A Life Cycle, Lesion Oriented Approach to Lameness Control

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Introduction

We have a global lameness crisis in our dairy industry. The worldwide prevalence of lameness in dairy herds (defined as a cow walking with noticeable weight transfer and a 'limp') is approximately 24% across studies based in Austria, Canada, China, Finland, Germany, Italy, Netherlands, New Zealand, Norway, UK and the US (e.g., Amory et al., 2006; Barker et al., 2010; Chapinal et al., 2014; Cook, 2003; Cook et al., 2016; Dippel et al., 2009; Fabian et al., 2014; Kielland et al., 2009; Popescu et al., 2014; Sarjokari et al., 2013; von Keyserlingk et al., 2012), with a trend toward lower prevalence in grazing or mixed housing and grazing systems (e.g. 16.5% in Amory et al., 2006; 8.3% in Fabian et al., 2014; and 15% in Haskell et al., 2006), and a higher prevalence in confinement housed freestall herds (e.g. 31% in Chapinal et al., 2014; 54.8% in North East US dairy herds in von Keyserlingk et al., 2013).

Despite research and a significant improvement in our understanding of the causes of lameness over the last three decades, we appear to be fighting a losing battle, and the problem has been associated with increasing intensification of the dairy industry, higher milk production and confinement housing, with the obvious conclusion that lameness is an inevitable consequence of these decisions. Consumers carry an expectation that cows should graze and appear to place considerable value on cattle having access to the outdoors, where they have fresh air and freedom to roam (Cardoso et al., 2016). They emphasize the need for humane care of the animals (Cardoso et al., 2016), so the sustainability of the industry is threatened when the general public learns that production systems do not meet their expectations – and lameness is an obvious problem that has been and should continue to be a high priority for us to resolve.

What Causes Lameness in Dairy Cattle?

The aetiopathogenesis of a variety of hoof lesions has been researched and reviewed extensively (eg. Bicalho and Oikonomou, 2013; Cook and Nordlund, 2009; Cook, 2015; Evans et al., 2015), centering on genetic, nutritional, hormonal, mechanical, infectious and environmental factors.

Across numerous surveys in different production systems, three lesions emerge consistently as the most significant contributors to lameness – digital dermatitis (DD), white line disease (WLD) and sole ulcer (SU) (eg. Barker et al., 2009; Defrain et al., 2013). Our ability to impact lameness globally will depend on developing effective control strategies targeted at these three lesions. I will concede that some differences do exist between production systems and some countries. For example, DD has yet to become a dominant hoof lesion in New Zealand and Australia, likely due to the absence of environmental risk factors. However, the disease has spread in association with confinement housed dairy systems around the world, and now even in these locations DD is appearing at a low prevalence (Chesterton, 2015). In grazing systems, WLD appears to dominate, with sole bruising and axial wall fissures often reported. It is however important to note that a healthy sole is unlikely to 'bruise' unless the sole thickness is compromised, suggesting this as an underlying cause. We know thin soles emerge as a significant problem in larger confinement dairy systems in association with toe ulceration (Shearer et al., 2006), where cows are asked to walk long distances to and from the parlor for milking. It is therefore likely that hoof wear is the underlying issue in both, due to exposure to the track (grazing herds) or concrete alley (confinement herds).

I will contend that we now know more than ever what causes lameness, and while we still have more to learn, we know enough currently to solve the global lameness problem in our dairy industry.

A Life Cycle Approach

No matter what the causation of lameness, once the cow develops a lesion, they are at much greater risk for developing the same lesion again in the next lactation (Oikonomou et al., 2013), likely due to permanent anatomical changes to the structure and function of the claw – including the fat pad, the suspensory apparatus and the pedal bone itself (Table 1).

We are also aware that while claw horn disease is relatively uncommon in heifers, DD infection may impact 20-30% of heifers after breeding age in many rearing facilities, likely as a result of the same poor leg hygiene risk factors that have exacerbated the problem in mature cows. Laven and Logue (2007) and Holzhauer et al. (2012) have demonstrated the importance of the pre-partum period in affecting DD occurrence during the following lactation, and Gomez et al. (2015) were able to demonstrate increased risk for DD in primiparous cows when they suffer one or more episodes of DD during the rearing period, compared to heifers that are unaffected during the rearing period.

DD affects younger cows, with incidence peaking typically in the 1st or 2nd parity, while SU and WLD incidence increases with age to around the 4th lactation (Oikonomou et al., 2013).

These data therefore support an approach to lameness control that encompasses the life-cycle of the cow, starting during the heifer rearing period, with strategies that are lesion specific and age-specific, tailored to the type of lesions that are most prevalent on each farm. Understanding the motivation for farmers to implement change is critical for consultants (Leach et al., 2010). However, it would seem likely that with the growth and expansion of welfare audits globally, they will have little choice but to comply. Ultimately, producers that have succeeded in their control of lameness will become the best salesmen of prevention programs to the others that lag behind, and these producers will increasingly need an effective roadmap to expedite change.

Herd Risk Factor Oriented Strategies

Herd level risk factors for lameness have been studied in multiple countries and in a variety of production systems in recent years. A number of consistent findings have emerged from these studies. Factors which appear to be associated with lower lameness risk include; less time standing on concrete (Bell et al., 2009), deep bedded comfortable stalls (Chapinal et al., 2013; Cook, 2003; Dippel et al., 2009; Espejo et al., 2006; Rouha-Mulleder, et al., 2009; Solano et al., 2015), access to pasture or an outside exercise lot (Chapinal et al., 2013; Hernandez-Mendo et al., 2007; Popescu et al., 2013; Rouha-Mulleder, et al., 2009), prompt recognition and treatment of lameness (Barker at al., 2010), higher body condition score (Dippel et al., 2009; Espejo et al., 2006 Randall et al., 2015), use of manure removal systems other than automatic scrapers (Barker at al., 2010), use of non-slippery, non-traumatic flooring (Barker et al., 2010; Sarjokari et al., 2013; Solano et al., 2015a), use of a divided feed barrier (rather than a post and rail system), with a wider feed alley (Sarjokari et al., 2013; Westin et al., 2016).

It should be expected that routine professional hoof-trimming, access to a trim-chute for treatment and use of an effective footbath program would deliver improvements in lameness (eg. Pérez-Cabal and Alenda. 2014), but these effects are often confounded in associative observational studies (eg. Amory et al., 2006). It is also true that many poorly trained hoof-trimmers cause more harm than good, and many footbath routines are ineffective through poor design and management (Cook et al., 2012; Solano et al., 2015b). Similarly, several studies point to restrictive neck rail locations, high rear curb heights, and lunge obstructions as risk factors for lameness (eg. Chapinal et al., 2013; Dippel et al., 2009; Rouha-Mulleder, et al., 2009; Westin et al., 2016), however correct neck rail location and curb height is stall design specific and care should be taken in interpretation of these findings. Most recently, stall width has emerged as a significant factor impacting lameness (Westin et al., 2016)

High Production and Low Levels of Lameness

While we know that Holstein cows are perhaps more susceptible to lameness (eg. Sarjokari et al., 2013), and there appears to be a genetic component to the development of DD, SU and WLD, with a link to higher milk production (Oikonomou et al., 2013), I do not believe failure is inevitable.

We had the opportunity to visit 66 high performance Wisconsin herds that have been implementing strategies to prevent lameness for over a decade (Cook et al., 2016). These herds had a mean herd size of 851 cows, were confinement housed in freestalls and produced more than 40 kg energy corrected milk per cow per day on average. The prevalence of clinical lameness averaged 13.2% - which would rival the degree of lameness identified in grazing herds (e.g. 8.3% as reported by Fabian et al., 2014), and mixed housing and grazing or organic management systems elsewhere (e.g. 16.5% in Amory et al., 2006; 17.2% in Rutherford et al., 2008). Interestingly, it is lower than the prevalence found in similar herds in the Midwest a decade or more ago (e.g. 22.5% in Cook, 2003; 24.6% in Espejo et al., 2006), suggesting that the overall degree of lameness in the region may be improving. Severe lameness was also uncommon at a mean of 2.5%. This average is lower than that found in the majority of previous freestall surveys (e.g. 5.3% in Barker et al., 2010; 16% in Dippel et al., 2009; 4.8% in Husfeldt et al., 2012). Thus it would appear that high performance can be compatible with ac-

ceptable lameness levels, if we manage cows correctly. Table 2 highlights some of the management characteristics of these herds pertaining to lameness management.

When examining the management strategies with high levels of adoption in Table 1, there are consistencies with the herd level risk factors documented previously. These herds use deep loose bedded stalls, have 2-row pen layouts with headlocks, have solid flooring with strategic use of rubber floors, especially around the milking center. Notably, these herds were not using rubber flooring in their pens to control lameness. They clean manure from the alleys when the cows are outside the pen, and have aggressive hoof care, heat abatement and footbath programs. Two thirds of herds use rBST and milk three times daily, and perhaps surprisingly, 9% let their high producing cows outside the barn strategically – not to graze, but to spend time away from concrete floors inside the barn. In a multivariate model, deep bedded stalls, pasture access and fewer cows per FTE worker significantly reduced the risk for lameness overall.

Lameness management will continue to be refined, but these herds prove that we know enough right now to implement positive change in the dairy industry and achieve acceptably low levels of lameness, even in cattle which may be inherently more susceptible.

A Structured Approach to Lameness Prevention

When troubleshooting lameness problems, I use a structured approach starting with locomotion scoring, lesion analysis and assessment of the routine hoof-trimming and lame cow surveillance program. It is essential that the hoof-trimming is a component of prevention rather than a risk factor in itself. I then examine the risk factors for each of the key hoof lesions and finish with a review of feeding practices. From this examination, we can create a herd specific action plan designed to maximize impact on the key hoof lesions on the farm.

For DD prevention, we focus on the early identification of acute lesions (before the cattle are lame) and prompt effective treatment, starting around breeding age in replacement heifer pens and continuing throughout the life of the animal, coupled with an effective footbath program to control the chronic lesions and hold them in check. Trace mineral supplements have a significant role to play, particularly during the rearing period. For SU prevention we target risk factors that extend daily standing times – stall design and surface cushion, stocking density, milking times, heat abatement and lock up time for management tasks. We optimize the transition period to maximize rest and reduce BCS loss in early lactation. Finally, for WLD control, we examine areas of the farm where flooring puts the cow at risk of slipping, trauma and excessive hoof wear, and watch workers to ensure low stress handling – especially around the parlor operation.

The overall approach is summarized in Figure 2. Each assessment results in a problem list which can then be used to develop a targeted action plan for the herd.

Conclusion

In this article, I have made the case, that while we still have knowledge gaps to fill in our understanding of lameness, the global crisis that we face with 1 in 4 cows walking with a painful limp can be solved by implementing our current knowledge targeted at the key hoof lesions; DD, WLD and SU. The challenge we face is one of creating a simple roadmap targeted at an individual producers most significant problems and motivating that producer to implement the changes necessary. Dairy producers that have already achieved success in lameness prevention will serve an important role motivating others to follow in their foot-steps.

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Figure 1. Worldwide prevalence of lameness in dairy herds by location from the peer reviewed literature since 2003

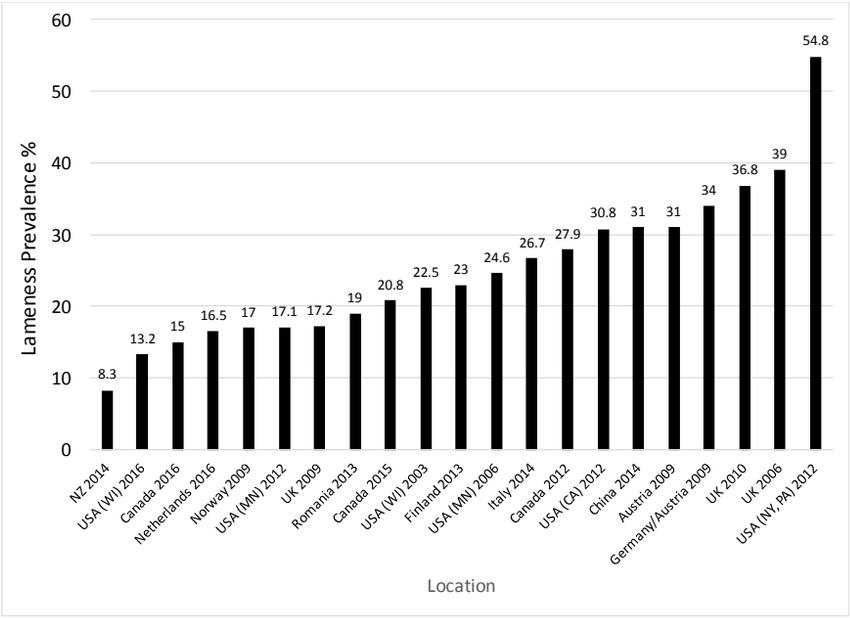

Figure 2. Herd lameness troubleshooting plan

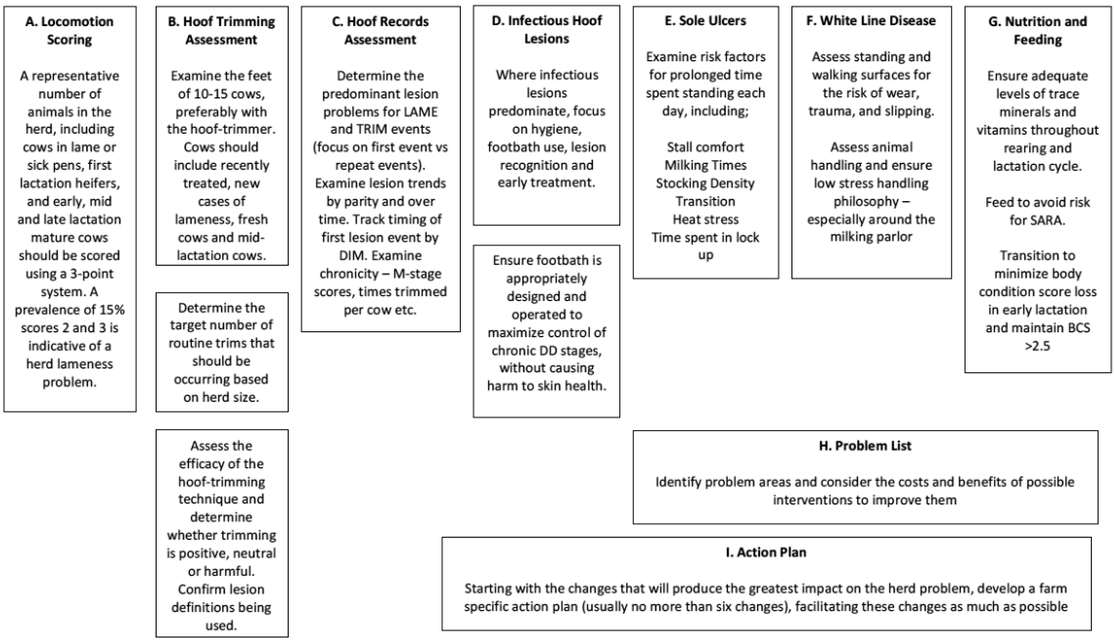

Table 1. Lactation adjusted incidence of lameness lesion (white line disease = WLD, sole ulcer = SU and digital dermatitis = DD) by lesion status (0 = no lesion, 1 = lesion) in the previous parity (1-3). (from Oikonomou et al., 2013)

Lesion	Parity	Lesion Status	Lactation Adjusted Incidence			P-value
			2	3	≥4	
WLD	1	0	6	11	15	<0.01
		1	20	21	24	
	2	0		9	13	<0.01
		1		20	18	
	3	0			10	<0.001
		1			21	
SU	1	0	12	20	26	<0.001
		1	44	32	23	
	2	0		15	24	<0.001
		1		40	30	
	3	0			18	<0.001
		1			41	
DD	1	0	7	7	8	<0.001
		1	32	15	10	
	2	0		5	7	<0.001
		1		19	12	
	3	0			5	<0.001
		1			14	

Table 2. Management characteristics of the high producing multiparous group cows in elite housed dairy herds in Wisconsin (from Cook et al., 2016).

Management Characteristic	***% Herds or Mean***
% Sand bedded stalls (deep loose bedding including manure solids)	62 (70)
% 2-row stall layout pens (vs 3-row)	61
% Use of headlocks at the feedbunk	83
Milking Frequency (% 3 times a day)	67
% Use of rBST	67
% Solid floor (vs slatted)	100
% Rubber floors in freestall alleys	5
% Rubber floors in transfer lanes	15
% Rubber floors in holding areas	41
% Rubber floors in parlors	68
% Manual manure cleaning from the alleys	73
% Use of fans over the resting area	96
% Use of water soakers in the pens	79
% Allow access to the outside to roam	9
% Trimming at least once per lactation	88
% Trim cows at least twice per lactation	65
% Trim heifers before calving	49
Mean footbath frequency (milkings per week)	4.5
Mean cows per full time equivalent (FTE) worker	62