Lactation failure in domestic swine is considered to be a worldwide problem. Within Illinois, a recent survey of swine producers has shown that lactation failure is considered to be a significant problem (present in 50% or more of farrowings), in about 7% of herds.

In another field survey of the 1974 syndrome in Illinois the incidence of the disease was 6.5% (preliminary results). Other earlier surveys have attributed more than 7% of neonatal pig deaths directly or indirectly to lactation failure.

Recently much effort has concentrated on coliform infections and endotoxin involvement in lactation failure.1,4,5 The purpose of this study is to examine actual cases of lactation failure for the presence of detectable levels of endotoxin in the blood and to ascertain likely sources of endotoxin absorption.

At the time of writing, preliminary results are reported.

Materials and Methods:

Some sows in the project were selected from privately owned swine producing units in Illinois described by the attending veterinarian as having a high incidence of lactation failure. At each production unit studied two to three animals were selected from each of two groups; those affected clinically with symptoms of MSA and those which appeared to be clinically healthy. The affected sows each showed one or more of the following clinical signs: pyrexia (rectal temperature >103°F), hunchback depression, clinical signs of inflammation of the udder, or vaginal discharge. A few sow exhibited hyperemia or edema or edema, diagnosed by the unseized condition of the litter of pigs and the hungry behavior of that litter. To provide accuracy in selection of cases, any sow with a litter that showed signs of diarrhea before examination by the investigator was eliminated from the study. Control sows exhibited none of the above clinical signs.

To evaluate each case for the presence of detectable endotoxin in blood, a sample was obtained from the antebrachial vein after sterile preparation of the jugular vein. All methods used for handling and processing of blood samples for endotoxin analysis were rendered pyrogen-free by careful washing followed by boiling at 100°C for four hours. The blood samples obtained were analyzed for endotoxin using the Limulus Amoebocyte Lysate (LAL) test. Plasma was heat-extracted eliminating inhibition of the LAL test.6

To enable bacterial culturing of the intestinal tract and also to ensure uterine bacterial culturing without contamination from the cervix or vagina, a laparotomy technique was utilized. Each case received epidural analgesia and was tied in right lateral recumbency for additional restraint. The left parametrial fossa was prepared for surgery and a laparotomy performed. The ovaries, uterus and lymph were each elevated and flushed with sterile saline with a needle and syringe in standardized locations to obtain cultures for bacteriological analysis.

The udder was prepared by scrubbing dorsal-lateral to the teats for sterile acquisition of milk samples. With a syringe and 20g, needles the teat cleft was entered and milk aspirated for bacteriological culturing. The above techniques precluded contamination of the milk from pooling of milk on the rough end of the teat as occurs in usual sampling methods. Six glands of the udder were cultured from each case in which milk could be obtained.

Results:

To date 24 affected and 13 control sows have been evaluated. Eight (33.3%) of the affected and 2 (15%) of the control sows have had demonstrable levels of endotoxin in blood samples as analyzed by the LAL test. Bacteriologic results demonstrated a higher incidence of Gram-negative "pathogens" in the uterus, teat, and udder, 13.5%, 6%, and 21%, respectively, of the affected cases, than in controls, 0%, 0%, and 0%, respectively. Final results of the study are not available at this time of writing.

Conclusions:

Gram-negative bacteria and endotoxin are normally present in the intestinal tract of animals, but are prevented from reaching peripheral circulation by the intestinal mucosal barrier and the recto-vaginal system. Endotoxin is not expected to be normally present in the healthy udder or uterus. At the time of farrowing, increased stress may reduce the immunological capability of the sow. With a reduced immune response, farrowing and post-farrowing sow become more susceptible to bacterial infections from the environment. There may also be a change in intestinal bacterial growth patterns. Greater than normal exposure to endotoxin coupled with a reduced capability to eliminate endotoxin that enters the blood may lead to endotoxia.

Preliminary results of work by Drs. W. Wagner and colleagues at the University of Illinois have shown an effect on prolactin in the post-farrowing sow following intravenous endotoxin administration.7 The study reported here demonstrates that endotoxia may indeed be a factor in actual clinical cases of lactation failure in sows. The intestinal tract is suggested as the primary likely source of endotoxin absorption, followed by the uterus and udder in cases of mastitis or mastitic caused by Gram-negative bacteria.

References: