RESEARCH REPORT

PORK SAFETY

**Title:** Factors Affecting *Campylobacter* Status of Swine, **NPB #98-151**

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## I. Abstract:

Four Yucatan miniature gilts, naturally infected with *C. jejuni*, had cecal cannulas surgically implanted. For 7 days prior to and for 7 days following a 48 hr. fast, cecal samples were collected and mean values were determined for pH, volatile fatty acids (VFA), and colony forming units (cfu) of *C. jejuni*. This was replicated 3 times. To study the effects of transport stress, full-fed gilts were loaded onto an open stock trailer and transported for 3-5 hr. Cecal samples were collected for 48 hr. before and for 48 hr. after transportation, and pH, VFA, and cfu were measured. A diet formulated with 50% lactose was fed for 48 hr. and cecal samples were evaluated for changes in pH and VFA. *C. jejuni* bacteria from the gilts in this study were sensitive to the antibiotic erythromycin. In an attempt to eliminate *C. jejuni* from the gastrointestinal (GI) tract, gilts were fed an oral suspension of 1.6 g erythromycin/pig/day (800 mg twice daily) for 10 days. Cecal contents were collected every second day, and values for pH, VFA, and cfu were determined. Pigs were euthanatized and ileocolic lymph nodes and cecal contents cultured for *C. jejuni*. Following the 48 hr. fast, cecal pH increased by 1.66 units (approximately 50 times); acetic, propionic, and butyric acids decreased by 61%, 71%, and 19%, respectively; and the cecal concentration of *C. jejuni* increased by over one hundred-fold. Values returned to pre-fast levels within 5 days of full feed. We observed no changes in measured cecal values following transportation or following a diet of 50% lactose. Concentrations of cecal bacteria progressively decreased during antibiotic treatments and cecal samples were culturally negative for *C. jejuni* by 7 days. Lymph node and GI tract cultures for *C. jejuni* were also negative upon necropsy.
These data are important for food safety considerations because feed withdrawal, commonly associated with shipping and slaughter, can increase the shedding of *Campylobacter* in swine. Experimentally, it is possible to eliminate *Campylobacter* from the GI tract of swine.

## II. Introduction:

In recent years, *Campylobacter* has emerged as one of the most common causes of human enteric disease in developed countries. In the United States, *Campylobacter* is considered by many to be the chief culprit of enteric illness; in the Netherlands,

*Campylobacter* is responsible for 12% of all cases of acute gastroenteritis; and in Denmark, the number of cases of campylobacteriosis doubled during the four years from 1992 to 1996. Secondary conditions associated with *Campylobacter* infections include miscarriage, pneumonia, meningitis, and a highly debilitating form of Guillain-Barre' syndrome. While poultry, raw milk, and untreated surface water are assumed to be the chief sources of infection in the United States, the actual source of infection often goes unidentified. *Campylobacter* has been isolated from raw beef, pork, lamb, cooked meats and seafood. Pigs are probably one of the natural reservoirs of *Campylobacter* with slaughterhouse isolation rates ranging from 66% to 95%, yet very little is known about the epidemiology of *Campylobacter* in swine. *Campylobacter* has repeatedly been isolated from pork and other meats. *Campylobacter* incidence studies have been identified as providing critical information by FSIS in their 1997 Food Safety Research Agenda--Directions for Future Food Safety and Inspection Service. This directly supports the identification of emerging pathogens along any point of the farm-to-table continuum to be implemented by FSIS and ARS as outlined in the Presidential Food Safety Initiative.

**III. Objectives:**

The objectives of the proposed research are to evaluate some of the factors that influence the pathogenesis of *Campylobacter* carrier status in pigs and to explore some intervention strategies affecting *Campylobacter* infection. Specifically, we will look at the impact of antimicrobial treatments on carrier status and how feed withdrawal and feed manipulation affects *Campylobacter* populations in pigs.

**IV. Procedures:**

The authors will surgically implant cannulas into the ceca of research pigs which will allow multiple cecal samples to be withdrawn over a relatively long period of time to evaluate how the cecal environment affects *Campylobacter* populations. Items to be evaluated include dietary alterations and management practices and their effects on cecal pH, volatile fatty acid profiles, and bacterial populations (both *Campylobacter* and natural microflora). In addition, the influence of feed withdrawal times versus cecal *Campylobacter* status can be monitored. A biometrician is on our staff and appropriate statistical analysis of data will be employed. All animal experimentation project proposals will be submitted to and approved by the Laboratory Animal Care and Use Committee and we will abide by recognized guidelines for humane care and treatment of experimental animals.

**V. Results:**

Feed Withdrawal—A 48 hr. fast caused a multitude of changes in the intestinal tracts of our experimental gilts. The “good” VFA’s (important building blocks formed during the digestive process) went down dramatically; the pH increased (acidity went down) approximately 50-fold; and *Campylobacter* concentrations in the digestive tract increased by 100-fold. Feed withdrawal, commonly associated with shipping and slaughter, can therefore increase the shedding of the food-borne disease organism *Campylobacter*. This could be of public health significance.

Transportation Stress—In an effort to separate the influence of feed withdrawal from the stresses of shipping on *Campylobacter* shedding, we transported full-fed gilts in a livestock trailer for 3-5 hrs. *Campylobacter* concentrations were evaluated before and after transporting. Under our conditions, transporting had no effect upon bacterial populations, VFA, or pH.

Intervention and Dietary Manipulation—Because low pH (more acidic) conditions in the lower intestinal tract appear to inhibit growth of *Campylobacter*, we fed our gilts diets of 50% lactose. The theory was that if an appreciable amount of the lactose was not digested, a higher level of lactic acid would be present in the lower intestinal tract. We saw no effects of this diet on the cecal contents. The *C. jejuni* of our gilts was sensitive to erythromycin. We fed an oral suspension of erythromycin for 10 days and we were able to eliminate *Campylobacter* from both the lymph nodes and lower digestive tract of our gilts. This could be a possible method of *Campylobacter* reduction in pigs prior to slaughter.