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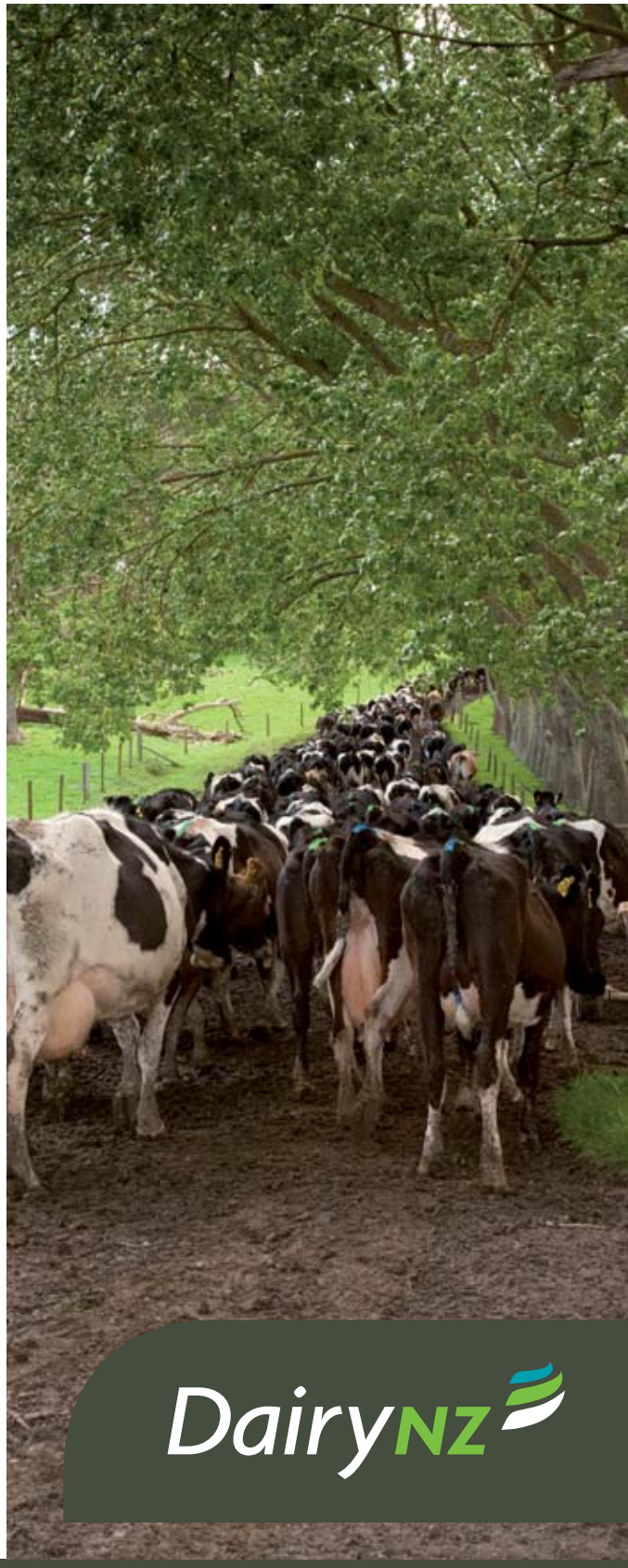

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Dry cow treatment

– how can it be used?

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Summary

Dry cow treatment is infusion of a special formulation of antibiotic into the mammary gland after the last milking of a lactation:

- It is highly effective in curing existing infections
- It is highly effective in preventing new infections. The vast majority of new infections occur just after dry-off or just before calving
- It must always be used on all quarters of the cow
- Low cell count cows, and those with no other history of mastitis, can be protected by using an internal teat sealant at dry-off
- Teat sealant may protect the udder for a few months but will not cure infections so is not for high cell count cows
- Organic producers may be able to use teat sealants depending on which standards are in use.

What is dry cow treatment?

Dry cow treatment (DCT) is the infusion of each quarter of the mammary gland (through the teat canal and into the teat sinus) with an antibiotic preparation immediately after the last milking of the lactation. The product is usually a formulation of antibiotic designed to persist for three to seven weeks. Infusion of antibiotic preparations into heifers before calving is also considered DCT.

Why was it developed?

Antibiotic treatment of bacterial mastitis in cattle first became available more than 65 years ago[1]. Soon after penicillin was obtainable, Jim Pearson in Northern Ireland used a solution as a prophylactic (preventive) treatment for summer mastitis, a severe suppurative condition mostly seen in dry cows at pasture during summer in northern Europe. He was able to reduce the incidence of summer mastitis from 10% to less than 1%[2,3]. Control was achieved but the preparations only provided 14-18 days of protection.

The need for a longer-acting product was obvious because new infections occur at a much higher rate during the dry period than in lactation. Some 48% of cows were found to become infected in the dry period, most in the first three weeks, and half of the infections persisted into the next lactation[4]. Most were *Staphylococcus aureus* and non-agalactiae streptococcal infections and 50-80% of all UK cows were found to be infected[5]. A New Zealand survey at that time found 52-60% of cows were infected with subclinical mastitis[6]. DCT was further developed since the risk of contaminating milk with antibiotic was minimal, treatment costs could be reduced, and expensive diagnostic services could be avoided. Oliver et al[7]., found they could eliminate 90% of streptococcal and 50% of staphylococcal infections using experimental penicillin-streptomycin mixes, or sodium cloxacillin.

Beecham Laboratories then produced a slow-release formulation of the relatively insoluble salt, 1% benzathine cloxacillin in an oil base, which they claimed was active for the first 21 days of the dry period. This preparation reduced the number of infected quarters by more than 80%[8]. They reported 90% efficacy in preventing new infections by *Staphylococcus aureus* in the dry period but only 58% efficacy in the prevention of new streptococcal infections. Many other studies subsequently reported similar results e.g. for use of 500 mg benzathine cloxacillin from Australia[9] and from the United States[10].

What is DCT today?

Dry cow therapy is predominantly applied as a blanket treatment of all quarters of all cows dried off and intended to remain in the herd, and has been part of the SAMM recommendations since 1995[11].

A number of products based on long-acting penicillin or cephalonium groups are available in New Zealand by prescription from veterinarians. While these products vary in their persistence and thus the duration of protection against new infections, they vary little in their efficacy in curing existing infections. All available products are active against staphylococci and streptococci but not against Gram negative bacteria e.g. *Escherichia coli*, Klebsiella species or Pseudomonads.

Why do we use DCT?

Therapy

A mastitis control scheme that included recommendations to use DCT on all cows in the herd was first proposed by Dodd & Neave[12] on the basis that the prevalence of infection is a product of the rate of new infections and the duration of existing infections. Field studies in the UK showed that, irrespective of treatment of clinical cases, new infections were occurring at a rate of two infections/cow/year and that 70% of infections survived longer than 12 months[13]. The rate of new infection could be reduced by limiting exposure to bacteria through improved hygiene, treatment of clinical cases in lactating cows and better machine milking technology. Treatment of clinical cases during lactation often resulted in good clinical cure but poor bacteriological cure, but treatment of sub-clinical mastitis is not practical or economically justified. Dry cow treatment achieved significantly higher rates of bacteriological cure.

Efficacy varies with the type and dose of antibiotic used. Storper & Ziv[14] showed that effective cure rates (number of eliminated infections less number of new infections) varied from 80-88% (between herd variability). This is similar to other reports using persistent formulations of the original product, benzathine cloxacillin (Table 1).

Table 1. Elimination of intra mammary infections by use of dry cow treatment, compared with untreated controls, data from some early studies.

Study	Treatment	% cure		Effect
		Treated	Control	Reduction
Smith et al.[8]	1g benzathine cloxacillin	82	9.5	88%
Eberhardt & Buckalew [33]	Procaine penicillin + streptomycin	46	11.6	75%
Sinkevitch et al.[34]	0.5g benzathine cloxacillin	78	6.5	91%
Postle & Natzke[10]	0.5g benzathine cloxacillin	93	42	55%

Many dry cow trials have been conducted using a wide variety of antibiotic preparations. These are difficult to compare and, not surprisingly, occasional reports of ineffectiveness have appeared. The first of these from Bratlie[14,15] has been widely quoted. Field studies are difficult to design; most have some faults and a number have given perverse results. Difficulties arise in field studies from limited sampling and bacteriology (suggested earlier by Morris[9] to be a key limiting factor), or because the formulations and doses of antimicrobials may not be administered accurately. In the Bratlie studies, quarters were incorrectly assumed to be independent and the control group had a different proportion of infections to the treatment groups.

The overwhelming majority of trials with DCT indicate that the rate of elimination of infection over the dry period is at least 50% and in some instances may be as high as 90%, compared with an average background of 10% spontaneous elimination in untreated quarters[8,14,17].

Prevention

In 1975, at the beginning of a now-polarised international debate on DCT, Prof. Funke of Sweden said that 'prophylaxis is the most important part of mastitis control…'[18]. Overall, mastitis control strategies have been very successful, and DCT in particular. Using the relationship defined by Eberhardt et al.[19] between bulk milk cell count and infection rate, it can be shown that the prevalence of mastitis in good to average herds in many countries has been reduced approximately six-fold, to fewer than 10% of cows infected.

Historically, studies on the prevention of new infections in the dry period are fewer than studies of cure rates, because negative control groups are essential to demonstrate effectiveness and large numbers of uninfected cows are needed.

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Early studies on prophylaxis in herds with a high prevalence of infection may also have been affected by variation amongst cows in susceptibility to infection in that cows that are less susceptible (and so uninfected) may add bias to the negative group. Untreated control groups in commercial farm studies are rare as few farmers want an untreated group when they expect good success from DCT, and the herds used were small making it hard to find sufficient uninfected cows to give a viable trial design.

While early studies suggested that DCT could achieve a useful prophylactic effect could be achieved (Table 2), somewhat perversely when the prevalence of infection is low the effect seems less. One Australian study[20] points to many quarters being treated for no possible therapeutic benefit. Where a herd has a low prevalence of infection, the probability of new infection is an order of magnitude less than in the original work of Smith et al.[8], despite more quarters being open to infection. Neave et al.[4] had shown that uninfected quarters in cows with one or more already-infected quarters were more susceptible to new infections in the dry period. Bratlie[15] confirmed this observation. In the absence of milking, the level of exposure may be different when a herd has only a low prevalence of infection. Understanding how the risk of infection varies between herds and dairy systems is clearly important when determining the effectiveness of DCT as a prophylactic treatment.

Table 2. Prevention of new intra mammary infections by use of dry cow treatment, compared with untreated controls, data from some early studies.

Study	Treatment	% infection		Effect
		Treated	Control	Reduction
Smith et al.[8]	1g benzathine cloxacillin	5.4	30.9	82%
Sinkevitch et al.[34]	0.5g benzathine cloxacillin	1.4	6.5	78%
Postle & Natzke[10]	0.5g benzathine cloxacillin	6	12	50%
Browning et al.[20]	0.5g benzathine cloxacillin	2.1	3.8	45%

What else can we do?

The original work on prevention and cure of mastitis during the dry period is still relevant even though many of the risk factors have changed, not least being that *S. aureus* may now account for less than 15% of clinical mastitis. Nevertheless, the many changes in dairying systems in the past 50 years mean that the blanket application of DCT requires review. Blanket application has long been controversial. Even in 1975, Dodd commented ' …in case someone has the mistaken view that the NIRD group believe that antibiotic therapy for all cows at drying off will always be an essential part of mastitis control. This is not so'[18]. He also said that some means to reduce the duration of infection would always be necessary. If this is false then there is value in selective dry cow treatment, simply for protection. Without protection from DCT, the prevalence of infection may well increase progressively to the high levels of 30 years ago.

Selective DCT

Australian studies[20] suggested that a selective approach to use of DCT might be warranted. Østerås et al.[21] concluded that if individual cows are selected for DCT then all quarters should be treated, with later analysis leading to a proposal that particular risk factors might be useful to determine whether short-acting or long-acting preparations should be applied[22].This approach was developed in the particular circumstances of the Norwegian dairy industry, and requires detailed knowledge and investigation of the animal and herd background.

Sealants

A plethora of non-antimicrobial products have been 'trialled' both inside and outside the udder as alternatives to DCT but the only products for which credible benefits have been proven are the internal teat sealant products based on a 65% suspension of bismuth subnitrate as a viscous paste. As they are infused into the mammary gland they are a medicine i.e. must have proven efficacy for the benefit claimed and be shown to be safe. Thus they have been tested extensively in recent years. This has given much new information on prevention of new infections in the dry period, in this century, and allowed a re-evaluation of the relevance of DCT today.

This non-antibiotic product is infused into the teat sinus just like DCT, but sterile technique is critically important. It remains in the teat sinus above the teat canal entrance acting as a physical barrier to bacteria entering through the teat canal from moving further into the gland. It is removed when the calf suckles or by teat stripping after calving.

Recent work closer to our current problems

Various studies over the past 10 years have addressed situations in which the environmental causes of mastitis are more common, especially *Streptococcus uberis*, as opposed to contagious mastitis e.g. *S. aureus or Streptococcus agalactiae*, over which good control can be achieved.

Berry & Hillerton[23] showed that when cows were not treated with DCT, 11-22% of cows in the study herds became infected, with half of the new infections occurring soon after dry-off and half around calving. This is very similar to 50 years ago, but with *S. uberis* now as the principal cause of infection. When cows received DCT, the herd level rate of new infection was lowered by 66-100%. Williamson et al.[24] also showed that DCT reduced infection by S. uberis by 90%, from 12.5% to 1.2% quarters at risk.

When an internal teat sealant was compared with no treatment, results were essentially similar (66% reduction in new infections; [25]. Huxley et al.[26] found no obvious differences between DCT and teat sealant in preventing new infections. These studies support New Zealand findings that DCT was essentially the same as a prototype teat sealant formulation at preventing new infections[27], in line with a number of studies from other countries (Table 3). Overall, the various approaches to testing the efficacy of treatments provide support for the continued need for DCT and teat sealing.

Table 3. Percentage of quarters with a new intra mammary infection in the dry period as determined at calving in recent studies. Different symbols within a row indicate a statistically significant difference.

Study	Dry period prophylaxis			
	None	DCT	Teatseal	DCT + Teatseal
Smith et al.[8] for comparison	9.5†	3.3*	-	-
Williamson et al.[24]	12.5†	1.2*	-	-
Berry & Hillerton[23]	13.4†	4.5*	-	-
Huxley et al.[26]	-	15.4†	11.1*	-
Berry & Hillerton[28]	-	6.0† 11.4†	-	3.7*(<10 weeks) 3.8* (>10 weeks)
Berry & Hillerton[25]	11.6†	-	3.4*	-
Gooden et al.[35]	-	21.1†	-	17.5*
Woolford et al.[27] for comparison	13.1†	2.3*	2.4*	1.6*

Combining DCT and a teat sealant has been shown to be more effective than DCT alone in preventing new infections[28,29]. These benefits may be greater in cows with a high cell count before drying off, i.e. those cows more susceptible to infection[29]. Moreover, when administered in combination, the effects of the teat sealant persist for up to 16 weeks even though the antibiotic is only claimed to be active for seven weeks. This finding is highly relevant to New Zealand where the dry period can last for several months.

Both teat sealant and DCT have a marked effect in preventing new infections in first-calving heifers. Teat sealant reduced the risk of a post-calving infection by *S. uberis* in New Zealand heifers by 84%[30].

In a recently reported New Zealand experimental infection model a novel teat sealant containing a disinfectant was shown to reduce the prevalence of new infections at calving in cows[31], although the overall rate of infection in the study cows, even in the DCT-treated group, was several-fold higher than is usual under natural farm conditions.

Economic considerations

The value of dry-off treatment strategies depends on their efficacy in controlling infection and the resulting cost:benefit ratio in economic terms. An analysis by Berry et al.[32] showed that, for a cow that is uninfected at dry-off, both DCT and teat sealant incur a small cost, but both were financially beneficial compared with no treatment which incurred losses from the effects of subsequent infection, e.g. lost milk, labour etc. If the cow was infected (i.e. a cow with >200,000 cells/ml) then benefits only accrued from using a DCT with a cure rate of 88%, such a cure rate being in line with historical and current data for DCT.

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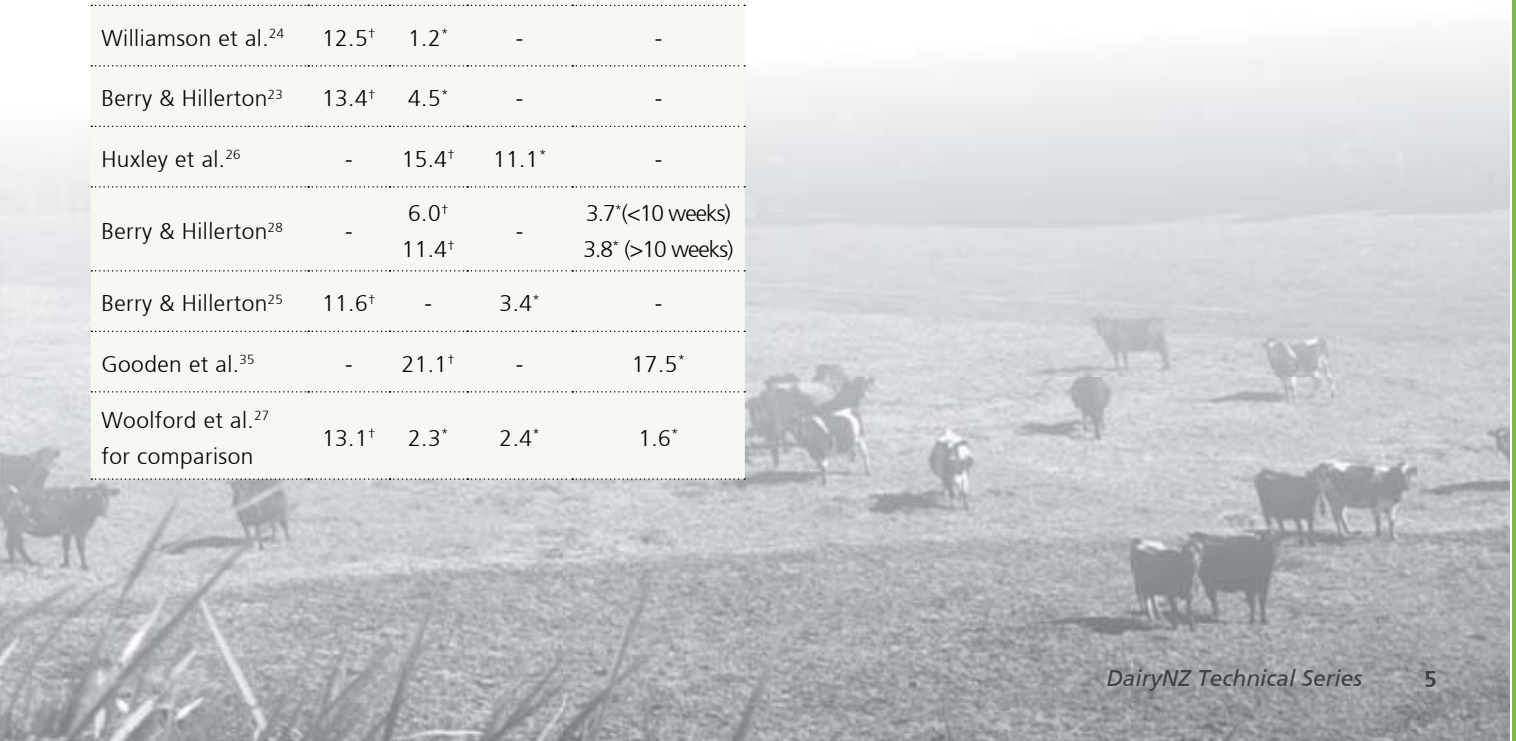

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Managing dry period infections

The dairy farmer has four options to manage existing and new infections in the dry period. Before making any decision, good information is essential including historical experience at herd-level (to assess risk) and individual cow infection status (i.e., latest herd test records or RMT results, and clinical records over the whole lactation). Decisions should be made at the cow level, never at individual quarter level.

No dry-off treatment

Doing nothing to the udder at dry-off will result in up to 25% of the herd developing a new infection sometime in the dry period, with many infections developing into clinical mastitis. For some organic production systems, e.g. those following USA guidelines, this is the only option available, and strict hygienic management of cows with low mastitis susceptibility is essential to manage the risk.

Internal teat sealant

This the best option for those who wish to limit use of antibiotics, and it is acceptable under organic rules in some European countries. Teat sealant will not cure existing infections so is not suitable for high cell count cows or those that suffered clinical mastitis during the previous lactation. It is the best choice for those cows that have had persistently low cell counts and may also be useful for first-calving heifers. A significant advantage of teat sealant is that its protective benefit is effective for at least 16 weeks and possibly longer, making it suitable for low cell count cows that are dried off early or calve late.

Dry cow antibiotic treatment

Blanket use of DCT (i.e. all quarters of all cows to be kept to calve) is one of the main reasons why contagious mastitis has reduced so radically over the past 40 years, and also why New Zealand milk quality (based on bulk milk cell counts) meets high market standards.

Dry cow antibiotic treatment will always remain the best way to remove existing infections and protect cows in high exposure situations such as wintering on stand-off pads and crops. For low cell count cows, it is equally protective as teat sealant. It does not need to be used on all cows in the herd, but when used should be applied to each quarter of the cow.

Dry cow antibiotic and teat sealant

Infusion of DCT followed immediately by teat sealant can be used to 'cure' existing infections and provide extended dry period protection against new infections. Generally it will be too expensive to consider for more than a few cows but it may be useful for high value, high cell-count cows that will have an extended dry period, or in farms dealing with dry period mastitis crises.

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Milking your cows once-a-day throughout lactation

Kerst Stelwagen, Scilactis Ltd; Claire Phyn, DairyNZ Scientist; Jane Kay, DairyNZ Scientist; and John Roche, DairyNZ Principal Scientist

Key messages

- Whole season once-a-day (OAD) milking can be successfully adopted as a profitable farm system. Key benefits include lower labour requirements, improved animal health, lower farm working expenses, and a more flexible lifestyle
- Milking OAD reduces milksolids production per cow, due to a decrease in the activity and number of milk-producing cells in the udder. It appears, however, that the udder "resets" itself during the dry period and there are no carry-over effects in subsequent lactations
- Production losses during whole season OAD are less in Jerseys than Holstein-Friesian cows; however, this is only partially due to their more concentrated milk
- In Holstein-Friesian cows, heifers and three-year olds have greater production losses than mature animals
- Cows milked OAD have a less persistent lactation curve and days in milk may be limited by low milk yields and increased somatic cell counts (SCC)
- Cows milked OAD for a full lactation have greater live weight and body condition compared with those milked TAD; this may improve production and reproduction in the next lactation
- Animal health and welfare can be improved by OAD milking, and although milk somatic cell count increases, there is no increase in the prevalence of mastitis as long as good management practices are used.

Background

Conventionally, cows are milked twice a day (TAD); however, the concept of milking cows only once-a-day (OAD) has been around for a considerable time in New Zealand[1] and can fit well in low-cost, pasture-based systems. Farmers have many reasons for adopting an OAD milking system[2], but managing labour requirements, farm infrastructure and topography, and farm costs are common drivers. This article reviews available data on the factors affecting the performance of whole season OAD systems.

Milk production

There is undeniable scientific evidence that OAD milking results in lower milk production per cow compared with TAD milking. Data from short-term studies indicate that milk production losses average 21%, but can range from 7 to 40% depending on factors such as stage of lactation, parity and breed[3, 4]. Milk production losses tend to be greater in full lactation studies, with an average loss of 34% (range 22 to 50%)[5-8].

In a Massey University trial[6], milk and milksolids production were reduced by 34 and 31% in grazing cows milked OAD for a whole season, compared with their TAD counterparts. In a more recent DairyNZ farm systems trial, milk and milksolids production per cow were about 20% lower in Jerseys and about 30% lower in Holstein-Friesian cows managed in separate OAD and TAD farmlets for each breed (Table 1)[8]. This result indicated that cows with more concentrated milk (i.e. higher milksolids content) and overall lower milk volume, such as Jerseys, may be more tolerant to OAD milking, possibly due to being able to store more concentrated milk in the udder[3,9]. A subsequent experiment; however, demonstrated that there were no milksolids production differences between Holstein-Friesians with high or low milksolids content when milked OAD, indicating that the difference in OAD milk yield loss between breeds is not solely due to different milksolids content (Dalley pers. comm).

Table 1. Annual milksolids (MS) production for Holstein-Friesian (F) and Jersey (J) cows milked once (OAD) or twice (TAD) daily in a four-year DairyNZ farm systems trial[8].

	FOAD	FTAD	JOAD	JTAD
Cows/ha	3.5	3.0	4.2	3.6
kg MS/cow	237	336	222	278
kg MS/ha	879	1051	979	1045
Days in milk	230	244	229	242

There is limited evidence that parity may be a factor in determining the milk production response to OAD milking. French studies indicated that Holstein-Friesian heifers lost more milk production than older cows[10]. Similarly, DairyNZ data indicated that the milk production drop was 45-55% greater for two- and three-year old Holstein-Friesian cows than mature cows aged four years and older; however, there was no such difference in Jerseys, with similar milk production losses irrespective of age (Table 2)[8]. Milk production differences across parities in OAD and TAD herds registered in the national database are consistent with these experimental results (A. Winkelman, LIC, pers. comm.). Nevertheless, there is no evidence to indicate that milking heifers OAD negatively affects their lifetime milk production potential, as milk production losses in mature cows milked OAD are similar regardless of whether they were milked OAD or TAD as heifers[8].

Table 2. Annual milksolids yield (kg/cow) for different ages of Holstein-Friesian (F) and Jersey (J) cows milked once (OAD) or twice (TAD) daily in a four-year DairyNZ farm systems trial[8].

Cow age	FOAD	FTAD	JOAD	JTAD
2 years	174	280	173	227
3 years	221	345	224	267
4+ years	271	362	247	307

Regardless of breed or parity, the negative effect of OAD on milk production is due to changes in the regulation of genes in the cow's udder that determine milk secretory cell activity and number, and are similar to changes in the udder that occur during drying-off[11]. It is, therefore, no surprise that cows milked OAD for a full season have a lower lactation persistency than cows milked TAD[12] and may be dried-off earlier (Table 1)[7,8] particularly if feed quality or quantity is reduced during mid-late lactation[8]. It appears, however, that the udder "resets" itself during the dry period, as there are no carry-over effects of OAD milking on the ability of the udder to produce milk in subsequent lactations[8,10].

Milking cows OAD also alters milk composition. A common observation is that the concentrations of milk protein and fat are higher, resulting in greater milksolids content, whereas lactose concentrations are lower[3,4,8,10]. Unfortunately, due to the decrease in milk yield, there is still a significant reduction in milksolids yield per cow. Moreover, the increase in protein content in milk is partially due to a greater concentration of less desirable blood proteins leaking into the milk[3, 9] which may affect milk processing characteristics. Nonetheless, secretion of high-value, bioactive milk proteins, such as lactoferrin, is increased, resulting in greater concentrations in OAD milk compared with TAD milk[13]. Thus, changes to milk composition during OAD milking can be both advantageous and detrimental to the manufacturing of dairy products.

It is possible to reduce the on-farm impact of production losses per cow due to OAD milking by slightly increasing stocking rate and/or by selecting high-performing cows tolerant of OAD. Milksolids production per hectare was 6% lower for Jerseys and 16% lower for Holstein-Friesian cows in OAD relative to TAD farmlets when stocking rate was increased by 17% (Table 1)[8]. Increasing stocking rate, however, only makes economic sense if pasture is not limiting[14] as underfeeding OAD cows further decreases their production[4].

(cont'd p10)

(cont'd from p9)

Furthermore, increasing stocking rate may reduce some of the benefits of a whole season OAD system by increasing cow-associated variable costs. Despite the negative effects of OAD on milk production, there may well be other benefits of whole season OAD that outweigh the negative effects of OAD or that reduce the negative impact of other factors (e.g. walking distance, hilly land) on milk production.

Nutritional status

Metabolically the process of synthesising milk is very demanding and requires a significant amount of energy, protein and other nutrients. Thus, it would be reasonable to assume that if cows milked OAD produce less milk, they will require less energy and thus eat less. It is, therefore, surprising that there is very limited information on how OAD milking affects the feed intake of cows. The only accurate feed intake data from cows milked OAD for a whole season are from a French experiment in which measurements were taken for 14 weeks post-calving in fully-fed cows offered a total mixed ration and kept indoors[7]. There was no reported difference in overall feed intake between OAD and TAD milked cows, but during week 7 to 14 post-calving, cows milked OAD ate between 1.3 to 2 kg DM/d less than cows milked TAD, who consumed 22 kg DM/d[7,10]. Feed intakes gradually, but only partially, adapted to the lower nutritional requirements of OAD milking from calving, and it is unknown what happened to dry matter intake in mid- and late lactation.

Under grazing conditions it is difficult to obtain accurate feed intake data for individual cows and, in many studies, the impact of OAD milking on feed intake has been confounded by stocking rate. In a Massey University study[6], where cows were milked OAD or TAD for a full lactation but otherwise managed identically, energy requirements estimated from milk production and changes in live weight indicated that OAD cows ate, on average, 1.4 kg DM/cow/d (15 MJ ME/cow/d) less than their TAD counterparts. A difference of about 1 to 1.5 kg DM intake/d between cows milked OAD and TAD was supported by individual feed intakes calculated from a small number of cows using an intake marker during November (16.6 and 18.1 kg DM/cow/d, respectively) and January (11.3 and 12.3 kg DM/cow/d, respectively)[6]. Further research, however, is required to determine the effect of OAD milking on feed intake at various stages of lactation.

Changes in live weight and BCS may provide an indirect insight into the nutritional status of the dairy cow. Cows milked OAD have an improved energy status and body condition score (BCS) during early lactation, although there is generally no effect on BCS loss until five to six weeks post-calving[4,7,10].

Differences in live weight and BCS between cows milked OAD and TAD gradually increase during the course of lactation[7,8,10].

In one study[6], cows milked OAD gained 40 kg live weight and 1.6 BCS units during the lactation, whereas cows milked TAD lost 17 kg live weight and 0.2 BCS units. A greater live weight and BCS at dry-off relative to TAD milking was also reported in other whole season OAD milking experiments[7,8]. These data indicate that cows milked OAD are able to partition additional energy into BCS and, furthermore, that any reduction in feed intake relative to TAD is not as great during lactation as the reduction in milksolids yield.

A greater BCS during late lactation can enable cows milked OAD to be dried off later and reduce the need for supplement during the dry period, as they do not need to gain as much BCS to meet targets at calving; however, in practice, days in milk may be limited by low daily milk yields as live weight and BCS gain during lactation is inversely proportional to milk production. For example, Holstein-Friesian cows, and particularly heifers, produce substantially less milksolids but gain large amounts of live weight and BCS during whole season OAD milking[8]. Therefore, high-producing cows tolerant of OAD milking (i.e. those that have a smaller production loss) are likely to have smaller reductions in feed intake and lower live weight and BCS gain.

Reproduction

Improvements in energy status and BCS in cows milked OAD for an entire season may enhance reproductive performance. In the four-year DairyNZ trial[8], OAD farmlets used fewer CIDRS and had improved three-week submission and pregnancy rates relative to TAD farmlets, despite having lower pasture allowances due to greater stocking rates. Final in-calf rates were not altered, but the number of days from calving to conception was reduced. Greater numbers of cows are required to assess the impact of whole season OAD on reproduction, but on commercial farms these effects may result in fewer late-calving cows and/or empty cows, less need for hormonal intervention and a tighter calving pattern. If an earlier or more compact calving pattern occurs following several seasons of OAD, care must be taken to ensure that it does not result in cows being underfed due to insufficient pasture (Dalley et al., unpublished data).

Cow health and welfare

Given that milking OAD differs from what is generally considered normal practice, it is important to look at any possible effects on the health and welfare of the dairy cow. An obvious concern with OAD is around the "fullness" of the udder, especially during peak lactation, but research indicates that there is no significant increase in udder discomfort[15, 16].

Although the increased udder firmness did not affect the OAD cows' welfare, one study reported a significant increase in the incidence of milk leaking from the udder prior to milking[15].

Any time the teat orifice is open, the likelihood of bacteria entering the udder is increased; however, a four-year DairyNZ trial demonstrated that there was no increase in the incidence of clinical or subclinical mastitis when cows were milked OAD relative to TAD[17].

Cows milked OAD may exhibit more obvious clinical signs of mastitis if an infection does occur because milk is less frequently "flushed" out of the udder. Dedicated attention to mastitis prevention, detection and treatment is required as OAD milking potentially magnifies existing udder health problems. Therefore, it is generally recommended that steps are taken to reduce clinical and subclinical mastitis in herds (e.g. using appropriate dry cow therapy) prior to switching to whole season OAD.

Adhering to best-practice mastitis management will also help to maintain a low bulk tank SCC in a whole season OAD milking system. Milking cows OAD approximately doubles individual SCC relative to milking TAD[6,8,] regardless of whether cows are uninfected or have subclinical or clinical mastitis[17]. The increase in SCC in mastitis-free cows is not usually detected until after peak lactation and increases relative to TAD during the course of lactation[17]. This may require high SCC cows to be dried off early to prevent exceeding the bulk tank SCC penalty threshold of 400,000 cells/ml.

Milking OAD also means that cows walk shorter distances on farm tracks, which may reduce lameness[18], allow greater time for grazing and possibly reduce heat stress, as they spend less time walking in the afternoon heat and standing in yards[19]. These benefits are likely to be greatest under conditions where cows are required to walk long distances or across hilly terrain to be milked.

Conclusions

There are many reasons for adopting a whole season OAD system, including lower labour requirements, improved animal health, lower farm working expenses, and a more flexible work schedule. However, milksolids production per cow is reduced by about 20 to 30% when cows are milked OAD relative to TAD. Nonetheless, nutritional status, BCS and reproductive performance are likely to be improved. Milksolids production per hectare may be optimised by using high-producing cows suitable for whole season OAD along with a small increase in stocking rate.

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What should I know about Johne's Disease?

Guidelines on Johne's Disease for New Zealand Dairy Farmers

Kaylene Larking, Johne's Disease Research Consortium Manager; Lindsay Burton, Fonterra Risk Management Programme Specialist; Eric Hillerton, DairyNZ Chief Scientist.

Johne's Disease (or paratuberculosis) is a wasting disease that affects cattle, sheep and deer. No cure is known and clinically affected animals die. The disease affects production, by causing reduction in body weight, lower milk yield and losses at calving.

JOHNE'S DISEASE RESEARCH CONSORTIUM

What do I need to know about Johne's Disease?

- The bacterium *Mycobacterium avium paratuberculosis (MAP)* causes Johne's Disease. Infection is spread primarily through contaminated faeces, but also occur in the uterus or via colostrum and milk fed to calves
- Calves and young stock are the most susceptible groups. Control programmes should be targeted at limiting exposure of these animals to MAP
- Once infected, an animal may appear healthy for its entire lifetime, but few will develop clinical disease as adults and become sick and die. This will usually happen one to five years after becoming infected
- It is not fully understood why some animals develop clinical disease and others do not, but the level of exposure to MAP is critical and animal genetics, strain of MAP and stress triggering the onset of clinical disease are all thought to be factors
- As the disease progresses, an animal will normally shed increasing numbers of MAP in faeces, so that animals in the late stages of the disease are the major source of infection. Late stage animals are often known as "super shedders"; releasing up to 10 million bacteria in every gramme of faeces
- The signs of clinical disease are diarrhoea and wasting. Clinical animals will eventually die from dehydration and severe malnutrition unless culled. The intestines of diseased animals are swollen and corrugated. Lesions may also be found in lymph nodes and elsewhere in the body
- MAP in faeces of infected animals contaminate pastures and act as a source of infection for other animals on the property
- Exposure to sunlight will kill MAP on pasture, with contamination largely gone in three months. However, pockets of MAP may survive for up to 18 months in wet and shaded areas
- Many wildlife species, including rabbits, hares and hedgehogs, have been shown to be infected with MAP but their role as a spreader of MAP to domestic animals is unclear.

Why be concerned?

- Johne's Disease has effects on animal welfare and animal productivity
- If unmanaged, the level of infection within a herd may increase
- It has been widely suggested that MAP may have a role in the human disorder Crohn's Disease. Crohn's Disease is a chronic degenerative disease of the human bowel that is not fully understood and has no known cause or no cure. Its symptoms resemble those seen in Johne's Disease in animals. While evidence of an association is increasing no research has proven that MAP causes Crohn's Disease. Whether or not MAP infects and causes disease in humans remains a complex and controversial area of investigation.

Dairy cattle facts

- MAP is reported to be widespread in New Zealand. It is believed that more than 60% of dairy farms have infected animals, but the level of clinical disease continues to remain low in the majority of herds. Disease prevalence is reported to be higher in the South Island of New Zealand than the North Island
- Jersey cows appear to be more susceptible to the disease than Holstein-Friesians
- The onset of clinical signs increases in times of stress, e.g. calving, drought, poor nutrition
- Most MAP transmission occurs from adult infected animals to young calves through the faecal-oral route. The organism is swallowed in manure-contaminated milk, water or feed; or direct ingestion of faeces. MAP is also shed directly into the colostrum and milk of infected dams in later stages of the disease providing a significant route of exposure for calves
- There is no vaccine registered for use in cattle in New Zealand. Control is by preventing exposure in young stock.

Table 1: How common is the disease in New Zealand?

	Herds/flocks with infected stock	Herds/flocks reporting disease
Beef cattle	~31%	~4%
Dairy cattle	~60%	~10%
Deer	~60%	~30%
Sheep	~68%	~20%

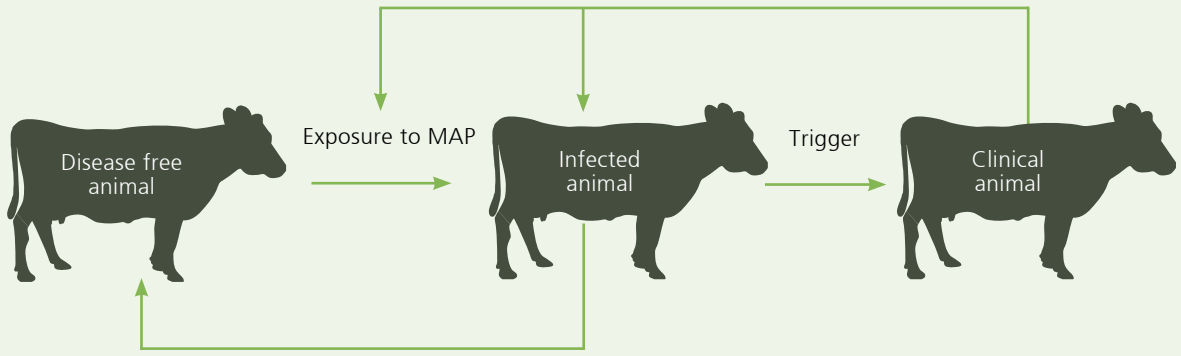
Table 2: Effects on production. Overseas data from studies of **clinically** affected cows have reported the following effects.

Parameter	Impact of clinical Johne's Disease
Milk production	Losses average 15%
Slaughter value	Reduced by 20-30% with clinical disease
Slaughter weight	Reduced by ~60 kg in infected animals
Life expectancy	~5 years compared to ~7.5 years for uninfected cows
Feed conversion efficiency	Reduced from ~60 to ~40%
Other	Longer calving intervals and more infertility. Frequent mastitis

(cont'd p14)

(cont'd from p13)

Figure 1: How Johne's Disease spreads



How do I know if I have Johne's disease in my herd?

If you have adult cattle that are scouring or continue to lose weight even with adequate nutrition, it is possible that Johne's Disease may be the cause.

Always work with a veterinarian to confirm the diagnosis. Testing is of little value in younger animals but blood, milk or faecal testing can be used to determine the presence of MAP in adult animals. The disease is most effectively detected in clinical animals or post mortem. None of the tests are 100% accurate and may not always be needed depending on what you are looking to achieve in your herd. Veterinarians will be able to advise which tests are most suitable for diagnosing the disease.

What do I do? I am concerned about Johne's Disease

Eradication of Johne's disease is not feasible but a risk management approach should be taken to minimise the infection rate.

The following are suggested as controls to aid in reducing the impact of Johne's Disease on high risk farms with advanced MAP infection (high test positive herds):

- Know the status of the herd by using tests to identify positive cows and "super shedders"
- Manage test positive cows to minimise the spread of infection. It is recommended not to breed from high test positive cows and that they should be isolated from young cattle

- Remove super shedders from the herd by culling
- Calves are most susceptible to infection in the first six months of life and this decreases with age. They should be removed from dams within 12 hours and not graze pastures grazed by adult cattle or where effluent is spread
- Make sure colostrum and milk from high test positive cows are not fed to calves
- Use grazing management to reduce exposure. Avoid set stocking calves on the main areas of the dairy farm grazed by adult stock and areas where effluent is routinely spread.

I do not have Johne's Disease in my herd

MAP is widespread in NZ, so even if Johne's Disease is not apparent in the herd, there are some practical steps that you should take to reduce the chances of Johne's Disease affecting your livestock:

- Apply best management for calves and young stock
- Avoid purchasing from high prevalence herds
- Watch for clinical signs and isolate stock that are unwell
- Animals with clinical Johnes Disease should be culled.

Remember that eradication is technically very difficult, if not impossible in most situations, but application of good herd risk management practises can keep the incidence of clinical disease and infection rates at low levels within the herd.

This information represents the best advice currently available for controlling the spread of Johne's Disease in dairy cattle under pastoral grazing conditions in New Zealand.

Further technical information and any new developments can be found by visiting the Johne's Disease Research Consortium website **www.jdrc.co.nz**.

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Focus on international research

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

Kronqvist and others (2012) Relationship between incidence of milk fever and feeding of minerals during the last 3 weeks of gestation. *Animal 6: DOI:10.1017/S175173111200033X.*

This study evaluated whether differences in milk fever incidence among Swedish dairy herds could be explained partly by differences in mineral feeding during the last weeks of gestation. Increasing dry matter intake pre-calving was associated with an increased risk of milk fever. Dietary calcium and phosphorous content did not affect incidence of milk fever but there was evidence that increased potassium may increase the risk. They recommended increasing magnesium intake pre-calving.

DairyNZ comment: These data are consistent with New Zealand data. Cows at BCS 5.0 a month pre-calving are less likely to get milk fever if fed slightly less energy than requirements (approximately 20%) and magnesium is the most important mineral in preventing milk fever.

O'Brien and others (2012) Evaluation of the effect of accounting method, IPCC v. LCA, on grass-based and confinement dairy systems' greenhouse gas emissions. *Animal 6: DOI:10.1017/S1751731112000316.*

Life cycle assessment (LCA) and the Intergovernmental Panel on Climate Change (IPCC) guideline methodology were evaluated using a dairy farm greenhouse gas (GHG) model. The model was applied to estimate GHG emissions from a seasonal calving pasture-based dairy farm and a total confinement dairy system, where cows were fed a total mixed ration. The IPCC method indicated that the cows in the confinement system produced 8% less GHG emissions per unit of product compared with the pasture-based system, but the LCA approach predicted that the confinement system increased emissions by 16% when off-farm emissions associated with primary dairy production were included. Their modelling also suggested that changes to a single factor had little impact on GHG emissions, but that multiple changes could have large effects (15-30% reduction in GHG). Systems that reduced GHG/ha did not necessarily reduce GHG/kg milksolids, and vice versa.

DairyNZ comment: Full life-cycle analysis is the preferred approach to assess the effect of management changes on GHG emissions, as this accounts for all contributors to GHG emissions.

Soberon and others (2012) Pre-weaning milk replacer intake and effects on long-term productivity of dairy calves. *Journal of Dairy Science 95:783-793.*

The effect of calf pre-weaning growth rate and milk production was investigated in 1,868 heifers. Increasing pre-weaning average daily gain from 0.5 kg/day to 1.0 kg/day increased post-calving milk production by 4% in cows from research and commercial herds (i.e. 16 kg milksolids/heifer/year from a herd averaging 400 kg milksolids/cow). For every 10 MJ metabolisable energy consumed above maintenance per day pre-weaning, heifers produced 5% more milk. They also reported a 1% increase in heifer milk production if ambient temperature at birth was increased 5^{o}C, highlighting the importance of providing shelter for calving cows.

DairyNZ comment: These data highlight the importance of pre-weaning liveweight gain on subsequent heifer milk production. These results need to be validated in New Zealand dairy systems but highlight the importance of adequate colostrum and shelter for new-born calves and, potentially, the importance of pre-weaning feeding level on subsequent milk production.

Rajala-Schultz and others (2011) Milk yield and somatic cell count during the following lactation after selective treatment of cows at dry-off. *Journal of Dairy Research 78:489-499.*

Selective dry cow therapy (DCT) has received increasing attention in recent years owing to concerns regarding agricultural use of antibiotics and the development of antimicrobial resistance. This research from the USA evaluated the use of selective dry cow treatment (SDCT) on subsequent milk production and somatic cell count (SCC). In four commercial herds, cows with low SCC at the end of lactation and no history of clinical mastitis randomly received DCT or not. On average, DCT reduced SCC the following lactation (~35,000 cells/mL), but the effect varied between farms. Similarly, the positive effect of DCT on milk yield varied between farms. Results suggest that although selective dry cow therapy could be effective on some farms, other farms benefited from whole herd DCT.

DairyNZ comment: This study highlights the lack of predictability about the outcome of selective DCT across different herds. These herds had a low prevalence of contagious and environmental bacteria, so the risk of new infection was relatively low. In New Zealand, farmers and vets are encouraged to develop a DCT strategy that is appropriate for an individual herd and its level of risk for mastitis.