf1 via a series of serial dilutions and was maintained under the same conditions as described above for Pcf1.

In the first experiment baby pigs were provided 5 ml of Pcf1 within 4 hr after weaning, and again 24 hr later. Pigs were challenged with 10³ CFU *S. cholerasuis* 24 hr after the second dose of Pcf1. Fecal *Salmonella* shedding was measured for 7 days post *Salmonella* challenge via rectal swabs. 100% of the possible 72 rectal swab samples obtained from the untreated control pigs (non Pcf1 treated) were *Salmonella* positive versus 10/56 or 18% *Salmonella* positive rectal swab samples in the Pcf1 treated group. In a second experiment piglets were provided 5 ml of Pcf3 at farrowing, and challenged with either 10⁵ or 10⁶ CFU *S. cholerasuis* 24 hr later, then at weaning provided a second dose of pCF3. Fecal *Salmonella* shedding was measured for 7 days post-*Salmonella* challenge. Untreated controls in the 10⁵ challenge group had a *Salmonella* shedding rate of 32% compared to 0% in the Pcf3 treated group. Untreated piglets challenged with 10⁶ CFU *Salmonella* had a *Salmonella* shedding rate of 81% compared to 44% in the Pcf3 treated group.

These experiments suggest that by establishing a robust microflora in the gastrointestinal tract of baby pigs, *Salmonella* shedding in the feces can be decreased, and competitive exclusion may be useful in decreasing *Salmonella* colonizaton in young pigs.

REFERENCES


