FATE OF ESCHERICHIA COLI ENDOTOXIN INFUSED INTO JEJUNUMS OF PIGS

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Although over 30 different etiologies are recorded in the literature for lactation failure in swine, the pathogenesis of the causative agents is not currently known. It is postulated that lactation failure is the result of an interaction between endotoxins from gram negative bacteria, altered endocrine functions, and any of the many stress situations listed in the literature as predisposing factors. A high percentage of field cases are associated with Escherichia coli and other gram negative organisms. Likely 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 (1,2), it has not been demonstrated that absorption can or does occur 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 followed by the addition of a bacterial culture of E. coli or B strept. to the feed (3). Constipation is a common finding in sows with lactation failure (3). It has been stated that finely ground feed or overfeeding allows packing in the sow's stomach in such a way that bacteria are not destroyed by the action of the hydrochloric acid (3). It has been suggested that the bacteria may then pass through the intestinal tract where their toxins may be absorbed. Although lactation failure has been reproduced experimentally by altering normal feeding programs, the pathogenesis of these alterations has not been elucidated. The relationship between altered dietary regimes and bacterial toxin absorption in the small intestine has only been postulated, not demonstrated.

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 (5-9). Previous studies in our laboratory demonstrated that sows and gilts limit-fed during the periparturient period had less stable hormonal 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 our postulate 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. This 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 particular portions of the gut, a method had to be devised to place the E. coli endotoxin directly into the small intestines. A variation of the Maydl jejunostomy 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 12 cm. distal to the transection using an end to side crushing technique, and the distal portion of the transection was sutured into the skin incision in the paralumbar fossa. Because the direction of peristalsis was away from the skin only minimal leakage occurred. No external or artificial devices were used to close the fistulae.

Twelve pregnant sows were prepared for this experiment by placing jejunal fistulas as described. Control blood samples were obtained by cranial vena cava 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/E. coli 0:27:38, Difco Laboratories, Detroit, Mich.); 3 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 for 8 hours, and at 24 hours. Baby pigs weights were recorded daily. None of the nine sows infused intrajejunally 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 the 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:

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