# Technical Series

### Issue 10

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# The best preparation you can do



Eric Hillerton, DairyNZ Chief Scientist

## Key actions to make your dry cows fit for purpose:

- Make dry period 60 days or a little more
- Chose best dry cow option
- Get foot health right
- Service milking machine properly
- Train heifers in yards and dairy
- Fully milk out newly calved cows within 12 hours
- Think hygiene.

We are all aware of opportunities to 'prep' our cows for milking, whether it is to clean teats or check for mastitis or get good milk let down. The dry period is an opportunity to 'prep' the cow for the coming lactation so that she is optimally fit and healthy, and so minimise many of the problems associated with calving and the following critical period. General guidance is given here on what should be done, why and when.

#### Drying off

When to dry off is usually a decision based on estimated calving date, cow's body condition and availability of feed. Dry off is the best opportunity to clear up mastitis problems hanging over from the whole lactation and reduce risk for the coming lactation. Options and how to choose what is most appropriate are described in *DairyNZ Technical Series Issue 9, Dry cow treatment – how can it be used*<sup>1</sup>. Be aware of the length of the dry period.

#### Dry period – part 1

In recent years much has been written about short versus long dry periods. Overall, the evidence suggests that short and extralong dry periods affect milk yield and udder health adversely<sup>2</sup>. So aim for a dry period of about 60 days. This gives the cow a rest, allows optimum regeneration of mammary tissue and still leaves the 21 days necessary for the gland to initiate the full secretion processes.

The first four weeks of the dry period are when body condition can be added. Cows will respond to good quality feed and supplements to reach BCS 5.0 (5.5 for heifers)<sup>3</sup>. This is also the ideal time to treat and prepare feet. Resting cows, with minimal walking to do, are good subjects for treating foot problems especially of the sole and getting the hooves in 'shape' for trekking on races and concrete.

Shape is important, this includes length of the toe, the angle of the hoof wall, the thickness of the sole and the height of the heel. Proper trimming is a skill so a professional trimmer or undergoing good training yourself are the better options. When getting your boots for the coming season get your cows properly shod too. The *DairyNZ Healthy Hoof Programme* is your starting point<sup>4</sup>.

At this time, cows are often out of sight, don't let them be out of mind. Daily or at least five inspections a week, are necessary to spot the early calvers, a predicted calving date is only that – a prediction.

Early dry period is when your vet still has time to think, plan and prepare. Revise your herd health plan – get vaccinations done, sort out treatment protocols for the coming lactation and train appropriately, existing as well as new staff. Do not forget the milking machine. It needs to be serviced by replacing consumable parts, especially oils and rubbers, and it needs to be tested to demonstrate that all parts function properly. Any deficiencies need to be remedied now! Replace all liners. It is nonsense to milk for the first few weeks with old liners as they are "softer". They may be softer because they are old (maximum 2500 milkings) but they do not operate properly<sup>5</sup>, especially when unused for several weeks over the dry period. 'Breaking in' liners occurs, if such a thing even exists, with one hot plant wash and that is an essential anyway before the first milking of the season.

#### Dry period – part 2

This time is four weeks to calving and with the heifers home stocking rate is at its highest. The weather is at its wettest so stock are standing off as much as possible; good job the feet are at their best standing in wet mud, on a sodden feed pad or on a hard surface for a long period. Feet kept wet for a long period have softer hooves so care is needed. If they also stand in faeces contaminated materials then hoof deterioration may become an issue. The highest prevalence of digital and interdigital infections occur in the northern hemisphere when cows stand in wet and faecal material for long periods<sup>6</sup>. Stand off and feed pads may increase risk in New Zealand, so feet should be managed to be as clean and dry as possible.

Cows know their way around the farm but everything is strange to the heifers. Start running the heifers through gates, into handling and holding areas, also walk them across concrete and round corners. At least three weeks before calving train them to load into the dairy. Getting in is one thing, backing out from a rotary bail is another. When the heifers load for real they will have just calved and their pelvic ligaments will be soft and pliant so acute turning in a fearful situation is less than desirable. Training takes much of the fear away.

When loaded in the dairy the heifers and cows can have tails trimmed to improve hygiene and to prevent that much dreaded matted tail brush that swats the milker. Maybe excess udder hair can be removed. This is common in many countries, using an electric trimmer or by singeing with a yellow flame, to keep the udder clean and to stop trapping hair in the liner mouthpiece. The latter can lead to liner slip and air ingress during milking.

Teat spraying the heifers during the training period gets them used to the process and can have benefits in reducing mastitis at calving<sup>7</sup>. Finally run the milking plant during the later training sessions so the heifers can be customised to the sound of vacuum pump and pulsators (and the radio?). Keep the dog away now, and always.

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#### She's calved

Contradictory advice and lots of folklore exist on dealing with the newly calved cow. First, aim to get the calf feeding on the dam's colostrum well within six hours of calving, then milk the cow targeting within 12 hours, not 24 hours. This is to get the mammary gland to produce milk and not colostrum. Before calving, the cow is in the colostrogenesis phase, she must have completed lactogenesis, making proper milk, before day five so that you can avoid grading. This requires milking the cow out completely at least twice a day. Never take off the cluster when only part of the milk has been removed. A further benefit is to reduce the risk of grading for inhibitory substances. The cow may well be in the milk withhold period after dry cow treatment, and complete milk out removes natural inhibitory substances found in colostrum/early milk<sup>8</sup>. Complete and regular milking helps remove any mastitis-causing bacteria that may invade the udder immediately post-calving when the cow's immune system is compromised. Mastitis is most common just after calving, anything to reduce the risk has major benefits.

If necessary, clean up the cow from mud and faeces, washing without chilling her. This benefits milk quality and cow health from hygiene, the environment and you from cleaner conditions. You know how much better you feel when spruced up.

You now have a cow that is clean, healthy and producing quality milk with minimal stress. Enjoy the benefits of a little investment made during the dry period.

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# Growing good calves

# - health and rumen development



Nita Harding, DairyNZ Team Leader for Animal Husbandry and Welfare; Gwyneth Verkerk, DairyNZ Senior Scientist; Kevin Macdonald, DairyNZ Senior Scientist; Garry Waghorn, DairyNZ Senior Scientist

Proper rearing of calves affects the survival and the quality of heifers at first calving. Management practices for rearing calves vary, but reducing the risk of disease and ensuring early development of the rumen are key to successful production. If the calf is healthy and the rumen well developed, calves will better utilise pasture when weaned off milk and suffer no set-backs.

#### Keeping your calves healthy

Once the calf is born it is exposed to diseases, particularly from bacterial and viral infections. Management must help calves to withstand pathogenic organisms but allow others to colonise the developing rumen. The cow (or calf) does not digest pasture; this is done by the bacteria, protozoa and fungi in the main stomach (the compartments known as the rumen and the reticulum – the Rumen). However, unwanted organisms entering the body are a problem, and calves must deal with these threats through their own immune system. Their immune system is at first poorly developed and the calf relies on maternal antibodies in colostrum, then the immune system develops by gradual exposure to organisms. It can be further stimulated by vaccination. All are equally important to survival and growth.

#### Colostrum

Colostrum has a high concentration of immunoglobulins which provide early protection from disease. These immunoglobulins are not digested and pass into the small intestine where they are absorbed as intact molecules through cell gaps in the mucosa. The antibodies carried by the immunoglobulins confer passive immunity to the calf against diseases to which the dam has some immunity. If the dam has been vaccinated against some calf diseases the calf gains passive immunity from colostrum. The small intestine is permeable to immunoglobulins for 24-36 hours following birth, but the permeability decreases from about six hours after birth, so the calf needs to receive adequate amounts of colostrum as soon as possible. About half of New Zealand dairy calves may not receive colostrum from their dams even when they are together for 24 hours<sup>1</sup>. This is why the calves should be removed from their dams after six hours and fed colostrum to guarantee that they receive sufficient immunoglobulins. This may be a greater problem in large herds where staff have responsibility for large numbers of animals, and where there are many cows calving together. This may reduce the chances of successful mothering and adequate colostrum intake (see also *DairyNZ Technical Series Issue 4 – Colostrum for calves: why is it so important*?).

#### Disease

The most common health problems with calf rearing are:

- Scours nutritional and infectious
- Pneumonia
- Navel infection
- Coccidiosis
- Intestinal parasites.

These are a result of bacteria, viruses, protozoa and nematodes – all affect the health of the young animal, but problems are preventable if some thought is put into planning how the calves are reared and if adequate housing and feed are supplied.

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#### Vaccination

Calves benefit from vaccination to counter the risk of diseases, some of which can strike very quickly and affect a large proportion of animals. Many of the bacteria and viruses responsible for disease are present in the environment, meaning exposure is common. Disease may be triggered by dietary stress, injury, changes to management or climatic conditions. In some instances the first sign of infection is death!

A vaccination programme needs to be developed for each farm in consultation with the veterinarian as the risk will vary between farms and regions. Booster vaccinations are just as important as the initial vaccination. High risks for calves include pneumonia, enteritis, Clostridial diseases such as Black Leg and Pulpy Kidney, Leptospirosis and Bovine Viral Diarrhoea (BVD). Other risks include nasal catarrh, salmonellosis and Johne's Disease, but the list of possible problems is much longer.

#### Developing the rumen for early weaning

The main compartment of the new-born calf's stomach is the abomasum, or 'true stomach' which secretes enzymes and acid to digest milk. At birth the abomasum comprises 70% of total stomach volume and the other parts (rumen, reticulum and omasum) are small and non-functional. In the first weeks of life the stomach undergoes a remarkable transformation, so that by four months of age the abomasum is only 10% of the total volume (Figure 1). The rumen and reticulum ("Rumen") undergo an extensive physical remodelling, along with colonisation by bacteria, fungi and protozoa. Calf feeding and management during the first few weeks of life will affect the success of this transformation.

When a young calf drinks milk, the "sucking reflex" causes a muscle in the stomach wall to contract, creating the oesophageal groove, which sends the milk directly to the abomasum. Solid food and water, on the other hand, go into the developing rumen. If the oesophageal groove does not close properly or if the milk flows too quickly, it can spill into the rumen. The developing rumen can cope with small amounts of milk, but excessive spill-over leads to ammonia production, rumen stasis (lack of contractions) and bloat, which reduces appetite and growth, and can be hard to overcome. Ensuring that the young calf is somewhat excited in anticipation of being fed, providing warm milk for the youngest calves, ensuring that feeder teats are in good condition and having the calf work a little to get the milk from the teat all help the sucking reflex reduce the chance of spill-over.

To avoid growth checks at weaning, the rumen must be fully functional before the calves are weaned off milk. In natural situations, such as rearing beef calves, they remain with their dams for four to six months so there is more time for the rumen to develop before the calf is weaned. However, dairy calf rearing systems often aim to accelerate rumen development for earlier weaning, to reduce the costs of using milk and milk replacers, and the workload for staff. This is done by limiting the amount of whole milk or whole milk replacer, and offering a free-choice starter mix and limited amounts of good quality forage such as hay.

To develop digestive activity in the rumen requires water, feed and microbial activity<sup>2</sup>. Rumen development is not fully understood, but it can be manipulated to accelerate the conversion of the milk-drinking calf into a pasture-digesting ruminant.

#### The three key factors that need to be provided are:

 Volatile fatty acids (VFA). These result from microbes digesting feed, so as the calf nibbles at meal and hay from about two days of age, microorganisms begin to colonise the rumen and digest the feed. From about 10 days of age, calves can ferment solid food, and VFA production becomes stable when they are one month old<sup>2</sup>. The VFA stimulate growth of the rumen papillae (see photo) on the mucosal (inside) surface of the rumen. The papillae increase the surface area of the rumen mucosa, which helps with nutrient absorption.

Three main VFA are formed; acetic acid, propionic acid and butyric acid. The most potent stimulator of rumen development is butyric acid and to a lesser extent propionic acid. These come from digestion of starch and simple sugars, so feeding grain-based supplements will increase VFA production and promote rumen development<sup>3</sup>.

2. Solid fibre particles ("scratch"), especially from good quality hay. These will stimulate muscle development in the rumen<sup>4</sup>. Muscle development increases mixing of the rumen contents and enables cudding. The large amount of saliva produced during cudding contains bicarbonate, which buffers the acidity of rumen contents and provides an ideal environment for microbial fermentation. Provision of solid fibre may also protect calves against the potentially negative effects of milk spill-over and bloat<sup>4</sup>. However, solid fibre alone does not accelerate rumen development to the same extent as feeding grain-based supplements. Providing about 70% concentrate: 30% roughage appears most beneficial for developing rumen function and enabling good growth rates. Calves fed straw rather than hay as the roughage source (in combination with a concentrate) had lower intakes and lower daily gains<sup>4</sup>. Providing only hay or straw to very young calves before rumen motility is established can lead to overconsumption and impaction of the undeveloped rumen, with less appetite and poor growth. Insufficient milk and boredom may both contribute to this sort of development.

 Separate drinking water ("free water"). There is a common view that milk or milk replacer is sufficient to provide the calf's daily water requirement, but calves grow better when they are provided with drinking water. This is because the water provided from milk is diverted to the abomasum, while drinking water goes into the developing rumen. Water is mixed with solid food in the rumen and helps rumen development. Good quality drinking water should be available at 2-3 L/calf/day<sup>5</sup>.

Calves offered ad libitum water eat more starter mix, especially from the third week of life, and have better weight gains than calves without free water<sup>6</sup>.

The photographs (figure 1) of rumens from calves slaughtered at 6 weeks of age demonstrate developmental in calves fed only milk, milk and grain, or milk and hay<sup>7</sup>. Calves fed milk and grain had the greatest rumen development.

#### When is the right time to wean calves off milk?

Two schools of thought prevail – whether calves should be weaned on age or on liveweight, and there is no clear answer. The important point is that the rumen must be fully functional and the calves have good resistance to disease before weaning.

In practical terms, the best guide to whether a calf is ready to wean is the amount of meal eaten in an indoor system. A study conducted in Switzerland<sup>8</sup> reduced daily milk available as meal intakes increased above 0.7kg/day, and calves were weaned when they ate 2kg meal/day.

So, the main criterion was intake of meal, and this is good providing farmers know the intakes of individuals – but this is difficult in a pen feeding situation. TLC (tender loving care) is essential for young calves, and they should be managed as individuals, rather than the average of a group.

#### Calves that are ready to be weaned will:

- Have a broad belly at the bottom, indicating a full rumen
- Show cudding behaviour calves can be seen chewing and swallowing their cud in a regular pattern, and regular patterns of ripples across the left flank showing the rumen contracting
- Readily eat a large amount of meal.

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**Figure 1:** Images of the rumen wall from 6 week old calves fed (a) milk only, (b) milk and grain, and (c) milk and hay. The ridges and papillae of the rumen wall are more developed in (b), the calf fed milk and grain than the other two examples. The darker colour of the rumen wall in image (b) is due to greater vascularisation and blood supply indicating that it has advanced functionality (pictures reproduced with permission from Dr Jud Heinrichs, Penn State College of Agricultural Sciences; source: http://www. das.psu.edu/research-extension/dairy/)



a. 6 weeks - milk only



b. 6 weeks - milk and grain



c. 6 weeks - milk and hay

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### Are there risks with feeding mastitic or antibiotic-containing milk?

Although mastitic milk is unfit for human consumption, many farmers elect to feed it to their calves as a matter of economy.

It is generally accepted that there are no direct effects of feeding mastitic milk on calf health<sup>9, 10</sup> and no effects were reported from milk with a high somatic cell count (SCC) on calf health or growth<sup>11, 12</sup>. In a recent review of heifer mastitis<sup>13</sup> it was concluded there were no effects of feeding milk with high SCC or mastitic milk on udder development.

Two important contagious diseases that may have an indirect effect on SCC are Johne's Disease and Bovine Viral Diarrhoea (BVD)<sup>14</sup>. Studies in several European countries have shown that SCC is higher in herds infected with BVD virus, and North American studies have shown that farms with a high prevalence of cows testing positive to Johne's Disease have higher SCC and incidence of clinical mastitis<sup>14</sup>. Farms that are working to control these diseases should not feed mastitic milk to calves because it increases the risk of transfer of these diseases. (For further information on BVD and Johne's Disease control programmes, refer to *DairyNZ Technical Series* – Issues 8 and 9). The biggest problem from feeding mastitic milk arises from the fact that it usually contains antibiotics used to treat the dam's udder. Feeding milk that contains antibiotic residues can contribute to the development of bacterial resistance to antibiotics. This has become an important public health concern and international regulatory bodies such as the European Union Commission and the US FDA have recently banned or restricted the use of some antibiotic classes in food-producing animals (see article in *Inside Dairy*, March 2012 for further information).

While there is no definitive proof that use of milk containing antibiotics to feed calves leads to a bacterial resistance to antibiotics, it seems incongruous that feeding a substance containing antibiotics directly into an organ in which bacteria are being encouraged, is practised.

Antibiotics are an important tool for human and veterinary medicine, and good product stewardship must be applied to their use in animals. This means their use should be limited to responsive conditions, and the dose rate, route of administration, and with-holding periods should be strictly observed. Feeding milk containing antibiotic residues to calves does not constitute good product stewardship!

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# Feeding your calves for rapid growth and early weaning



Kevin Macdonald, DairyNZ Senior Scientist; Garry Waghorn, DairyNZ Senior Scientist and Gwyn Verkerk, DairyNZ Senior Scientist.

#### Nutrient requirements of young calves

Newborn calves are not ruminants. They are monogastrics and have monogastric requirements for nutrients, especially crude protein (CP), which should be true protein, rather than nonprotein nitrogen (NPN) such as urea. Energy and protein are two components of the diet but non-structural carbohydrate (sugars and starch), structural carbohydrate (fibre) – measured as neutral detergent fibre (NDF), fats and minerals are equally important as calves grow and develop.

The change in the calf digestive system and the combination of monogastric as well as ruminant digestive processes during rumen development makes it difficult to provide specific nutrient requirements for calves growing from four to 10 weeks of age. However, good husbandry and good nutrition will ensure an adequate (or excess) supply of protein and essential nutrients for the young calf.

#### Published requirements

Detailed recommendations for calf nutrient requirements are given by the U.S. National Research Council (NRC, 2001) and are summarised in Table 1. These guidelines suggest an introduction to starter rations soon after birth, with some reduction in milk supply to encourage calves to consume the ration. Objectives are two fold; ensure the calf is healthy and grows rapidly to target weight and to achieve this at minimum cost. Unfortunately, the latter can lead to problems so the first objective may not be achieved.

The NRC nutrition guidelines apply to calves under all environments, but when they are raised in confinement without access to pasture their ration composition and availability can be completely controlled. In the New Zealand situation, where pasture forms an increasingly large portion of the diet, the diet composition and quality is affected by pasture composition (and sometimes availability) and these additional variables influence animal growth.

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Although some calculations given here are based on North American data<sup>1</sup> the principles and values apply equally to Australian<sup>2</sup> and New Zealand recommendations.

Milk is used most efficiently by young calves. The efficiency of Metabolisable Energy (ME) use from milk for maintenance is about 86% and for growth about 69%<sup>1</sup>. In contrast, the efficiency of energy use from a good quality starter ration is about 75% for maintenance and 57% for growth. Typical daily intakes (of dry matter) by a three week old calf will be 0.8-1.0% of bodyweight and this will increase to 2.8-3.0% of bodyweight by eight weeks of age (Table 1). For Friesian calves, these equate to about 0.45 and 2.1 kg DM/day at three and eight weeks of age, respectively.

Dietary crude protein requirements depend on the quality (biological value; true versus. NPN) of the protein.

- The dietary requirements for crude protein are highest for new-born and very young calves and decline with age (and rumen development) (Table 1);
- Animals having high rates of weight gain require higher concentrations of crude protein in the diet than those with slower growth.

In other words, high concentrations of good quality crude protein will enable high rates of gain, especially in young calves (Table 1).

It is, therefore, essential that rations contain sufficient crude protein to meet calf requirements when fed with pasture. The crude protein content of pasture is equally important, as both components combine to provide nutrients for the growing calf. As indicated previously, the actual requirements for crude protein will depend on calf age, maturity and rumen development. This value changes as the calf grows and develops.

**Table 1.** Calculated daily intakes of dry matter (DM), metabolisable energy (ME, MJ), crude protein (CP, g) and the concentration of CP in the DM. Data are from NRC<sup>1</sup>; Tables 10.2 & 10.4.

Calf weight (kg)	Live- weight gain (g/day)	DM intake (kg/day)	ME (MJ/day)	Percentage CP in the DM
45	600	0.9	15	25
55	800	1.2	21	23
70	800	2.0	26	18
90	800	2.5	30	16

#### Feed system costs

Milk replacers (MR) are often used due to the unavailability of whole milk, and a cereal-based starter-meal in the diet is often used to encourage rumen development<sup>3</sup>. Because of the high cost (\$) of milk products relative to cereal grains, meal may also be used to reduce the quantity of milk required. Restricting the energy intake from milk or MR will generally increase meal intake and encourage rumen development (see "Growing Good Calves–health and rumen development" this publication), and reduce the total cost of feed required<sup>4</sup>.

To attain weaning weights of 70kg (Jersey) and 90kg (Friesian) at nine to 10 weeks the calves need to gain 0.65 to 0.70 and 0.75 to 0.85kg/day for Jersey and Friesian, respectively.

To rear a 37kg calf gaining 0.80kg/day to 90kg at weaning at 10 weeks on milk will require approximately 1150 MJ ME: and if fed a mixture of milk and concentrate 1200 MJ ME.

Using these values, it is possible to compare the cost of the different feeding systems (Table 2). In these calculations, no allowance has been made for:

- Housing for the calves reared indoors (estimated at \$30/calf)
- Loss of pasture through grazing or spoiling, so cows will not eat it
- Extra feeds such as hay or straw.



Table 2. Approximate cost of different	feeding systems based or	n energy inputs, based on NRC <sup>1</sup> .
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	Ratio of feeds (MJ ME)	Whole milk (L)	Milk replacer (kg)	Concentrate (kg)	Approx. feed cost (\$)
Whole milk	-	400	-	-	195
Whole milk <sup>a</sup> + Conc <sup>c</sup>	3:1	300	-	25	180
Whole milk <sup>a</sup> + Conc <sup>c</sup>	1:1	200	-	50	150
Milk replacer <sup>ь</sup>	-	-	60	-	275
Milk replacer <sup>ь</sup> + Conc <sup>c</sup>	3:1	-	42	25	230
Milk replacer <sup>ь</sup> + Conc <sup>c</sup>	1:1	-	30	50	185

<sup>a</sup> Cost of whole milk: 51 c/L (milk 4.5% fat, 3.5% protein 3.4 MJ Net energy/L and supplying the calf with 3.0 MJ ME/L at \$6.35/kg MS (milk price)<sup>5</sup>.

<sup>b</sup> Cost of milk replacer: \$4.60/kg (\$4600/tonne) and 20.0 MJ ME/kg DM.

<sup>c</sup> Cost of concentrates (Conc) - 90c/kg (\$900/tonne) and 12.0 MJ ME/kg DM.

NB: Since protein content is important for the growing calf, the concentrate must have a protein level of 20-24%.

#### Whole milk

Whole milk is the preferred feed on dairy farms because it is readily available and easy to feed. It is cheaper than MR, there is no extra work with mixing, and colostrum can be easily added to the milk. To consume 1150 MJ ME as whole milk will cost \$195.

#### Whole milk and concentrates

This method takes advantage of the low cost of whole milk and concentrates. If whole milk and concentrates are fed at a ratio of 3:1 (milk replacer and concentrate on a MJ ME basis) then the cost is \$180, this can be further reduced by using a 1:1 ratio (MJ ME) for a total cost of \$150 (Table 2).

#### Milk replacer

This is generally the only choice for calf rearers who do not have access to whole milk. The cost is greater than for whole milk (\$275), but the advantage is that the calves can be fed away from the dairy and there is no need to transport milk to the calves.

#### Low volume milk replacer plus extra concentrates

This method has received some acceptance because the calves do not need to be close to the dairy. In this system the calves are fed a concentrated milk replacer from about two weeks of age. It is mixed at 150-200g/L of water instead of the usual 125g/L of water and costs \$185 to \$230 to rear a calf to weaning, depending on the milk replacer : concentrate ratio.

#### Which system and method to choose

All these systems can work successfully. The most appropriate choice will depend on cost, the amount of labour available and the required performance. Probably the most important cost consideration is that the protein and ME provided by concentrates generally cost only 40 and 60%, respectively, of those in whole milk. Therefore it makes good economic sense to have a high proportion of the calves' diet made up of concentrates.

#### Economic consequences of underfeeding

The economic consequences of under feeding calves is difficult to predict because some instances of compensatory growth have been reported and there is a general paucity of data on the impact of inappropriate feeding over a cow's lifetime.

Macdonald and Penno<sup>6</sup> reported that calves that were weaned early following restricted milk replacer (i.e. 22 kg milk replacer and weaned at six weeks of age) were partially compensated by increased pasture intakes but post-weaning live-weights were reduced. This supports conclusions by Everitt and Jury<sup>7</sup> who also found underfeeding in the first 16 weeks of life reduced subsequent growth, and compensatory growth did not make up the difference. The net result is that if calves are poorly grown pre-weaning, they may never make up the difference.

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## Affect of pre weaning growth on subsequent milk production?

Studies that have examined pre-weaning growth rate on future milk production have produced equivocal results but the overall evidence from a review by Heinrichs and Jones<sup>8</sup> was that there were no detrimental effects on production from first-lactation heifers reared for fast growth rate (up to 1 kg/day). The main benefit of accelerated growth was in their economic efficiency because calves are very efficient at converting feed into growth.

Furthermore, a recent study in the United States<sup>9</sup> suggests that calves with high growth rates had higher milk yields than their slower growing counterparts. So, lifetime production performance can be influenced by early life development, but the relationships established with overseas concentrate feeding regimes may differ from the New Zealand situation.

#### Growth from weaning to calving

This was fully covered in the DairyNZ Technical Series Issue 2 – Replacement heifers-rearing the next generation. If reared outdoors calves should always have access to leafy material. Calves are fussy about what they eat and drink. They must have continual access to fresh pasture and water. Dehydration can be a problem if the water is dirty as they will refuse to drink. In general, target liveweights should be: at 3 months 20%, at 11-12 months 50%, at 14-15 months 60% and at 22 months 90% of mature liveweight.

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# Avoiding metabolic diseases around calving



John Roche, Principal Scientist Animal Science.

#### Summary

- On average, the prevalence of metabolic diseases is low; however, on individual farms, they can be a costly and time consuming problem. Most metabolic diseases occur shortly before or in the four weeks after calving.
- The most common metabolic diseases in New Zealand are milk fever and ketosis, but other diseases may become more common with system intensification.
- Prevention involves:
  - achieving BCS targets (5.0 for mature cows and 5.5 for heifers and second calvers)
  - supplementation with magnesium during the weeks before calving
  - supplementation with calcium during the colostrum period
  - managing the cow's diet before calving so that she is not over fed.



#### Introduction

Metabolic diseases are complex disorders that occur when the cow's ability to adjust to a major change (e.g. calving) is compromised. They have been a persistent problem for farmers for centuries, with milk fever first documented in 1793 in Germany<sup>1</sup> and ketosis reported in the USA as early as 1849<sup>2</sup>.

Although, on average, the prevalence of metabolic diseases is low in NZ herds<sup>3</sup>, many farmers face annual problems that are both costly and frustrating. In addition, clinical cases are only part of the problem, with many more cows suffering subclinical problems. For example, DairyNZ data indicate that for every downer cow, two more cows have milk fever and 16 more cows have subclinical milk fever<sup>4,5,6</sup>.

The majority of metabolic diseases occur during the three weeks either side of calving (the Transition Period). Although genetic factors influence the risk of these diseases, with reported genetic effects on susceptibility to milk fever<sup>7,8</sup> and on the rate of body condition score (BCS) loss, farm management in the weeks around calving has a major effect on the risk of disease. The principal causes of, and strategies to avoid, these metabolic diseases will be reviewed here.

#### Metabolic diseases in New Zealand

The two most common metabolic diseases in New Zealand are milk fever and ketosis, although it is likely that "fatty liver syndrome" and left displaced abomasum will become more common with higher levels of supplement use and higher milk production/cow. All of these diseases have secondary effects, with the occurrence of one disease increasing the risk of another (Figure 1). In addition, they predispose cows to infectious diseases<sup>9</sup>, particularly of the udder and uterus, and reduce milk production and fertility. Conservative estimates indicate that the failure of cows to transition properly through calving costs the New Zealand dairy industry in excess of \$1 billion/year.

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**Figure 1.** One metabolic disease can increase the risk of other metabolic and infectious diseases and reduce milk production, health and reproduction.

#### Milk fever

Milk fever is best described by its technical name – parturient hypocalcaemia – which means lowered blood calcium around the time of calving; ~90% of milk fever occurs in the 24 hours after calving<sup>8</sup>. On average, only 2% of cows are diagnosed with milk fever (i.e. "downer cows")<sup>3</sup>. However, laboratory analyses indicate that double this number have less than the clinical threshold of calcium circulating in blood (i.e. have milk fever but do not become "downer") and 33% of cows are subclinically affected (i.e. low dry matter (DM) intake, reduced milk production)<sup>4,5,6</sup>.

Clinical milk fever is reported to reduce milk production by 14%, while cows suffering subclinical hypocalcaemia produce 7% less milk<sup>10,11</sup>. There is also evidence that milk fever increases the risk of ketosis and uterine infections<sup>9</sup> and that 5% of downer cows do not recover<sup>1</sup>. The cost of milk fever on the average farm (i.e. 2% downer cows, 5% clinical hypocalcaemia and 33% subclinical hypocalcaemia) is approximately \$8,000/100 cows.

Although a cow has substantial stores of calcium in her skeleton (~6 kg) and consumes a considerable amount of calcium in food (i.e. a cow eating 10 kg of pasture and pasture silage has 40-80g calcium in her intestine), blood calcium is under very strict hormonal control; a cow only absorbs from food and resorbs from bones what she requires. This can create an issue at the onset of lactation, when a cow's requirement for calcium in blood can increase by more than 400% in a day. This requires a rapid increase in the absorption of calcium from the intestines and in

the resorption of calcium from bone. Anything that interferes with these processes will increase the risk of milk fever.

### Many factors affect the cow's ability to maintain blood calcium during this period.

- Genetics: Jersey cows are 2.5 to 5 times more likely to get milk fever than Holstein-Friesian (HF) cows<sup>7,8</sup>. This effect of breed is well published, with Channel Island breeds having less ability to absorb calcium and secreting more calcium in milk. Within breed, HF cows from North America have lower blood calcium and magnesium than New Zealand HF cows<sup>12</sup>, indicating a greater risk of milk fever.
- Body condition score (BCS): Cows that are excessively fat (greater than BCS 6.0) or excessively thin (less than BCS 3.0) at calving are at an increased risk of milk fever<sup>8</sup>.
- 3. Weather: Milk fever is more likely to occur during wet days and nights, probably because of lower DM intakes and increased stress. In addition, frosty nights increase the risk of milk fever<sup>8</sup>.
- **4. Diet:** Many dietary factors can contribute to the risk of milk fever:
  - <u>Magnesium</u>: Magnesium intake is, arguably, the single greatest dietary factor determining the risk of milk fever<sup>7,8</sup>.
    Magnesium is essential for the efficient absorption and resorption of calcium; therefore, cows that have low blood magnesium around calving are more likely to get milk fever. In an analysis of 30 years of data from the DairyNZ No. 2 Dairy,

milk fever prevalence dropped from more than 10% to less than 5% following the introduction of pre-calving magnesium supplementation in the late 1970s/early 1980s (Figure 2)<sup>8</sup>. Pre-calving cows should be supplemented with 15-20g magnesium/cow per day (double the dose if applying MgO to pasture). DairyNZ data indicate that magnesium sulphate and magnesium chloride are more effective than magnesium oxide. However, their use is not practical on all farms<sup>13</sup>.

<u>Calcium:</u> Cows only absorb as much calcium as they require and it takes several days for a cow to alter the proportion of calcium she absorbs from her diet<sup>14</sup>. Therefore, if a diet is high in calcium before calving, the proportion of calcium absorbed will be low because the cow needs very little for maintenance requirements and foetal growth. When the cow calves and her requirements for calcium increase rapidly, she can't increase the proportion of dietary calcium absorbed sufficiently or quickly enough and milk fever occurs.

Because of this effect of dietary calcium on calcium absorption, traditional recommendations have been to feed a low calcium diet pre-calving. This will stimulate the cow to absorb a higher proportion of calcium from her diet, such that, when she calves and is fed a high calcium diet, she will absorb enough calcium to prevent milk fever<sup>15</sup>. This strategy has been demonstrated to be effective. However, it is very difficult to reduce dietary calcium low enough precalving to stimulate calcium absorption sufficiently postcalving to prevent milk fever.

Oetzel<sup>16</sup> and Lean et al.<sup>17</sup> noted that very low and very high levels of dietary calcium appeared to prevent milk fever, with the greatest risk of milk fever being between 0.5 and 2.0% DM calcium. These reports have led some to suggest that milk fever can be prevented by supplementing cows with calcium pre-calving. This is a very dangerous recommendation and should only be considered as a last resort for milk fever prevention. The data presented<sup>7,16</sup> indicate that dietary calcium would need to be greater than 2.0% DM to be effective in milk fever prevention: on average, a dry cow eating 9kg DM of a pasture and pasture silage mix would have to eat a further 400g ground limestone (limeflour) each day for dietary calcium to be sufficiently high to reduce the risk of milk fever. This is obviously not practical. Calcium supplementation (200-300g/day of ground limeflour) while cows are in the colostrum herd along with magnesium supplementation is one of the most practical ways to prevent milk fever<sup>13</sup>.

 Potassium: Dietary potassium also contributes to the prevalence of milk fever, but it is not as important as many people claim. Recommendations based on research undertaken in the USA suggest that potassium is the primary nutritional factor contributing to milk fever through its effect on the dietary cation-anion difference (DCAD). Because of this, high potassium forages should be minimised in the weeks before calving<sup>17</sup>. If this research were appropriate for New Zealand, 100% of cows would get milk fever. In comparison, incidence of milk fever is low<sup>3</sup> and New Zealand data<sup>13</sup> indicated no difference in blood calcium around calving when cows were fed pastures varying from 3.3 to 4.2% DM potassium. This does not mean that potassium is unimportant. Potassium interferes with the absorption of magnesium in the rumen and, as magnesium is important for calcium absorption, thereby increases the risk of milk fever. However, it is secondary in importance to magnesium supplementation.

- <u>Phosphorus:</u> Feeds that are high in phosphorus increase the risk of milk fever by interfering with the production of hormones essential for calcium absorption<sup>18,19</sup>. Because of this, feeds that are high in phosphorus (e.g. palm kernel extract, distillers grains) should be used with caution in the weeks prior to calving in milk fever-prone herds.
- Dietary Cation-Anion Difference: The DCAD is calculated from the amount of potassium, sodium, chlorine and sulphur in the diet. The proportion of these minerals in the diet influences the acidity/alkalinity of blood (blood pH)<sup>20</sup> and blood pH affects calcium absorption from the intestine and bone calcium homeostasis<sup>21,22,23</sup>. Blood pH drops when DCAD is less than -100 meq/kg DM and calcium absorption from the small intestine increases<sup>21</sup>. Such a low DCAD is generally not achievable when pasture is a part of the ration. Lowering the DCAD through removal of potassium from the ration, however, will improve magnesium absorption and reduce the risk of milk fever in some circumstances.
- Level of feeding: DairyNZ data<sup>4,6</sup> indicate that the more feed cows are offered in the two weeks before calving, the lower the concentration of calcium in blood at calving. The reason for this is unclear; it may be because well fed cows are receiving more calcium in their diet, reducing the proportion of calcium absorbed, or because they are consuming more potassium pre-calving, or it may be because well fed cows have lower DM intakes after calving<sup>6</sup> and, therefore, reduce their intake of calcium at the time when it is most needed.

#### Ketosis

Ketone bodies (acetone, acetoacetate, B-hydroxybutyrate: BOH) are intermediates in the breakdown of fat they can accumulate in blood when large amounts of body fat are mobilised and there is insufficient carbohydrate to facilitate use of fat for energy (fatty acid oxidation). The clinical state of this disease is referred to as ketosis.

Ketosis is sometimes referred to as "Sad Cow Syndrome", with one of the first indications being that the cow is lethargic, goes off feed, and drops in milk production and there is often a sweet

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**Figure 2.** Prevalence of milk fever at No 2 Dairy between 1970 and 2000. Note the reduction in the prevalence following the introduction of magnesium supplementation pre-calving.



**Figure 3.** Prevalence of clinical and subclinical ketosis in NZ dairy cows as defined by blood BOH concentrations (i.e. >1.2 mmol/L BOH = subclinical ketosis and >2.0 mmol/L BOH = clinical ketosis). 0 = day of calving



**Figure 4.** Plasma  $\beta$ -hydroxybutyrate (BOH) concentrations in dairy cows being fed the same amount of energy as either fresh pasture or fresh pasture and a starch-based concentrate. Milk energy output and body condition score change was not affected by treatment.



smell (acetone) on the breath<sup>24</sup>. In a proportion of cows, the ketone bodies affect the brain and they can become excitable, eat soil or lick fence posts and gates, walk around in circles or stand with their heads raised and pushed into a corner<sup>24</sup>.

International data indicate that both clinical and subclinical ketosis reduce milk production and conception rate. Based on these effects, ketosis costs approximately \$3,500/100 cows.

There is no information on the actual prevalence of clinical ketosis in New Zealand, but internationally the prevalence of clinical ketosis is reported at 4%<sup>25</sup>, although Oetzel<sup>26</sup> also reported the detection of severe clinical ketosis in 4% of cows that were not detected as sick. New Zealand data collected over several years (Figure 3) <sup>4,5,6</sup> indicate that:

- 8% of cows have greater than 2.0 mmol/L BOH in blood; this is defined internationally as clinical ketosis.
- 10-12% of cows have greater than 1.2 mmol/L BOH in blood; this is defined internationally as subclinical ketosis.

However, it is unclear whether these international definitions are appropriate for New Zealand dairy farms as blood ketone body concentrations are influenced by feed ingredients in otherwise healthy cows. Cows fed fresh pasture alone have greater blood BOH concentrations than those supplemented with a starchbased concentrate supplement (Figure 4)<sup>5</sup>, even when BCS loss and the fat content of blood are the same, and the cows are secreting the same amount of energy in milk<sup>6</sup>. Therefore, the prevalence data for ketosis based on blood BOH are probably not accurate for pasture systems.

#### There are three types of ketosis<sup>26</sup>:

- Type 1 ketosis is a result of an unexpected drop in DM intake, particularly in high producing cows (spontaneous ketosis). This can be caused by not allocating feed properly or because adverse weather prevents cows from eating allocated feed (e.g. heavy rain, snow). Because the underfeeding is spontaneous, the cow continues to secrete large amounts of energy in milk and must mobilise BCS to meet her energy demands. The mobilised fat cannot all be used for energy because of insufficient oxaloacetate to complete the process and, so, ketone bodies accumulate. This type of ketosis can be prevented by ensuring cows are adequately fed or by ensuring any feed restriction is imposed gradually (e.g. over a week). Proper use of the spring rotation planner along with appropriate use of supplementary feeds should prevent the occurrence of Type 1 ketosis. Once-a-day milking may also reduce the risk.
- **Type 2 ketosis** occurs to a degree in all cows 3-4 weeks after calving (Figure 3), but the clinical condition generally occurs in over-conditioned cows, particularly those that have been well fed in the springer mob. The risk of Type 2 ketosis doubles when calving BCS increases from 5.5 to

6.0<sup>27</sup>; therefore, carryover cows tend to be particularly at risk. Fat cows tend to have a lower DM intake after calving and greater BCS mobilisation. These factors combine to reduce the liver's ability to utilise fat fully as an energy source, leading to the accumulation of ketone bodies. Type 2 ketosis is also affected by the level of feeding precalving. Cows that are fed well in the weeks before calving tend to have reduced liver function and an increased risk of Type 2 ketosis post-partum<sup>28</sup>.

Prevention should be through feeding management in late lactation and during the dry period. Mature cows should be fed to achieve a calving BCS of 5.0 one month before calving (heifers and second calvers at 5.5), with no cows greater than BCS 5.5. If these targets are achieved, springing cows should consume 80% of their energy requirements (i.e. make allowances for wastage)<sup>29,30,6</sup>. Internationally, Rumensin has been reported to reduce plasma BOH concentration in early lactation<sup>31,32</sup>, indicating a positive effect on early lactation cow health; however, the appropriateness of these findings for grazing cows in New Zealand is not clear.

 Silage ketosis: In addition to the two main types of ketosis, cows can also get ketosis from consuming poor quality silage. Silage that has not been properly covered (i.e. air not properly excluded, torn plastic, tyres not touching) can undergo a secondary fermentation, wherein remaining sugars and lactic acid can be fermented to butyric acid. If consumed, this butyric acid will be absorbed and converted to BOH, increasing blood BOH and the risk of ketosis. Silage should be tested for organic acids and silage high in butyric acid should not be fed to transition dairy cows<sup>26</sup>.

Successful treatment of clinical ketosis will involve veterinary intervention to provide cows with drugs that stimulate an increase in blood glucose. In addition to this, oral administration of sodium propionate or monopropylene glycol in a "starter drench" will provide energy that allows the cow to utilise the ketone bodies and a change in diet to provide more energy will ensure the cow can recover<sup>24</sup>.

#### Fatty liver

Fatty liver is a condition wherein large amounts of fat (triglycerides) are stored in the liver. When liver triglyceride concentration is greater than 5mg/100mg liver tissue, the cow is deemed to have fatty liver. The increase in liver fat content reduces the liver's ability to produce glucose and detoxify ammonia, thereby increasing the risk of ketosis and, very likely, contributing to liver dysfunction and a systemic activation of the immune system<sup>28,33</sup>. This whole body inflammation has been linked to reduced milk production and increased reproductive failure. Fatty liver occurs to a certain degree in all cows in early lactation, when BCS mobilisation is rapid and much of the mobilised fat is transported to the liver. The cow is unable to utilise it all for energy and the system for exporting fat from the liver to other tissues is not very efficient in dairy cows. Therefore, some of it accumulates<sup>24</sup>.

There are limited data on liver triglyceride content in New Zealand grazing dairy cows. DairyNZ data indicate liver triglyceride levels of 6mg/100mg of liver tissue in the first week post-calving, declining to 3.5 and 2mg/100mg of liver tissue in week four and eight post-calving, respectively. There was no difference between New Zealand and North American HF cows and no effect of concentrate supplementation on liver triglyceride levels. These data do not indicate a problem. However, cows calving at greater than BCS 5.5 and well fed precalving are likely to have high liver triglyceride concentrations and reduced liver function. This may contribute to low milk production and poor reproduction and an increased incidence of mastitis and uterine infections. Research is on-going in this area to determine the implications for pasture-based dairy cows.

#### Conclusions

Although, on average, the prevalence of metabolic diseases in dairy cows is low in New Zealand, when they occur, they are both costly and frustratingly time consuming. Many metabolic diseases are inter-related, with the occurrence of one disease increasing the risk of another. Care must be taken to ensure mature cows are at BCS 5.0 one month before calving (heifers and second calvers should be 5.5), and that springer cows are adequately supplemented with magnesium (15-20g magnesium/cow/day: double the dose if applying MgO to pasture). Springer cows that have achieved the calving BCS targets should consume 80% of their daily metabolisable energy requirements during the last two weeks before calving. Colostrum cows should receive supplementary calcium (200-300g limeflour/cow per day) and magnesium (15-20g magnesium/cow per day: double the dose if applying MgO to pasture).



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## Recently published by DairyNZ

DairyNZ researchers publish their findings in high calibre national and international journals, so they remain at the leading edge of dairy industry research.

#### Peer reviewed publications

Bryant, R. H., P. Gregorini and G. R. Edwards. 2012. Effects of N fertilisation, leaf appearance and time of day on N fractionation and chemical composition of Lolium perenne cultivars in spring. *Animal Feed Science and Technology* 173(3):210-219.

Green T.C., and D. J. Mellor. 2012. Extending ideas about animal welfare to include assessment of quality of life and whether or not farm animals have lives worth avoiding or living, or good lives. *New Zealand Veterinary Journal* 59(6):263-271.

Gregorini, P. 2012. Stobbs Memorial Lecture - Diurnal grazing pattern: Its physiological basis and strategic management. *Animal Production Science* http://dx.doi.org/10.1071/AN11250.

Higgs, R. J., L. E. Chase, and M. E. Van Amburgh. 2012. Development and evaluation of equations in the Cornell Net Carbohydrate and Protein System to predict nitrogen excretion in lactating dairy cows. *Journal of Dairy Science* http://dx.doi. org/10.3168/jds. 2011-4810.

O'Brien, B., J. Jago, J. P. Edwards, N. Lopez-Villalobos and F. McCoy. 2012. Milking parlour size, pre-milking routine and stage of lactation affect efficiency of milking in single-operator herringbone parlours. *Journal of Dairy Research* 79:216-223

Pryce, J. E., J. Arias, P. J. Bowman, S. R. Davis, K. A. Macdonald, G. C. Waghorn, W. J. Wales, Y. J. Williams, R. J. Spelman, and B. J. Hayes. 2012. Accuracy of genomic predictions of residual feed intake and 250-day body weight in growing heifers using 625,000 single nucleotide polymorphism markers. *Journal of Dairy Science* 95:2108-2119.

Rius, A. G., H. A. Weeks, J. Cyriac, R. M. Akers, B. J. Bequette, and M. D. Hanigan. 2012. Protein and energy intakes affected amino acid concentrations in plasma, muscle, and liver, and cell signaling in the liver of growing dairy calves. *Journal of Dairy Science* 95:1983-1991.

Sun, X. Z., G. C. Waghorn, J.-H. B. Hatier and H. S. Easton. 2012. Genotypic variation in in sacco dry matter degradation kinetics in perennial ryegrass (Lolium perenne L.). *Animal Production Science* http://dx.doi.org/10.1071/AN11290

Sun, X. Z., and G. C. Waghorn. 2012. Improving in sacco incubation technique to evaluate fresh forage for selecting fastdegrading perennial ryegrass (Lolium perenne L.). *Grass and Forage Science* DOI: 10.1111/j.1365-2494.2012.00860.x.

Waghorn G., K. A. Macdonald, Y. Williams, S. R. Davis and R. J. Spelman. 2012. Measuring residual feed intake in dairy heifers fed an alfafa (Medicago sativa) cube diet. *Journal of Dairy Science* 95(3):1462-1471.

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# Focus on international research

#### The following is a brief summary of some key science papers recently published

Bernier-Dodier and others. 2011. Effect of dry period management on mammary gland function and its endocrine regulation in dairy cows. Journal of Dairy Science 94:4922-4936.

The effect of dry period length on mammary function and milk production in the subsequent lactation was evaluated. Cows dry for 31 days produced 9% less milk, 10% less milk fat and 7% less milk protein than cows dry for 65 days. Mammary cell development and death and the expression of genes involved in milk production were not different between the groups. Results indicate that the negative effect of a short dry period is probably due to differences in mammary development during the dry period, rather than an effect on mammary cell function post-calving.

**DairyNZ comment:** These data are consistent with previous international studies, that dry periods of less than 40 days reduce production in the following lactation. This study provides possible reasons for this, which mean that the negative effect of a short dry period cannot be overcome by post-calving management.

Alawneh and others. 2012. The effect of liveweight change on reproductive performance in a seasonally calving, pasture fed dairy herd. *Livestock Science* 145:131-139.

The effect of liveweight (Lwt) change on reproductive performance was evaluated in NZ. Cows that lost the most Lwt after calving took longer to conceive; 1% less Lwt loss resulted in a 2% reduction in the interval from planned start of mating (PSM) to conception. Live weight change in the three weeks before PSM was also associated with time to conception; cows that lost the least Lwt or gained Lwt before PSM conceived earlier. Effects were additive (i.e. cows that lost the most Lwt after calving and lost Lwt before PSM took even longer to conceive).

**DairyNZ comment:** Data are consistent with previous studies highlighting the effect of body condition score and Lwt loss postcalving and Lwt gain pre-mating on reproduction. However, little can be done about post-calving BCS loss and the effects of Lwt gain in the weeks before PSM are small, although linear. Graugnard and others. 2012. Blood immunometabolic indices and polymorphonuclear neutrophil function in peripartum dairy cows are altered by level of dietary energy prepartum. *Journal of Dairy Science* 95:1749-1758.

The immune system of cows is suppressed around calving, making them susceptible to mastitis and uterine infections. The effect of feeding level pre-calving on immune function was investigated. Cows that were overfed pre-calving were in greater negative energy balance in the first week post-calving, but there was very little difference in measured blood metabolites and indicators of inflammation between treatment groups. Phagocytosis capacity of polymorphonuclear cells was less in the week after calving in cows that were fed more than requirements pre-calving.

**DairyNZ comment:** Although there was very little difference between the treatments, results indicate a possible negative effect of overfeeding cows in the weeks before calving on the cow's ability to withstand a bacterial challenge.

Deguillaume and others. 2012. Effect of endocervical inflammation on days to conception in dairy cows. *Journal of Dairy Science* 95:1776-1783.

This French team investigated the effect of an inflammation of the cervix (cervicitis) and the uterus (endometritis). Of the cows examined before 35 days post-calving (168 cows), 11% of cows had cervicitis without endometritis, 13% had endometritis without cervicitis, and 32% had both cervicitis and endometritis. The results indicate that these are independent conditions and have additive and negative effects on days to conception.

**DairyNZ comment:** Although cervicitis was not measured, DairyNZ data also indicate a high incidence of endometritis in early lactation cows and this significantly reduces conception rate (by up to 20%). Further work is needed to determine the factors predisposing cows to these independent conditions and ways to prevent and treat them.

