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Review

A descriptive review of the prevalence and risk factors of hock lesions in dairy cows

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ABSTRACT

This article reviews the literature on hock lesions in dairy cattle, focusing in particular on their prevalence and associated clinical signs, as well as the scoring systems used to assess them and the data on risk factors. This analysis was limited to hock lesions where there was inflammation and damage of the skin and the subcutaneous tissue only without involvement of the joint.

The presence of hock lesions, or tarsal peri-arthritis, is strongly related to time spent lying on abrasive surfaces, prolonged high local pressure or friction of the hock on hard surfaces, and collisions of the hock with cubicle fittings. Since hocks have almost no fatty tissue or muscles between the bones and skin, there is no protection against these types of trauma and skin damage occurs (resulting in hock lesions). The risk of these lesions becoming infected is strongly dependent on the hygiene of the lying area.

The prevalence of hock lesions in dairy cows is generally reported as high (>50%). As hock lesions are often correlated with lameness, they are associated with economic losses and impaired welfare, as well as negative societal perception of the dairy sector. Alterations in cubicle characteristics, bedding material, pasture access and lameness prevention may all lower the prevalence of hock lesions; nevertheless, the actual relationship between housing design and other cow- and management-related risk factors on the occurrence of hock lesions appears to be complex and interrelated.

Introduction

Hock damage of dairy cattle is a problem in many herds. The term 'hock lesions' describes multiple clinical presentations of hock damage, which may vary in severity from mild hair loss to ulceration and swelling (Laven and Livesey, 2011). Although the term includes more than just cases with damaged skin and subcutaneous tissue (i.e. tarsal peri-arthritis or peri-tarsitis), this review focuses principally on those cases.

The aetiology of hock lesions is not yet fully understood, although a deteriorative process from hair loss to ulceration and from ulceration to swelling is assumed. Many factors, such as lameness, lack of grazing and low levels of cubicle bedding, have been reported as having a significant influence on the prevalence of hock lesions, but published research is often contradictory. Clearly, as hock lesions have serious economic and welfare consequences, are not always easy to cure, and reduce the longevity of dairy cows (Barberg et al., 2007), we need to better understand their aetiology, control and prevention.

Our aim here is to review the literature on hock lesions with particular attention to the published findings on their prevalence, the associated clinical signs, their causes and the scoring systems used. We also give an overview of the key risk factors associated with hock lesions categorised as cow-, management- and housing-related factors, in order to create more awareness, a better understanding, and effective prevention of the condition.

We have used peer reviewed papers published after 2000 in English, one German written paper from Switzerland, a paper in Dutch about advised cubicle dimensions, a recent abstract from a conference proceeding and various cited anatomy and lameness textbooks.

Causes and consequences of hock lesions

Damage to the hock region is the most commonly recorded skin injury in the dairy cow (Veissier et al., 2004). It is generally assumed that hock lesions arise from the animal lying on abrasive surfaces, prolonged high local pressure or friction of the hock on hard surfaces (such as a curb), or collisions of the hock with cubicle fittings (Van Amstel and Shearer, 2006; Brenninkmeyer et al., 2012; Nuss and Weidmann, 2013). Such damage can occur, for example,

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Table 1
Overview of studies reporting cow-related risk factors for hock lesions.

Cow-related factors	Clinical presentation	Comparison	Effect measure	*P*-value	Reference
Lameness	Hair loss and ulceration	Locomotion score 3 vs. 0	OR = 1.65 (hair loss) OR = 1.93 (ulceration)	≤0.05	Potterton et al., 2011a
	Hair loss, ulceration and swelling	Lame vs. normal gait	OR = 1.21	≤0.05	Burow et al., 2012
	Scabs, wounds and swelling	Association between hock lesion prevalence and lameness prevalence	$r = 0.48$	≤0.0001	Brenninkmeyer et al., 2012
	Hair loss, swelling, wounds	Lame vs. not lame	OR = 5.76	≤0.05	Kielland et al., 2009
	Hair loss	Lame and recovered vs. non lame Lame vs. non-lame	OR = 8.65 OR = 7.01	≤0.001	Lim et al., 2013
Age/parity	Hair loss and ulceration	Lactation no <5 vs. ≥5	41 vs. 50%	≤0.001	Rutherford et al., 2008
Age at insemination	Hair loss and ulceration	<14 vs. ≥16.5 months	54.5 vs. 24.9%	≤0.05	Rutherford et al., 2008
BCS	Hair loss, swelling, wounds	Continuous	OR = 0.65	≤0.001	Kielland et al., 2009
	Swelling	3.5–4.5 vs. 1–1.5	OR = 0.56	≤0.05	Potterton et al., 2011a
	Hair loss, swelling, ulceration	Higher BCS	$r = 0.4$	≤0.01	Regula et al., 2004
Cow size	Swelling	Hip distance <50 vs. >55 cm	13 vs. 31%	≤0.01	Haskell et al., 2006
Cleanliness	Ulceration	Score dirty vs. clean	OR = 0.76	≤0.05	Potterton et al., 2011a
Milk yield	Hair loss and ulceration	<7108 vs. ≥ 8548 L	28.8 vs. 50.4%	≤0.05	Rutherford et al., 2008
	Hair loss and ulceration	33.9–58.1 vs. 2.4–20.7 kg	OR = 1.55 (hair loss) OR = 1.31 (ulceration)	≤0.05	Potterton et al., 2011a
Days in milk	Swelling	270–979 vs. 2–101 days	OR = 1.84	≤0.05	Potterton et al., 2011a
	Hair loss, swelling, wounds	30–59 vs. ≥270 days	OR = 0.57	≤0.01	Kielland et al., 2009
Holstein breeds	Hair loss	No Holstein vs. (partly) Holstein	OR = 0.48	≤0.05	Potterton et al., 2011a
Cow-related factors	Hair loss, swelling and lesions	Danish Holstein vs. other breeds	OR = 1.61	≤0.01	Burow et al., 2012

BCS, body condition score.

because cubicles are too narrow or because bedding is too hard or too rough (Brenninkmeyer et al., 2012). Additionally, if protrusions are present on the lying surface, hock lesions will develop when the skin rubs against them (Kielland et al., 2009).

Freedom from pain, injury and disease is one of the internationally-recognised 'Five Freedoms' so hock injury status is often included in welfare assessment protocols (Burow et al., 2012). Indeed, as injuries due to housing systems occur mostly around the hock (Rutherford et al., 2008), the prevalence of hock injuries may be a fair indication of the degree of discomfort induced by the housing system. There is also a close link between hock lesions and lameness (Table 1; Brenninkmeyer et al., 2012; Burow et al., 2012; Lim et al., 2013), so reducing hock lesions may also reduce lameness.

In addition, positive correlations between hock lesions and other health problems have been reported. These include: callosities at the carpal joint ($r = 0.6$), teat injuries ($r = 0.2$), skin injuries other than hocks and teats ($r = 0.3$), and somatic cell count ($r = 0.3$) (Regula et al., 2004; Fulwider et al., 2007). Nevertheless, the specific impact of hock lesions on welfare is unclear because the scores for the clinical presentations are usually combined in studies and little account is taken of severity and duration. Similarly, although it is clear that hock lesions indirectly cause economic losses, principally via their relationship with lameness (Brenninkmeyer et al., 2012), there are no published data on the specific costs of hock lesions.

Clinical signs and pathology

The term 'hock lesions' includes the clinical presentations of hair loss, swelling, and skin lesions and ulceration (Laven and Livesey, 2011). Although a process of development from mild to severe lesions seems logical and is often assumed, it is, however, still unproven. Potterton et al. (2011a) evaluated many potential risk factors for hock lesions and found that none were common to all three presentations (i.e. hair loss, ulceration, and swelling). The mildest stage (hair loss) and the most severe stage (swelling) had only one risk factor in common (cubicle-bedding material). As a result, Potterton et al. (2011a) concluded that ulceration was not a direct extension of hair loss and that the underlying aetiology and development of hock lesions differed between all three presentations.

Despite the findings of Potterton et al. (2011a), the clinical presentations are still most commonly described as a progressive process (Nuss and Weidmann, 2013) where hair loss is designated as the first indicator of abrasion, and where further abrasion may lead to haemorrhages, swelling and scabs (Livesey et al., 2002). The non-infectious inflammation (hair loss) occurs due to repeated pressure or friction of the hock on abrasive surfaces. This results in hairless areas of skin over the bony extremities of the hock and localised swelling of the skin. If no visible entry points on the skin occur then this process can continue for a long period. When penetration of the skin occurs, an access point for bacteria to invade the existing inflamed area can arise, shifting the infection from non-bacterial to bacterial (Van Amstel and Shearer, 2006; Aiello and Moses, 2010). Infectious inflammation occurs under non-hygienic circumstances when the abraded skin and wounds on the hock become necrotic and subcutaneous tissue becomes colonised by bacteria (Brenninkmeyer et al., 2012).

When the process is restricted to skin inflammation, hock lesions will result in mild lameness only. In fact, when such lesions are bilateral, gait may not be affected at all (Aiello and Moses, 2010; Budras et al., 2011). When the lesions are accompanied by severe swelling or become infected and suppurating, severe lameness can be observed (Brenninkmeyer et al., 2012).

Hock swelling predisposes to arthritis (Aiello and Moses, 2010). In the most serious cases tarsal peri-arthritis can manifest itself as a serous arthritis or 'puffy hock'; this is a severe inflammation with excessive fluid in the tarsocrural joint but is rarely seen. In such cases, the skin on the joint itself is swollen and warm and, in the most serious stage, synovial effusion can be noticed. A reduction in the use of the joint can be noted, although the degree of lameness varies greatly according to severity and accompanying swelling and pain (Whay, 2002). Septic arthritis, where bacteria invade the local joint, results in villous hypertrophy of the joint capsule and fibrin deposits. In chronic cases, degenerative joint disease (DJD) may be present (Van Amstel and Shearer, 2006). Septic arthritis is accompanied by severe swelling with peri-articular inflammation, general malaise and severe to extreme lameness (Budras et al., 2011).

Other complications of hock lesions can be quite diverse. Systemic spread of the infection via the blood or lymph may be more common than tarsal arthritis. This can result in diseases such as