Balancing High Production and Health of Cows

Roger Blowey

Wood Veterinary Group, St Oswalds Road, Gloucester GL1 2SJ, UK
Email: rblowey@gloucs-vet.demon.co.uk

Take Home Message

- The majority of common health disorders in dairy cows are associated with calving. Milk fever, retained placenta and retained placenta are obvious examples. Sole ulcers, mastitis and displaced abomasum less so.
- It is therefore how we feed and manage cows over the period from 3 weeks before calving to 3 weeks after calving that has such a major influence on subsequent losses from disease
- Post–partum integration will be discussed in relation to housing, environmental hygiene, socialization and stocking density.

Maintaining adequate herd health remains a major issue for the majority of dairy herds, and is an area where constant attention to detail is required. Over the past 25 years we have seen a dramatic reduction in the number of dairy herds, an increase in yield per cow, and an increase in the number of cows in each herd, often all cared for by one stockman. This has clearly put significant strains onto the maintenance of herd health, and is one possible reason for the dramatic reduction in herd fertility. One study (Royal, 1999) has suggested that first service calving rates have fallen from 56% in the late 1970s to early 1980s, to an average of less than 40% today. In fact pregnancy rates are said to be falling by 1% per year, and if the trend continues, conception rates to first service will be as low as 28% by 2010! This paper examines some of the current health issues that are important to the high yielding dairy cow, and suggests ways of maintaining high health in the face of continuing economic pressure. Fertility remains a major problem and will be discussed as the basis for general improvements. Mastitis remains an issue for many herds. Lameness has been discussed in a separate presentation.

Level of Yield, Nutrition and Fertility

Several studies have shown an association between increasing yield and reduced fertility (O'Farrell, 1998), and the recent increase in the use of high genetic merit bulls may therefore be a contributory factor. Coffey and Price (2000) compared the fertility of 'selected' cows where intense sire selection had been used with the fertility of control cows (Table 1).

Table 1. Comparison of the fertility of 'selected' cows where intense sire selection had been used with the fertility of control cows. (Coffey and Price 2000)

	Selected	Controls
Yield (kg)	10,355	7,300
1st service conception rate	39%	45%
Days to first heat	53	42

The higher yielding cows had poorer fertility despite the fact that both were kept in the same environment and management. Similar results were obtained comparing selected and non-selected cows on high forage rations.

The standard 'early lactation energy gap' cannot be the whole cause however, because similar results were obtained comparing selected and non-selected cows on high forage rations, and studies on extended lactations have shown that fertility does not dramatically improve even when service is delayed.

Another potential cause is the feeding of high protein/high nitrogen diets (Laven and Drew, 1999). High protein diets will undoubtedly boost milk production, but fertility may be adversely affected. The cause is unknown, but possible reasons include:

- the increased energy requirement needed to eliminate excess protein as urea
- when protein is fed to excess, part of the protein will be used as an energy source, with the release of yet more nitrogen which has to be eliminated.
- increased blood urea levels can affect all stages of the cycle, from the suppression of normal ovarian cycles to a cause of embryo death.

The problem may get worse as we move toward a higher proportion of forage in the ration. With fresh forages up to 90% of the dietary nitrogen intake may have to be excreted as urea. This is because the nitrogen in plants, and especially in

rapidly growing plants, is very soluble and rapidly released into the rumen. If sugar levels are low, then the problem is further compounded because nitrogen uptake by the rumen micro-organisms is even slower. A careful balance between the energy and protein fractions of the diet is therefore necessary to optimise both food utilisation and fertility. Perhaps the answer is to bring back meat and bone meal!

Maize silage is notoriously low in vitamin E, and if high levels are being fed, additional supplementation may be required. This is especially the case with high fat diets, where the vitamin E requirement is increased even further. For optimal performance and fertility, cows need 600 to 1000 mgs of vitamin E daily.

■ Irregular Oestrous Cycles

Another interesting feature of the change in fertility over the past 25 years is the marked increase in abnormal cycles (Royal, 1999) (Table 2). One data set showed that the incidence of delayed luteolysis, (also known as 'persistent corpus luteum'), increased from 6% to 17% of ovarian cycles, the incidence of prolonged anovulatory phases from 11% to 13%, and the overall incidence of atypical cycles rose from 32% to 44%!

Table 2. Increase in abnormal cycles over the past 25 years (Royal 1999)

	1975-1982	1995-1998
Oestrous cycle length	20.2	22.3
Luteal phase (days)	12.9	14.8
% Cows with prolonged anovulatory phase	10.9	12.9
% Persistent corpus luteum	6.4	16.8
One or more atypical pattern	31.7%	43.7%
Calving rate to first service	55.6%	39.7%

With approaching half of the cycles irregular, it is perhaps surprising that we get as many as 40% of the cows pregnant! My own anecdotal experiences and involvement in herd fertility programmes demonstrate the increasing number of cows which 'hover', close to oestrous but do not quite make it to oestrus and ovulation. These are the cows with prolonged anovulatory phases. Treatment is needed when the cow is re-examined two weeks later and found to be at the same stage of the cycle, viz. close to oestrus. This is perhaps one of the

reasons why cycle lengths are getting longer (current mean 22.3 days versus 20.2 days 25 years ago).

One of the major benefits from the administration of prostaglandin to groups of animals is that they all come on heat at roughly the same time and as a consequence the manifestation and detection of oestrus becomes much easier. Put more simply, cows that come bulling in a group are likely to have 'stronger heats' than those animals which come into oestrus singly (Britt 1987). Using DAISY data from recorded herds within our own practice Chris Watson (personal communication) has shown that over the 15 years from 1983 to 1998 heat detection rates (based on the interval between services) have fallen steadily from just over 60% in 1983 to only 40% at present. The rate of heat detection is, however, assessed by the inter-service interval, and hence as the proportion of irregular oestrous cycles increases, the heat detection rate will fall. Is the declining rate of heat detection simply a reflection of the number of irregular cycles therefore? This seems unlikely. Increasing cow numbers, increased numbers per group and a decrease in available labour will also play a part.

■ Parturition as a Major Cause of Health Disorders

Parturition must be the single most hazardous incident in the productive life of the modern dairy cow. Not only does the process itself have inherent dangers, but the immune suppression associated with parturition has a pronounced influence on the incidence of disease for a major part of the lactation.

It has long been recognised that many new intramammary infections are contracted during the dry period. The most important of these are Streptococcus uberis and E. coli, with the first and the last two weeks of the dry period being the major time of increased susceptibility (Williamson et al 1995; Bradley and Green 1998). The infections remain dormant in the non-lactating udder to cause clinical disease in early lactation. In fact almost 60% of all cases of clinical coliform mastitis that occur in the first 100 days after calving originate from dry period infections, and the most severe of these are often at the time of parturition. Perhaps even more surprising is the fact that only 8% of new coliform infections contracted during the dry period are ever translated into clinical mastitis. Put another way *self-cure occurs in 92% of cases*, and this presumably occurs in those cows least affected by the immune suppression associated with calving

Cows carrying latent subclinical infections such as IBR or Salmonella typhimurium are also most likely to break down with clinical disease during the periparturient period. The problem seems to be one of reduced immune competence in the periparturient cow, rather than one of increased susceptibility to new infections at this critical period. Selectin molecules on the

capillary wall are responsible for the attachment, activation and subsequent diapedesis (= migration) of neutrophils to combat invading pathogens. During the periparturient period selectins become less active, and this is combined with a reduced phagocytic activity by the neutrophils, viz they have a reduced ability to ingest and destroy invading pathogens. In addition there is a reduced antibody output by the lymphocytes, and this includes both IgM and gamma interferon production. Although the consequences of the immune suppression are well recognised, it is interesting to speculate what evolutionary significance they might have. One theory is that the foetus is 'foreign' to the dam, and hence in late pregnancy the dam must suppress her immune system to allow the foetus to develop. In addition, at the birth process there will be extensive uterine damage leading to the release of toxins and potentially paternal antigens, all of which could enter the maternal circulation to produce hypersensitivity in the dam.

■ Physiological Changes in the Transition Cow

There are additional physiological reasons for an increase in disease incidence in the post parturient period. The rate of rumination decreases in the periparturient cow, and this leads to a depression of food intake of around 30%, especially of forage, and hence to an increased risk of rumen acidosis. Food intake falls at the very time when the nutrient requirements of the cow (for foetus and milk production) are increasing, and this leads to excess fat mobilisation and development of 'fatty liver', both of which further suppress immune function. Finally management often then imposes radical changes in feeding, housing, husbandry and social grouping, all of which subjects the periparturient animal to further stresses

There is of course, a range of diseases directly associated with parturition. These include nerve damage associated with dystocia, trauma to the birth canal and subsequent infections, severe blood loss, retention of the placenta, and specific periparturient disorders such as hypocalcaemia and endometritis. Probably equally as important however are the many disorders whose incidence increases as a consequence of parturition, especially when changes in housing management and nutrition are less than optimal. Examples include mastitis, ketosis, and lameness. The rings on a cow's horns, one for each calving, clearly demonstrate that there is a disruption in horn and hoof formation at the time of calving (Blowey 1998), and this occurs when there are no other changes in housing and feeding. Many authors have shown that there is then a peak incidence of hoof lameness (sole ulcers and white line disease) some 10-14 weeks after calving, when the damaged horn has grown to the surface of the sole.

The Influence of Nutrition

The final piece in the jigsaw is the effects of nutrition. The reduced rumination in periparturient cows is one of the reasons why dry matter intake can fall by some 30%. Because the rate of rumination decreases, there is a decrease in saliva production. Saliva contains sodium bicarbonate and so there is then an increased risk of rumen acidosis. An acid rumen is known to increase the fragility of the corium, predisposing the cow to white line and sole haemorrhage at the critical time around calving. Some recent data (Da Costa Gomas et al 1998) has shown that acid rumen conditions suppress the synthesis of biotin, and cows with acidosis may be producing only half the level of biotin compared to a cow with normal rumen function. In addition, in cows receiving high starch diets, the requirements of biotin for gluconeogenesis increase. There is therefore the potential for even ruminants to become B vitamin deficient during the critical periparturient period. This can lead to poor quality horn formation and consequently increased lameness later in lactation.

The Importance of Stress

Stress is an inability of the animal to cope easily with its environment, and once again this is most commonly seen when heifers are introduced into the dairy herd, especially if they have received no prior acclimatisation. It is also the time when they are least able to cope with new situations. Management at the time of calving affects conception rates in the first lactation. The best system is to keep heifers in a separate group for the whole of their first lactation. If this is not possible, then second best is to keep them housed in a separate group or at low stocking density until they are pregnant, or at least for the first few weeks of lactation. Just a few days in a maternity yard seemed to help, with the worst effects on conception rates being the heifers which are introduced into the main herd immediately after calving, especially if they have had had no previous cubicle training. It is, unfortunately, not uncommon to see freshly heifers four to six weeks after calving really struggling with the system. Not only have they just come through parturition, which is one of the most traumatic periods of their life so far, but their immune system becomes less competent, and their food intake drops by some 30%, further increasing their susceptibility to disease and their inability to cope with a stressful situation. At the same time they are mixed with a large number of animals (cows) with which they have not previously interacted, and fighting or bulling takes place. They are often the last to be milked and the last to feed, and as a consequence they are on their feet for much longer – and all this at a time when the corium is at its most fragile. Their feet are painful, and hoof horn production is reduced.

Diseases are more common around the time of calving, and this further reduces subsequent conception rates (Borsberry and Dobson 1989). Dobson (1998) assessed the social status of individual cows within a herd by monitoring

their ranking as they approached a feed area. The observations were repeated four weeks later, and the subsequent fertility of those cows which moved up the social scale by five places was compared with the fertility of the cows which moved down two places. Cows moving down the scale not only had poorer fertility, but they also had more lameness and higher cell counts.

The Impact of Mastitis

Although mastitis is a major problem for dairy farmers world-wide, we should not forget that the industry has made enormous strides towards controlling the disease. The thirty years since the 1960s has seen a dramatic decrease in the incidence of clinical mastitis, from about 120 cases per 100 cows per year in 1968 to a figure closer to 40 cases per 100 cows p.a. at present (Hill, 1990). The most striking change has been the decrease in the incidence of Staphylococcus aureus infections, which has fallen from 55% of all clinical cases in 1968 to around 5% at present. This followed, the introduction of improved milking hygiene routines, post-milking teat disinfection and dry cow therapy. The reduction was largely associated with a reduction in 'contagious' mastitis pathogens however, and as a consequence the percentage of 'environmentals' has increased considerably, with Streptococcus uberis and E. coli being currently the two most important pathogens.

Teat End Defenses

The teat is a complex structure, as it must allow milk to flow at the same time as preventing the entry of bacteria. At the base of the teat, adjacent to the udder, there is a ring of erectile tissue. When this tissue becomes engorged with blood it holds the teat base open so that milk can flow from the udder into the teat. Failure of this process would immediately stop milk flow as the teat would be sucked dry and fail to refill. The process of milk let-down therefore consists of three parts:

- contraction of the myoepithelial cells surrounding the alveoli forces milk from the mammary tissue and into the udder cistern.
- engorgement of the errectile tissue at the base of the teat allows milk to flow from the udder cistern and into the teat,
- relaxation of the sphincter muscle at the teat end allows milk to flow from the teat end into the milking machine liner,

Disruption of any of these processes can lead to poor milk flow, teat end damage and potentially a new mastitis infection.

The outside of the teat and the inside of the teat canal is lined by keratinised squamous epithelium that is a normal skin surface. When closed, the inner lining of the teat canal is folded, and the folds interlock with each other to improve the effectiveness of the seal. In addition, a 180nm layer of *surface lipid* covers the keratinocytes, holding them in place and further improving the teat seal. When milking starts and 'let-down' occurs:

- the teat end unfolds and the canal opens
- milk flows over the lipid, removing milk residue from the previous milking, thereby cleaning the surface of the teat canal
- the surface layer of keratinocytes slough away, carrying any adherent bacteria with them. This 'keratin slough' is extremely important in the prevention of new infections.

At the end of milking the teat must reseal. A peristaltic wave of muscle contraction passes from the teat base to the apex. The teat and the teat canal shorten, the contracting sphincter refolds, and most of the residual milk is squeezed out. Because of the hydrophobic nature of the lipid surface of the teat canal, any milk remaining in the canal exists in small 'lakes' of approximately 3ul. There is therefore no solid column of milk, and hence the risk of infection tracking up through in a 'wick' affect is considerably minimised. However if the teat closure takes place in a highly damp or humid atmosphere, the static column of milk persists for longer and hence the risk of mastitis is greater.

If the teat end becomes damaged in any way, then these delicate closure mechanisms become compromised, and there is a risk that mastitis will result. A cracking of the inner lining of the teat canal could lead to:

- an incomplete lipid seal. Any residual milk could then exist as a solid column and not as small 'lakes',
- serum oozing from the cracked canal surface would act as nutrients for bacteria,
- milk flow at the next milking would not result in complete flushing of residual bacteria.

It is therefore vital that cows are housed, managed and fed to optimise these teat end defences. Poor housing will increase the challenge of environmental infection at the teat end, and may contribute to teat end damage through trauma. Poor management, and especially poor milking machine management, will lead to teat end damage, thereby predisposing to mastitis. Poor feeding could lead to loose faeces and excess environmental challenge; and a deficiency of dietary sulphur or zinc could potentially lead to poor keratin production and hence a failure of the teat end defence mechanisms.

Causes of Teat End Damage

There are numerous causes of damage to the teat end, all of which increase the mastitis risk, although by far the most common is the milking machine. There is a tendency to consider that a 'good' milking machine is one which extracts milk rapidly from the teat end, thereby minimizing time spent in the milking parlour. This has been achieved by changes such as larger volume claw pieces and bigger bore pipelines. Moving milk away from the teat end more quickly does have some advantages in reducing contamination rates and reducing the risk of a reflux of milk back into the open teat canal (ie., minimizing teat end impacts), but it also means that teat end vacuum during milking is higher because it is closer to plant vacuum, and there is therefore an increased risk of teat end damage. The causes of teat end damage are numerous. They all must lead to an increased risk of mastitis and include:

- excessively high teat end vacuum,
- inadequate pulsation, especially an inadequate 'massage' phase in the pulsation cycle,
- worn liners,
- overmilking, especially when milk flow has ceased,
- poor ACR function, for example an inadequate delay between vacuun shut-off in the claw and ACR activation. Pulling the cluster off creates a vacuum at the teat end which can produce damage.

Hyperkeratosis, sometimes referred to as eversion of the teat sphincter, is a particularly common form of teat end damage. It is related to suboptimal plant function, and is most commonly seen in higher yielding cows, where the milking unit is on for longer; in older cows where the elasticity of the teat tissue is waning; at or after peak lactation, when the stresses of the machine have been at their greatest and in pointed rather than flat ended teats. The problem is becoming so important that many herds are now having their teat ends 'scored' on a routine basis to monitor the incidence of damage.

Host Response

Even when infection has penetrated the teat canal and has entered the teat cistern, mastitis is by no means inevitable. The cow has an excellent cellular response mechanism which is able to repel the majority of infections. The approximate sequence of events is as follows

- macrophages in milk identify, engulf and destroy the invading pathogen by a process of phagocytosis

- by-products produced as a consequence of phagocytosis stimulate an 'alarm' reaction in the host.
- the response to the alarm is seen as a dilation of blood vessels in the affected quarter, producing increased blood flow (and hence a hot swollen and painful quarter), followed by a massive invasion of neutrophils (white blood cells) from the adjacent blood vessels into the teat.

This response can be so effective that the white cell count in the blood may fall to almost zero within a few hours of the entering the teat! Unfortunately not all cows are able to mount such an effective response. Some early lactation animals appear to be less able to rapidly mobilise neutrophils, and as a consequence they may become very sick following an E. coli infection. The reason for their poor response is not fully understood, and is an area that needs investigating in the future.

Milk Flow Rates and Susceptibility to Mastitis

Work by Grindal and Hillerton (1991) has demonstrated an interesting relationship between teat end milk flow rates and susceptibility to mastitis. Over the past 30 years milk flow rates have doubled from approximately 0.8 litres per quarter per minute to the current level of 1.6 litres per quarter per minute, and this has lead to a *twelve fold* increase in susceptibility to mastitis. Slow milking cows do not fit into the milking routine of a herring bone parlour and are culled. In addition there is a positive correlation between level of yield and milk flow rate. As milk yields will continue to rise in the future, then mastitis susceptibility will also rise. Future cases of mastitis are therefore inevitable. However if we can learn to house, manage and feed our cows such that the mastitis risk is reduced and their immune response is not compromised, then the problem could be minimized.

Dry Period Infections

Infections contracted during the dry period may remain dormant in the udder and cause mastitis any time up to three months after calving. It has been estimated that at least 50% of all clinical mastitis that occurs in the first 100 days after calving is associated with dry period infections. The cow is at her most susceptible to new infections during the first and last two weeks of the dry period, when the natural keratin plug is forming and then opening again, respectively. In addition, it has been shown that many cows fail to form an adequate keratin seal. For example New Zealand workers found that 20% of cows had failed to form an adequate seal by 40 days after calving, whereas in the higher yielding cows in the US some 50% of cows had failed to form a proper seal by 40 days. Management of the dry cow is therefore critical in the

prevention of clinical mastitis during lactation. Factors leading to a poor teat seal include

- high yields at drying off
- fast milking cows
- teat end damage, e.g. from machine milking

An estimate of the prevalence of dry period infections can be made from an analysis of cell counts. If more than 5% of cows move from below 200,000 cells per ml at drying off to above 200,000 at calving, or if more than one in twelve cows develop mastitis in the first 30 days after calving, then dry period infections should be suspected. Environmental infections with coliforms and Streptococcus uberis are the most common organisms involved. Control measures include dry cow therapy, internal teat seal products, paddock rotation every 4 weeks if dry cows are outside, and low stocking densities, eg 10 sq metres per cow, if housed in straw yards. If teat seals have not been used, then walking transition cows through the parlour twice a week, applying a thick barrier dip, and then through the foot bath reduces both mastitis and digital dermatitis.

Conclusions

Parturition remains one of the most stressful periods of an animal's life, and as a consequence there is a marked increase in the incidence of disease. The majority of important health disorders in dairy cows in fact arise from the consequences of parturition, and yet many people continue to inadequately integrate their heifers into the main herd. Inherent changes in rumen function could lead to B vitamin imbalances, and this in turn will increase the incidence of disorders such as white line disease, ketosis and fatty liver. As yields increase management will have to improve if we are to keep production disorders under control.

Acknowledgements.

Much of this information has been previously published in A Veterinary Book for Dairy Farmers**,** published by Old Pond Books*.* Copies are available from the author at the address given above, and from Diamond Farm Books, Burlington, Vermont.

References

Blowey (1998), in Cattle Lameness and Hoofcare, published by The Farming Press, Ipswich, pages 75-76

Borsbery S and Dobson H (1989) Periparturient diseases and their effects on reproductive performance in five dairy herds Vet Rec 124 217-219

Bradley AJ and Green MJ (1998), A prospective investigation of intramammary infections due to *enterobacteriacae* during the dry period: a presentation of preliminary findings. Cattle Practice 6, 91-94

Britt JH, (1987) Detection of oestrous in cattle Vet Ann. 27, 74-80

Coffey M and Price J (2000), The effect of breeding on fertility in the dairy cow, in 'Profit from improved fertility', Kingshay Dairy Conference, Stafford, UK, February 2000.

Dacosta Gomez C, al Masri M, Steinberg W & Abel HJ (1998).Effect of varying hay/barley proportions on microbial biotin metabolism in the rumen stimulating fermentor RUSITEC. Proc. Soc. Nutr. Physiol., no.7, pp 14-28

Dobson H (1998) Stress and subfertility reprod. Dom. Animals 33 107-111

Green LE, Hedges VJ, O'Callaghan C, Blowey RW, and Packington AJ (2000), Biotin supplementation to dairy cows – multivariate analysis of the prospective longitudinal study, in Proc 11th Symposium on Disorders of the Ruminant Digit, Parma Italy, September 2000, pp 305-307

Grindal RJ, and Hillerton JE (1991) Influence of milk flow rate on new intramammary infection in dairy cows J Dairy Res. 58 263

Hill AW (1990) Proc. British Mastitis Conference (1990) page 49

Laven RA and Drew SB, (1999) Dietary protein and reproductive performance of cows Vet Rec 145 687-695

O'Farrell KJ, (1998) Changes in dairy cow fertility, Cattle Practice 6 387-392

Royal MD (1999) To what extent has dairy cattle fertility declined in the UK? Cattle Practice 7 395-396

Williamson JH, Woodford MW, and Day AM, (1995) The prophylactic effect of a dry cow antibiotic against *streptococcus uberis*. New Zealand Vet J. 43 228-234