Although over 30 different etiologies are recorded in the literature for lactation failure in swine, the exact causative agents is not currently known. It is postulated that lactation failure is the result of an interplay of disease processes, altered endocrine functions, and many of the stress situations associated with lactic acidosis. The potential sites of endotoxin absorption in naturally occurring cases are the mammary glands, the uterus, and the small intestine. Although E. coli endotoxin absorption from the mammary glands and uterus has been demonstrated, it has not been demonstrated that absorption occurs from the small intestine.

An increased incidence of lactation failure has been associated with disturbed or altered feeding programs, both overfeeding and underfeeding (3-4). Experimentally the disease syndrome has been reproduced by drastic feed changes (3), feeding of poor quality feed (3), administration of antiperistaltic agents (1), by feeding large quantities of skim milk (3), and by overfeeding with a finely ground feed following the addition of a bacterial contamination of the feed (3). Constipation is a common finding in sows with lactation failure (3). It has been stated that endotoxin enters the lymphatic system and is carried by the lymphatic system to the liver where it is detoxified (5, 6). The significance of this process is not known. The pathological changes associated with lactation failure are thought to be the result of shortening of the crypt length (6), decreased mitosis of the crypt epithelium (7), increased permeability of the small intestine (6), and increased blood flow to the small intestine (6). It is not known whether these changes are the result of endotoxin absorption from the small intestine or from other sources.

Several workers have suggested that stress, for example parturition or altered feeding programs during the periparturient period, may alter the barrier function of the gut epithelium such that endotoxin from the intestinal flora may enter the systemic circulation (9-10). Previous studies in our laboratory demonstrated that sows and gilts fed limit-fed during the periparturient period had lower serum endotoxin levels than sows and gilts allowed free choice diets. Limit-fed sows had a higher incidence of lactation failure than free-choice sows. In the study, it is postulated that any of the thirty predisposing factors listed in the literature for lactation failure may alter the natural resistance of the inner lining of the small intestine and allow endotoxin absorption. A study was conducted to help elucidate the significance of endotoxin absorption from the small intestine in the pathogenesis of lactation failure in swine.

Because of the uncertainty of the effects of gastric secretions or orally administered endotoxin and because of the importance of determining the absorption from different portions of the gut, a method had to be devised to place the endotoxin directly into the small intestine. A variation of the McCall jejunal fistula for dogs was used (9). After the site for fistulation was located, the intestine was transected, the proximal portion of the gut was anastomosed to the intestine approximately 2 cm distal to the transaction using an end to side crushing technique, and the distal portion of the transaction was sutured into the skin incision in the paralumbar fossa. Because the direction of peristalsis is away from the skin only minimal leakage occurred. No external or artificial devices were used to close the fistula.

Twelve pregnant sows were prepared for this experiment by placing jejunal fistulas as described. Control blood samples were obtained by caval vein puncture at weekly intervals for 3 weeks prior to farrowing. Total white blood cell counts (WBC) were determined, and the Limulus Amebocyte Lysate (LAL) assay was used to detect plasma endotoxin (10). Within 48 hours following farrowing 6 sows were infused with endotoxin (200 mg Lipopolysaccharide W/5 E. coli 0127:B8, Difco Laboratories, Detroit, Mich.). 2 sows were infused with pyrogen free water; 2 sows were infused intraperitoneally with endotoxin; and 1 sow spontaneously developed lactation failure. The sows were bled at 0, 1, 3, and 8 hours post-infusion. Rectal temperatures were taken at 15 minute intervals for the first hour, then hourly, until 20 hours. Daily pig weights were recorded daily. None of the nine sows infused intraperitoneally showed significant declines in WBC, increases in rectal temperatures, or positive LAL's. The two sows infused intraperitoneally did have declines in WBC, increases in rectal temperatures, and positive LAL's following infusion. The sow with spontaneous lactation failure had low WBC, high rectal temperatures, and positive LAL's.

Conclusion:

There was no evidence that the exogenous E. coli endotoxin infused directly into the small intestines of early postpartum sows caused any adverse effects.

References: