Lactation Failure in the Sow – Pork Industry Handbook
Michigan State University Extension Service
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pork industry handbook

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Lactation Failure in the Sow

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In the past, lactation failure in swine was known as "MMA" or "mastitis, metritis, agalactia." Now it is identified as the Periparturient Hypogalactia Syndrome (PHS). Symptoms vary from death of baby pigs from starvation to increased susceptibility to other newborn pig diseases, or in its mildest form, poor performance of the litter. It is referred to as a syndrome to indicate that there may be several risk factors and clinical diseases that are referred to collectively as lactation failure or, more exactly, a temporary dysgalactia. Lactation failure is a major concern to the pork producer because of the economic impact.

The usual swine dysgalactia syndrome occurs in the very early stages of lactation. If the herdsman is trained to recognize the signs, the disease may be evident at farrowing when it severely affects the sow and/or the piglets. However, the most common form of this syndrome is not easy to recognize because clinical signs are related only to relatively poor growth performance of the litter. Under the title of lactation failure, a sow can have a variable degree of lactational impairment from a total lack of milk production (agalactia) to the more commonly encountered hypogalactia (reduction in the normal amount of secreted milk). In many cases, the sow has only a temporary reduction in the normal amount of secreted milk (hypogalactia) beginning with dysgalactia, which means that there is an impairment in the initiation of lactation. The number of sows or gilts affected with this disease syndrome during a farrowing period varies from 10% to 100%. It may be important to identify susceptible sows, i.e. primiparous (first litter) and/or multiparous sows, young and/or oldest sows.

Clinical Signs

Lactation failure can be present at farrowing time or it can appear within the first few days after parturition, or even after 10 to 14 days of successful lactation. Most sows appear normal; however, some hypogalactic sows exhibit rapid breathing, depressed attitude, lack of desire to eat or drink, fever, reluctance to move about, and refusal to allow nursing. Restless baby pigs are an indication of the need for closer evaluation of the sow.

Lactation failure is a multifactorial syndrome, and many pathways have been identified. The origin of cause may differ from herd to herd. It can start in the mammary glands, uterus, urinary tract, or gastrointestinal tract. When the primary site of disease is in the mammary glands, they may appear enlarged, firmer, warmer, and more sensitive than usual, and often discolored when compared to the other glands. Some hypogalactic sows will, however, show little or no apparent change. Mastitis, an inflammation of the mammary glands usually caused by bacterial infections, is a frequent component in this disease syndrome. All mammary glands are rarely affected with mastitis. Careful palpation of the mammary glands of each sow several times within the first 12 to 36 hours after farrowing may reveal developing hypogalactia and allow for treatment early in the syndrome. In some sows, the affected glands may show typical signs of regression or drying off similar to that seen at weaning. In these instances, the glands probably are not mastitic. Palpation prior to farrowing can be helpful in detecting sows with excessively hard or edematous (caked) glands.

When the primary site of the disease is an infection located in the uterus and/or a urinary tract infection, there is not always a discharge. The difference between normal and abnormal vulvar discharges is difficult to determine. The presence of purulent discharge (pus) from the birth canal does not necessarily indicate that the sow or gilt has a uterine infection (metritis). Although studies have revealed that over 60% of clinically normal sows have vulvar discharge for 24 to 36 hours, this discharge is not correlated with impaired piglet growth. Discharges that persist past 36 hours often are accom-

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panied by decreasing thrift of the litter and indicate a need to examine and treat the sow.

If the disease starts in the gut, it is generally associated with constipation. This condition may be linked with feed intake or, more often, with low water intake before farrowing.

Tissue changes within the affected sow are variable because of the different causative agents or management factors involved. Lactation failure can be a primary disease or secondary to other diseases, such as mycotoxicosis (zearalenone toxicity) and PRRS (Porcine Reproductive and Respiratory Syndrome).

The decrease in milk secretion is the result of alteration in the hormonal sequence of events necessary for normal mammary gland secretion. The hormones involved primarily in mammary gland development and function–estrogens, progesterone, and prolactin–along with a number of direct and indirect synergistic hormones, are essential to full development and secretion of milk. Each of these hormones must be present at the proper time in the proper amount to initiate and maintain lactation. Anything altering the levels of the hormones, such as environmental stress, poor nutrition, bacterial endotoxin, low water intake and/or poor water quality, can decrease lactation.

The precise role of bacterial endotoxin in the lactation failure syndrome is still unresolved. Endotoxin is a large lipopolysaccharide molecule released from the cell wall of bacteria such as *E. coli* after the bacteria die. This endotoxin can be readily absorbed from the mammary gland, uterus, urinary tract, and/or intestines into the blood vascular system. It can cause many changes in pigs, including fever, increased adrenal corticoid secretion, and in early lactation, a decrease in prolactin secretion which depresses milk production.

Despite the fact that many infectious causes have been identified, mastitis caused by *E. coli, Klebsiella,* or related bacteria is common. In the affected sow, however, a multifactorial approach to lactation failure evaluation seems to be more appropriate (Figure 1). Numerous factors can be involved allowing the absorption of sufficient endotoxin to be present, or an endocrine (hormonal) imbalance to occur. The role of genetics is not known. In a given herd, lactation failure may affect either primiparous sows, the oldest sows, or all parity sows. It is important to maintain accurate health records to clearly identify the affected sows within a herd and initiate preventative measures and treatment when the syndrome occurs.

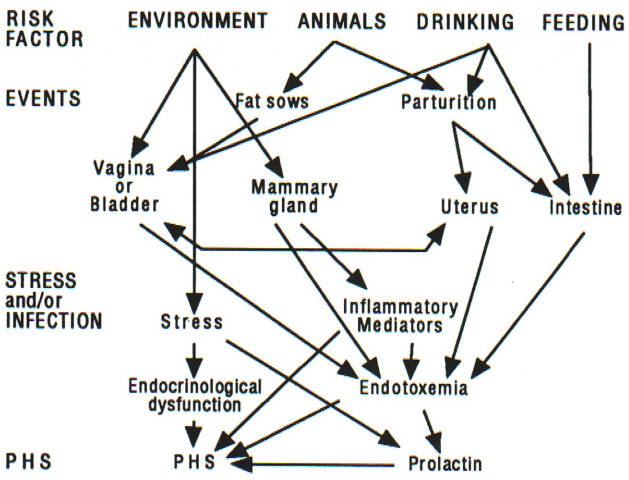

Figure 1. Factors that lead to decreased mammary gland function or PHS.

Diagnosis

Diagnosis of the lactation failure may be relatively easy. Proof of the cause, however, is difficult. Agalactia must be differentiated from other diseases such as PRRS, transmissible gastroenteritis (TGE), or pseudorabies (Aujezky's or PRV) to allow for effective treatment and/or control measures to be instituted. The history, clinical signs, palpation of the mammary glands, postmortem examination of piglets, and the weight of the piglets and the uniformity of the litter usually allow a definitive diagnosis.

Culturing of milk samples and/or uterine discharge should be considered but may not yield diagnostic results even when properly performed. Attention should be given to coliform bacteria. Although treatment should begin immediately, treatment can be changed after culture results are known if the initial treatment is not appropriate. Elevated temperature is a good predictor of affected sows. Although blood count findings may be useful, they are not always economically justified. Determination of urine concentration and composition may be useful in identifying problems of low water intake and urinary tract infection.

The farrowing unit manager and the swine veterinary practitioner should work together to characterize a particular situation and develop a program of corrective action as quickly as possible. An accurate identification of the risk factors leads to effective treatment(s), the initiation of preventive measures, and the ability to make an accurate prognosis.

Common associations between risk factors and PHS seen in practice are as follows:

1. Locomotor disturbances, obesity and changes in housing and floor-type.
2. Urinary tract infection and locomotor disturbances.
3. Changes in housing, flooring and/or ambient temperature.
4. "Fat Sow Syndrome," leg weakness and slow farrowing.
5. Low water intake, both during gestation and a few days before farrowing.

Prognosis

The prognosis for the life of the affected sow or gilt is good; however, the prognosis for the reestablishment of normal lactation is guarded. The affected sow usually recovers in two to five days with or without return of lactation. Culling the sow should be considered if she has several hard, nonfunctional mammary glands after recovery. Unless lactation is rapidly reestablished or supplemental feeding is successful, the chances for baby pig survival are low. Chilling, secondary diarrhea, and other neonatal diseases must be considered in estimating the survival of individual piglets. Cross fostering may be the only practical solution to reduce piglet mortality if recipient sows are available.

At the herd level, prognosis is a function of the identified risk factors (Figure 1). Minimize or eliminate risk factors as soon as they are identified to help correct and prevent lactation failure in the future.

Treatment

Treatment of the affected sow must be directed toward establishing milk flow. No other single treatment is superior to oxytocin. This hormone expresses or releases the milk within the mammary glands which can then be taken by the hungry

pigs. Multiple injections of oxytocin may be necessary. Because the biological half-life of oxytocin is very short, injections can be given every 2 to 4 hours and repeated over a 24 to 36 hour period, without being harmful to the sow. If the oxytocin preparation is 20 units/ml, then 0.25 to 0.5 ml should be injected per treatment. Injecting oxytocin in this manner coincides, in part, with the normal suckling pattern of newborn pigs. Although oxytocin is the single superior treatment choice, there are some agalactic sows and gilts that respond poorly, or not at all. Reasons for the lack of response are related to the cause and effect of the disease within the individual group of sows. Oxytocin only can cause release of milk that has been secreted and is present in the alveoli of the mammary glands. Research results have proven that many mammary glands are not functional and no milk is present to be released. Do not give sows an injection of oxytocin until they have farrowed a pig.

Antibacterial drugs should be used in treating the affected sow since there is no immediate method for determining the presence of bacterial pathogens. Use of antibacterials increases producer costs and forces withholding treated animals from slaughter. The added economic burden is justified because of the high prevalence of bacterial infections, whatever the target organ–mammary gland, uterus, bladder, intestine.

Because bacterial endotoxin can suppress prolactin secretion and lactation, reduction of coliform bacterial populations and correction of prolactin deficiencies are indicated. A major difficulty is that drugs which release prolactin in other species, such as phenothiazine tranquilizers, are not effective in the lactating sow. Thyrotropin-releasing hormone (TRH) does stimulate prolactin release in lactating sows, but it is not very effective when used to counteract the effect of endotoxin on prolactin secretion. Similarly, estrogen injections have not proven effective in increasing prolactin secretion. Tranquilizers may be clinically useful in quieting excitable sows and improving the normal suckling response.

Because endotoxin also causes large increases in prostaglandin secretion, administration of antiprostaglandin drugs, such as flunixine meglumine, may be helpful. Data are not available to confirm that this treatment restores prolactin secretion, but reduction of prostaglandin release should improve the general status of the sow. Corticosteroids are used frequently and may have supportive benefit. It must be remembered that bacterial endotoxin invariably stimulates a very pronounced rise in corticosteroid secretion. In such cases, additional corticosteroids may have limited benefit.

The use of vaginal or uterine infusions, douches, or pessaries to combat suspected uterine infections may be useful as they stimulate a neurohormonal reflex action that could result in the release of oxytocin from the posterior pituitary gland. Be very careful when using this approach because it can lead to genitourinary tract contamination with microoganisms if not done sanitarily.

Treatment programs should include supplementary feed for the pigs, because they have small energy storage capabilities which make their food intake critical for sustained life. Supplemental heat for the baby pigs during the first few days is energy conserving to them and aids in survival as part of routine treatment. Cross fostering to lactating females may be the ultimate solution for pigs on affected sows.

Prevention

Prevention should center around herd health management, water and nutrition. In order to identify probable causes of PHS, the veterinarian should be familiar with the management practices of a particular farm before attempting to offer suggestions for prevention of lactation failure in sows. The veterinarian who is consulted only when a sow is ill is at a disadvantage in offering constructive suggestions.

It is controversial whether vaccines to minimize lactation failure from infectious agents are efficacious.

Efforts to reduce stress throughout gestation, especially near parturition, are important preventive measures. An example would be sow acclimatization to the farrowing facility, especially the water distribution system. Low water intake in gestation is a common risk factor for cystitis (inflammation of the bladder) and, secondarily, for postpartum uterine infection. Low water intake just before farrowing is a common risk factor that leads the sow to conserve water at the expense of the water content of the intestine, which may lead to constipation.

Nutritional disorders are a significant factor in the development of PHS. Feeding levels during gestation have an influence on the incidence of agalactia. The underfed sow apparently cannot maintain blood glucose levels as well as the adequately fed sow; therefore, resistance may be lower. Supplementation of the sow diet with vitamin E and selenium may be helpful in geographic regions deficient in selenium. See PIH-23, Swine Diets and PIH-129, Mycotoxins and Swine Performance. Level and quality of fiber and protein are important factors to take into account. Laxative materials such as wheat bran, beet pulp, potassium chloride or epsom salts may be included in the diet if constipation is a problem. Enemas may be given to constipated sows that are off feed prior to farrowing.

Management attention to environmental factors in the farrowing house is critical. Maintenance of clean, dry floors during gestation and parturition can reduce bacterial populations and udder or uterine infections.

Preventive research efforts using various drugs and hormones (fed and/or injected) prior to farrowing have occasionally produced excellent clinical results. However, critical evaluations and continued study prove that what works in one herd may have no value in another herd. Research efforts are continuing and results will be reported when available.