Fertility in Dairy Herds - Advanced

Part 5 : The impact of mastitis and lameness on fertility

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The impact of mastitis and lameness on fertility

Mastitis is one of the commonest diseases of dairy cattle; in the UK recent research showed that the average rate of clinical mastitis was between 50 and 70 cases per 100 cows per year (cow-years), with many farms having significantly more.

In addition, the average herd bulk milk cell count of 200,000 means that ~20% of the national herd has subclinical mastitis at any one time.

Mastitis is also one of the most costly diseases, costing the UK dairy industry ~£200 million per year. One of the ways in which mastitis costs money is in its impact on fertility of affected cows. Multiple studies have shown that cows with mastitis have significantly poorer fertility. Clinical mastitis has been shown to:

1) Increase the time between calving and when the cow is first inseminated
2) Increase the time between calving and when it gets pregnant
3) Increase the number of services per pregnancy
4) Increase the proportion of cows which are culled because they are not pregnant
5) Increase the abortion rate in early pregnancy

Subclinical mastitis can also affect fertility having been shown to:

1) Reduce the conception rate (the probability an inseminated cow will become pregnant)
2) Alter the timing of ovulation relative to the start of oestrus
3) Result in irregular interoestrus intervals.

These effects occur in cows with mild and moderate mastitis as well as those with severe mastitis; in cows with Gram negative pathogens (e.g. E. coli) and Gram positive ones (e.g. Staph. aureus); in cows which get mastitis before breeding and in cows which get mastitis afterwards. In fact for the last comparison, some studies have shown that cows which get mastitis after they have been bred for the first time have poorer fertility than cows which get mastitis which occurs (and is cured) before they are inseminated.

There are many ways in which mastitis may be producing its impact on fertility. Probably the most important is through inflammation; inflammation produces prostaglandins, one of which, PGF\(_{2α}\), is important in the regulation of the cow’s oestrus cycle. So reducing the inflammation due to the cow’s oestrus cycle.

Fig 1: Clinical mastitis, particularly around the time of mating can reduce fertility in affected animals, but at a herd level the proportion of cases of clinical mastitis is not associated with reduced reproductive performance.
After mastitis, lameness is the most economically important disease of dairy cattle (in the UK it affects over 45% of cows every year and costs more than £200 per case). One of the key economic impacts of lameness is its impact on fertility. One major route by which lameness influences fertility is its impact on body condition; lame cows lose considerable amounts of weight and condition (principally because lame cows eat less); this starts even before the cow is observed as being lame. This means that lame cows have longer and deeper periods of negative energy balance, which significantly reduces fertility, particularly by increasing the interval between calving and first service and calving to conception interval.

Lameness may also directly affect fertility by altering behaviour - lame cows are less likely to be seen in oestrus and if they do show oestrus they will show it less intensely and for a shorter period than non-lame cows. These effects may also be mediated by the effect of lameness on feed intake. Lameness, like mastitis, is highly stressful so also has significant effects on ovarian function. It is also an inflammatory process, so just like mastitis, is associated with reduced risk of conception and increased number of services per pregnancy. However, in contrast to mastitis, where tackling the inflammation associated with mild and moderate cases has been shown to be effective at reducing the impact of mastitis on fertility, no similar data have been produced for lameness. This is because even simple cases of white line disease or sole ulcer involve significant changes in the hoof which will not respond to treatment anywhere as quickly as an udder with mild to moderate mastitis.

So both mastitis and lameness can have significant effects on fertility, with lame or mastitic cows getting pregnant more slowly, requiring more inseminations and being more likely to be culled for being not pregnant. Control and prevention of mastitis and lameness will thus have significant benefits on fertility at the individual cow level.

However, just because lameness and mastitis affect fertility it doesn't mean that reducing them will have a significant effect on fertility at the herd level. This is important as when developing a fertility programme for a farm, it is essential to be able to identify how much impact a decision (such as reducing lameness) will have on a herd's reproductive performance and thus identify the potential benefit of such a programme (in terms of reproduction) as well as its cost.

Recent published research has looked at the effect of lameness and mastitis on fertility at the herd level, using a computer-based simulation. The lameness simulation used data from 39 herds across the UK and included the records of 12,515 dairy cows which had a total of 22,319 lactations, while the mastitis one was based on a model built from data from 80 herds which included 39,590 lactations in 21,068 cows.
For lameness the research was based on records from 4,360 lactations which had at least one lactation when a cow was treated for lameness. Treatment for lameness within 14 days of breeding reduced the odds of pregnancy by almost 25%; lameness further away from breeding still had a significant negative impact on the odds of getting pregnant of around 15%. Their analysis, therefore, clearly showed the negative impact of lameness on fertility.

However, the authors then looked at the effect of lameness on a herd fertility score which combined the average interval from calving to pregnancy and the proportion of cows which got to 300 days in milk without getting pregnant. No association was found between the proportion of lame cows and fertility score, whereas the herds ‘background’ submission rate and conception rate were significantly correlated, and in fact accounted for 75% of the differences between herds in fertility score.

The analysis showed that increasing submission rate from the median (i.e. the submission rate exceeded by 50% of the farms) to the value at the bottom of the upper quartile (i.e. that exceeded by 25% of farms) resulted in increased income of around £100/cow/year due to improved reproductive performance. In contrast, reducing lameness from the bottom of the top quartile to the median (decreasing the number of treatments per 100 cow years from 115 to 80) only resulted in gains of £5/cow/year due to improved reproductive performance. So, even though in this dataset lameness was common (50% of farms had more than 80/100 cows treated for lameness per year), and lameness reduced fertility (reducing the odds of getting pregnant by up to 25%), the amount of improvement in reproductive performance from reducing lameness! treatments by 35 cases per 100 cow years was the same as that produced by an increase in submission rate of <1%, even though reducing lameness treatment by 35 cases per 100 cow years would probably require significant investment of time and resources, whereas improving the submission rate by 1% could be achieved with only small changes.

This simulation was based on data from non-seasonal herds, so as the authors pointed out, the impact could be different in herds with block calving as the risk of a lame cow being empty at the end of a short breeding season may be significantly greater than the risk of a lame cow being empty 300 days after calving in a non-seasonal system. However, the differences in the figures suggest that even on seasonal farms improving lameness is likely to have much less impact on herd fertility than improving submission rate.

For clinical mastitis (i.e. cows showing signs of mastitis such as change in milk or udder), in the dataset used for the simulation, the average incidence rate was 55 cases per 100 cow years, with the average somatic cell count being 205 000/mL. Analysis of the association between mastitis and fertility confirmed the findings of the earlier studies that mastitis had a negative effect on fertility. Clinical mastitis significantly reduced the odds of a cow becoming pregnant; if a cow developed clinical mastitis within one week of insemination, the odds of pregnancy were around 75% of that of a cow which did not get clinical mastitis. Mastitis after insemination affected the risk of pregnancy for a longer period than mastitis before insemination: clinical mastitis up to 70 days after insemination decreased conception rate, but no such association was found if clinical mastitis occurred more than 28 days before insemination. Subclinical mastitis also affected fertility, though the impact was less than that of cows with clinical mastitis. There was an association between cell count and fertility, with higher cell counts tending to be associated with poorer fertility; for example, the risk of pregnancy in cows with a cell count of ≥400 000 mL in the month after insemination was 80% of that of a cow with a cell count of ≤20 000 /mL, while the equivalent figure for cows with a cell count between 200 000 and 400 000/mL was 85%.

So the dataset used for the simulation clearly showed that mastitis, at the individual level, significantly reduced fertility. However the analysis at the herd level found no association between mastitis and herd level reproductive performance (as measured using fertility score); herds at the extreme ends of the udder health spectrum (i.e. very good vs very poor) had very similar ranges of reproductive performance. Therefore the model showed that, at the herd level, reducing mastitis would have no or very limited impact on reproductive performance.

These studies both show that focussing on the impact of disease on fertility may not pay dividends in terms of improving herd reproductive
performance. At the herd level, the most important factors are the underlying submission rate and conception rate. Focusing on improving these will have the greatest impact on the reproductive performance of the herd.

This does not mean that reducing lameness and mastitis are not of value; both of these diseases have significant welfare and economic consequences and their reduction can have significant benefits for a dairy herd. It also doesn't mean that focusing on improving the fertility of cows with mastitis or lameness is not of economic value - giving a cow with moderate mastitis meloxicam is economically beneficial because it results in improved fertility. The findings of the two simulation studies, simply mean that if your focus is on improving fertility, then you need to focus on underlying reproductive performance (i.e. how quickly your cows get pregnant and how many cows you cull because they are not pregnant) rather than on reducing the number of cases of lameness or mastitis.

Summary

Both mastitis and lameness can have a significant negative impact on the fertility of affected cows. Compared to normal cows, affected cows are likely to be inseminated later, have a lower chance of getting pregnant per insemination, and are more likely to be culled because they fail to get pregnant.

However, although this impact on fertility adds significantly to the cost of an individual case of lameness and mastitis, at the herd level the influence of lameness and mastitis on herd reproductive performance is small. Improving herd reproductive performance should be based on improving submission rate (the proportion of eligible cows which are mated) and conception rate (the proportion of inseminated cows which become pregnant).

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