Milking Problems in Sows and Gilts

For the smaller pig keeper who keeps breeding animals, the ability of each sow to milk and rear a litter is crucial.

Whereas the larger producer can resort to cross fostering, this is often not feasible for the smaller farmer where sows farrow individually. Furthermore, there is no transfer of immunity prior to birth and the newborn piglet is totally dependent upon early intake of colostrum. Failure of colostral supply is, therefore, often fatal to newborn piglets.

In emergency situations, hand rearing with bottled milk can be used but is highly labour intensive and fraught with difficulty. Thus every effort should be made to ensure production and maintenance of colostrum and milk supply from each sow, with early recognition and action essential.

Conditions affecting milk supply

There are many colloquial terms used to apply to milking problems that occur around farrowing such as MMA (Mastitis, Metritis, Agalactia) and farrowing fever and there is often confusion over the causes. The aim of this paper will be to address the principle problems seen which are:

- Primary agalactia due to a range of causes.
- Acute mastitis due to ascending infection in the udder.

Brief mention will also be made of chronic mastitis (Actinobacillus) which tends to only affect individual glands, and whilst compromising milk production from the affected glands, does not usually lead to a complete loss of milk.

Agalactia

Failure of udder tissue to develop

Seen primarily in gilts and recognised in the period immediately leading up to and immediately following farrowing as a failure of the mammary tissue to enlarge so there is no functional milk producing tissue. This can result from:

- Inadequate nutrition - particularly gilts that are maintained on a low protein, low lysine diet at basal levels right up to farrowing. (In group fed sows the same can occur if individuals are bullied out of feeding in late pregnancy.) Use of inappropriate diets can be an issue for the small producer, where small volume usage necessitates limited range of diets in use
- Toxins. Ergot poisoning is a classic cause of inhibition of mammary growth but other mycotoxins can have similar effects. Feeding mould affected food or bedding should be avoided at all times.
- Lack of water supply.
- Individual (?genetic) abnormality including inverted or damaged teats

Failure to produce milk at farrowing

If sufficient udder tissue has developed, colostrum and milk will be produced, unless there is a major limit placed on the sow. Inadequate water is the most likely cause of failure, although extreme heat stress may also be implicated

Weak or inadequate numbers of piglets can provide insufficient stimulus for milk letdown. Furthermore, high levels of stress and excitability (adrenalin release) can impede chemical effect of oxytocin preventing milk let-down. Pain and discomfort at farrowing and teat damage from piglets’ teeth may also play a part.

Initial lactation with later agalactia

The primary stimulus for continued milk production is removal of milk by the suckling piglets, and lactation can persist beyond 8 weeks post farrowing if allowed. Anything that interferes with sucking (weak or premature pigs born, severe piglet disease, litter desertion and mis-mothering) will reduce the stimulus to produce milk and can lead to agalactia.
Similarly, a severe limit on nutrition can simply lead to a failure to produce milk as a result of protein and energy deficiency.

The most commonly seen cause of agalactia in sows arises as a result of excessive colostrum/milk production around farrowing. In this circumstance, even a strong, healthy litter will not remove the milk, leading to pressure build-up in the udder and resulting tissue damage that limits milk production. In these circumstances, typically the litter will appear well but will start to "run off" at 7-10 days as the increased demand for milk is not met. A hardening and tapering of the udder tissue can be detected at the point of connection with the belly at 24 hours post farrowing. Hardening of the udder close to the teats will not occur until later and once detected it is likely that it is too late to 'save' the udder for that lactation. The sow may be reluctant to allow sucking by this stage.

**Fig 2:** Pictorial cross-section of normal and congested mammary glands showing pointing of tissue on the right lateral margin

**Fig 3:** A full udder at farrowing can lead to congestion and tissue damage.

Oedema (fluid build up) will often be evident between the back legs and down into the posterior glands when congestion of this nature occurs.

As excessive milk is produced and not removed, the pressure builds up in the udder with the result that damage is caused to the cells that produce the milk limiting further production. Moreover, as the cellular damage increases endotoxins will be produced in the udder, which can lead to pyrexia (high temperature) loss of appetite and general lethargy. The udder may become hot and painful as a true mastitis ensures, but this is very much an end point of the condition.

Overfeeding prior to farrowing - particularly with high protein lactator ration is the most common cause of this problem and a specific feeding regime must be tailored to each farm, taking into account feed used, genotype, housing provision and various managemental factors. Where this condition is recognised as a problem the immediate response must be to relieve the pressure in the udder. This can be done:

- By manually milking out the sow (if necessary aided by oxytocin injection); any colostrum collected in this way can be stored in a freezer for later use in weakling pigs.
- Use of a human breast pump to achieve the same end.
- If older piglets are available, boxing the sow’s own litter away and temporarily placing strong 10-14 day old pigs onto the sow (having prevented them from suckling their own mother for 2 hours) for 1 hour to take the milk away. Care is needed not to transfer disease such as scour by this technique.

Simply injecting a sow with antibiotics is not appropriate or useful. As a general rule, sow feed levels need to be reduced in the immediate pre-farrowing period. A typical protocol would be:

- At 12-13 weeks gestation increase feed level by 50% (using dry sow diet)
- On entry to farrowing houses (15 weeks +) reduce to previous base level but using lactator ration.
- 48 hours prior to farrowing reduce to 1kg/sow/day (measured), with the lack of dietary bulk made up with soaked bran.
- Day of farrowing give bran only or very limited amounts of feed.
- 24 hours after farrowing commence the steady building of feed on a daily basis with no preset maximum.
If problems still occur, consider delaying the change to lactator ration until after farrowing is complete.

High feed intake pre-farrowing can also lead to bacterial endotoxin production in the gut, which can have an adverse effect on the muscle cells that trigger milk let-down with the same results and make the sow unwell. In this case failure of milk release can occur at farrowing with later udder tissue atrophy (shrinkage).

Many pigs kept on smallholdings will be offered supplementary morsels. These may include grass and vegetables. Such foods help maintain gut movement, reduce toxin absorption and avoid constipation. It is important if such material is available to sows during pregnancy that it is not withdrawn prior to farrowing or during lactation. Equally the old fashioned idea of supplying bran around farrowing time can be a valuable technique to reducing the risk of agalactia but ensure plenty of water is available at all times.

**Acute Mastitis**

**Clinical Signs**

The clinical presentation of a true acute infectious mastitis is nearly always concentrated around or just after farrowing and the sow will be obviously ill - depressed, reluctant to rise or suckle, inappetant and will have a raised temperature. Great care must be taken in interpreting temperatures around farrowing as they are highly variable and can rise to 40°C (104°F) without any obvious abnormality. The truly acutely mastitic sow will have a temperature above this and up to 41.5°C (107°F).

The udder will be hard all over - not just at the edges - hot to touch, usually reddened and very painful - the sow reacting instantly to the touch. She is likely to lie on her belly to protect the teats but will occasionally lie laterally but fail to release any milk.

If it is possible to express milk from the teats, this may show an altered form - watery, clotted, discoloured and, in severe cases, even blood stained. The condition can be life threatening.

Obviously the litter will display signs of starvation as they fail to suck.

**Causes**

The causes of acute mastitis are usually bacterial and gain access to the udder via the teat as an ascending infection. The agents most commonly implicated are E coli, Klebiella and occasionally Pseudomonas, which are contaminants of the udder arising from the faeces or the environment. Thus, the hygiene of the pen and the overall conditions have a part to play. Warm, moist conditions favour penetration of the teats and thus solid floor systems are more likely to be involved than fully slatted systems. Wood based bedding, especially shavings, are particularly implicated - a parallel with peracute mastitis in housed dairy cows. The sharp needle teeth of piglets can damage the teats and udder and allow infection to penetrate.
treatment selected must have good penetration to the udder.

- Anti-inflammatory (NSAID’s) e.g. ketoprofen, meloxicam which will reduce both the pain and inflammation and also the raised temperature. Cortisone is best avoided as it can reduce milk production.

- Oxytocin. If milk flow can be established, the "poisoned" milk can be stripped out by hand.

- The sow must be encouraged to drink by regularly stimulating her to rise.

In some cases, death will occur despite treatment. Often the best that can be achieved is survival but complete drying off with the obvious need to foster the litter elsewhere or hand rear it.

Control

Hygiene is the key. Washing and disinfection of the farrowing area is a standard requirement but the pen must be dry prior to entry and it is best to avoid allowing disinfectant to dry on the floors. In its concentrated form it can damage the teat and make it more prone to ascending infection. This is particularly true of phenolic disinfectants. Always ensure that disinfectants are cleared from water troughs before sows are housed to avoid them drinking what are potentially highly toxic chemicals. Similarly, if lime washing is undertaken, ensure it is fully cured (4 days) before re-stocking.

In many smallholdings dedicated pregnancy and farrowing accommodation is not available with sows spending all their time in a single pen, sty or paddock. Hygiene is still vitally important and regular cleaning of the pen - especially prior to farrowing - is necessary. Do not allow manure to build up in the living area.

If acute mastitis is recognised as a significant problem on the farm, avoid the use of wood based bedding around the farrowing period. Chopped straw or shredded paper are preferable.

Washing sows prior to farrowing can be beneficial but only if they are allowed to dry thoroughly before housing. The veterinary surgeon may advise teeth clipping at birth and in high risk situations, preventative treatments at farrowing may be prescribed.

Chronic Mastitis

Hardened lumps in the udder tissue, especially in the rear half of the udder, are commonly seen in sows and are termed ‘Actinomycosis’ or lumpy udder. Actinomyces suis is the most common cause but other bacteria may be implicated.

Discrete swellings are usually first seen after weaning and in the first month after service and appear to be associated with damage and fight wounds to the udder particularly whilst it is still engorged with milk. The condition appears to be more common in sows housed on straw than on slats and may be a feature of the heavy levels of contamination that occur in the weaned sow pen. It is also seen outdoors where soil becomes contaminated. As such the typical smallholing may be particularly vulnerable.

Alternatively the infectious agent may penetrate the udder during the previous lactation as a result of teeth damage but only becomes apparent once the milk producing tissue atrophies after weaning. The initial swelling is hard and painless and there is no systemic illness.

In some cases the hardened lump remains dormant and is of little consequence, although there is probably a reduction in milk production in the affected glands. As the sow approaches her next farrowing the lesions can become active, increasing in size and ulcerating such that purulent material exudes from the ulcer. The udder can become so enlarged that it drags on ground. When weaned, it will be prone to further damage and contamination.

Treatments

The key to controlling chronic mastitis is dealing with the initial lesions. Once the lesion becomes enlarged, cure is impossible and the best that can
be hoped for is healing of the ulcer, shrinkage of the swelling and premature culling.

Efforts are need during suckling and at weaning to reduce udder injury and contamination. Weaning into smaller groups with additional floor space (4m²/sow) will help, as will regular cleaning and disinfection of the weaning pen. In persistent problem herds, medication of sows around weaning, using long acting injectable antibiotics may be indicated to prevent seeding of infection.

Routine teeth clipping of pigs in the first 24 hours after birth may be appropriate.

Once lesions have developed they will persist. Sows with swellings that are larger than a small melon, and any ulcerated lesions, are not appropriate for routine culling - transportation and slaughter - and often require on farm euthanasia. On no account should such animals be presented at market. Whilst a short course of antibiotics may be prescribed for an early stage lesion they are likely to be ineffective once substantial tissue enlargement and suppuration have become established.

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