THE EPIDEMIOLOGY AND CONTROL OF LAMENESS IN PASTURE-FED DAIRY CATTLE

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Abstract

A series of studies was conducted on lameness in dairy cattle in New Zealand. All cases of lameness that occurred in three dairy herds over a 12-month period were studied to establish the types of lesions that commonly result in lameness, and to monitor the recovery process. White line disease (39%), traumatic pododermatitis or sole bruising (42%) and septic pododermatitis or sole abscess (9%) were the most common lesions. There was a close association between the onset of lameness and time since calving, regardless of season of calving. Lameness was associated with wet weather conditions. Total lactation yields of the lame cows were significantly less than matched herd-mates of similar age and calving date.

A longitudinal study of the feet of cows from a spring calving dairy herd with a low incidence of lameness (2% over 12 months) was conducted over a 12 month period to measure the occurrence of subclinical hoof lesions. Sole haemorrhage, erosion of the heel bulb and minor white line separation were the most commonly observed lesions. Waves of each type of lesion affected different digits and passed through the herd at specific times of the year and breeding season.

Methods were developed for assessing physical hoof properties such as hardness, moisture content, elastic modulus, compressive strength, resilience and sole concavity. A series of epidemiological studies was conducted to determine to what extent these properties varied between digits on different cows, between digits on the same cow, with changes in environmental moisture conditions, and with changes in husbandry over the course of a dairy herd's production year. There was a clear tendency for hoof moisture to vary in concert with various measures of environmental moisture and for the other physical hoof properties measured to be affected by hoof moisture content.

Hoof growth and wear studies were also conducted on both autumn- and spring-calving cows. Rates of hoof wall growth were lower in autumn and winter than during spring and summer and were greater in 2-year-olds than in mature cows. Both wall wear and sole wear were greater in lateral digits than in medial digits. Lateral digits almost always had less sole concavity than medial digits. Cows suffered a rapid and substantial loss of sole concavity following calving regardless of season of calving. Lame digits tended to

have less sole concavity than non-lame digits, providing some evidence that lack of sole concavity is causally associated with occurrence of lameness.

The effect of two different surface hoof treatments (daily formalin footbathing and bimonthly Hoof Bond application) on hoof characteristics and occurrence of lameness was examined. Both treatments failed to prevent lameness, reduce the incidence of subclinical hoof lesions or substantially alter hoof moisture, hardness or sole concavity changes.

A tentative hypothesis is proposed to explain the well recognised association between lameness onset and rainfall. Mechanisms involved in the development of common claw lesions in pasture-fed cattle are discussed.

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CHAPTER 1

Introduction

Introduction

Lameness is one of the most common diseases of dairy cattle and is a major cause of economic loss to farmers. However, as is discussed in the review of the literature that follows, despite its importance, it has been inadequately researched. Further, most work that has been done has been conducted in the northern hemisphere under systems where cows are housed and are fed large amounts of concentrates. Relatively limited work has been performed in Australia and New Zealand.

In an attempt to answer questions relating to the epidemiology of lameness in pasture-fed dairy cattle and to assist with development of methods for prevention and control a series of studies was conducted in New Zealand. Initially a case study of lameness in three dairy herds was performed to establish the types of lesions that commonly result in lameness, and to monitor the recovery process. Risk factors possibly associated with lameness were also investigated. At the same time a longitudinal study of the feet of cows from a large seasonal dairy herd was conducted over a 12 month period to determine the incidence and prevalence of subclinical hoof lesions and to assess the association of such subclinical lesions with clinical lameness.

Because there is general belief that lameness in pasture-fed cattle is largely caused by traumatic injury to the hooves, studies were then undertaken to ascertain if variation in the physical nature of cows' hooves is associated with their ability to resist external damaging influences. Methods were developed for assessing physical hoof properties such as hardness, moisture content, stiffness, compressive strength, resilience and sole concavity. In a longitudinal study using 10 cows, the effect of environmental moisture and of changes in husbandry over the course of a dairy herd's production cycle on these physical hoof characteristics was examined. In another study, a case-control design was chosen to determine which, if any, of these physical hoof properties might be associated with an individual cow's susceptibility to lameness.

Finally, attention was directed to methods that might be used to modify the susceptibility of hooves to trauma and penetrating injury. The ability of surface hoof treatment to alter physical hoof characteristics and occurrence of lameness was evaluated.

Because this work was done as a sequence of studies, all of which are either in the process of publication or have already been published, this thesis is presented in the form of a series of papers. References have been combined into a single bibliography.

CHAPTER 2

Literature review

Introduction

Lameness remains one of the most important health problems of dairy production. Financial losses resulting from lameness are substantial and arise in a variety of ways (Weaver, 1984; Politiek *et al.*, 1986; Esslemont, 1990). Milk production from affected cows is often reduced, reproductive performance can be lowered and the value of cows culled because of lameness may be affected by lower cow weights at slaughter. Involuntary culling as a result of lameness can reduce the genetic level in the herd as the selection pressure for production traits is reduced. If lameness results in higher culling rates, there are extra costs associated with rearing extra replacement heifers. The cost of veterinary attention and treatments, although usually only a small proportion of total costs due to lameness, can be considerable. Milk discarded when antibiotics are used represents a further loss. Treatment and management of lame cows by the herdsman also involves a substantial labour input and does have an opportunity cost that cannot be ignored.

Various workers have attempted to estimate the financial effects that can be attributed to lameness. Whitaker *et al.* (1983) reported that the average annual financial loss due to hoof problems in the United Kingdom amounted to £1175 for every 100 cows. At a national level, estimates of loss for the British dairy industry have ranged between £15 million and £89 million per year (Baggott and Russell, 1981; Pinsent, 1981; Whitaker *et al.*, 1983; Booth, 1989; Esslemont, 1990). Digital disease in Canada is estimated to cost $10million per annum (Choquette-Lévy *et al.*, 1985).

Little reliable information on financial effects of lameness is available for Australia and New Zealand. Dewes (1978) reported that lame cows in two herds had shorter lactations than non-lame cows and their production over one lactation was approximately half that of non-lame cows in the same herds. The number of animals in each herd is not stated. In a more recent Australian study, Harris *et al.* (1988) estimated the mean cost due to loss of production, treatment, the culling of lame cows, and the extra man hours required to manage lame cows as $42.90 per lame cow. These figures did not take into account possible reduced fertility, the cost of bail feeding, track construction and maintenance or other factors influencing lameness. In this study the extent of loss of milk due to a lower level of milk production in lame cows and the amount of milk withheld from sale because

of possible antibiotic contamination following treatment was based solely on estimates made by participating farmers.

From the findings of Collick *et al.* (1989) and Lucey *et al.* (1986) it is clear that lameness can have a marked effect on fertility. However, the magnitude of the effect depends on the type of lesion, severity and duration of lameness and the time after calving when lameness occurs. Kirton (1982) postulated that if the lame cows in an average 300 cow New Zealand herd with a 10% annual lameness incidence miss one oestrous cycle the cost to the farmer would be $1536. While this is a simplistic approach and ignores lost milk, drugs and veterinary costs as well as ignoring culling options it does highlight the potential effect of lameness on profitability.

Lameness can result in increased culling rates (Collick *et al.*, 1989) and this contributes substantially to the overall cost of the disease (Esslemont, 1990). Estimates of annual culling rates for lameness have been published and have ranged between one and six percent (Renkema and Stelwagen, 1977; Andersson and Lundström, 1981; Whitaker *et al.*, 1983).

When estimating losses associated with lameness, it is not possible to take account of subclinical lameness which in many herds may cost more than clinical disease. In addition to the economic importance of lameness, welfare effects are gaining increasing recognition (Potter and Broom, 1990).

Despite the magnitude of the losses, lameness has received less attention from research workers than most of the other major disease syndromes of dairy cattle. In July 1989, the Veterinary Record reported it had published only 8 articles on lameness in dairy cattle during the previous five years compared with 278 articles on other health problems of dairy cattle such as parasitism and mastitis. Some progress in understanding the epidemiology of lameness has occurred in the 1980s. The holding of international symposia on disorders of the ruminant digit approximately every two years has helped in this regard. However, most research that has occurred has been conducted in the northern hemisphere and relates to production systems in which cows are often housed and fed large amounts of concentrates. Very little work has been conducted that can be readily extrapolated to the pasture-based farming systems of Australia and New Zealand.

Clinical Lameness

Lameness is a clinical sign of a disorder that causes a disturbance in locomotion, which is in turn observed as an aberration of gait (Greenough, 1991). Numerous different lesion types can cause lameness each with its own set of risk factors that predispose to lesion development.

Annual incidence

The incidence rates for lameness reported by various authors are summarised in Table 2.1. Some studies have included only those cases of lameness treated by veterinarians based on veterinary practice records, while others have provided details of cases treated by farmers as well. Those based on veterinary practice records only give unreliable estimates of total lameness incidence since many lame cows are not attended by a veterinarian unless chronic lameness develops. In the study of Whitaker *et al.* (1983) 25% of cases were treated by a veterinarian. McLennan (1988) used this proportion to make an estimate of total lameness incidence in Queensland herds of 10.8%. Another shortcoming of the majority of reported surveys is the lack of information on the severity and duration of cases. A method has been developed recently to quantify the degree of severity of lameness cases using a system of "locomotion scoring" (Manson and Leaver, 1988a). These authors used this method to measure the "quantity" of lameness using "a measure of cow incidence (proportion of cows with score 3 or over), the severity (the score) and the duration (number of weeks with score 3 or more)". This method for measuring the extent of a lameness problem may be valuable in future studies.

Large differences in the incidence of lameness exist between herds. Eddy and Scott (1980) reported an incidence of 60% on one British farm. In New Zealand, 33% of farmer respondents involved in a lameness survey considered they had a herd lameness problem (New Zealand Dairy Board, 1974). In another New Zealand survey involving large herd owners (owners milking 300 cows or more), 48% of 224 respondents indicated that lameness was a problem in their herds (Bridges, 1985a).

Table 2.1. Annual incidence of lameness in reported surveys

Authors	Year	Country	No. of farms	Total No. at risk	% annual incidence
Surveys that include only cases treated by veterinarians:					
Prentice and Neal	1972	United Kingdom	7	521	30
Cagienard	1973	New Zealand	229	36754	1
Eddy and Scott	1980	United Kingdom	150	13588	7.3
Russell *et al.*	1982	United Kingdom	1821	136800	5.5
McLennan	1988	Australia	83	9675	3.6
Jubb and Malmo	1991	Australia		58880	2.5
Surveys that include all cases of lameness:					
Arkins	1981	Ireland		3150	23
Anderson	1985	New Zealand	36	7056	3.6
Whitaker *et al.*	1983	United Kingdom	185	21000	25
Bartlett *et al.*	1986	Michigan, USA	21	2474	9.5
Harris *et al.*	1988	Australia	73	9097	7.5
Philipot *et al.*	1990	France	160	4896	8.2

Lesion types

The introduction of accurate and appropriate terminology for digital disease (Espinasse *et al.*, 1984) and meaningful terminology for digital anatomy (Greenough, 1978; Weaver, 1981; Mills *et al.*, 1986; Mills *et al.*, 1988) has helped considerably to reduce the confusion that was a feature of some of the early literature on lameness.

Numerous studies conducted in a wide range of environments have confirmed that the majority of lameness (83% - 97%) in dairy cattle involves lesions of the foot as opposed to the upper limb, with hind feet being much more commonly affected than front feet (Prentice, 1972; Eddy and Scott, 1980; Baggott and Russell, 1981; Arkins, 1981; Choquette-Lévy *et al.*, 1985; McLennan, 1988; Jubb and Malmo, 1991).

Several comprehensive descriptions of the common lameness-causing lesions, some with excellent illustrations, have been published (Greenough *et al.*, 1981; Baggott and Russell, 1981; Espinasse *et al.*, 1984; Petersen and Nelson, 1984; Toussaint Raven *et al.*, 1985; Greenough, 1987a, 1987b, 1987c, 1987d; Malmo, 1991). More detailed discussions of specific lesion types with recommendations for their control and treatment are also available.

Interdigital necrobacillosis (footrot, foul in the foot) is an acute necrotising cellulitis of the interdigital dermis caused by *Fusobacterium necrophorum* (Clark *et al.*, 1985) and *Bacteroides melaninogenicus* (Berg and Loan, 1975). The typical lesion begins with superficial necrosis which then extends to the deeper tissues and is associated with a foul smelling exudate. In most surveys it has been responsible for 10% - 20% of clinical lameness (Eddy and Scott, 1980; Russell *et al.*, 1982; McLennan, 1988; Jubb and Malmo, 1991). Interdigital dermatitis (scald) is another infectious disease involving inflammation of the interdigital skin, but without extension to deeper tissues, caused by *Bacteroides nodosus* (Thorley *et al.*, 1977; Laing and Egerton, 1978; Clark *et al.*, 1986). It is widespread in moist housing and wet climatic conditions when the ground is wet and muddy. Several sequelae to this condition are recognised including verrucose dermatitis and separation of the soft horn overlying the heel (Toussaint Raven, 1973; Laing and Egerton, 1978). Other conditions that can affect the interdigital space include systemic viral diseases, traumatic injuries due to puncture wounds and interdigital fibroma (Weaver, 1974). Digital dermatitis is a diffuse or circumscribed erythematous lesion of the skin proximal to the coronary margin of the heel (Blowey, 1990). The interdigital skin is rarely affected. Its occurrence is widespread in parts of Europe (Cheli and Mortellaro, 1986) although it has only recently been recognised in Britain (Blowey and Sharp, 1988; Bassett *et al.*, 1990).

White line disease involves disintegration of the junction between the sole and hoof wall and its penetration by debris (Edwards, 1980). The penetration commonly leads to infection of the corium and abscess formation. Bacterial invasion often tracks up the laminae of the wall leading to sinus formation at the coronary band. Most lesions are observed in the region of the abaxial border of the lateral hind digits near the sole-heel junction.

Excessive hoof wear occurs when cows walk long distances each day along gravel or concrete raceways and spend lengthy periods standing on concrete at milking time (Dewes, 1978; Malmo, 1991). It can be associated with bruising of the sole (aseptic traumatic pododermatitis) or with underrun sole or sole abscess (septic traumatic pododermatitis). In bruised sole the horn of the sole becomes very thin and pliable and maybe easily depressed with the thumb. This can be accompanied by patchy haemorrhage extending over a varying area of the sole (Malmo, 1991). Sole abscess occurs when a foreign body penetrates the horn. Dirt and bacterial organisms accompanying penetration result in a purulent and necrotic infection of the damaged tissue. Similar infection can occur through very small cracks or defects in the hoof (Malmo, 1991). Weaver (1971) suggested that acute and chronic laminitis can predispose cattle to traumatic injury of the sole; he considered road walking, together with nutritional factors, only as factors that exacerbate the lesions.

Sole ulcer (pododermatitis circumscripta) is a circumscribed lesion located in the region of the sole-heel junction, usually nearer the axial than the abaxial hoof margin (Smedegaard, 1964a; Edwards, 1973). It is one of the more common conditions that affect the claws of cattle kept under confined conditions in the northern hemisphere but is only seen occasionally in pasture-fed cattle (Jubb and Malmo, 1991). The lesion consists of a defect in the horn, extending from the solar corium to the weight-bearing surface. In advanced cases, granulation tissue may protrude through the defect. Since lesions tend to occur in hind lateral claws rather than in medial hind or front claws, and since the lesion site is so specific, anatomical and mechanical factors are considered important in the pathogenesis (Zantinga, 1973; Toussaint Raven, 1973; David, 1983; David, 1986). Much attention has also been given to the role of laminitis in predisposing animals to these lesions (Nilsson, 1966; Peterse, 1982; Greenough, 1985; Peterse, 1987; Bradley *et al.*, 1989; Colam-Ainsworth *et al.*, 1989).

Laminitis is a diffuse aseptic inflammation of the corium of the hoof, usually in several digits (Espinasse *et al.*, 1984). It occurs in acute, subacute and chronic forms (Weaver *et al.*, 1981). The clinical signs of acute and chronic laminitis have been extensively described (MacLean, 1965; MacLean, 1966; Greenough *et al.*, 1981; Greenough, 1982). In the acute and subacute stages of the disease an aseptic inflammation coincides with systemic illness in the animal. At this stage the claw shows few if any visible changes. In chronic laminitis there are no generalised signs but the shape of the claw is invariably

altered and lameness may persist. The dorsal wall becomes elongated and concave with grooves and ridges caused by irregular episodes of horn growth. In addition, the sole becomes flatter and broader than normal and the horn of the sole is often extremely soft (Greenough, 1987a). Another form of laminitis known as subclinical laminitis was first described in dairy cattle in 1979 (Peterse, 1979) and has since received an increasing amount of attention. The predominant features of subclinical laminitis are haemorrhages of the sole and abnormal horn production (Livesey and Flemming, 1984; Bradley *et al.*, 1989; Greenough and Vermunt, 1991). Several comprehensive descriptions of the pathology and arteriographic changes that are associated with laminitis hypothesise that the syndrome is evoked by disturbed digital circulation (Andersson and Bergman, 1980; Mortensen, 1986; Boosman *et al.*, 1989; Boosman, 1990).

Vertical fissures in the hoof wall (sandcracks) usually cause lameness only if they extend into the coronary band. They occur more commonly in the axial wall of hind digits or the abaxial wall of the front digits (Malmo, 1991).

Infection of the deeper tissues of the digit, often termed deep sepsis, usually occurs secondarily to other conditions such as white line disease, interdigital necrobacillosis, sole ulcer or sole abscess. It can be extensive, involving several structures, or localised to one structure causing septic pedal arthritis or septic navicular bursitis (Baggott and Russell, 1981). Infection of the distal interphalangeal joint usually enters via one of three common routes, namely the interdigital space, the abaxial coronary band or the retroarticular space (Greenough, 1987c). Retroarticular abscesses arise from inflammation and infection in the space immediately posterior to the intermediate phalanx and are confined posteriorly by the deep flexor tendon (Greenough, 1987c).

Incidence of different lesions

The incidence and importance of each lesion varies with the environment and production system. Eddy and Scott (1980), in a survey of lameness cases treated by their veterinary practice in England, reported that white line disease accounted for 34.9% of cases, pricked sole (septic pododermatitis) for 20.4%, foul in the foot for 14.3% and sole ulcer 11.4%. In the large study of Russell *et al.* (1982), which involved over 1,800 herds in the United Kingdom, white line disease was responsible for 20.3% of lameness, foul in the foot for 16.7%, sole ulcer 13.6% and punctured sole with pus (septic pododermatitis) for

10.4%. The results of these surveys prompted Baggott (1982) to conclude that white line disease is the most common cause of lameness in the U.K. dairy cattle population.

Sole ulcer was the most common lesion observed in the study of Choquette-Lévy *et al.* in Quebec, Canada (1985) accounting for 48.6% of cases. White line disease (11.8%), erosion of the bulb (11.8%), traumatic pododermatitis (8.6%) and interdigital necrobacillosis (7.4%) were the other common lesions. Sole ulcer has also been reported as being the most frequently observed lesion in the dairy herd at the University of Saskatchewan in Canada (Vermunt and Greenough, 1990).

In Australia, two studies involving veterinary treatments only have been documented (McLennan,1988; Jubb and Malmo, 1991). Sole ulcer and laminitis were rarely observed. Interdigital necrobacillosis accounted for 13 -15% of cases and white line disease for 7 - 9% of cases in both studies. The incidence of other lesions appears to vary with area. Jubb and Malmo (1991) reported that axial wall cracks and underrun soles are common (21.7% and 14.6% of cases respectively) in the Macalister irrigation area of East Gippsland. In McLennan's report from Queensland (1988) deep sepsis accounted for 8.9% of lesions, axial groove fissure for 8.4% and bruised sole for 5.6% while axial wall cracks and underrun soles were not mentioned.

In another Australian study, the herd was the unit of observation (Harris *et al.*, 1988). Farmers' opinions were sought to determine the clinical signs usually present in lame cows in their herds. In 87% of the herds (27 out of 31 herds) the farmer's opinion was that either overworn soles (9 herds), interdigital stones (9 herds) or both (9 herds) were usually present. Since this study was based entirely on the retrospective opinions of farmers as to whether the affected feet of lame cows usually showed any of a list of clinical signs that was provided it contains little reliable information on the types of lameness in the area under survey.

The only New Zealand study with any information on lesion types involved in lameness is that of Dewes (1978). Abscess beneath the horn was the most common lesion. From descriptions included in the discussion of his findings, feet with these lesions usually had overworn and bruised soles.

Subclinical hoof lesions

In one of the earliest reported surveys of lameness in dairy cattle Prentice and Neal (1972) observed some degree of foot abnormality in 76% of 504 cows from west Cheshire and northern Wales whose feet were examined after slaughter. In Sweden, Andersson and Lundström (1981) found 75% of slaughtered cattle had some form of digital disease. Haemorrhages of the sole, sole ulcer and heel erosion were the most common lesions observed. Haemorrhages in the sole were also commonly observed in another abattoir study of free range cattle in Tanzania (Mgassa *et al.*, 1984). 55% of 225 adult cattle examined had hoof lesions indicative of subclinical or chronic laminitis. The authors regarded the most likely cause of these lesions to be trauma as a result of overburdening of the digits during trekking.

A number of studies on live animals in several different countries have confirmed that the prevalence of subclinical hoof lesions in dairy cattle is frequently quite high. In a recent French study involving 4896 cows on 160 farms, 89% of cows surveyed had some foot lesion in the one hind leg examined (Philipot *et al.*, 1991). 55% of legs were affected by heel horn erosion and 49% had some haemorrhage of the sole. Heel erosion also occurs commonly in Danish herds (Enevoldsen and Grohn, 1991b). Its severity has been shown to increase with parity and the risk of cows being affected to increase with stage of lactation. In the Netherlands, lesions of the sole, ranging in severity from discoloration to ulceration of the sole and undermining of the horn of the wall and sole are common in heifers (Peterse, 1982). Smart (1986) observed a range of hoof lesions, many fitting the description of subclinical laminitis, in cows from a herd in Western Canada. In the study of Bradley *et al.* (1989) the finding of sole haemorrhages in the hoof horn of calves as young as 5 months and the consistent finding of lesions in heifers of varying ages lead the authors to conclude that subclinical laminitis is a common condition during the early growing period of young dairy heifers.

An increasing emphasis has been placed on study of subclinical hoof lesions since Peterse (1979) first described the syndrome of subclinical laminitis. Greenough and Vermunt (1991) have developed a method of scoring the presence and severity of sole haemorrhages for recording purposes. In their work, a high prevalence of sole haemorrhages was found in heifers prior to calving. Factors associated with nutrition and management such as rapid rearing, the sudden introduction of heifers into the dry cow

group after they became pregnant (leading to confrontations with dominant cows), and housing on concrete in a cubicle system appeared to influence the occurrence of claw haemorrhages. Livesey (1984/85) studied the effect of feeding a low fibre, high starch diet in early lactation on the incidence of laminitis and sole ulcers in Friesian cows in Wales. Although the diet influenced the incidence of clinical lameness almost all cows, regardless of whether they were fed a high or low fibre diet, had some hoof lesions by mid-lactation. Lesions varied from mild haemorrhage to classical sole ulcer. Diet was also found to influence the prevalence of toe and heel haemorrhage in the claws of feedlot cattle (Greenough *et al.*, 1990). Feeding a higher-energy ration increased the prevalence of toe and heel haemorrhages in calves and of heel haemorrhages in yearlings.

Greenough (1986) has suggested that subclinical laminitis occurs in New Zealand dairy cattle that rarely receive concentrates, being fed almost entirely on grass and silage. However no objective studies have been conducted, and whether the epidemiology of hoof lesions is similar in New Zealand to that described by European and North American workers remains unclear.

Aspects of claw quality

Since the majority of lameness in dairy cattle is associated with claw lesions, attention must be directed towards those aspects of claw quality that provide resistance to damaging influences. High claw quality has been defined as a low susceptibility to claw disorders and a low need for claw care. Different approaches have been taken to evaluate claw quality such as recording of hoof lesions, visual scoring for claw shape, measuring the physical and biochemical properties of claw horn and progeny testing of bulls for claw traits.

Politiek *et al.* (1986) have described claw quality as the product of claw shape, characteristics of the horn, and anatomy and physiology of the inner structure.

Claw shape

The relationship between conformation of the claw and incidence of lameness has been studied. Eddy and Scott (1980) found 21% of claw lesions in abnormally shaped claws while Russell *et al.* (1982) reported 42%. Overgrowth was most frequently associated with sole ulcers and white line disease. Peterse (1986) found that the length and angle of the dorsal wall of the lateral hind claw was correlated with the severity of claw disorders in heifers. A number of other associations have been reported. Steep claws have less numerous and less serious sole lesions (Smit *et al.*, 1986b). Short toe lengths and steep angles are needed for good locomotion (Manson and Leaver, 1989). Cows affected by digital disease have longer toes and deeper heels (Andersson and Lundström, 1981). However, further research is needed to determine whether claw shape is a predisposing factor for or a result of a claw disorder (Smit *et al.*, 1986b).

Visual scores of claw characteristics are used by cattle breeders in the process of genetic selection. However, they are not very useful as criteria to be used for improving claw quality as they show very low heritabilities and their genetic relationship with claw disorders is low (Hahn *et al.*, 1984a; Peterse, 1986; Politiek *et al.*, 1986; Smit *et al.*, 1986,a,b).

Objective claw measurements can be made more accurately and with higher repeatabilities than visual scores. Greenough (1991) lists the following as the most commonly used claw measurements:

1. The angle between the dorsal wall and the sole (angle of the toe)
2. The toe to heel ratio, ie vertical height from ground to the skin horn junction of the dorsal border to the vertical height from the ground to the skin horn junction at the heel;
3. The surface area of the sole
4. The length of the dorsal border.

Hoof moisture content

An association between wet weather conditions and the incidence of lameness in dairy cattle has often been reported (Eddy and Scott, 1980; Arkins, 1981; Williams *et al.*, 1986; Jubb and Malmo, 1991). Although changes in hoof moisture content have commonly been suggested as one of the reasons for this association, there are few documented reports of the degree to which hoof moisture changes. Hardness of the hoof horn has

been shown to be correlated with hoof moisture levels with hardness increasing as moisture content decreases (Martig *et al.*, 1980). This is not surprising as the rigidity of keratin is believed to be inversely related to its water content (Fraser *et al.*, 1972). A low water content also makes horn less prone to abrasion and to lesions (Dietz and Prietz, 1980).

MacLean (1971) found a significantly higher moisture content in laminitic horn and concluded that the increased water content is likely to be responsible for the physically softer horn in animals affected by laminitis. Similarly, Bodurov *et al.* (1981) reported that diseased hoof horn had higher water content than healthy horn.

Hoof hardness

Many hoof lesions are more common when the claw is soft (Greenough, 1991). Hoof hardness therefore, which is a measure of resistance to penetration, is usually regarded as an important property. Manson and Leaver (1988b) stated that their observation that there was a significant negative correlation ($r = -0.332$) between mid-sole hardness and locomotion score confirmed the importance of sole hardness in reducing lameness.

Various workers have studied factors that may influence hoof hardness. Clark and Rakes (1982) found that hoof hardness at some claw sites was lower in cows supplemented with methionine than in unsupplemented cows. In their study hoof hardness was unrelated to hoof colour. Baggott *et al.* (1988) showed that hoof hardness is related to the chemical composition of the horn. A high concentrate/silage ratio in the diet reduced hoof hardness in Manson and Leaver's study (1989). However, type of concentrate fed did not influence hoof hardness (Kelly and Leaver, 1990). Further, hoof trimming had no effect on horn hardness (Manson and Leaver, 1988b). Reilly and Brooks (1990) reported that biotin supplementation, previously shown to influence the physical properties of pig hoof (Webb *et al.*, 1984), produced significantly harder hoof tissue in cattle.

Resistance to abrasive wear

Lesions associated with excessive sole wear are common in pasture-fed cattle that have to walk long distances each day along gravel or concrete raceways (Dewes, 1978; Malmo, 1991). Hence resistance of hoof to abrasive wear is another physical property of interest.

Attempts have been made to measure abrasive wear but only on hoof samples collected from cattle after slaughter.

Camara and Gravert (1971) measured abrasive wear of cattle hoof by moving claw pieces collected from slaughtered cattle across a concrete surface. Pflug *et al.* (1980) measured abrasive loss of hoof wall strips by applying them against a rotating grinding disk at a constant pressure while Murphy *et al.* (1987) used a modified Martindale cloth-rubbing/wear testing machine. Abrasion was measured by weight loss following a specified number of revolutions on the wear testing machine.

Other physical hoof properties

The horny capsule surrounding the claw is responsible for protecting the sensitive tissues of the foot from infection and traumatic injury. Changes in the physical properties of the hoof that result in weakening of the horn and that make it more susceptible to breakdown are likely therefore to predispose cows to lameness. There is however no single ideal measure of hoof strength.

It has often been suggested that properties other than hoof moisture and hardness are likely to be important in providing the hoof with the ability to resist external damaging influences. Resilience, viscoelasticity, fracture toughness, concussion resistance, elasticity, strength, stiffness, and mechanical hysteresis are all properties of hoof that have been mentioned in reports of lameness-related research as being of importance. Some have been studied in the hooves of horses and pigs.

Webb *et al.* (1984) used compression tests to measure the compressive strength and elastic modulus of pigs' hooves after slaughter. They describe compressive strength of hoof as the amount of stress on the tissue which corresponds to the point at which cellular structures are ruptured. The elastic modulus is a measure of the rigidity or stiffness of a material.

Leach and Zoerb (1983) measured the elastic modulus of hoof wall in horses' hooves collected from the abattoir. They demonstrated that hoof samples from the inner hoof wall were less rigid than those from the outer hoof wall. Tensile tests were used by Bertram and Gosline (1986, 1987) to measure both elastic modulus under tension and

fracture toughness of horse hoof keratin, again in samples collected from dead animals. They showed that stiffness of hoof is inversely related to its water content. Decreasing hydration will increase the stiffness of hoof keratin but in many materials an increase in stiffness can adversely affect the fracture properties, making them brittle.

Resilience is a further characteristic which measures both the elasticity of a material and its strength and is yet another hoof property that is likely to contribute to the hoof's ability to resist traumatic damage. A method for measuring resilience was described by Zoerb and Leach (1978).

No reports could be found of studies of these hoof properties in cattle. Hence which properties are important when considering modes of mechanical failure that result in foot lesions of cattle is largely unknown.

Distribution of pressure on the sole

Methods have been developed to quantify the interactions between the ground and the limbs of livestock using force and pressure platforms (Webb and Clark, 1981; Ossent *et al.*, 1987; Scott, 1988b; Distl and Mair, 1990). The pressure distribution can be visualised under the limb in contact with the ground; and changes in pressure during movement, such as walking, can be recorded. While studying the gait of Friesian heifers, Scott (1988b) noted that the forefeet carried maximum loads equivalent to about 60% of bodyweight, while the hind legs carried loads equivalent to 50%. There were generally no significant differences between the contact areas and pressures applied by the front and back feet. In other work using pregnant cows, he observed that the load distribution during late pregnancy was not changed. The fore hooves always carried significantly greater loads than the hind hooves (Scott, 1988a). When he compared the gait changes of lame cows with the gaits of non-lame cows, he observed that the maximum vertical force was often reduced on lame limbs (Scott, 1989). Ossent *et al.* (1987) studied the variation in loads on the hind digits of stationary cattle. Barely discernable movements of the animals were sufficient to cause considerable changes in the magnitude of loads borne. Lateral digits displayed a far greater range of loads than medial digits.

Chemical composition of hoof

MacLean (1971) compared the amino acid composition of cows affected by acute laminitis with that of healthy herdmates. There was a significant reduction in sulphur-containing amino acids and lysine in laminitic horn. He concluded that the lower content of sulphur-containing amino acids must result in some loss of mechanical resistance as disulphide cross linking of polypeptide chains in keratin is considered to be responsible for its high mechanical strength. Bodurov *et al.* (1981) also reported that horn of diseased claws had a lower content of sulphur and of cystine, methionine and lysine than healthy horn.

Clark and Rakes (1982) postulated that if keratin growth is limited by the availability of substrates at the site of synthesis it may be possible to stimulate hoof growth through addition of sulphur to the diet. They studied the effect of dietary supplementation of methionine hydroxy analog - on hoof growth, hoof hardness and hoof keratin composition. The hooves of cows fed the analogue grew faster (but only during the spring-summer period), were softer at some measurement sites and had a different amino acid composition than those of unsupplemented cows.

The mineral composition of hoof horn varies between breeds, according to moisture content, health status of the horn and housing system (Dietz *et al.*, 1986). Baggott *et al.* (1988) reported that some inorganic elements (calcium, phosphorus, copper, zinc) are related to hardness of hoof and that variations in copper, zinc and magnesium appear to be associated with lameness. They suggested the higher concentrations of copper in the hoof horn of lame cows may be a consequence of foot lameness as concentration of copper is known to rise in inflamed tissue.

Horn growth and wear

An understanding of the normal processes of hoof growth and wear in cattle under different management systems is necessary since hoof disorders have been associated with both hoof overgrowth and excessive hoof wear (Dewes, 1978; Toussaint Raven *et al.*, 1985; Malmo, 1991). Numerous studies have been conducted in the northern hemisphere. These have mostly involved housed cattle. Unfortunately there is little published information available for grazed cattle.

Various factors have been reported to influence hoof growth and wear. Average rates for hoof wall growth lie between 0.9 and 1.4 mm per week (Prentice, 1973; Clark and Rakes, 1982; Manson and Leaver, 1988b). Hoof wall growth rates have been shown to decrease with increasing age of cattle. Prentice (1973) found higher rates in calves and yearlings than in mature cows. Hahn *et al.* (1978) observed higher horn growth rates in cows on their first lactation than those on their second, and Glicken and Kendrick (1977) also showed that the hooves of younger cows grow faster than those of older cows. However, Clark and Rakes (1982) found rates of horn growth not to be related to age.

Sex and breed had no influence on hoof growth rates in young calves (Schlichting, 1987). However, Murphy and Hannan (1986) reported that both growth and wear were greater in the digits of Friesian steers than in those of Herefords. They suggested that the faster turnover of horn in Friesians was due to a softer horn texture and that this accelerated the development of cow hocked conformation.

Prentice (1973) reported that there was no difference in hoof wall growth between lateral and medial digits but hoof wall of front digits grew faster than that of rear digits. In contrast, Hahn *et al.* (1986) found that hoof wall of hind digits grew faster than that of front digits while Clark and Rakes (1982) concluded that the rates of horn growth and wear for front and hind claws were not significantly different.

Hoof growth is increased by hoof trimming, suggesting that some compensatory mechanism stimulates hoof growth (Manson and Leaver, 1988b, 1989). It may also be influenced by nutrition but reports are conflicting. Manson and Leaver (1988b) reported higher rates of hoof wall growth in dairy cows fed high-protein rations than in cows fed low-protein diets. Sole horn growth rates, however, were decreased in yearling beef cattle as the level of dietary protein increased (Greenough *et al.*, 1990). Hoof wall growth and wear rates were higher in cows fed rations with a high concentrate/silage ratio than in cows fed rations with a low concentrate/silage ratio (Manson and Leaver, 1989). However, hoof growth and wear were not affected by the type of concentrate fed when rates were compared in cows fed a fibrous source of energy with those in cows fed rations containing a starchy source (Kelly and Leaver, 1990). Sole horn growth rates were not increased in either calves or yearlings by feeding high-energy rations (Greenough *et al.*, 1990). Clark and Rakes (1982) studied the effect of feeding a methionine hydroxy analogue on hoof growth and wear. The hooves of cows fed the analogue grew faster

than those of controls during spring and summer but wear rates were not affected by treatment.

Another factor that has been shown to influence hoof growth rates has been season of the year, with higher growth occurring during summer than in winter. Hahn *et al.* (1986) suggested the cyclical nature of hoof growth could be due to variation in environmental temperature, photoperiod, herd management or nutrition. Clark and Rakes (1982) demonstrated that periods of increased hoof growth coincided with increasing photoperiod length while decreased hoof growth coincided with decreasing photoperiod length. Periods of increasing hoof growth also coincided with increasing photoperiod length in Vermunt's studies (Vermunt, 1990). However, Wheeler *et al.* (1972), working with sheep, concluded that hoof growth was not influenced by photoperiod but that it was greatly reduced by low temperatures. Vermunt (1990) also raised the possibility of hoof growth being affected by changes to the circulation during extremely cold weather that involve constriction of arterioles and dilation of arteriovenous shunts.

The nature of the surface to which hooves are exposed has a large influence on the rate of hoof wear. Cows housed on abrasive concrete have been shown to have 35% greater hoof wear than cows at pasture (Hahn *et al.*, 1986). These authors concluded that cows confined on an abrasive surface during lactation should be placed on dirt or pasture during the dry period to allow for recovery of hoof length. In Vermunt's study (1990), hoof wall wear was lower in heifers housed outdoors on a dry feedlot than in heifers housed indoors on slats. Hoof growth and wear were both lower in calves reared on deep litter than in those housed on slatted flooring (Schlichting, 1987). Higher wear rates were also observed in steers kept on slats than in those kept on straw (Murphy and Hannan, 1987).

Risk factors associated with lameness

Foot lameness in dairy cattle is a problem where numerous risk factors are thought to contribute to the severity of the problem on any particular farm. However, it has been generally considered difficult to distinguish their separate contributions and the links between related variables, although progress in achieving this has been made (Chesterton *et al.*, 1989).

Cow factors

i) Digit distribution

Most surveys of lameness in cattle have confirmed that the majority of lameness occurs in the hind feet and that lesions occur more commonly in lateral digits of the hind leg than in medial digits (Prentice and Neal, 1972; Eddy and Scott, 1980; Andersson and Lundström, 1981; Arkins, 1981; Russell *et al.*, 1982; McLennan, 1988; Jubb and Malmo, 1991; Willyanto, pers. comm.). Various reasons have been suggested for this unequal distribution of lesions between digits. Pinsent (1981) has suggested that lameness is more common in the hind legs because hind legs are involved with propulsion, during which the hind digits are subject to extra pressure due to friction. This has been supported by Scott's work (1987). Russell *et al.* (1982) postulated that the difference in incidence between front and hind legs may be due to the fact that front feet can move easily in a vertical plane parallel with the midline of the cow and can be placed with precision, whereas the back feet are forced out of this plane by the udder and may be subjected to uneven loading and torsion. These authors also suggested that the extra loads on the hind feet during mounting activity associated with oestrous behaviour may also be a contributing factor.

The high proportion of hind leg lameness involving the lateral digits can be partly explained by Scott's work (1988a) which shows that higher pressures during load bearing occur mainly on the lateral digits and mostly around the heel region and abaxial walls and toes. He also showed that lateral digits have a significantly larger area of contact compared to medial digits (Scott, 1987). Toussaint Raven *et al.* (1985) has suggested that weight bearing by the lateral claws varies widely with successive movements of the body, whereas the loads on the medial claws are far more stable. The continual shift and alteration of forces is considered to be more detrimental than the absolute load. Ossent *et al.* (1987) recorded greater ranges for loads on the lateral hind claws of heifers than on medial claws using a force plate, adding support to this suggestion.

ii) Stage of lactation

Association of the onset of lameness with particular stages of lactation has been a common finding, with most studies reporting a high proportion of lameness

in the first 3 months of lactation (Dewes, 1978 ; Prentice and Neal, 1972 ; Eddy and Scott, 1980; Arkins, 1981; Russell *et al.*, 1982; Whitaker *et al.*, 1983; Rowlands *et al.*, 1985; Choquette-Lévy *et al.*, 1985; McLennan, 1988; Harris *et al.*, 1988; Collick *et al.*, 1989; Jubb and Malmo, 1991). The reasons that have been suggested for this association include changes in husbandry at calving (especially those associated with reintroduction into the milking herd and in some systems resumption of long distance walking to pasture), changes in nutrition at calving (which often involve a switch to high concentrate rations), metabolic stresses associated with calving disorders or high production that may result in soft or poor quality horn, seasonal calving patterns that coincide with wet weather, and an association with oestral mounting activity.

iii) Age

The influence of age on the incidence of lameness appears to depend on the type of lesion involved. Excessive hoof wear and bruising are often more common in younger cows, low in social ranking in the herd (Dewes, 1978; Harris *et al.*, 1988; Malmo, 1991). Others have reported that young cows are more susceptible to the acute and subclinical forms of laminitis (Bazeley and Pinsent, 1984; Colam-Ainsworth, 1989; Vermunt and Greenough, 1990). Chronic laminitis occurs more commonly in older cows (Weaver,1985). The common observation that laminitis is more often encountered in first lactation heifers than in cows could partly be attributed to the fact that many heifers are fed little or no concentrate from an early age until they enter the milking herd (Bazeley and Pinsent, 1984).

Rowlands *et al.* (1985) and Baggott and Russell (1981) reported that susceptibility to lameness increased with age. This was due largely to the increased incidence of white line abscess and sole ulcer in older cows. They suggested this may be due to a progressive deterioration in claw condition possibly involving deterioration in shape, internal structure or horn defects such as softening.

iv) Genetics

Poor characteristics of limb conformation, claw shape, size and quality may predispose to some diseases or lesions that cause lameness (Nelson and Petersen, 1984; Politiek *et al.*, 1986; Greenough, 1991). However, the role of genetics in the expression of such poor characteristics is unclear.

Glicken and Kendrick (1977) suggest that there is a genetic component in the expression of hoof overgrowth and they state that overgrown hooves are more susceptible to lameness. Eddy and Scott (1980) consider that some claw deformities may be heritable. Russell *et al.* (1982) suggested that the high rate of lesions in abnormally shaped claws is probably caused by the recent emphasis on conformation of legs rather than of claws in breeding stock.

The possibility of breed predispositions has been raised. Pinsent (1981) considered the hooves of Friesians were more prone to damage than were the small, hard and compact hooves of Jerseys or Shorthorns. He stated that the straight and upright hocks of the modern Friesian cow must accentuate impacts and frictional forces. Jubb and Malmo (1991) suggested that their observation that sole ulcers occurred predominantly in very large framed purebred Holstein-Friesian type cows, with almost complete sparing of other breeds, indicated a breed predisposition. Bazeley and Pinsent (1984) mentioned a possible inherited predisposition to laminitis.

Claw characteristics as assessed by visual scoring have low heritabilities (Smit *et al.* 1986b). However, moderate to high heritabilities for the angle of the toe, the length of the dorsal border and height at the heel have been recorded (Hahn *et al.*, 1984b; Politiek *et al.*, 1986; Smit *et al.*, 1986b; Baumgartner and Distl, 1990; Ral, 1990).

Nutrition

Great emphasis has been placed during the last decade on the role that nutrition plays in predisposing cattle to lameness. Laminitis due to digestive disturbances has become the centrepoint of most discussion on the epidemiology and aetiology of the major lameness lesions (David, 1990). Weaver (1979) stated that in the United Kingdom,

laminitis and its complications accounted for about half of the cases of lameness in the national dairy herd. The traditional explanation of the aetiology of laminitis involves an excess carbohydrate intake, followed by ruminal acidosis (Nilsson, 1963). As a result of an environment that is hostile to the normal rumen flora, toxic substances (such as histamine, lactic acid, serotonin and possibly endotoxin) are released into the circulation. These substances have a devastating effect on the blood vessels that supply the horn-producing tissues of the claw. This process causes irreversible damage to some of the structures that produce horn (Greenough, 1991).

The subclinical form of laminitis, which is believed to predispose the hoof to other important lesions such as sole ulcer and white line disease, is also commonly believed to be related to high carbohydrate intakes (Peterse, 1979; Moser and Divers, 1987; Manson and Leaver, 1988a; Greenough *et al.*, 1990; Greenough, 1990). Other aspects of nutrition have also been implicated. These have included the level of protein intake, the ratio of concentrate to forage, the quality of roughage intake, the rate of introduction of concentrate to the ration and the level of free ammonia in silage (Livesey and Fleming, 1984; Peterse *et al.*, 1984; Bazeley andPinsent, 1984; Manson and Leaver, 1988b; Manson and Leaver, 1989; Kelly and Leaver, 1990). However, the numerous herd feeding trials that have been conducted to study the effects of nutrition on lameness and hoof lesions have sometimes given conflicting results.

Smit *et al.* (1986a) failed to demonstrate any significant differences in the incidence of sole ulcer or sole haemorrhage between two groups of cows fed 4 kg and 12 kg of concentrate daily. These same authors showed no correlation between the incidence of sole ulcer or sole haemorrhage scores and the rate of increase in concentrate feeding. Similarly, Peterse *et al.* (1986) could not demonstrate any difference in lesion scores between two groups in which concentrates were increased at different rates. Bergsten *et al.* (1986) failed to show any association between the severity and incidence of sole haemorrhage and the amount of concentrate fed from three weeks prior to calving to 2 weeks after calving. Bee (1986) described a high incidence of both sole ulcer and white line disease in cattle fed low levels of concentrates.

It is clear that acute laminitis can be caused by nutritional disturbances and that feeding high levels of concentrates can increase the risk of lameness in some circumstances. However, there has been a tendency to over-emphasise the contribution of nutrition in

the aetiology of laminitis (David, 1990). There is an increasing recognition that behaviour, management and genetic factors are of considerable importance as well. Greenough (1986) has suggested nutrition could be a risk factor for lameness in New Zealand cattle. However, apart from possibly high protein intakes, few if any of the other nutritional factors that have been implicated in lameness studies conducted in the northern hemisphere are recognised as being present in our pasture-based farming systems.

Housing

In countries where cattle are housed for part or all of the year the type of housing can be expected to influence the incidence and severity of lameness. The literature dealing with the design and technical aspects of various housing designs is extensive. Factors that have frequently been implicated in predisposing cattle to digital lesions include the type of stall design, the quality of the floor surface and the amount of bedding used. Lesions are usually considered to be caused by mechanical overloading of the corium (especially of the lateral hind digits) associated with the unyielding nature of concrete floor surfaces and the amount of time cows spend standing (David, 1983). A form of overloading/rheumatic laminitis, as described by Nilsson (1963), occurs and this results directly in clinical lameness or predisposes to sole ulceration and white line disease. On several occasions, David (1984/85, 1986, 1989, 1990) has emphasised that changes to management and housing seemed likely to have been more important contributing factors in precipitating many of the herd lameness problems that he has investigated than have nutritional imbalances. It is interesting to note that in pigs, Penny (1990) considers that trauma from the external environment is more likely to be responsible for claw bruising and hoof haemorrhage than are vascular changes within the claw associated with aspects of nutrition.

Many accounts of increased incidence of lameness and/or increased occurrence of sole haemorrhages associated with aspects of housing have been published (Weaver, 1971; Rowlands *et al.*, 1983; David, 1986; Murphy *et al.*, 1987; Maton, 1987; Colam-Ainsworth, 1989; Faye and Lescourret, 1989; Greenough and Vermunt, 1991).

The incidences of interdigital dermatitis and heel horn erosion are also influenced by the type of housing (Smit *et al.*, 1986a; Thysen, 1987). Predisposing factors include a continuously dirty and moist environment which are common in some housing systems.

Raceway maintenance

In Australia and New Zealand, where cows in most herds graze pasture year-round and housing of cattle is rare, cows are frequently required to walk long distances each day along gravel or sometimes concrete raceways. In several studies lameness has been associated with aspects of race design and maintenance. In a case-control study of environmental and behavioural factors influencing foot lameness in 62 dairy herds, the average level of maintenance of the farm track was one of the most influential variables in explaining variation between case and control herds (Chesterton *et al.*, 1989). In an Australian study, the most important environmental factor affecting lameness was the farm track (Harris *et al.*, 1988). Lameness increased when the track had steep slopes, had coarse gravel on the surface or had broken sections. Raceway maintenance has also been identified as a risk factor for lameness in the United Kingdom. Clarkson and Ward (1991) reported that the incidence of lameness was higher on farms where tracks were less well maintained. Bee (1986) stated that the most important cause of white line disease is cows being forced to move on poor conditions underfoot. He considered concrete surfaces which have started to break up and which have loose sharp stones on the surface to be particularly damaging.

Chesterton (1989) has discussed the mechanisms by which poorly maintained tracks might affect lameness incidence. There is the effect resulting from direct injury to the feet of cows irrespective of other factors. An indirect effect also operates whereby a poorly maintained track slows the movement of animals along the track, causing the farmer to become less patient. Impatient handling results in jostling between cows and unplanned placement of their feet; both of which can potentially result in trauma to the feet.

Race construction methods on New Zealand farms have been examined by means of a postal survey of herd owners in which 495 valid responses were received (Bridges, 1985a). This survey indicated that the majority of maintenance work on raceways is performed in an unsatisfactory manner. Of the farms surveyed 22% undertook no race maintenance whatsoever. Of the remainder who did undertake maintenance work, lack of rolling was identified as the most serious deficiency, with less than 8% of farmers using a roller.

Lack of rolling results in the race surface being more susceptible to pugging and water damage. Less than 10% of respondents used a grader for shaping the race surface and only 16% had races which incorporated drains. The author of this survey identified the lack of appreciation of some farmers of the true costs of metalling and maintaining races as being another area of concern. He suggested that if true costs were known the use of contractors with proper machinery could be justified.

Herd management

Foot lameness is inextricably associated with management (Pinsent, 1981). Amstutz (1985) considered that one of the more frequently observed management errors resulting in lameness is the rough handling of stock such as crowding through narrow door ways or hurrying them across slippery floors. Although these comments were made with housed cattle in mind the same management errors have been identified as risk factors for lameness in cattle grazed at pasture. In the study of Chesterton *et al.* (1989) the degree of patience demonstrated by the farmer while bringing cows in for milking was important in explaining the variation between high and low lameness incidence herds. Use of a biting dog was another influential variable. The stockman's patience was also identified as important in Clarkson and Ward's recently reported study (1991). Factors such as impatient animal handling and use of a biting dog interfere with the social forces of leadership and dominance which control spacing and order within a moving cow herd (Dewes, 1978). If cows are under pressure from the farmer or his dogs crowding is likely to develop and thus the choice of foot placement may be restricted(Chesterton, 1989)

The feeding of meal in the dairy at milking time is associated with a lower risk of lameness, probably as a result of improving cow movement to the dairy and in the yards (Harris *et al.*, 1988; Chesterton *et al.*, 1989). Similarly voluntary entry of cows into the dairy for milking is associated with a lower lameness incidence (Harris *et al.*, 1988) confirming that herds with more content cows have a lower risk of lameness.

Sudden introduction of animals to concrete, cubicles, or a new housing design, and sudden introduction of new animals into an established dominance hierarchy of other animals have all been responsible for some of the reported outbreaks of lameness or increased occurrence of hoof haemorrhage (Bazeley and Pinsent, 1984; David, 1984/85; Greenough and Vermunt, 1991). Factors considered to be involved have been increased

activity and modified behavioural interactions following the introduction of new animals, and decreased resting time because of unfamiliarity with the new environment. Traumatic injury to the claws and concussive damage to the corium were responsible for the development of laminitis, sole bruising and sole ulceration. However, some workers prefer to emphasise the possibility that a nutritional aetiology is involved. In a discussion of the aetiology of claw disorders Peterse (1987) implied that inadequate space at a feeding barrier could result in laminitis in low ranking and weak, freshly calved cows because low roughage intake could affect the ratio of roughage to concentrate in the diet and alter the rumen pH. Toxic agents released into the circulation then cause laminitis by decreasing capillary perfusion of the pododerm.

Season and rainfall

In numerous lameness studies, an association between wet weather conditions and the incidence of lameness has been observed (Eddy and Scott, 1980; Arkins, 1981; Williams *et al.*, 1986; Jubb and Malmo, 1991). Williams *et al.* (1986) associated lameness incidence with amount of rainfall during the two weeks prior to onset, and with potential soil moisture deficit. They suggested that it might be possible to obtain stronger correlations between lameness incidence and wetness of the environment by using more refined measures of soil moisture. Wet conditions are thought to increase hoof moisture content and cause softening of the hoof, thereby predisposing cows to lameness. However, few objective studies have been conducted to verify these associations.

Seasonal influences have also been observed. Reasons for these have usually been ascribed to differences in housing between seasons (housing period versus grazing period) (Andersson and Lundström, 1981; Peterse, 1982; Rowlands *et al.*, 1983), the seasonal nature of calving patterns (Cagienard, 1973; Harris *et al.*, 1988) or to seasonal variation in rainfall (Eddy and Scott, 1980; Arkins, 1981).

Infectious agents

The role of *Fusobacterium necrophorum* in the aetiology of interdigital necrobacillosis footrot, foul in the foot) is unquestioned, for although several organisms have been isolated from such lesions it is the only one that is found to be consistently present (Baggott and Russell, 1981; Berg and Loan, 1975; Clark *et al.* 1985). Bacterial invasion

follows some form of interdigital trauma and an acute cellulitis follows. Spread of the disease is primarily related to the degree of contamination and quality of the environment. The work of Berg and Loan (1975) provided evidence that *Bacteroides melaninogenicus* was also involved in the aetiology but the role of this organism has been questioned by other authors (Espinasse *et al.*, 1984; Clark *et al.*, 1985; Emery *et al.*, 1985).

Interdigital dermatitis is a superficial inflammation of the interdigital skin. *Bacteroides nodosus*, the causative agent of footrot in sheep, has been isolated from such lesions on numerous occasions (Egerton and Parsonson, 1966; Morgan, 1969; Toussaint Raven and Cornelisse, 1971; Thorley *et al.*, 1977; Laing and Egerton, 1978; Richards *et al.*, 1980). Infection is facilitated by prior hydration and maceration of the interdigital skin. The efforts of Egerton to equate interdigital and heel horn lesions in cattle with footrot in sheep (Egerton and Laing, 1982; Egerton, 1989) and to encourage the use of alternative terminology for these lesions to that proposed by the international working group on Disorders of the Ruminant Digit (Espinasse *et al.*, 1984) has done little to improve general understanding of the pathogenesis and epidemiology of these conditions.

Bacteroides nodosus may also be involved in the aetiology of heel horn erosion. Some workers believe this condition occurs as a result of invasion of the heel horn with *Bacteroides nodosus* as an extension of interdigital dermatitis (Toussaint Raven and Cornelisse, 1971). Others consider interdigital dermatitis and heel horn erosion are two separate entities caused by non-specific bacterial and chemical agents originating from dung and urine (Smedegaard, 1964b). More recently Greenough and Vermunt (1990) have suggested that heel erosion could occur partly as a sequel to haemorrhages associated with laminitis.

Prevention of lameness

To develop effective strategies for preventing lameness, it must be approached as a disease affecting the herd rather than the individual, as is common practice for mastitis and infertility (David, 1989). General guidelines for prevention and control programs have been summarised (Weaver, 1979; Greenough *et al.*, 1981; McDaniel, 1983; Chesterton, 1989; Malmo, 1991). Aspects of management, husbandry, and housing and

environmental influences may all need to be considered depending on the farming system involved.

Management and patient animal handling

One of the most effective measures that can be adopted to prevent lameness is that of patient animal handling. The degree of patience demonstrated by farmers while herding cows for milking has been shown to significantly influence the incidence of lameness (Chesterton *et al.*, 1989; Clarkson and Ward, 1991). Cows should be allowed to "drift" while being herded for milking and, if allowed to do this, will seldom misplace their feet, even on a poorly maintained track (Chesterton, 1989). Pinsent (1981) states that if cows are allowed to move with grace and elegance they will not become lame. Others have also emphasised the need to handle cattle quietly and slowly to avoid injuries (Whitaker *et al.*, 1983; Amstutz, 1985).

Management of the herd to avoid competitive social interactions has also been recommended. Cows low in social ranking in the herd which as a consequence spend the most time on concrete since they are milked last, should be shunted out of the main holding yard until late in milking or, alternatively, be milked first (Dewes, 1978). Another possibility that has been promoted has been that of separating the subordinates (usually the younger cows) and managing them as a separate herd (Dewes, 1978; Pinsent, 1981; David (1984/85). Culling cows ranked high in dominance that are usually late in the milking order has even been suggested (Dewes, 1978). In seasonal systems heifers can be mated a few weeks ahead of the rest of the herd making it possible for them to calve earlier and become accustomed to the milking routine without the dominating influences of older herd mates. In non-seasonal systems heifers should be introduced to concrete floored yards several weeks before calving in order for them to become accustomed to the surfaces (Weaver, 1979; McDaniel, 1983; David, 1984/85).

Provision of adequate feeding space for all stock is important (Weaver, 1979). Welfare problems due to social factors are likely arise if there are insufficient feeding or lying spaces for all animals (Potter and Broom, 1987). Where cattle are housed, provision of adequate bedding is also essential (Colam-Ainsworth, 1989).

Nutrition

Aspects of nutrition that need to be considered in order to minimise the risk of lameness have been emphasised in many publications relating to lameness. Weaver (1979, 1988) has listed several rules of nutritional management that should be adopted to prevent the ruminal acidosis syndrome which is considered to be involved in the pathogenesis of laminitis. These focus on the need to make feeding changes slowly, to feed concentrates in smaller amounts more frequently or preferably feed a total mixed ration, and to provide immediate and adequate access to forage after concentrate feeding.

Feeding of trace minerals and vitamins has been investigated as a means of preventing lameness. Interest has been aroused in biotin supplementation since it is now well proven that hoof integrity in pigs can be improved by supplementing the diets of breeding and replacement stock (Brooks, 1986; Simmins and Brooks, 1988). For many years nutritionists perceived there was no need to supplement the diet of pigs with biotin since it was believed raw materials in feeds contained substantial quantities and microorganisms of the gut also produced reasonable amounts. However, its biological availability is limited in many feedstuffs and absorption of microbially-produced biotin from the lower gut is poor; hence the need for supplementation. The need for supplementation in cattle has not been studied although Reilly and Brooks (1990) have recently reported that biotin supplementation of cattle produced significantly harder hoof tissue in the centre of the abaxial wall and increased hardness of heel horn tissue.

Schugel (1982) states that zinc methionine supplementation, because of the role that zinc plays in wound healing, has proven to be successful in programs designed to combat lameness and related foot problems in cattle. However, apart from some evidence suggesting that zinc was effective in curing severe interdigital lesions (Demertzis and Mills, 1973) few reports could be found to support Schugel's statement. Supplementation of the ration with sulphur-containing amino acids is suggested to aid in the prevention of lameness and laminitis in particular. Feeding methionine hydroxy analogues to improve horn hardness and increase hoof growth rates has given inconclusive results (Clark and Rakes, 1982). Feeding iodine in the form of ethylenediamine dihydriodide to prevent experimentally induced footrot has also been studied (Berg *et al.*, 1976, 1984) but results were conflicting.

Housing design and layout

Where cattle are housed or confined to yards for lengthy periods to prevent lameness, attention must be directed at housing design, feeding facilities and floor surfaces. It is important that floors have non-slip surfaces but they should not be so rough that they are excessively abrasive. A method of measuring the skid resistance of floor surfaces has been developed that has allowed the characteristics of surfaces providing optimum skid resistance to be described (Albutt and Dumelow, 1987; Dumelow and Albutt, 1990). Stalls should be of sufficient size and length to be comfortable for the size of cattle housed (Pinsent, 1981). Potter and Broom (1987) and Metz and Wierenga (1987) have discussed aspects of housing design that influence cows' behaviour and welfare. Amstutz (1986) has also provided some detail on methods for floor construction and building design that should be adopted if lameness is to be prevented.

Raceway maintenance and yard design

Chesterton (1989) has discussed the importance of raceway maintenance in facilitating the flow of a dairy herd along the farm track when moving to and from the dairy for milking. Congestion points along the track should be eliminated including those at the entrances to holding yards. The track surface should be kept well crowned to promote good drainage and be constructed of non abrasive material such as limestone. Similarly problem areas such as junctions between metal and concrete and areas around water troughs should be kept well drained and free from highly abrasive material. Anything that could make cows fear entry to the milking shed environment, such as slippery concrete or stray voltage, should be eliminated.

Basic principles of road construction that should be used in race construction have been described by Bridges (1985a,b) who also provides recommendations on race width for different sized herds. Dewes (1978) emphasised the need to reduce angles in the approach to yards and suggested a walkover bridge made of perforated steel be placed at the entrance to holding yards to reduce the amount of surface rock material that is routinely carried in by cows. A suggestion for reducing the abrasive effects of concrete at strategic points (e.g. in rotary sheds at the points where cows back off the platform or in areas of concrete races where cows are forced to turn) has been to place carpet or rubber mats at these points (Malmo, 1991).

Selective breeding

Baggott and Russell (1981) suggested that selective breeding is likely to be important for the control of lameness. Selection of bulls based on susceptibility to claw disorders in their daughters may reduce the incidence of lameness (Russell *et al.*, 1986). However, the heritability of the visually assessed trait referred to as "feet and legs" is low (Greenough, 1991). Politiek *et al.* (1986) concluded that the heritabilities and added genetic variation for claw shape measurements and hoof disorders are high enough to obtain genetic change in these traits. Ral (1990) also found the heritabilities of objectively measured hoof traits were high but they were low for horn content of minerals and amino acids. It is clear that objective measurements will need to be used to assess limb angulation and claw conformation if these traits are to be used in the process of genetic selection.

Footbathing

Footbaths have been widely recommended to control and prevent lameness in dairy herds (Toussaint-Raven and Cornelisse, 1971; Weaver, 1974; Greenough *et al.*, 1981; McDaniel, 1983). Formalin and copper sulphate solutions are the most commonly used materials. They are used to remove erosive materials from the foot, to reduce bacterial contamination and to harden the hoof horn.

Footbaths have been advocated for the control of many different foot lesions including both interdigital conditions and disorders of the claws. Effectiveness in preventing and controlling interdigital dermatitis, interdigital necrobacillosis, heel horn erosion, white line disease and lesions involving injury or excessive wear of the claw horn have all been mentioned (Edwards, 1980; Baggott and Russell, 1981; Cornelisse *et al.*, 1982; Davies, 1982; Gibson, 1984/85; Amstutz, 1985; Toussaint Raven *et al.*, 1985). However, in a review of special procedures used for treatment and prevention of foot disease, Nelson and Petersen (1984) suggest that the effectiveness of footbaths in the prevention of lameness has not been thoroughly examined. Some reports have indicated that they do reduce lameness from diseases involving the interdigital skin but are less effective in treating diseases of the hoof horn (Arkins, 1981; Arkins and Hannan, 1983; Arkins *et al.*, 1986; Russell, 1984/85; Roztocil *et al.*, 1988).

Recommendations on frequency of application, concentration of active ingredient and contact time required for effect have also varied widely. Consequently a wide range of footbathing strategies has been adopted (Sumner and Davies, 1984). It is common knowledge that formalin is an irritant solution and that repeated applications with solutions that are too concentrated will have a necrotising effect. Arkins *et al.* (1986) reported twice daily bathing produced hyperaemia and skin necrosis. Baggott and Russell (1981) recommended that formalin not be used more than once or twice per week or the hoof horn will become too brittle. In studying the harmful effects of formalin in work with sheep, Littlejohn (1972) found too frequent application was more damaging than concentration, as weekly applications of 10 % solutions were well tolerated whereas the same exposure twice weekly produced hyperaemia, hyperkeratinisation and necrosis of the interdigital skin.

There are no published reports of controlled trials conducted in New Zealand or Australia using protective hoof treatments. However, in a herd case-control study Chesterton *et al.* (1989) did observe that herds with a high lameness incidence were twice as likely to be using a formalin footbath than were low lameness incidence herds. This positive association between the presence of a footbath and lameness was suggested by these authors to be more probably an effect of high lameness incidence than a cause. They proposed that farms encountering more problems with lameness tried to solve them by using a footbath but this did not solve the underlying causes of the lameness problem.

Hoof trimming

In dairying systems where cattle are housed or kept in confinement for lengthy periods hoof trimming once or twice per year is recommended (Baggott and Russell, 1981; Pinsent, 1981; Russell, 1984/85). Amstutz (1986) recommends that examination and trimming of the hooves should begin in calves at the early age of six months. However, Arkins (1981) considered hoof trimming in Irish herds would not be justified unless the incidence of lameness is high. Manson and Leaver (1988b, 1989) reported that trimming claws, to maintain a short dorsal border and steep angle, prior to parturition, reduced significantly the number and duration of clinical lameness cases in their studies. The timing of hoof trimming can be important (Enevoldsen and Grohn, 1991a). Where cattle graze pasture year-round and walk long distances each day on gravel or concrete, hoof

trimming is rarely required. In fact excessive hoof wear can cause problems (Dewes, 1978; Malmo, 1991).

The Dutch method of hoof trimming as described by Toussaint Raven *et al.* (1985) is gaining increasing favour (Collick, 1990). It has as its objectives: a) the correction of the overburdening of the hind lateral claw, b) restoration of a normal bearing surface within each claw, and c) correction of developing claw lesions and defects at an early stage.

Vaccination

Formalised *Fusobacterium necrophorum* vaccines have been helpful in controlling outbreaks of interdigital necrobacillosis (Dawkins, 1969) but their use has not been widespread and has now largely been abandoned (Weaver, 1974; Malmo, 1985). Immunity from these vaccines is short lived if achieved at all (Egerton, 1979). An attempt has been made to improve the immunity against interdigital necrobacillosis with experimental vaccines containing fractions of *F.necrophorum* in a mineral oil adjuvant Clark *et al.* (1986).

Aspects requiring further research

This review raises several questions relating to the epidemiology and control of lameness in pasture-fed cattle that require investigation. Some of these include:

1. Is the epidemiology of subclinical hoof lesions similar in New Zealand to that described by European and North American workers? This is addressed in Chapter 4.

2. What are the common lesions responsible for lameness in New Zealand herds and what cow factors are associated with lameness? Apart from the work of Dewes (1978) little information has been published. This question is the focus of the study described in Chapter 5.

3. Excessive hoof wear is regarded as a common predisposing cause of lameness in Australia and New Zealand dairy-farming systems but there is little

information available on the dynamics of hoof growth and wear in cattle kept in these systems. (Chapter 6).

4. Which hoof properties are important when considering modes of mechanical failure that result in foot lesions of cattle and do these properties vary with changes in environmental moisture conditions? (Chapter 3 and 7).

5. To what extent do the physical properties of hoof vary between cows and digits and is variation in these properties associated with lameness? (Chapter 8).

6. To what extent do available hoof treatments alter the physical properties of hoof and are they effective in preventing claw disease in cattle? (Chapters 9 and 10).

CHAPTER 3

Methods used for evaluating the physical characteristics of bovine hoof[1]

[1] Submitted as: Tranter WP, Morris RS, Morgan DE, Cann B. New Zealand Veterinary Journal, 1992.

Abstract

Methods used to measure the physical characteristics of the hooves of cattle in epidemiological studies of lameness are described. An engineer's profile gauge was used to measure sole concavity, a hand-held moisture meter for hoof moisture measurements and a Shore Type D Durometer for hoof hardness. Sole wear was evaluated by monitoring changes in sole concavity and by recording the length of time taken for 1.5 mm deep grooves, placed in the weight-bearing surface of hooves, to disappear. Hoof wall growth and wear were studied by monitoring the movement of a small hole, placed in the cranial toe wall, towards the end of the claw. Methods were developed for obtaining biopsy samples from wall and sole of the hooves of cows in commercial dairy herds without injury to the cows. Elastic modulus, compressive strength and resilience of hoof wall and sole were measured using compression tests on samples collected by these methods.

Introduction

The horny capsule which forms the outer part of the claws in cattle is largely responsible for protecting the foot from the external environment. The ability of the claw to withstand the environment depends mainly on its physical properties. However, it remains unclear which hoof properties are important for protecting cows against lameness.

Various workers have shown that an association exists between rainfall and the incidence of lameness in dairy herds (Eddy and Scott, 1980; Arkins, 1981; Rowlands *et al.*, 1983; Williams *et al.*, 1986). Most veterinarians and farmers theorise that hooves become wetter and softer during wet weather and that this softening predisposes to lameness. Hence hardness and dryness are commonly considered to be important protective factors for lameness. However, hooves that are too dry become brittle. Under some pasture-based grazing systems, where cows are forced to walk long distances and excessively worn soles can produce lameness (Dewes, 1978; Malmo, 1991), the resistance of hoof tissue to abrasive wear must be considered as a factor influencing hoof disease. Resilience, which is a measure of the ability of a material to absorb energy up to the point where tissue failure occurs, is another property worthy of consideration. In their studies of the effects of dietary supplementation of biotin on pig hoof horn, Webb *et al.* (1984) have suggested that other properties of hoof, such as its compressive strength and elasticity, are also important when considering modes of mechanical failure that result in foot lesions of livestock.

Studies have been undertaken to ascertain if variation in the physical nature of cows' hooves is associated with their ability to resist external damaging influences and they are described elsewhere (Tranter *et al.*, 1992b, 1992c; Tranter and Morris, 1992). It has proved difficult in reviewing the literature to determine with adequate precision the methods which have been used to measure various physical characteristics of hooves. This paper therefore amplifies the brief description of methods given in other papers in this series, to document the techniques and equipment which have been used to measure the physical properties of the hooves of live animals. It also describes methods used to obtain and process hoof samples from animals in commercial herds, without adverse effects.

Cowside measurements and collection of hoof samples

1. Colour

Sole, heel and wall colour were recorded using a 1 to 5 scoring system (1 = 100% white, 2 = 25% black, through to 5 = 100% black). As the pigment in the wall horn of Friesian cows is often deposited only in the superficial layers, this colour score referred to the colour of the surface layer of horn.

2. Sole Concavity

Few reports of sole concavity measurements have been published. Gilmore (1978) measured the area of sole concavity in trials comparing cows kept in comfort stalls with those kept in free stalls. However the methods used were not clearly described.

In our studies a profile gauge (Figure 3.1), which is a device used in engineering to reproduce erratic profiles, was used to measure the contour of the weight-bearing surface of hooves at their widest point. A tracing of this contour was then drawn on paper. From this record, the depth of the sole was measured at 10 mm, 20 mm, and 30 mm from the abaxial hoof margin of each digit, using vernier callipers (Figure 3.2).

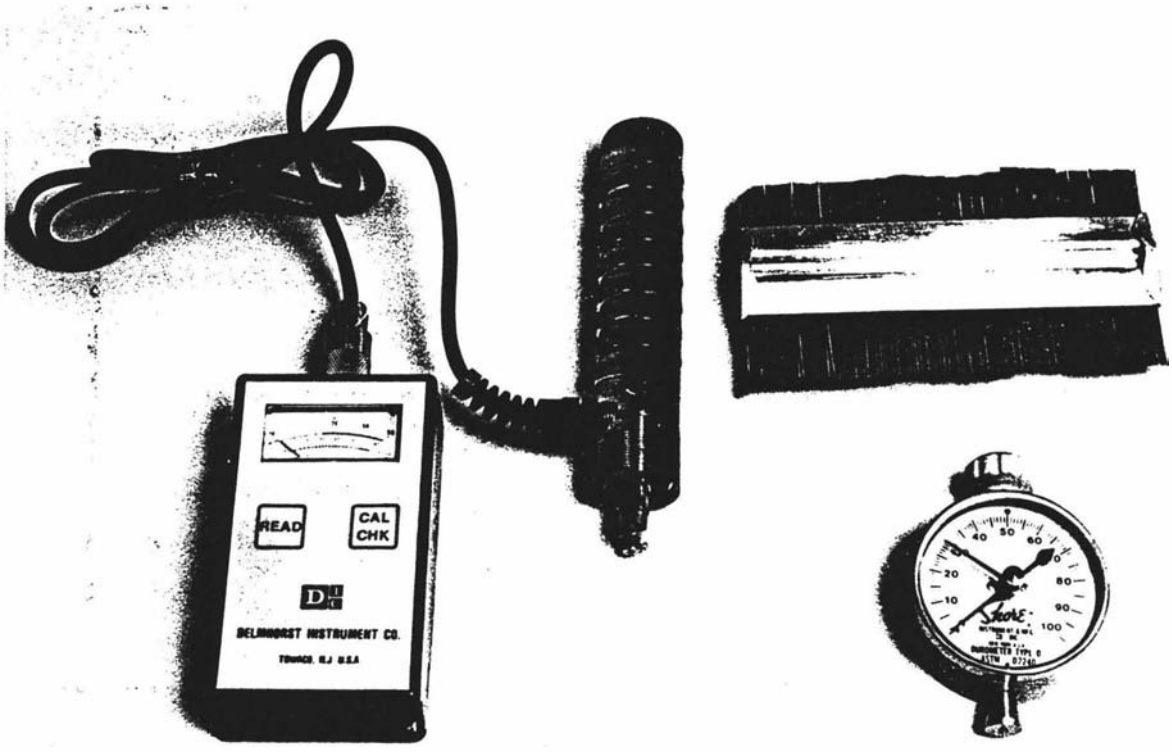


Figure 3.1. Instruments used to measure sole concavity, hoof moisture content and hoof hardness of the digits of live cows. Left - Delmhorst moisture meter. Top right - profile gauge. Bottom right - Shore Type D durometer.

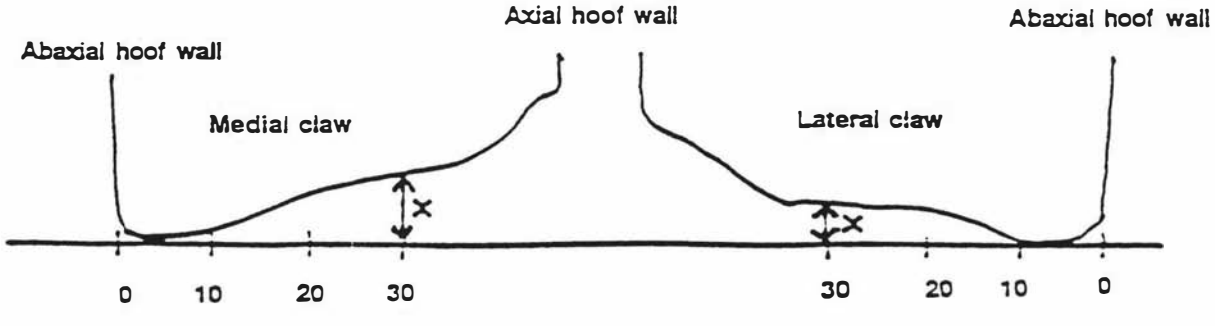


Figure 3.2. An example of a tracing of the contour of the weight-bearing surface of a cow's hoof. Measurement x is the sole concavity in mm.

3. Hoof wall growth and wear

Many workers have measured hoof growth and wear by placing a mark in the hoof wall and monitoring its displacement, from either the periople line or a reference point tattooed in the skin above the coronary band, toward the end of the claw. Wear and growth have been obtained by repeated measurements over time. Slight variations of technique have been used. Some have used a soldering iron to make small marks in the wall (Hahn *et al.*, 1984, 1986); some have drilled a small hole (Clark and Rakes, 1982; Manson and Leaver, 1988b; Vermunt,1990); while others have used branded marks or kerfs made with a saw blade or file (Prentice, 1973; Pflug *et al.*, 1980; Murphy and Hannan, 1987; Schlichting, 1987; Reilly and Brooks, 1990).

In our studies, cows were first tranquillised by injecting 12-15 mg Xylazine hydrochloride intravenously and measurements made on the standing animal. The hooves were scrubbed and a small hole was placed in the cranial toe wall 10-20 mm below the periople line with a soldering iron. The distance from the periople line to this hole and the distance from this hole to the bearing surface of the claw were measured at approximately 3-monthly intervals using vernier callipers. On each occasion all measurements of distances between the two points were repeated and the two distances averaged. Changes in these distances represented hoof wall growth and hoof wall wear. The original holes used as reference points were replaced with new ones closer to the periople before they grew out of the distal wall surface.

4. Sole wear

In contrast to the large numbers of studies on hoof wall growth and wear, there is little published information on sole growth and wear. Greenough *et al.* (1990) calculated net growth rates for sole horn by measuring sole horn thickness in hooves of beef steers after slaughter.

Sole wear was monitored in our studies by recording the number of weeks taken for 1.5 mm deep grooves to disappear from the weight-bearing surface. Three parallel lines were drawn (abaxially to axially) across the sole surface - one in the toe region, one in the mid-sole and one on the heel bulb. A battery powered drill, with the drill head adapted to penetrate only 1.5 mm, was used to drill along the lines placing three grooves in the sole. These digits were examined each week and the presence or absence of each groove at 10

mm, 20 mm and 30 mm distances from the abaxial hoof margin was recorded until all grooves had completely worn out (Figure 3.3).

Another measure of sole wear used was the change in sole concavity with time. Under some management systems, sole concavity decreased after calving when cows entered the milking herd and began walking long distances to pasture.

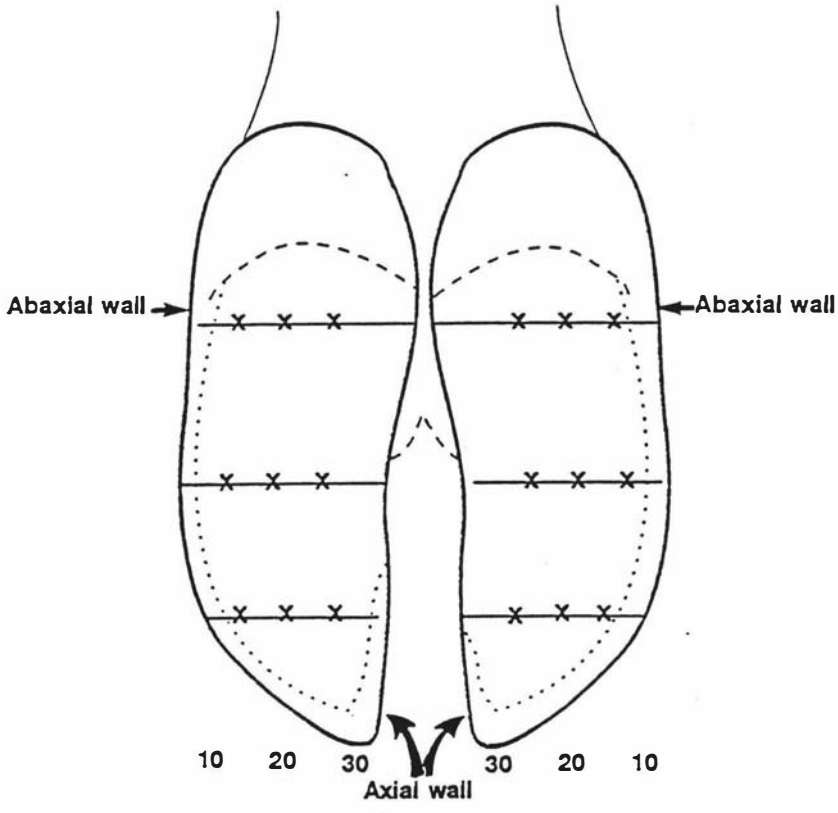


Figure 3.3. **Position of the grooves placed in the weight-bearing surface of the cows' hooves. x indicates the sites 10 mm, 20 mm, and 30 mm from the abaxial hoof margin at which observations for groove disappearance were made. The dotted line represents the white line.**

5. Hoof hardness

Various workers have measured hoof hardness both on live animals and on hooves collected from the abattoir. A modified penetrometer, an instrument for evaluating the degree of compaction of soils, was used by Clark and Rakes (1982) to measure hoof hardness in Friesian cows. In most other studies Shore Durometers have been used (Martig *et al.*, 1980; Webb *et al.*, 1984; Distl *et al.*, 1984; Baggott *et al.*, 1988; Manson and Leaver, 1988b, 1989; Reilly and Brooks, 1990).

A durometer is a hand-held instrument designed to determine the indentation hardness of materials such as rubber or leather. Hardness is determined by indentor penetration into the material with a calibrated known force, and the result is displayed on a scale graduated equally from 0 to 100. Different types of durometers using different calibration scales are available with main-springs of varying deflection, and indentors of varying contour to allow for the testing of a wide variety of materials. Readings below 10 and above 90 are not considered reliable.

For our work, a Type D Durometer (Model XDMXHAF) (Shore Instrument and Manufacturing Co., 80 Commercial Street, Freeport, New York) with a maximum reading pointer and a sharp 30 degree angle indentor was used (Figure 3.1). This model was chosen as the most suitable as hoof hardness readings ranged from 25 Shore units for soft heel hoof to 85 for hard wall material, all within the range of reliable function of the instrument. Another reason for choosing this model of durometer was the small size of the presser foot (0.5 inch diameter) which required only a small area to be prepared prior to measurement.

Because hoof exhibits the characteristic known as cold flow or creep (whereby the dial hand of the durometer recedes after the durometer is applied), it was necessary to read the gauge at a specified time after compression. Ideally this would have been at a fixed time interval, e.g. five seconds, after the start of the test. However, it was anticipated that it would often be easier in live cows to take readings at time 0 seconds using the instrument's maximum reading hand. To evaluate the relationship between readings taken at time 0 seconds (using the maximum reading hand) and readings taken 5 seconds after compression, pairs of hardness measurements were performed on hooves collected from six cows after slaughter. The hooves were stored overnight at 4°C and were submitted to hardness tests the following day. Hardness was measured at the three sites on each digit as shown in Figure 3.4. The mean fall in hardness between readings taken at time 0 seconds and time 5 seconds was 2.8 ± 1.5 Shore units. Simple linear regression was used to evaluate the relationship between the two different hardness readings (Table 3.1). There was a high correlation between the two readings ($r = 0.99$) and it was concluded that readings taken using the maximum reading hand would be suitable for routine measurement of hoof hardness in live cows.

Table 3.1. **Regression relationship between hoof hardness measured at time 0 seconds (independent variable) and hoof hardness measured at 5 seconds after the start of the test (dependent variable)**

Correlation coefficient	0.99
Regression constant	- 1.62
Regression coefficient[a]	0.98 ± 0.01
ANOVA: DF	143
F ratio	15343
p	< 0.001
Coefficient of determination	0.99

[a] Values are quoted as means ± standard error

To assess the repeatability of measurements made using the Durometer, a second hardness measurement (using the maximum reading hand) was performed at each of the three sites on the same hooves. Differences within pairs of measurements were small as shown in Table 3.2.

Table 3.2. **Sole, heel and wall hardness of the hooves of all eight digits of six cows collected after slaughter**

Site of measurement	n	Mean[a,b]	Mean difference between pairs of readings taken on each digit[a]
Sole	48	52.8 ± 3.1	1.2 ± 1.1
Heel	48	35.8 ± 8.5	1.6 ± 1.5
Wall	48	69.9 ± 3.3	1.5 ± 1.4

[a] Values are quoted as mean ± standard deviation.
[b] Values are the means of the means of pairs of hardness readings taken at each site of measurement.

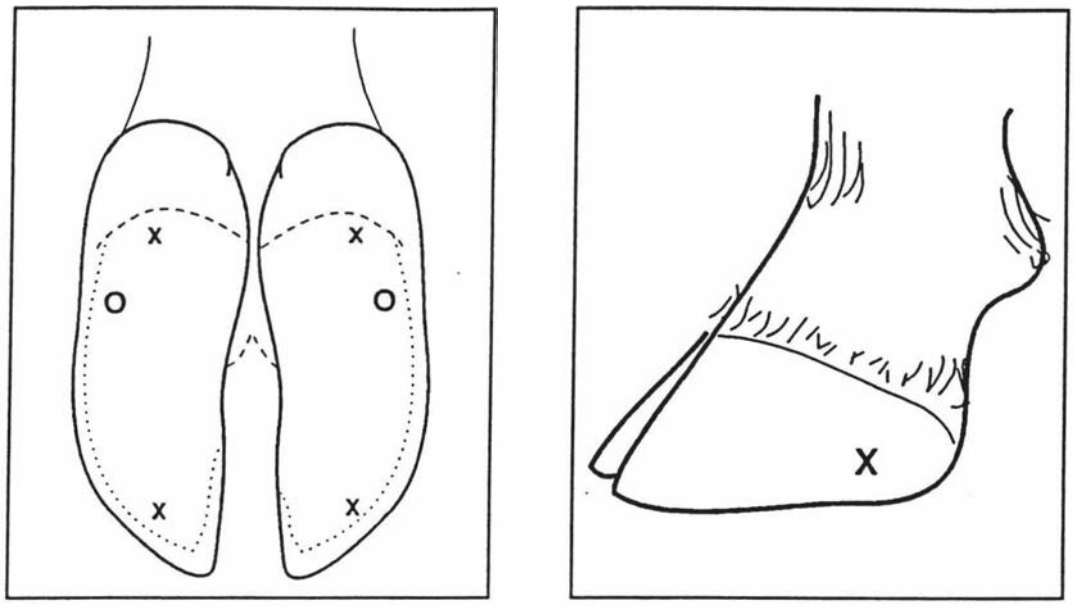


Figure 3.4. **Sites of moisture and hardness measurements and hoof biopsy. X indicates sites of moisture and hardness measurement; O indicates sites for collection of sole samples.**

When measuring hoof hardness in live cows, hooves were first lifted and washed, then dried with paper towelling. In order to provide a reasonably smooth and flat surface for the base of the durometer, the hoof surface was lightly sanded over the sites of measurement, using a battery-powered sander, to remove dirt and surface irregularities. Two hardness readings were taken on the sole, heel and the abaxial wall at the positions shown in the Figure 3.4. Each pair of readings was averaged and the results used as measures of sole, heel and wall hardness.

6. Hoof moisture

Previous workers have assessed hoof moisture by collecting hoof shavings from the sole surface and oven drying the samples until they reached a constant weight (Martig *et al.*, 1980; Arkins *et al.*, 1986; Thysen, 1987). Others have collected full depth hoof samples from abattoir feet and again subjected these samples to drying. Hoof moisture levels increase from exterior to interior (Leach and Zoerb, 1983) so in measuring moisture, reproducibility of depth of measurement is critical.

A method was sought that would allow for measuring hoof moisture content quickly and reliably in live cows under field conditions. A Delmhorst Moisture detector, Type BD-7, with 2E electrodes (Pacific Scientific, Gardner/Neotec Instrument Division, 1100 East-west Hwy, Silver Spring, MD 20910, USA) was used (Figure 3.1). This moisture detector is a portable instrument for measuring the moisture content of materials such as wood, plaster, or asbestos shingles to ascertain if moisture levels are too high for safe painting or application of adhesives. Moisture levels were measured at the same three sites used for measuring hardness (Figure 3.4), using electrodes adapted to ensure they always penetrated to a depth of 3 mm.

Hooves were washed, dried with paper towelling and then lightly sanded to remove the surface layer of dirt and moisture. The probes of the instrument were then inserted to a depth of 3 mm and one reading taken at each site. Care was taken when measuring heel moisture to gently insert the probes to the correct depth since it was possible to force the probes deeper than the desired 3 mm due to the softness of hoof at this site.

Values obtained for hoof moisture using the moisture meter were compared with those obtained by oven drying hoof biopsy samples using hooves collected from the abattoir. The hooves were stored overnight after slaughter in plastic bags at 4°C and were submitted to moisture tests the day following slaughter. Moisture levels were measured at three sites on 32 digits (sole, heel and abaxial wall) as shown in Figure 3.4. Moisture readings were taken at each site using the moisture meter. Since the probes of the meter were 20 mm apart, two hoof samples were collected from each site (one from each site of electrode penetration) for drying. Each sample was collected by first making a circular cut with a 10 mm diameter surgical trephine to a depth of 4 mm and then removing the core using a wood carver's gouge. The internal surface of each sample was then sanded back until the sample's thickness was 3 mm, this being the full penetration depth of the electrodes of the moisture meter. Samples were weighed, placed in an oven at 100°C for 10 days and then reweighed. The difference between the two weight measurements for each sample was its actual moisture content and this difference expressed as the proportion of the sample's initial weight was its moisture percentage. Mean values for each pair of samples were compared with values obtained using the moisture meter prior to sample collection.

The simple linear regression relationship between actual moisture percentage based on oven drying (independent variable) and hoof moisture percentage obtained using the meter (dependent variable) was evaluated (Table 3.3). When measurements from all three sites were included in the analysis meter readings were highly correlated with actual sample moisture percentages (r = + 0.85). Analysis for measurements within each site revealed that the correlation between the two variables was lower for heel measurements than for those from the sole or wall. The scatter plot shown in Figure 3.5 also demonstrates that the moisture meter readings were highly correlated with hoof moisture values obtained by drying except where hoof moisture content was higher than 35%. Above this level there was much greater variability around a 1:1 relationship between moisture measurements obtained using the two different methods. However, moisture levels above 35% are usually only found in heel hoof. It was concluded that the meter was a useful tool for measuring hoof moisture content but readings taken from heel hoof would need to be interpreted with caution.

Table 3.3. **Regression relationships between actual hoof moisture percentage as determined by oven drying (independent variable) and hoof moisture percentage measured using a hand-held moisture meter (dependent variable)**

	Sole	Heel	Wall	All sites
Correlation coefficient	+0.83	+0.65	+0.81	+0.86
Regression constant	-7.00	-34.18	0.82	-19.89
Regression coefficient[a]	1.24 ± 0.16	2.12 ± 0.46	0.95 ± 0.13	1.69 ± 0.10
ANOVA:				
DF	30	30	30	92
F ratio	62.39	21.29	57.38	263.02
p	< 0.001	< 0.001	< 0.001	< 0.001
Coefficient of determination	0.68	0.42	0.65	0.74

[a] Values are quoted as means ± standard error

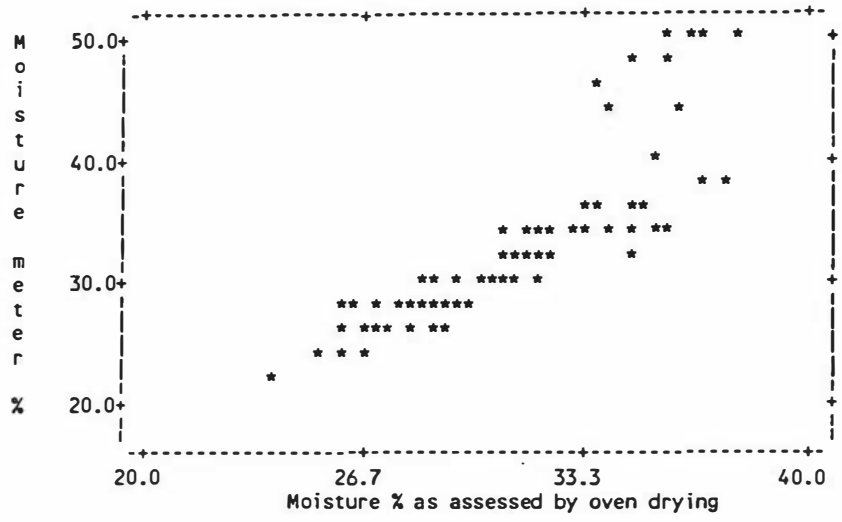


Figure 3.5. **Scatter plot of moisture percentage obtained using the hand-held moisture meter vs moisture percentage as obtained by oven drying**

7. Collection of biopsy samples

Methods were developed for collecting hoof samples from live cows for further *in vitro* testing. A toe clipping was taken with foot trimmers and, after preparation as described below, was used to provide a sample of hoof wall. A sole sample was collected from near the sole-heel margin (Figure 3.4) by first making a circular cut with a 10 mm diameter surgical trephine to a depth of approximately 4 mm. A core sample was then removed from the foot using a wood carver's gouge, taking care not to penetrate the sensitive corium. After removal of the sample the edges of the resulting hole and surrounding sole hoof were pared and smoothed to prevent dirt impacting in the biopsy site. In cases where this was not done, the biopsy site remained identifiable for much longer, and posed a risk of initiating a lesion at the site. All toe clippings and sole core samples were placed in capped plastic bottles immediately after collection and were stored overnight at room temperature.

These techniques have been used successfully in over 400 digits, in some cases on up to four occasions over the course of a year, without causing any detriment to the cows from which samples have been collected.

In vitro methods

1. Compression tests

a) Sample preparation

From the samples collected, small rectangular blocks of hoof were carefully prepared by standardised procedures. Sole samples were held in custom made holders, and sanded back to a depth of approximately 3 mm by removing the interior surface using a power sander. This produced a circular core of hoof which was then machined into a rectangular block approximately 6 mm x 4.5 mm x 3 mm using the equipment displayed in Figure 3.6.

Toe clippings were first mounted using a polyester resin in petri dishes (Figure 3.7). Using a small, purpose-designed jig saw (Figure 3.8), blocks of hoof wall were cut from the mounted specimens (Figure 3.7). They were then machined to a size similar to that of the sole samples. During preparation care was taken to make saw cuts parallel to the horn tubules of the wall.

The dimensions of each sample were measured with vernier callipers and they were then placed in capped vials. Earlier work had shown that weight loss due to drying with the samples contained in such vials was 0.5% - 1.0% of the initial sample weight per 24 hours. Apart from capping the vials, no other attempts were made to control humidity during sample preparation and storage but all samples were subjected to similar conditions. Practical constraints meant that compression tests had to be performed on the day following field measurements and sample collection. It is accepted that some minor changes may have occurred in physical hoof properties between sample collection and performance of the tests but this delay was unavoidable and was kept constant. Sample preparation was always performed on the day following collection and compression tests were always performed within 6 hours of machining.

Compression tests were performed at room temperature using an Instron Universal Testing machine. Sole samples were compressed in the exterior-interior direction while wall samples were compressed vertically. This meant that for all samples compression was parallel to the direction of the horn tubules. All samples were compressed using a crosshead speed of 5 mm per minute. Force deformation curves were charted on a synchronously-driven recorder. Typical curves produced for tests on both sole and wall hoof are shown in Figure 3.9.

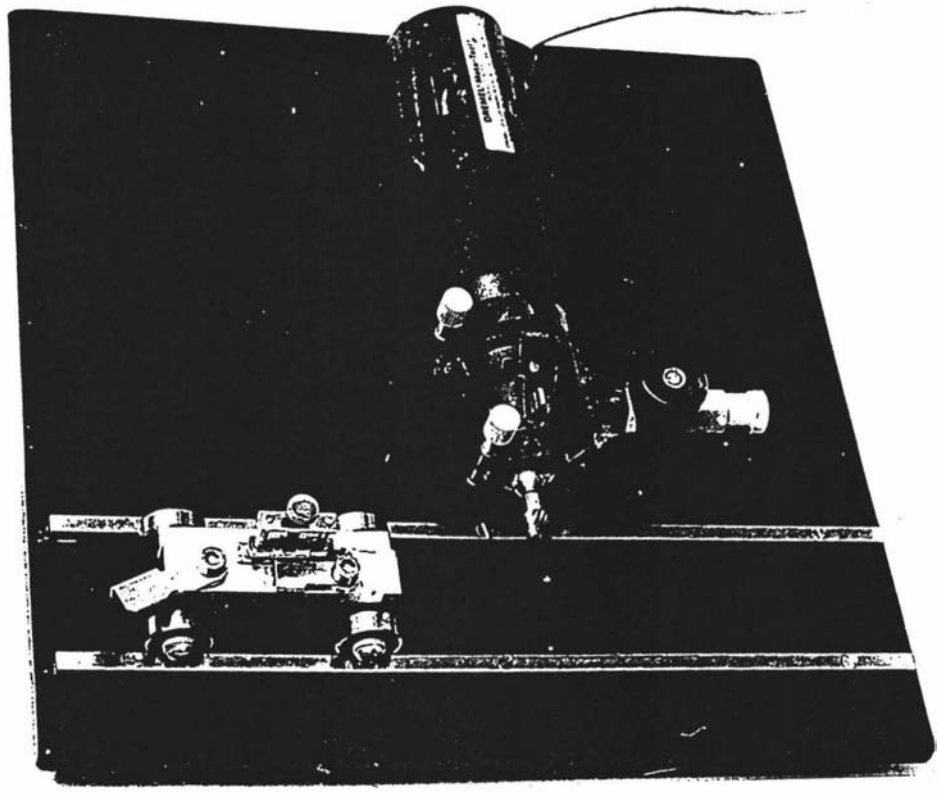


Figure 3.6. Equipment used to machine circular hoof biopsy samples, collected from the soles of the digits of cattle, into rectangular blocks for compression tests

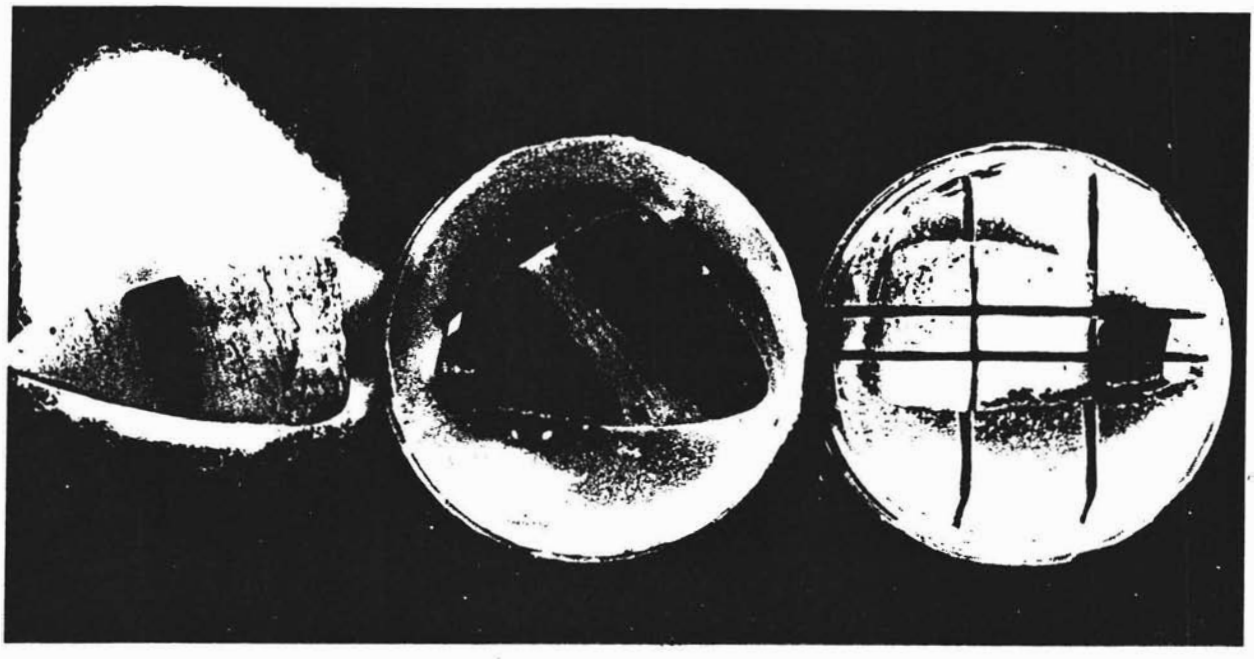


Figure 3.7. Toe clippings from which rectangular blocks of hoof wall were collected for compression tests. Left - toe clipping as first collected. Centre - toe clipping mounted in a petrie dish using a polyester resin. Right - toe clipping on which saw cuts have been made parallel and perpendicular to the horn tubules of the wall.

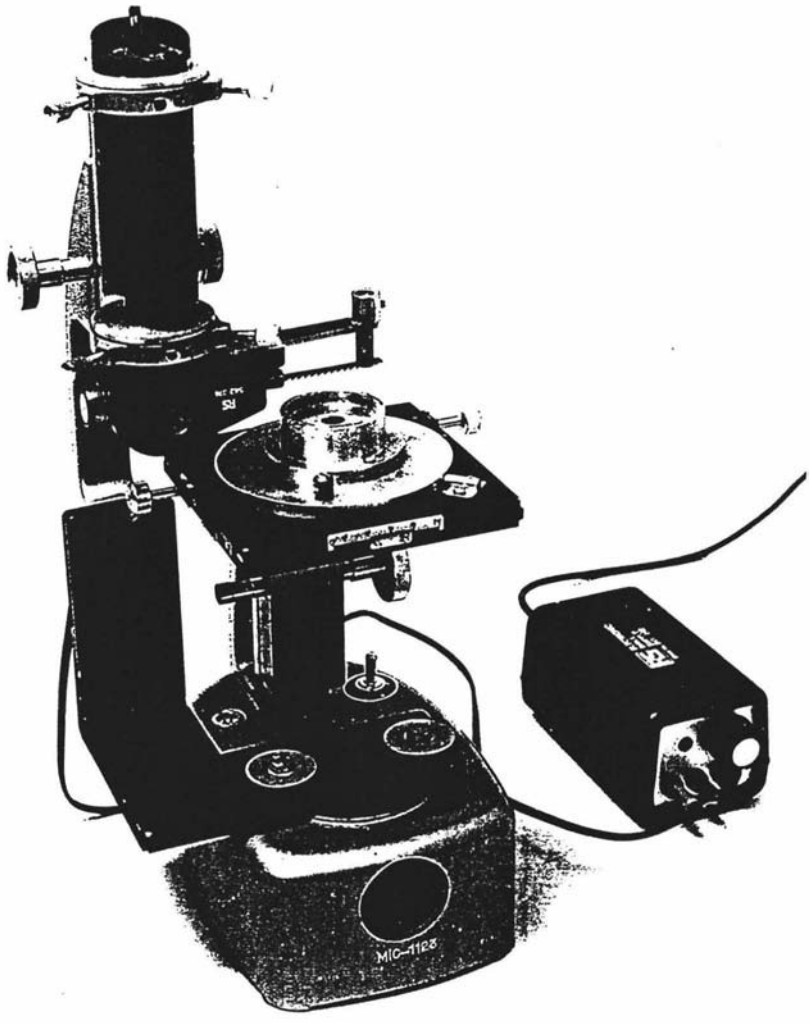


Figure 3.8. **Purpose-designed jig saw used to make cuts in toe clippings during the preparation of rectangular blocks of hoof wall for compression tests**

In preliminary work, some compression tests were performed on the circular biopsy samples prior to their being machined into rectangular blocks. These samples were more difficult to work with as on some occasions compression produced a continuously sloping curve from which no straight line portion could be used for slope measurement. This was considered to be due to the relative width of the samples when compared to their thickness. When assessing the shearing properties of a material, the sample width should be substantially less than twice its thickness since failure usually occurs along the 45° shear lines. Compressing samples that are much wider than they are thick, such as the circular core samples were, results in much of the sample undergoing pure compression with only the edges suffering failure. In such instances typical curves as shown in Figure 3.9 may not be produced.

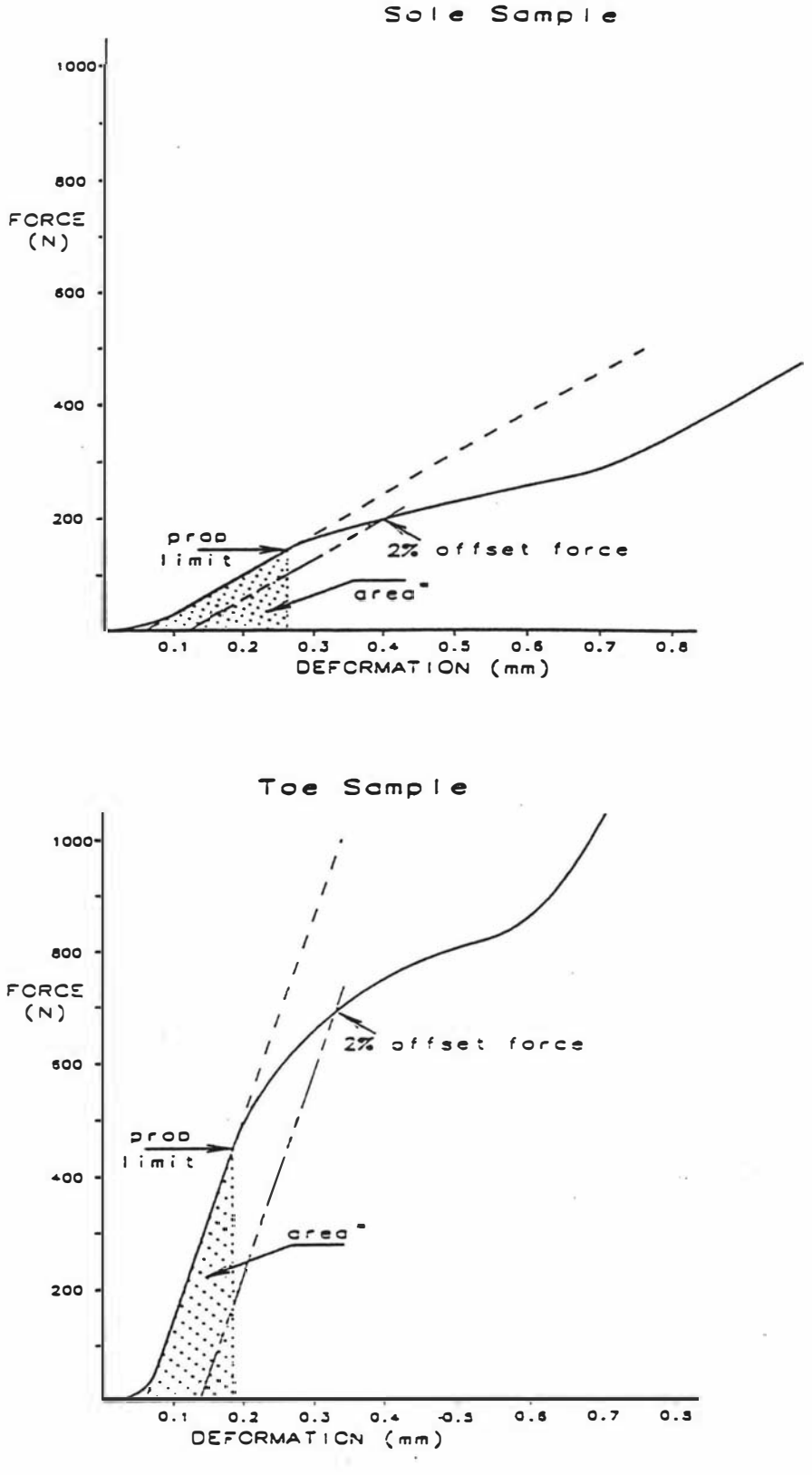


Figure 3.9. **Example force-deformation curves produced when hoof samples were compressed. Resilience is represented by the area under the curve (area*) up to the proportional limit corrected for volume of the sample. The elastic modulus for each sample was derived from the slope of the straight line portion of the curve and the compressive strength taken as the stress corresponding to a 2% offset strain of the sample.**

b) Calculation of elastic modulus

The elastic modulus is a measure of the stiffness or rigidity of the material and is derived from the slope of the initial straight line portion of the force-deformation curve. This is well described in the work performed on equine hoof wall tissue by Leach and Zoerb (1983). The formula used is:

$$E = \frac{\text{stress}}{\text{strain}}$$

where E = elastic modulus

$$\text{stress} = \frac{\text{force}}{\text{sample surface area}}$$

$$\text{strain} = \frac{\text{change in depth}}{\text{original depth of sample}}$$

c) Compressive strength

The compressive strength is a measure of the force required to produce tissue failure and has been measured previously by Webb *et al.* (1984) in their studies of pig hoof. Webb *et al.* (1984) describe this property as the stress corresponding to the point at which cellular structures are ruptured. In keeping with the convention used by these workers, the yield point was taken as that point on the force deformation curve corresponding to a 2% offset strain of the hoof sample. Division of the yield force by the cross sectional area of the sample before compression gave the yield stress or strength.

d) Resilience

Resilience is a measure combining both elasticity and strength. As measured in our study it represents the energy required to deform the hoof sample up to the point on the curve where it becomes non-linear (Figure 3.9). It was represented by the area under each curve up to the proportional limit corrected for volume of the sample. For simplicity of calculation and since the elastic modulus of each sample had already been derived, the formula used for resilience was:

$$R = \frac{(\text{PL stress})^2}{2E}$$

where R = resilience

$$\text{PL stress} = \frac{\text{force at proportional limit}}{\text{surface area of sample}}$$

and E = elastic modulus.

e) Abattoir studies

Hooves were collected from all four legs of five cows after slaughter and were stored overnight in plastic bags at 4°C. The sole and hoof wall hardness of each claw were measured using a durometer and then pairs of rectangular block samples were collected from toe clippings and from adjacent positions on the sole at the site shown in Figure 3.4. They were then prepared as described above. Elastic modulus was measured on 39 pairs of sole samples and 15 pairs of wall samples. Compressive strength and resilience were measured on approximately 15 pairs of both wall and sole samples. The results of the mean values of each pair of samples and the mean values of the differences within each pair are presented in Table 3.4.

Table 3.4. Elastic modulus, compressive strength and resilience of pairs of samples collected from toe clippings and from the sole of hooves harvested after slaughter

	n	Mean[a]	Mean difference between pairs[a]
Sole pairs:			
Av. elastic modulus (MPa)	39	66 ± 16	13 ± 2
Av. compressive strength (MPa)	14	7.3 ± 1.4	1.8 ± 1.4
Av. resilience (kJ/m^3)	15	231 ± 84	88 ± 56
Wall pairs:			
Av. elastic modulus (MPa)	15	343 ± 66	92 ± 80
Av. compressive strength (MPa)	15	29 ± 5	3.5 ± 2.6
Av. resilience (kJ/m^3)	15	770 ± 285	419 ± 449

[a] Values are quoted as mean ± standard deviation

The associations between hoof hardness and each of these three properties was evaluated using simple linear regression analysis. For sole hoof, elastic modulus and compressive strength were positively correlated with hardness and the relationships when tested by ANOVA were significant ($p < 0.05$) (Table 3.5). In contrast, resilience of sole samples was poorly correlated with sole hardness. For wall hoof, all three properties were poorly correlated with wall hardness (Table 3.6).

Table 3.5. **Regression relationships between sole hardness (independent variable) and other sole properties measured by compression test (dependent variables)**

	Elastic modulus	Compressive strength	Resilience
Correlation coefficient	+0.62	+0.44	+0.09
Regression constant	-107.5	-3.06	100.7
Regression coefficient[a]	3.60 ± 0.69	0.23 ± 0.07	2.69 ± 4.25
ANOVA:			
DF	45	43	45
F ratio	26.87	10.27	0.40
p	< 0.001	0.003	0.53
Coefficient of determination	0.38	0.20	0.009

[a] Values are quoted as mean ± standard error

Table 3.6. **Regression relationships between wall hardness (independent variable) and other wall properties measured by compression test (dependent variables)**

	Elastic modulus	Compressive strength	Resilience
Correlation coefficient	+0.13	+0.18	0
Regression constant	-277.6	-19.7	802
Regression coefficient[a]	7.82 ± 11.12	0.62 ± 0.66	-0.40 ± 52.14
ANOVA:			
DF	29	29	29
F ratio	0.49	0.89	< 0.001
p	0.49	0.35	0.99
Coefficient of determination	0.02	0.03	0

Values are quoted as mean ± standard error

2. Resistance to abrasive wear

Camara and Gravert (1971) measured abrasive wear of cattle hoof by moving claw pieces collected from slaughtered cattle across a concrete surface. Pflug *et al.* (1980) and Murphy *et al.* (1987) have also measured abrasive wear on hoof wall samples but again these tests were performed on samples collected after slaughter.

Attempts were made to develop a method for use on biopsy samples collected from sole, heel and wall hoof. Hooves were collected from cows after slaughter and stored overnight in plastic bags at 4°C. Circular core samples were collected by first making circular cuts with a trephine and then removing them from the foot with a wood carver's gouge. Each sample was further prepared by sanding the inner surface back to a depth of 5 mm. Abrasive wear tests were performed using a Model 5130 Teledyne Taber Abrasion testing machine (Teledyne Taber, 455 Bryant Street, North Tanawanda, NY 14120, USA). Samples were first weighed and then subjected to 100 cycles of abrasion, using sandpaper (grit size = P150) fastened to the rotating wheel of the abrasion testing machine. Samples were then reweighed and the weight loss for each sample during the test was used as a measure of wear.

Tests were done on pairs of samples from adjacent positions on the hoof. Within pairs there was considerable variation in the results obtained especially for samples obtained from the heel region. During the abrasion test, heel hoof tended to crumble unpredictably due to its softer texture whereas the harder hoof from other sites was worn down steadily by the typical sanding process. When results of abrasive wear tests on samples from adjacent positions on the hoof were compared using Student's paired t-test there was a significant difference between the samples of each pair ($t = 4.48$; $n = 96$; $p < 0.001$). Developing a method to hold each sample during the test also proved difficult due to their small size yet it would not have been possible to collect larger samples from live animals. Since this test did not give reproducible results it could not be used to obtain an indication of the resistance of hoof to abrasive wear. No other test was devised.

Discussion

Although lameness is one of the most common diseases of dairy cows, it has received far less attention from research workers than many other less important diseases. It is also one of the few major diseases for which curative treatment remains the main response to a lameness problem, because no methods have been widely adopted for prevention or control of the condition in individual cows. At the herd level, changes in management of cows and in the physical environment imposed on them offer the only practical

approach to reducing the severity of the problem in severely affected herds (Chesterton *et al.*, 1989).

Various reasons can be postulated for the paucity of research and the lack of progress in developing specific control procedures which the veterinarian can institute to reduce the likelihood of clinical lameness in individual cows. Firstly, lameness research almost always involves lifting the feet of many thousands of cows to examine them, and this is not seen as an attractive research activity. Secondly and more importantly, there have been few ways in which measurements could be made of the physical characteristics of hoof, especially on animals in commercial herds. Thirdly, it appeared to many that there was little prospect of progress in developing effective control procedures because lameness was seen as a largely random injury, more bad luck than bad management.

Earlier papers in this series have shown that under New Zealand conditions lameness is far from being a random event; in fact it is predictable in the digits affected, when they become affected with various types of lesions, and in the herd-level factors which influence the risk of a particular cow developing lameness (Tranter and Morris, 1991; Tranter *et al.*, 1991). Thus, since there is a pattern to the disease, it is likely that effective control measures can be identified by a structured program of appropriate epidemiological research.

One of the limiting factors on such research has been the difficulty of making satisfactory measurements of hoof characteristics which influence the physical performance of the foot during locomotion. Considerable progress has been made over recent years in developing or adapting methods of measuring features of hoof material which influence its biomechanical behaviour.

This paper describes various techniques which have been used for measuring the capacity of hooves to meet the physical demands put on them. In some cases methods have been adapted from those described by earlier workers, while in other cases totally new techniques have been developed. They are reported here in order to record their application for future workers in the field, and to offer practitioners some guidance on the features of hoof which they could practically measure under field conditions.

The concavity of the sole is an important feature associated with the occurrence of lameness in a particular digit (Tranter *et al.*, 1992b), and this can be measured very simply by the profile gauge. When a cow calves after a dry period, or especially when a heifer enters the herd for the first time, the weight-bearing surface of the sole is quite markedly concave, especially on the inner digit. After calving the hoof generally loses much of this concavity as the rate of wear imposed by walking to milking and being held in an abrasive concrete yard for lengthy periods prior to milking exceeds the rate of horn growth to compensate for the loss. Late in lactation it starts to return. The relationship between hoof growth, wear and the conformation of the bearing surface of the hoof is a much more dynamic one than veterinarians may envisage. The techniques for independently measuring growth, wear and concavity which have been described in this paper allow this relationship to be quantified. They have demonstrated a rapid removal of horn, such that externally applied chemicals would have relatively short persistence on the animal purely because treated horn would be abraded away over a few weeks to months.

Environmental moisture conditions influence the water content of hoof and this, in turn, affects other physical hoof properties such as hardness and elastic modulus (Tranter *et al.*, 1992c). The only method previously described for measuring hoof moisture in live animals has involved collection of hoof shavings and oven drying the samples until they reached a constant weight. Although use of the hand-held moisture meter provides only an estimate of hoof moisture content, it is a much simpler method for use in live cows. It also has an advantage in that by modifying the electrodes it is possible to be consistent with the depth of moisture measurement. This is difficult to achieve when hoof shavings are collected.

Hoof hardness is readily measured with a durometer. However, since hoof hardness varies greatly with position on the claw, it is essential to be consistent with site of measurement.

In contrast to hardness measurements, collection of hoof samples and measuring their elastic modulus, compressive strength and resilience is time consuming and labour intensive. It is possible, however, that variation in these properties is associated with lameness as has been postulated in other work (Tranter *et al.*, 1992c). Further, review of results, both from our preliminary studies using abattoir material (Tables 3.5 and 3.6)

and from later work on live cows (Tranter *et al.*, 1992c), shows that the correlation between these properties, as measured by compression tests, and the simpler measurements of horn hardness is, in most instances, quite low. Hence the compression tests appear to measure physical characteristics of hoof material which are distinctively different from hardness. For these reasons, the extra effort involved in measuring resilience, elastic modulus and compressive strength was considered to be justified.

With the exception of the abrasive wear test, all of the techniques described have been used extensively in epidemiological studies designed to determine what individual cow factors are associated with lameness in dairy herds. Other physical hoof properties mentioned in reports of lameness-related work that may also be important include fracture toughness, tensile strength, concussion resistance, elasticity, viscoelasticity and mechanical hysteresis. However measurement of most of these would either require larger samples of hoof or be very time consuming to perform, thereby limiting their application for routine use in field studies.

CHAPTER 4

A longitudinal study of the hooves of non-lame cows[2]

[2] Published as: Tranter WP, Morris RS, Williamson NB. New Zealand Veterinary Journal 39, 53-7, 1991.

Abstract

A longitudinal study of the feet of cows from a seasonal dairy herd was conducted over a 12 month period to measure the occurrence of subclinical hoof lesions. Eleven 2-year old cows, entering the herd for the first time, and eleven mature cows were randomly selected from a herd of 415 cows at the end of winter (July 1989) prior to calving. The incidence of lameness in the herd over the 12 months was 2%. None of the trial cows became lame during the study.

Cows were examined monthly. The type, severity and location of any hoof lesions were recorded. Sole haemorrhage, erosion of the heel bulb and minor white line separation were the most commonly observed lesions. These lesions were observed with specific claw distributions in the cow and at specific times of the year and breeding season, with waves of each type of lesion passing through the herd at a particular time.

White line separation was observed commonly during spring (up to 37% of digits affected), disappeared almost completely over the dry summer months, and reappeared in autumn, increasing to 40% again over winter. Lesions were more commonly observed in the lateral digits, with fore feet being more commonly affected than hind feet.

Erosion of the heel bulbs was common in August (65% of digits affected, more common in mature cows than 2-year olds), disappeared quickly during spring, and reappeared in all cows during the following winter (88% of digits; no age differences). There were no differences in distribution between digits.

Sole haemorrhages were concentrated over the mid-sole and abaxial sole zones. They were more common in the hind feet than the front feet and more common in the outside claws ($p < 0.001$). Haemorrhages were not observed prior to or soon after calving, appeared in October and reached a peak of 40% in December, then gradually disappeared by the time of drying-off in May. The 2-year olds were more frequently affected than the mature cows ($p < 0.001$).

Introduction

Most surveys and studies of lameness in dairy cows have reported details of lesions causing clinical lameness. Several reports have included only those cases of lameness treated by veterinarians (Prentice and Neal, 1972; Eddy and Scott, 1980; Russell *et al.*, 1982; Choquette-Lévy *et al.;* McLennan, 1988; Jubb and Malmo, 1991). Some have been able to give complete details of the incidence and types of lameness treated by farmers as well (Arkins, 1981; Dewes, 1978; Whitaker *et al.*, 1983).

More recently, attention has been focused on subclinical hoof lesions involving haemorrhage and erosion of the heel (Livesey and Fleming, 1984; Mortensen *et al.*, 1986; Bradley *et al.*, 1989; Greenough and Vermunt, 1990).

The objective of this study was two-fold: to determine the incidence and prevalence of subclinical hoof lesions in cows from a large New Zealand dairy herd which was grazing pasture throughout the year, and to assess the association of such subclinical lesions with clinical lameness.

Materials and Methods

Eleven 2-year-old cows, entering the milking herd for the first time, and eleven 6-year old cows were randomly selected from a herd of 415 cows in July 1989 prior to calving. The herd, one of the Massey University seasonal supply dairy herds, was grazed on 140 hectares near Palmerston North. All cows calved in August and September.

The cows were not lactating during June-July 1989 and June-July 1990. During the non-lactating period when the weather was too wet for day-long grazing, the herd spent only a few hours per day at pasture. For the remainder of such days, cows were supplemented with grass silage and confined to a sawdust loafing pad. After calving, 24-hour grazing resumed.

Commencing in August 1989 the selected cows were examined monthly for a period of 12 months. The following details were measured and recorded:

For each cow, monthly:

- the condition score (using the 1-8 scoring system of Earle (1976))
- the heart girth
- the severity of lameness (if any)

For each hoof, monthly:

- any hoof abnormality or lesions
- the zone or location of each lesion
- the severity of each lesion

The lesion classification and scoring systems used were:

Foot Abnormality Classification

1. Interdigital necrobacillosis (IN)
2. Interdigital hyperplasia
3. Interdigital foreign body (IFB)
4. White line separation (WLS)
5. Axial groove fissure (AGF)
6. Sole ulcer
7. Hoof Haemorrhage (HH)
8. Excessive wear of sole
9. Hoof overgrowth (HO)
10. Foreign body in sole or heel (FBS)
11. Vertical fissure
12. Erosion of the hoof (HE)
13. Sole abscess (septic pododermatitis)
14. Deep sepsis
15. Heel bulb abscess
16. Laminitis
17. Deformity of hoof wall
18. Punctured sole (PS)
19. Other

Digit Classification

1. Lateral left front (LLF)
2. Medial left front (MLF)
3. Medial right front (MRF)
4. Lateral right front (LRF)
5. Lateral left hind (LLH)
6. Medial left hind (MLH)
7. Medial right hind (MRH)
8. Lateral right hind (LRH)

Hoof Zone

Zones 1 - 12 as detailed in Figure 4.1.

13. Interdigital

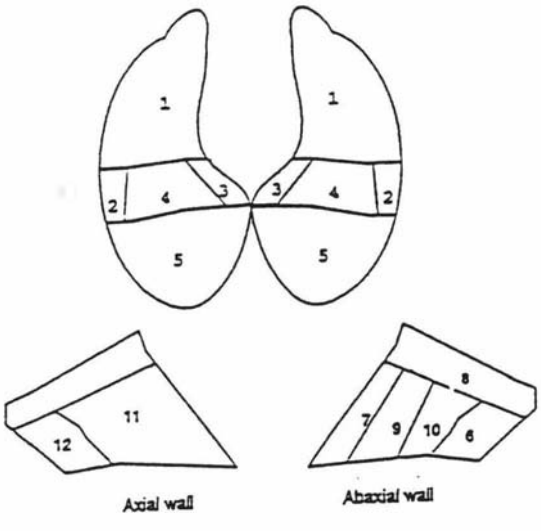


Figure 4.1 Hoof zone classification

Severity of Lameness

1. No abnormality of gait
2. Lameness hardly noticeable
3. Slightly lame
4. Markedly lame
5. Affected limb not weight bearing

Lesion Severity Score

1. Minor superficial lesion extending < 3 mm in depth and not causing lameness or pain reaction with hoof testers
2. More extensive lesion (> 3 mm in depth) but not extending to the sensitive corium and not causing lameness or pain reaction with hoof testers
3. Severe lesion extending to the sensitive laminae and resulting in either lameness or pain reaction with hoof testers

Results

Hoof Lesions

The types of lesions and the number of observations of each type of lesion are shown in Figure 4.2. 1094 lesions were recorded during 2097 digit examinations. As cows were examined monthly, in some instances the same lesions were observed and recorded on more than one occasion. With the exception of some cases of hoof haemorrhage, most of the lesions observed were scored as severity 1 only. None of the 22 cows showed any sign of lameness during the study.

White line separation, erosion of the heel bulb and sole haemorrhage were the most frequently observed lesions. Differences occurred between digits, months and age groups.

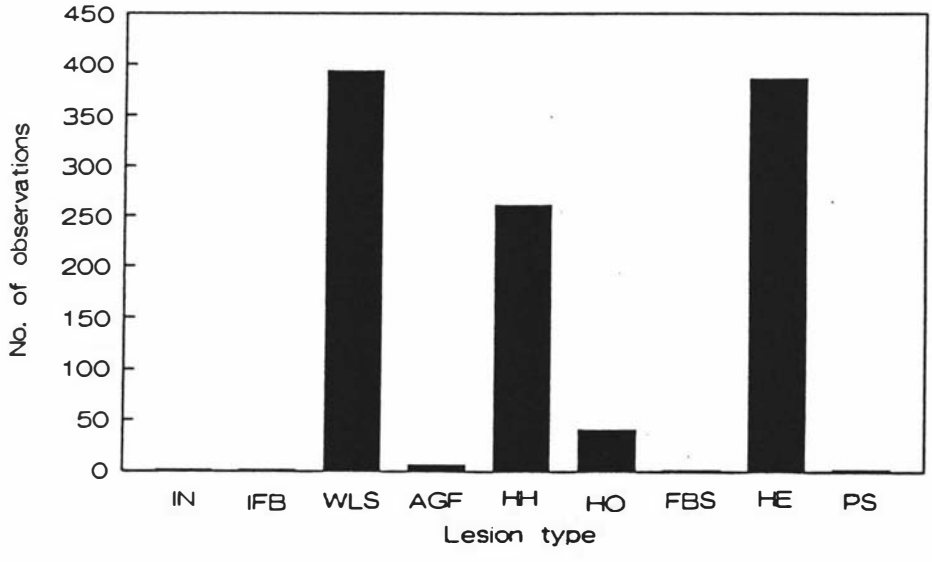


Figure 4.2 **Foot lesions in non-lame cows during a 12 month period of examination**

White Line Separation

Minor white line separation was the most commonly observed lesion (Figure 4.2). Very superficial cracks in the white line that could be scraped away with a few strokes of the hoof knife were ignored. Lesions were most commonly observed in the lateral digits with fore feet being more commonly affected than hind feet (Figure 4.3). These differences between digits were highly significant ($\chi^2 = 442$, df = 7, $p < 0.001$).

At the first examination in August, when the 2-year-old cows were calving and entering the milking herd for the first time, white line lesions were significantly less common in the 2-year-old cows than in the mature cows ($\chi^2 = 15.4$, $p < 0.001$). At the September examination this difference between ages was smaller ($\chi^2 = 1.56$, $p = 0.212$) and had disappeared completely by October ($\chi^2 = 0.004$, $p = 0.947$).

The distribution of white line lesions by month of examination is shown in Figure 4.4. These lesions were observed more commonly in the spring and winter months.

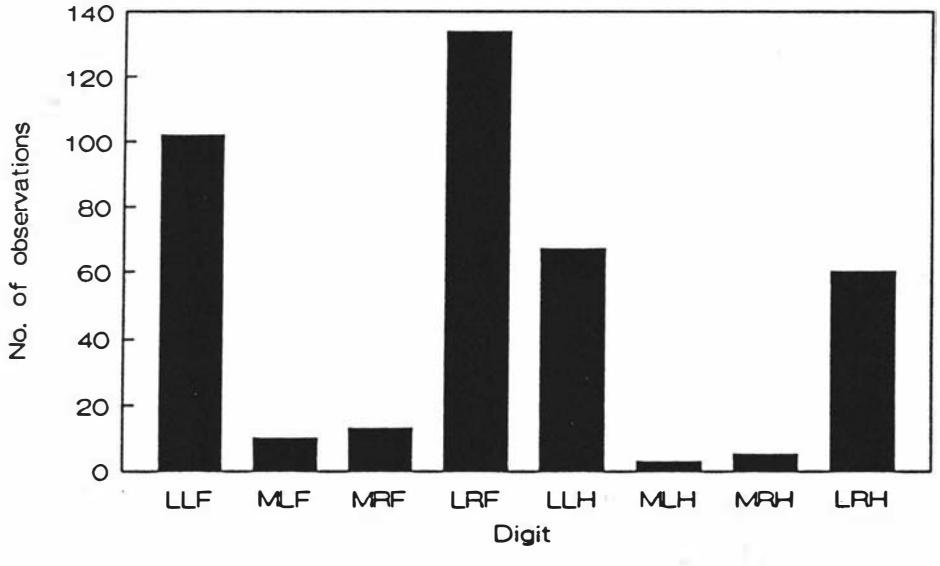


Figure 4.3. Distribution of white line separation by digit

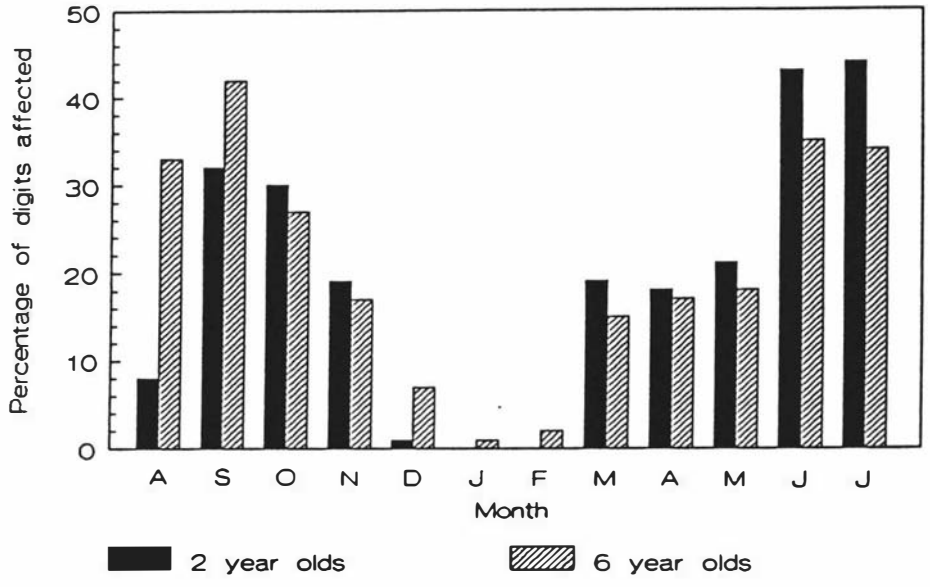


Figure 4.4. **Digit prevalence of white line separation over time, by age group**

Hoof Erosion

The next most commonly observed lesion was hoof erosion in the heel region and particularly on the axial border of the heel. This lesion was commonly observed in the mature cows in August, disappeared fairly quickly during the spring months, and then reappeared in all cows during winter (Figure 4.5). The difference between age groups was highly significant at the beginning of the study ($\chi^2 = 85.35$, $p < 0.001$) with the mature cows more commonly affected than the 2-year-olds. During the winter months, almost all digits were affected (88%), with no differences being observed between age groups.

There was not the same difference in distribution of hoof erosion (Figure 4.6) amongst digits as there was for white line separation (Figure 4.3) and hoof haemorrhage (Figure 4.8). The differences between digits were not significant ($\chi^2 = 5.58$; df = 7; $p = 0.59$).

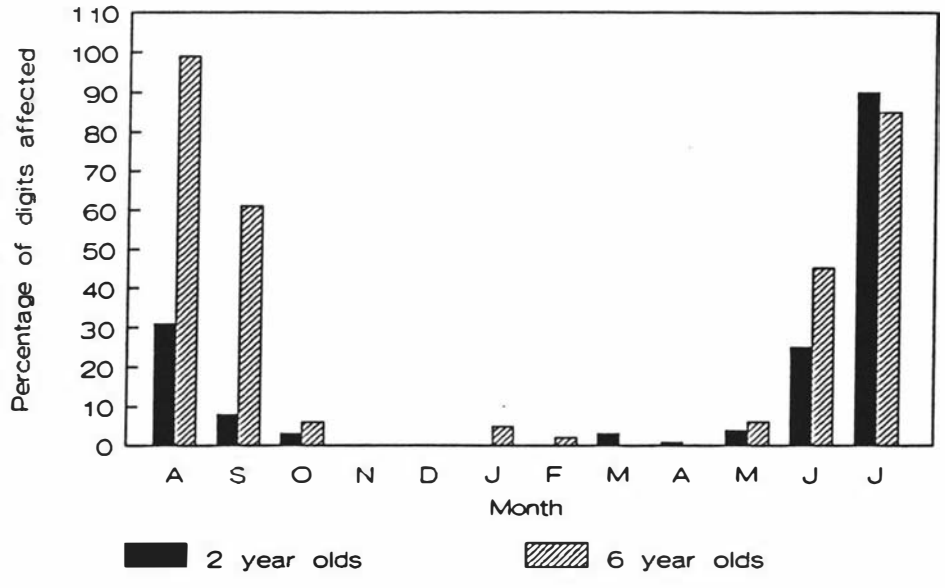


Figure 4.5 **Digit prevalence of hoof erosion over time, by age group**

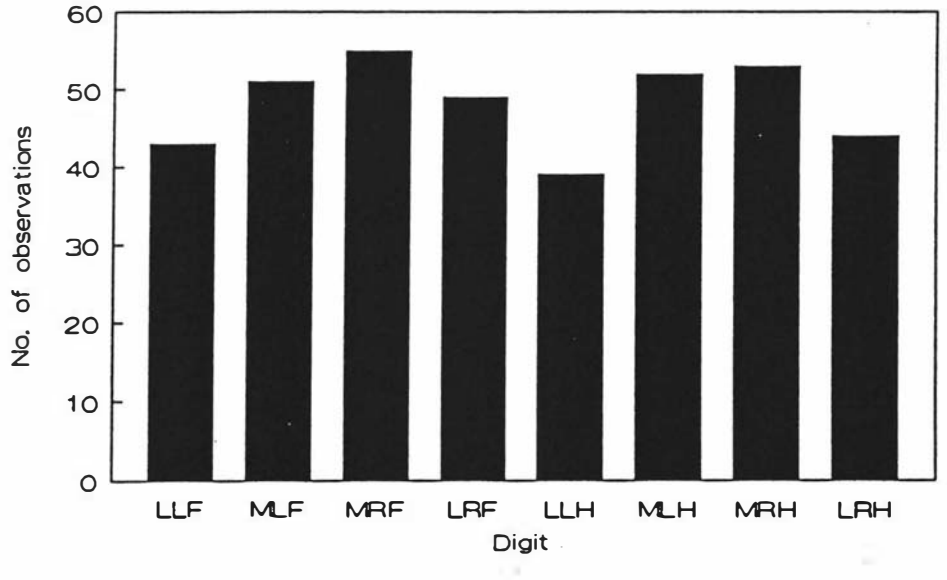


Figure 4.6. **Distribution of heel erosion by digit**

Hoof Haemorrhage

Hoof haemorrhage was the third most prevalent lesion (Figure 4.2). The severity and extent of haemorrhage varied from mild speckling to dark blotching. Figure 4.7 shows the observed distribution of haemorrhages over the bearing surface of the hooves; the mid-sole and abaxial sole were the most commonly affected.

Haemorrhages were more prevalent in the hind feet than the front feet, and more prevalent in the lateral claws (Figure 4.8). These differences were highly significant (χ^2 = 194.93; df = 7; $p < 0.001$).

Haemorrhages were not observed at the beginning of the study, prior to or soon after calving, but increased from September to December and then decreased until May. There were no haemorrhages in June or July (Figure 4.9). In December, 40% of digits contained some haemorrhages. The 2-year-old cows were more frequently affected, with the number of observations of haemorrhage being significantly greater in the 2-year-olds than the mature cows (χ^2 = 15.27 ; $p < 0.001$).

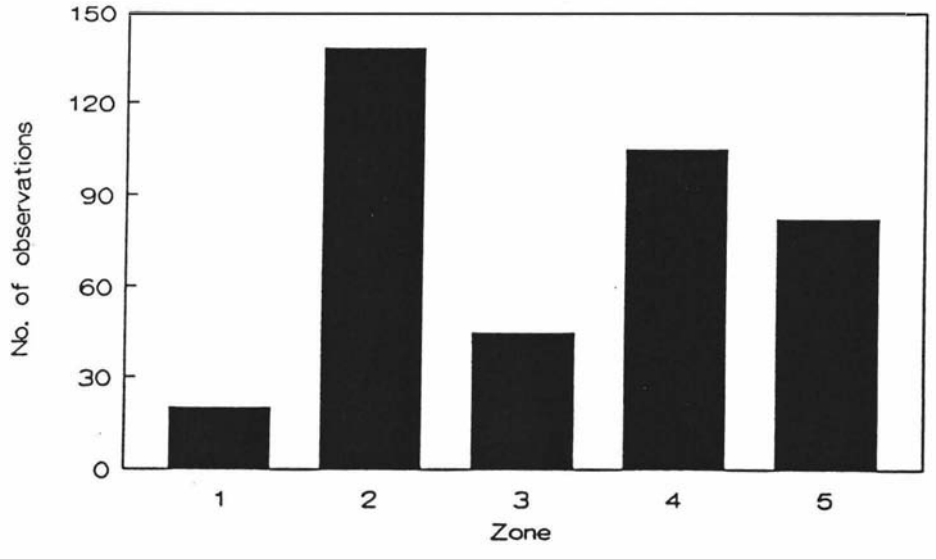


Figure 4.7. Distribution of hoof haemorrhage by zone

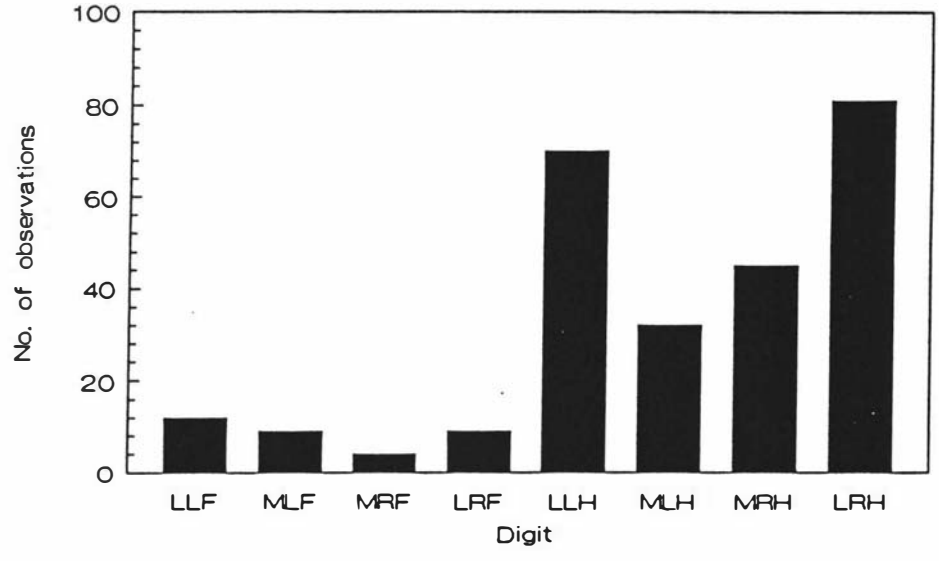


Figure 4.8. **Distribution of hoof haemorrhage by digit**

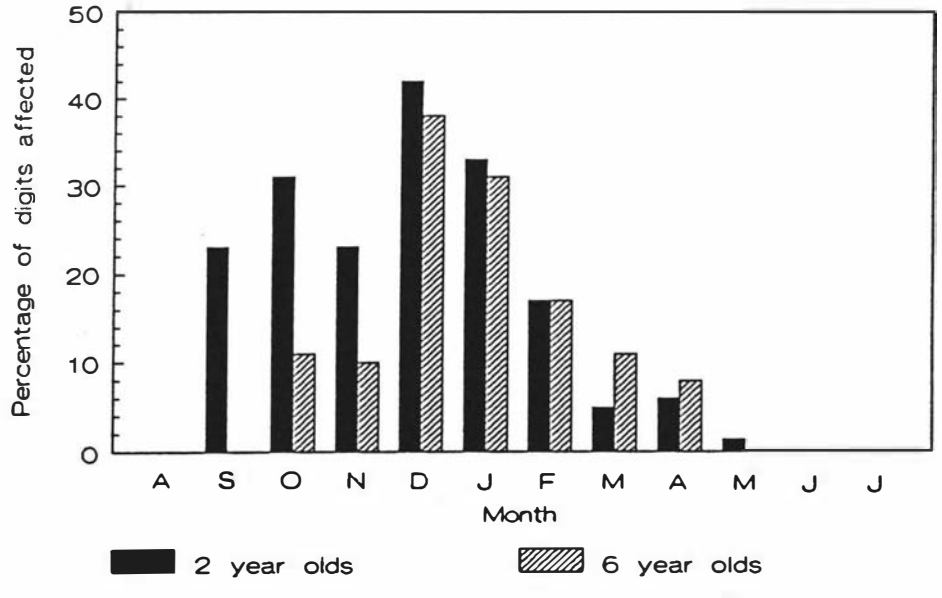


Figure 4.9. **Digit prevalence of hoof haemorrhage over time, by age group**

Discussion

A large number of lesions were seen in the study cow group, but were not associated with clinical lameness. Particular lesion types showed a specific cow distribution and were prevalent at specific times of the year and breeding season, suggesting that a number of animal and/or environmental factors strongly predispose to lesion development. There was a low incidence (2%) of clinical lameness in the herd.

White line separation is characterised by disintegration of the junction between the sole and wall and its penetration by debris. It has frequently been reported as a major cause of lameness (Eddy and Scott, 1980; Baggott, 1982; Russell *et al.*, 1982; Choquette-Lévy *et al.*, 1985). In this study, the lesions observed were very minor cracks and separation of the white line. However, it is logical to expect that they had the potential, given the right predisposing conditions, to develop into more severe lesions as grit and dirt were forced in deeper, thereby widening the crack further. White line separation resulting in lameness occurs most frequently in the lateral claws of the hind feet (Russell *et al.*, 1982). We can confirm that early disruption of the white line occurs at that site. We observed minor white line lesions commonly in the lateral fore digits as well. In fore legs, clinical white line disease is less prevalent.

Heel horn erosion is defined as an irregular loss of bulbar horn in the form of multiple irregular pit-like depressions or deeper grooves (Espinasse *et al.*, 1984). It is widespread in winter housed cattle and rarely causes lameness unless there is extensive underrunning of the heel and sole to the depth of the corium (Baggott and Russell, 1981). In a recent study of the hind feet of 4896 cows in France at the end of the winter housing period, Philipot *et al.* (1990) reported 55% of animals affected. In 80% of these animals the lesions did not involve detachment of the heel horn or other complications and were regarded as benign. The remainder were classified as serious and were associated with interdigital hyperplasia, detachment of the heel horn or sole ulcer. Russell *et al.* (1982) reported that underrun heel was responsible for 8% of all lesions causing lameness in a British survey.

Heel erosion is generally regarded as being associated with unhygienic conditions when feet are kept wet, in stalled rather than pastured cows. It is also more frequent in older cows. Toussaint Raven and Cornelisse (1971) have associated the condition with

interdigital dermatitis caused by *Bacteroides nodosus*. Smedegaard (1964b) implicated non-specific bacterial and chemical agents originating from dung and urine. More recently, Greenough and Vermunt (1990) have suggested that heel erosion could occur partly as a sequel to haemorrhages associated with laminitis. Our finding of heel erosion being most prevalent during winter (the period when dairy cows are heavily stocked on small areas of wet, boggy pasture and sometimes kept for days in wet boggy sawdust yards) is consistent with most previous reports. However, none of the lesions observed were severe, nor were they associated with horn haemorrhage. This lack of association lends little support to Greenough and Vermunt's suggestion that hoof haemorrhage may contribute to the development of erosions of the heel.

The sole haemorrhages observed appeared to be less severe forms of the haemorrhages and bruising commonly seen in clinically lame cows in other herds. In the study cows, haemorrhages appeared as red speckles, streaks or minor blotches. Bruising in clinically lame cows is usually more severe and more extensive, appearing as dark red blotching, with the horn of the sole sometimes thin and pliable. These lesions appear to result from traumatic injury and bruising of the solar corium. The highest recorded prevalence of haemorrhage was in December. With normal horn growth rates, it may take 1-2 months for haemorrhages occurring in the corium of the sole to reach the horn surface, indicating that October and November were the months when most injuries occurred. This coincides with wet conditions at a time of year when cows are walking long distances to pasture. It also coincides with the period of peak mounting activity associated with oestrus, when loads being applied to the hind digits increase dramatically.

The haemorrhages observed in this study have many characteristics in common with descriptions of subclinical laminitis reported in recent times (Greenough, 1982; Bradley *et al.*, 1989; Greenough and Vermunt, 1990). Haemorrhage in the sole of the hind lateral claws is considered by Peterse *et al.* (1986) to be a serious problem in Dutch cattle and by Smart (1986) to be a problem in Holsteins in Canada. Bradley *et al.* (1989) and Greenough and Vermunt (1990) have described a much higher prevalence of haemorrhages in outside than in inside claws of the hind feet in young heifers. These reported distributions of haemorrhage amongst digits are similar to that seen in this study. The major difficulty in associating the haemorrhages observed here with the subclinical laminitis syndrome is the lack in New Zealand herds of the commonly regarded predisposing cause of laminitis, namely high concentrate diets. Further, no

haemorrhages were observed prior to the second month of lactation. The 2-year-old animals, being low in social ranking in the herd, are the ones most subject to tussling and unplanned placement of the feet. This is the most likely explanation for the higher prevalence of haemorrhages in 2-year-old cows.

As none of the study cows became lame during the year, it is difficult to assess the full significance of the various subclinical lesions observed. However, given that white line disease and bruising of the sole are commonly observed lesions in clinically lame cows in other New Zealand herds, it seems likely that the white line separation and sole haemorrhages seen were minor manifestations or early stages of processes that may result in clinical disease. It is possible that the risk factors associated with the production of clinical lameness are similar to those associated with these subclinical lesions. Consequently a minor increase in exposure to key risk factors may have resulted in a much higher incidence of lameness. The work of Chesterton *et al.* (1989) showed that specific environmental and management risk factors could be identified as different between high-lameness and low-lameness herds. It maybe that differences between herds in the occurrence of lameness are mediated, partly or wholly, by differences in the probability of a subclinical lesion developing into a clinical lesion, rather than by differences in the initiation of lesions. Our study clearly indicates that hoof lesions do not occur sporadically as the result of chance events. In contrast, they occur with a definite epidemiological pattern. A greater understanding and awareness of the epidemiology of hoof lesions will be important in the development of improved methods for the prevention of lameness.

Acknowledgements

The authors are grateful to the staff of Massey No 4 Dairy for their cooperation with this project; to Mrs D Moore for technical assistance, and to Mrs F Dickinson for the illustrations.

CHAPTER 5

A case study of lameness in three dairy herds[3]

[3] Published as: Tranter WP, Morris RS. New Zealand Veterinary Journal 39, 88-96, 1991.

Abstract

All cases of lameness that occurred in cows from three dairy herds between August 1989 and July 1990 were examined every 2 weeks from the onset of lameness until the lesions resolved. The incidences of herd lameness were 38%, 22% and 2%. Some 186 clinical lesions were identified in 134 cases of lameness in 120 cows. Sole bruising (42%) and white line separation (39%) were the most frequently diagnosed conditions. Lateral digits of the hind limbs were the most affected. The mean time from the onset of lameness to clinical recovery was 27 days and to lesion recovery was 35 days.

The peak incidence of lameness occurred during winter for autumn-calving cows and during the late spring for spring-calving cows. The onset of lameness was associated with the stage of lactation and wet weather conditions. Survival analysis revealed that the probability of an individual cow lasting in the milking herd for any specified period of time without becoming lame was highly associated with both her herd environment and her age.

Total lactation yields of milk, milk fat and milk protein were lower for cows suffering from lameness than for herd-mates matched on age and proximity of calving date ($p < 0.05$). Reproductive performance was also poorer in lame cows than in their herd-mates.

Introduction

Lameness is considered to be among the most important health problems in dairy production. Dewes (1978) reported an incidence of 14% in New Zealand dairy herds. The 50th Farm Production Report of the New Zealand Dairy Board (1974) reported on a survey of lameness carried out in herds in the Levin-Otaki area, and concluded that lameness causes significant economic losses to New Zealand dairy production. In a case-control study of environmental and behavioural factors influencing foot lameness in 62 dairy herds in Taranaki, New Zealand, the average maintenance of the farm track and the degree of patience shown by the farmer in bringing the cows to milking were the most influential variables explaining variation between case and control herds (Chesterton *et al.*, 1989).

Numerous extensive surveys recording the incidence of lameness and the types of lesions involved have been conducted in the northern hemisphere (Prentice and Neal, 1972; Eddy and Scott, 1980; Arkins, 1981; Russell *et al.*, 1982; Whitaker *et al.*, 1983; Choquette-Lévy *et al.*, 1985). Recent reports have provided information on lameness in various regions of Australia (Harris *et al.*, , 1988; McLennan, 1988; Jubb and Malmo, 1991; Willyanto, pers. comm.). However, there is only scant documentation of the situation in New Zealand dairy herds (Cagienard, 1973; Dewes, 1978; Anderson, 1985).

All cases of lameness that occurred in three dairy herds over a 12-month period were studied to establish the types of lesions that commonly result in lameness, and to monitor the recovery process. Factors possibly associated with lameness, such as age, stage of lactation, claw distribution of the different lesions involved, rainfall, proximity to recorded heat dates and time of year, are also reported.

Materials and Methods

Three dairy herds, situated within 5 kilometres of Palmerston North and serviced by the Massey University veterinary practice, were selected for a case study of lameness. All herds had a concentrated calving pattern, with two herds calving all cows in August and September and the third calving all cows in March, April and May. The farm sizes are

137 ha, 100 ha and 112 ha (autumn calving), while the herd sizes at the start of the study were 422, 301 and 200 respectively.

Cows that became lame between August 1989 and July 1990 were examined soon after the farm manager detected the onset of lameness (usually within 1 - 2 days), and then at 2 weekly intervals until completely recovered. At each examination, cows were observed while walking and a lameness score allocated (0 = no abnormality of gait, 1 = lameness hardly noticeable, 2 = slightly lame, 3 = markedly lame and 4 = affected limb not weight bearing). Each cow was examined as she moved on concrete towards or away from the observer for 10-15 metres at a normal pace. The following items were noted : the degree of difficulty in deciding which leg was affected, the degree and timing of head movement (the head usually moves up as the affected limb contacts the ground); the degree of sinking of the hind quarter on the sound side; any abduction or adduction of the limbs; shortening or lengthening of the stride (the hind foot is normally placed in exactly the same place as the front foot).

The affected cows were then restrained, heartgirth was measured and a condition score (CS) allocated using a 1 (emaciated) to 8 (overfat) scoring system. The lame limb was examined and the lame digit(s) identified by using hoof testers. Details of the affected digit were recorded, including sole colour, wall colour, sole condition, lesion type(s), lesion severity and location of lesion. Lesion type and location were classified as outlined by Tranter *et al.* (1991) and following the system described by Mills *et al.* (1986). Lesion severity was scored from 1 to 3 (1 = minor superficial lesion causing no lameness or pain reaction with hoof testers; 2 = more extensive lesion but not extending to the sensitive corium and not causing lameness or pain reaction with hoof testers; 3 = severe lesion extending to the sensitive laminae and resulting in lameness or pain reaction with hoof testers).

Traditional treatment was administered. This included paring away all necrotic hoof horn and providing free drainage. Antibiotics were given when lesions were deep-seated. Lame cows were separated from the milking herd and kept in nearby paddocks adjacent to the dairy so that they did not have to walk long distances to pasture.

All cows were re-examined at about 2-weekly intervals until it was considered that the major lesion had completely resolved. This was when:

- There was no pain reaction to hoof testers
- Healthy soft hoof occupied the site
- There was no moistness or exudation of pus, fluid or serum from tracts or deeper lines of separation

At each examination, the cows were again examined as above. Lame cows were returned to the herd when the manager considered them completely healed.

The onset of oestrus and service dates were recorded. The intervals between the onset of lameness and oestrus were calculated. Because some heats were not detected or recorded by the farmers, the records of these cows were examined for likely unrecorded heats. Where this could be identified as highly likely to have occurred on a particular date, it was used together with recorded heats to determine the intervals between heat and lameness.

Production, reproduction and fate records were collected. Lame cows were compared with non-lame herd-mates matched for age and the proximity of calving dates.

Daily precipitation data was obtained from an official meteorological recording station in Palmerston North, situated within 5 kilometres of the three dairy farms.

The 12 month study period for the autumn-calving herd included two partial lactations for most cows. When the likelihood of individual cows becoming lame at specified intervals after entering the milking herd was calculated, only the 1990 lactation records were used. After the end of the study, the farmer recorded any cases of lameness until the end of lactation so that data could be gathered for a complete lactation.

Statistical Analysis

The data were analysed using the programs PANACEA 2 (PAN Livestock Services, Reading, England) and SOLO (BMDP Statistical Software Inc., Los Angeles, California). Linear regression analysis was used to evaluate the association between rainfall and the onset of lameness. The significance of the differences in production between lame and non-lame controls was tested using Student's paired t-test. Differences in the time taken for lameness to occur after calving, both between herds and between age groups within herds, were studied using survival analysis (Lee, 1984).

Results

Incidence of lameness

Of the 120 lame cows examined, 14 became lame more than once within the 12 months (the lameness was recorded as a separate case if the cow had previously been lame and had fully recovered before becoming lame again in the same or a different digit). Most cases were confined to the digits (154 digits in 123 cases), but 10 cases involved interdigital lesions, and two cases involved the upper leg. Each case was examined on average 3.5 times at 2-weekly intervals. The annual incidence of lameness ranged from 2% to 38% (Table 5.1).

Table 5.1. Incidence of lameness in the three study herds over 12 months

	Average herd size	No. of lame cows	No. of lameness cases	No. of lame digits	No. of severe lesions	Lame Case Incidence
Herd 1	181	60	68	77	85	38%
Herd 2	386	7	7	4	8	2%
Herd 3	271	53	59	73	93	22%
Total	**838**	**120**	**134**	**154**	**186**	**16%**

Lesion type

The types of lesions of severity score 3 recorded at first examination are shown in Figure 5.1. Frequently, each digit had more than one lesion with some having up to 4 different lesions. White line separation (39%) and traumatic pododermatitis or sole bruising (42%) were the most common lesions.

Distribution of lesions

Two-thirds of the cases occurred in the hind legs and one-third in the fore legs. Some 6.5% of cases were confined to interdigital inflammation, 1.5% to the higher leg and the remaining 92% to the digit itself.

The distribution by digit number of the three major lesions at the onset of lameness is shown in Figure 5.2. White line disease occurred most commonly in the medial front and lateral hind digits, bruising most frequently in the lateral hind digits and septic pododermatitis most frequently in the medial hind and lateral front digits.

The distribution of bruising, the most common lesion, by zone or location of the lesion, is shown in Figure 5.3. White line disease, the other most common lesion, occurred, by definition, along the white line in zones 1 and 2.

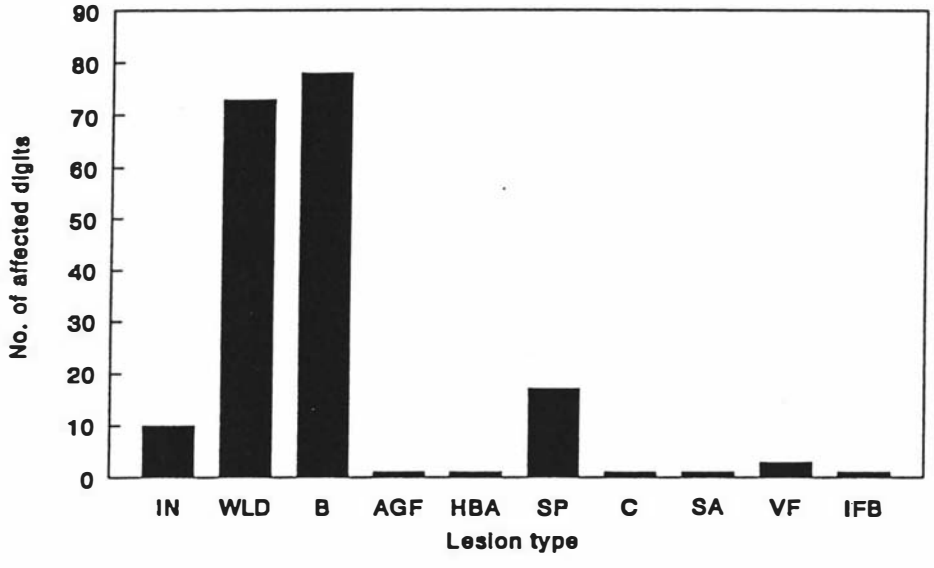


Figure 5.1. Severe lesions in lame cows (186 lesions).

IN	=	Interdigital necrobacillosis
WLD	=	White line disease
B	=	Bruising
AGF	=	Axial groove fissure
HBA	=	Heel bulb abscess
SP	=	Septic pododermatitis
C	=	Coxitis
SA	=	Stifle arthritis
VF	=	Vertical fissure
IFB	=	Interdigital foreign body

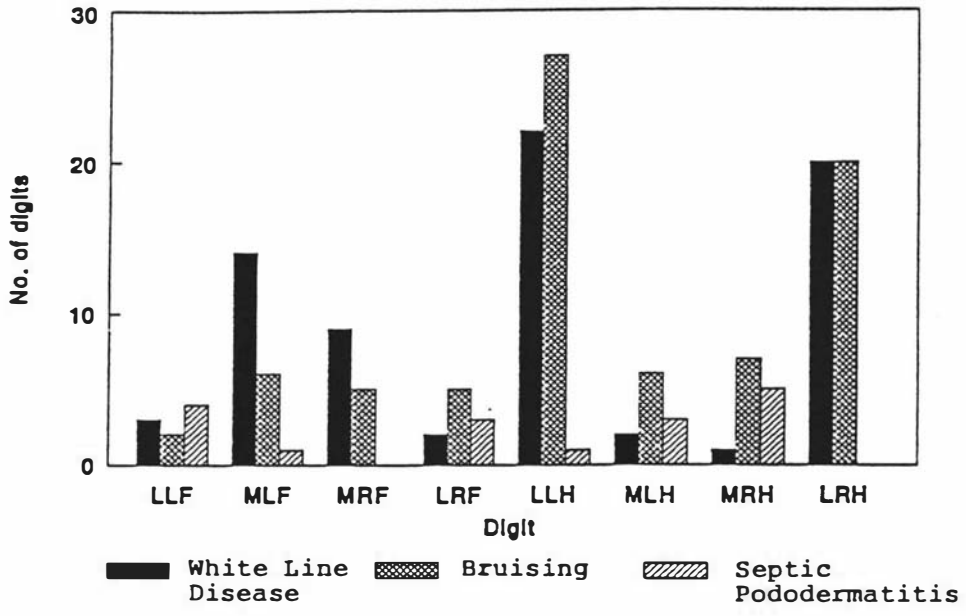


Figure 5.2. The distribution of the three most common lesions amongst digits (73 digits with white line disease, 78 digits with bruising and 17 digits with septic pododermatitis). Digits are coded lateral/medial, left/right, front/hind.

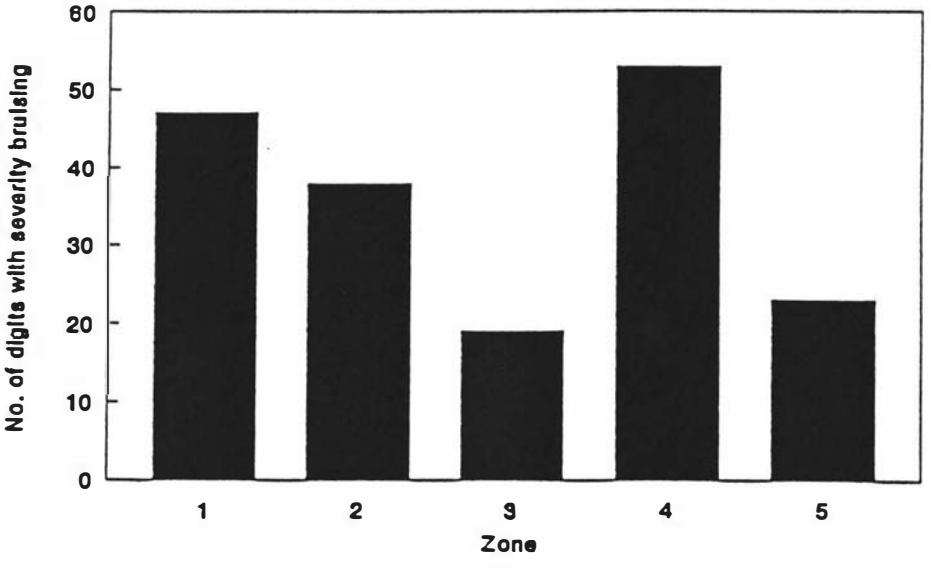


Figure 5.3. Distribution of severe bruising by zone at the onset of lameness. Zones are as defined by Tranter *et al.* (1991).

Association with rainfall and time of year

The peak incidence of lameness occurred during the winter in the autumn calving herd and in the late spring for the spring-calving herds (Figure 5.4). The onset of lameness was associated with wet weather conditions ($r = +0.53$). The simple linear regression relationship between the number of wet days in the week preceding lameness (independent variable) and the number of new lame digits each week (dependent variable) was tested by ANOVA. The coefficient of determination was 0.28 and the relationship was highly significant ($F_{1,50} = 19.78$; $p < 0.001$).

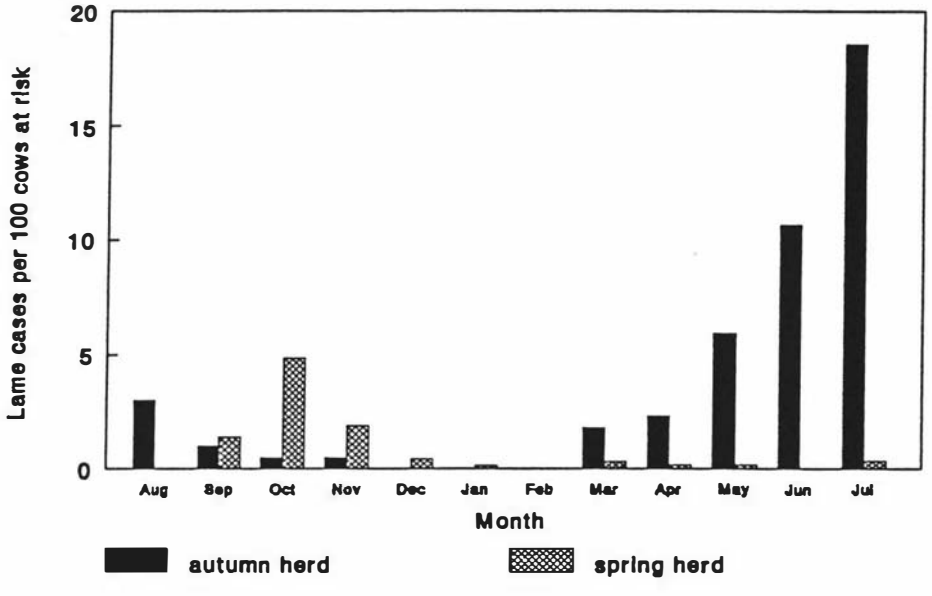


Figure 5.4. Incidence rates of lameness cases by month

Association with calving date and herd environment

There was also a close association between the onset of lameness and time since calving as shown in Figure 5.5. The average time from calving to the onset of lameness was 91.9 ± 54.0 days (mean ± SD).

The technique of survival analysis was used to further analyse the onset of lameness after calving in the three study herds. In Figure 5.6, survivorship of cows in the milking herd without developing lameness is plotted against time in weeks since calving. In this

instance, survivorship refers to the probability of cows lasting in the milking herd for a specified period after entering the milking herd at calving without becoming lame. S(t) = 1 - P_L where P_L is the probability of becoming lame and S(t) is survivorship. The differences in survivorship of cows between the three herds were highly significant when tested by Gehan's generalisation of Wilcoxon's two-sample rank sum test ($p < 0.0001$). Survivorship of cows in the two high-lameness incidence herds was similar until about 14 weeks after calving (Figure 5.6). However, for Herd 1 (the autumn calving herd) the probability of cows surviving without lameness continued to fall until 22 weeks after calving. Thus these autumn-calving cows were at continuing risk of becoming lame between weeks 14 and 22 after calving (which coincided with the wetter winter months of July and August), whereas spring-calving cows were at very low risk after week 14 (which coincided with summer and reduced oestrous activity).

Age Distribution

The age distribution of affected cows from all three herds combined is shown in Figure 5.7. However, there were substantial differences between herds (Figure 5.8) which could be partly explained by differing management systems. In Herd 3, 301 cows were run as one mob and the 2-year old cows (in their first lactation) had the highest incidence of lameness (36% compared with the overall herd incidence of 22%). In Herd 1, the younger cows (which included all 2-year olds) were run as a smaller herd, separate from the main mob, and grazed in paddocks closer to the dairy. In this herd, the 2-year olds had the lowest incidence of lameness (10% compared with the overall herd incidence of 38%).

These differences in the age distribution of the lame cows from the two herds with high incidences are very apparent in the survivorship functions (Figure 5.9). The survivorship function curves were somewhat similar for all three age groups in both herds until 8 - 10 weeks after calving. In Herd 3, survivorship of the 2-year old cows continued to fall substantially further than that for their older herd mates. In contrast, in Herd 1 where the 2-year olds were run separately, survivorship of the two older age groups continued to fall. Although most of the cows from these two older groups ran together as one herd, survivorship of the 8+ year-old group was significantly lower than that for the 3 -8 year old group ($p < 0.05$).

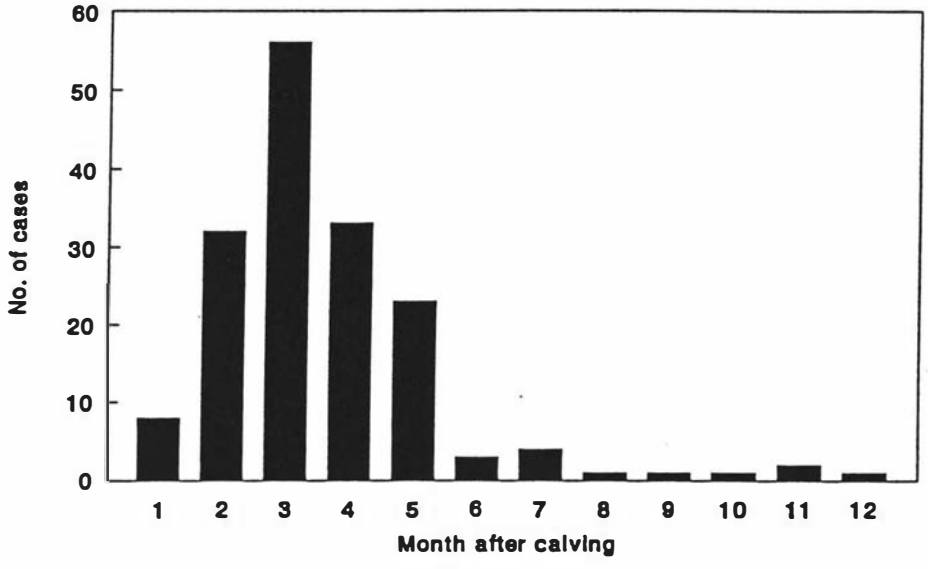


Figure 5.5. Onset of lameness vs time since calving

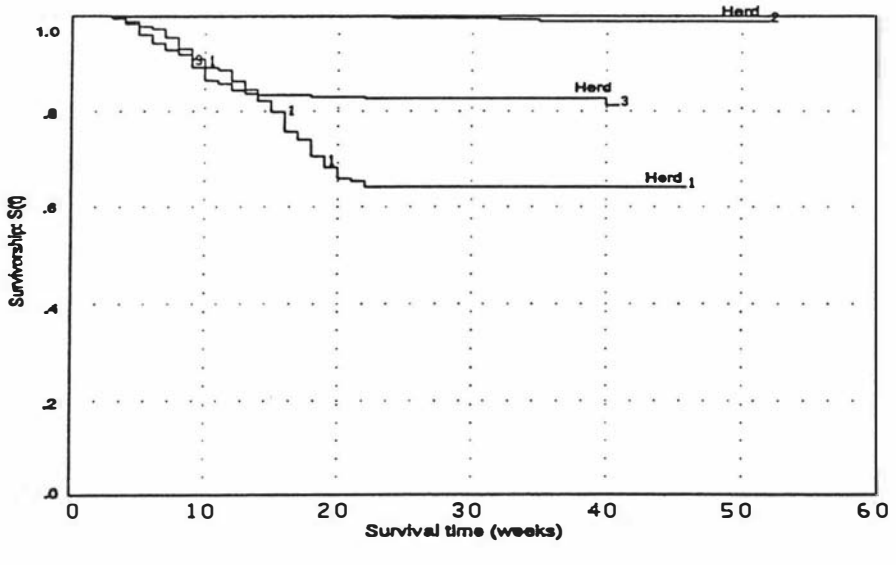


Figure 5.6. Survivorship functions for cows in three different herds after calving

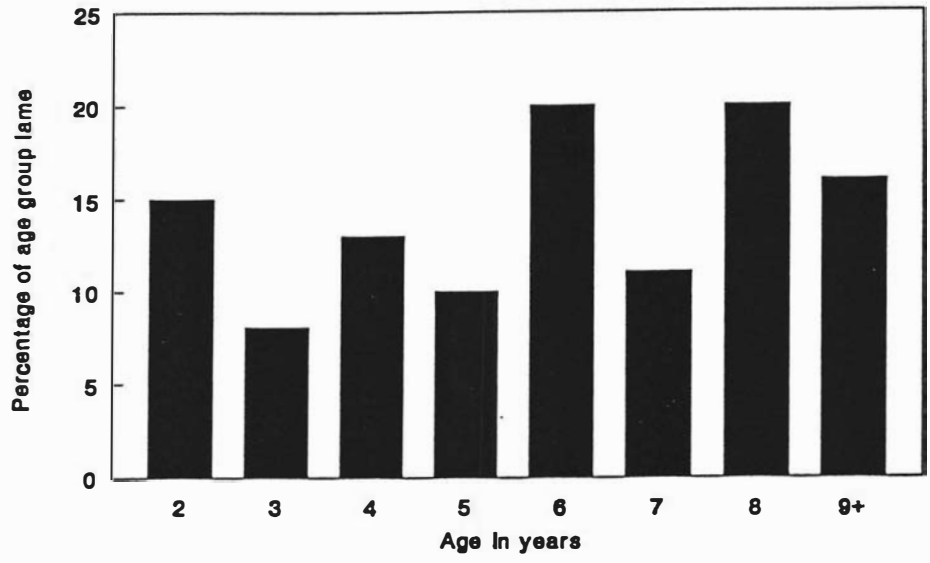


Figure 5.7. Age distribution of lame cow incidence

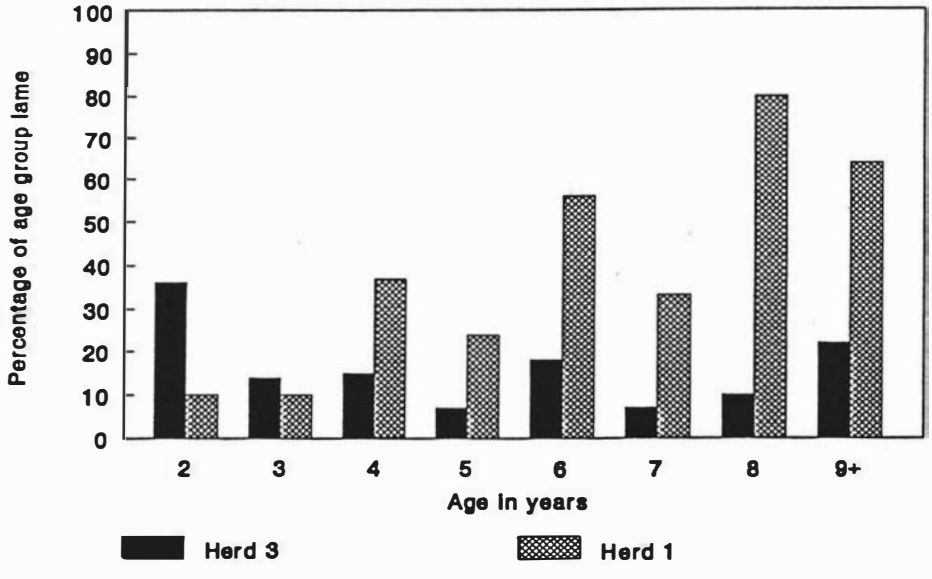
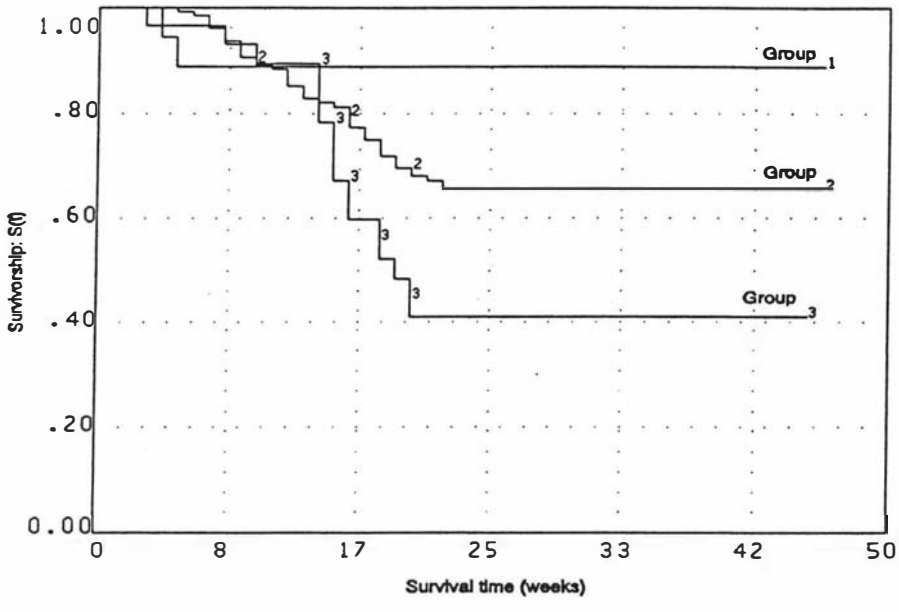


Figure 5.8. Age distribution of lame cow incidence in the two herds with a high incidence of lameness



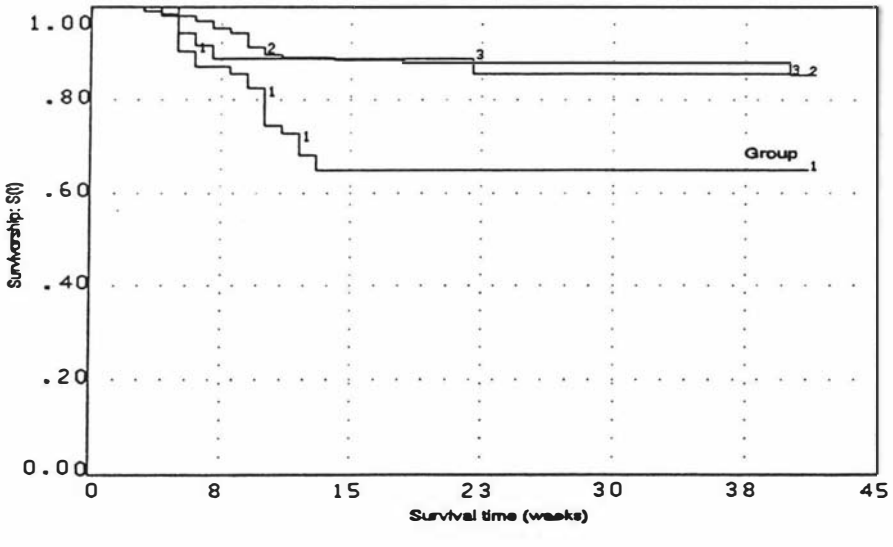


Figure 5.9. **Survivorship functions for cows of three age groups in the two high-incidence herds. Herd 1 - top; Herd 3 - bottom. Group 1 = 2-year-olds; Group 2 = 3-7 year olds; Group 3 = 8+ year olds.**

Association with date of recorded oestrus

The onset of lameness in 25% of all affected digits occurred within 14 days after a recorded heat. When the cases of lameness that occurred close to the estimated date of a possible heat were included, 36% of all lameness fell within 14 days after a recorded or possible heat period.

Some 95% of all lameness involving the bearing surface of the hoof occurred at the peak of oestrus activity extending from 4 to 18 weeks post partum (Table 5.2).

Table 5.2. Relationship of lameness onset to date of oestrus

Time when lameness occurred	Affected digits	% digits affected
Within 7 days of oestrus	16	11
Within 7 days of an estimated oestrus	13	8
Between 7 and 14 days after oestrus	22	14
Between 7 and 14 days after an estimated oestrus	4	3
During mating season but with no apparent association with oestrus	66	43
Greater than 2 weeks after conception	33	21

Development and resolution of lesions

Data on the duration of lameness and the time taken for the different types of lesions to heal are shown in Table 5.3. The average duration of lameness was 27 ± 19 days (range = 3-75). The mean time from the onset of lameness until resolution of the lesion was 35 ± 19 days (range = 12-80). Cows with interdigital necrobacillosis (footrot) were usually severely lame at onset but were lame for only a few days after treatment with antibiotics, whereas white line lesions and bruising often took 1 or 2 months to resolve.

White line disease and bruising were often both present at the onset of lameness or appeared together at subsequent examinations during the episode. In over half (54%) of the cases of lameness involving the bearing surface of the foot, these two lesions appeared in combination.

Lameness involving the bearing surface usually developed into some degree of underrunning of the sole. In this study, this occurred in 20% of severe white line lesions and in 29% of cases involving severe bruising. Pus and necrotic material from white line disease or blood and serum from bruising separated the horn from the secretory corium. After a time, under favourable conditions, the corium started to produce new horn which then formed a second sole. The old horn of the sole would lift and was removed by paring.

In three cases, severe white line disease developed into heel bulb abscesses and in one case extended into the deep tissues of the foot to produce septic pedal arthritis and deep sepsis. One case of bruising developed into deep sepsis.

Table 5.3. Duration of lameness and lesion healing

Lesion type at the onset of lameness	No. of lesions	Days lame (mean ± SD)	Days for lesion to recover (mean ± SD)
Bruising	78	30 ± 20	38 ± 19
White line disease	73	27 ± 19	35 ± 20
Septic pododermatitis	17	26 ± 12	32 ± 12
Interdigital necrobacillosis	10	5 ± 2	15 ± 7
Vertical fissure	3	17 ± 12	40 ± 13
Axial groove lesion	1	7	21
Heel bulb abscess	1	33	33
Stifle arthritis	1	No recovery	No recovery
Coxitis	1	40	77
Interdigital foreign body	1	3	7
Total	**186**	**27 ± 19**	**35 ± 19**

Production losses and costs of lameness

Lost production

The differences in production between cows that became lame and matched herd-mates of similar age and calving date appear in Table 5.4. Yields of the lame cows were

significantly less than those for the controls ($p < 0.001$) to 2 weeks after their last herd test for the lactation. Using the price paid for seasonal supply milk during the study ($3.20 per kg of milk fat plus $4.92 per kg of milk protein minus 3.5¢ per litre), these differences in production were valued at $94.40 per lame cow. The average lactation length of lame cows was shorter, due mostly to premature culling of some cows because of their poorer production and reproductive performance. Even when lactation-to-date production levels of the two groups were compared as at the last herd test prior to either one of each matched pair leaving the herd, differences were noted (Table 5.4). At the times of peak incidence in the two herds with the highest incidence of lameness, up to 40 lame cows were being grazed separately close to the dairy. This would have had some effect on the production of the non-lame cows as well, because they had to walk further to grazing, as the paddocks close to the dairy were needed for lame cows.

Table 5.4. Production in 113 lame cows and 113 matched herd-mates[a]

	Total lactation yields		Lactation-to-date yields (for similar days in milk)	
	Lame cows	Controls	Lame cows	Controls
Milk (l)	3296 ± 1156**	3615 ± 930	3283 ± 1150**	3520 ± 1049
Fat (kg)	145 ± 55**	158 ± 48	144 ± 55**	153 ± 49
Protein (kg)	110 ± 39**	123 ± 32	110 ± 39**	119 ± 36
Days in milk	241 ± 51**	253 ± 28	237 ± 59	237 ± 59

[a] Values are quoted as mean ± standard deviation
Student's paired t-test ** $p < 0.001$

Lost body condition

The mean loss of body condition per cow over the period of lameness was 0.1 CS units (range: -0.8 - +0.2). Attempts were made by the farmers to minimise losses in body condition by providing preferential grazing for lame cows and by, at times, milking severely affected cows only once daily.

Poorer Reproductive Performance

Table 5.5 shows the herd-in-calf rate following the mating start date (MSD) for the cows that became lame before the start of mating. The lame cows performed considerably

worse than the non-lame cows, with some not conceiving before the end of the mating season or before being culled.

Table 5.5 In-calf rates for cows lame before the MSD (Cumulative percentage of cows in-calf by the end of each week)

Week No.	1	2	3	4	5	6	7	8
Lame before MSD[a]	2	13	22	26	37	41	44	46
Rest of herd[b]	12	26	43	50	56	62	66	72
Target rate		**33**		**57**		**80**		**86**

[a] There were 54 cows to be mated at MSD
[b] There were 842 cows to be mated at MSD

Increased Culling

Although lameness was rarely nominated by the herd managers as the primary reason for culling, lame cows were culled more heavily than their herd-mates. Of 54 lame cows that suffered one or more cases of lameness through the season in Herd 3, 21 were culled (39%). This compares with a culling rate for the remainder of the herd of 16%. In this herd, because the incidence of lameness was highest in the 2-year old cows, culling was biased towards the young cows which were of higher genetic potential (average breeding index (BI) of 2-year-old cows = 120; average BI of 2+ year-old cows = 115).

Treatment Costs

All lame cows in the three herds were examined and treated by a veterinarian. This is not typical; most New Zealand farmers treat many lameness cases themselves. It is estimated that 20% of the lameness cases would typically have been treated by a veterinarian at an approximate cost of $25 per case. About 20% of the lame cows received antibiotics because of secondary infections at an approximate cost of $16 per cow. Assuming the equivalent of 3.5 kg of milk fat was discarded over 5 days for each cow treated because of drug residues, a further loss of $21.53 per cow was incurred, based on a milk price equivalent to $6.15 per kg of milk fat. Averaged over all cases, these treatment costs would typically have amounted to $12.51 per case. Treatment and

management of lame cows also involved a substantial farm labour input not included in these figures.

Discussion

The varying incidence of lameness between herds is consistent with the variation reported by Chesterton *et al.* (1989) in their case-control study of lameness. The predominance of lameness involving the foot (95%), as opposed to the upper limb is similar to that observed by previous workers (Prentice and Neal, 1972; Eddy and Scott, 1980; Arkins, 1981; Baggott and Russell, 1981; Russell *et al.*, 1982; Choquette-Lévy *et al.*, 1985; McLennan, 1988; Jubb and Malmo, 1991; Willyanto, pers. comm.). Similarly, our findings that a majority of lameness occurred in the hind legs (67%), and that over half (53%) of all hoof lesions occurred in the lateral hind claws is in agreement with most other studies (Prentice and Neal, 1972; Arkins, 1981; Anderson and Lundström, 1981; Russell *et al.*, 1982; McLennan, 1988; Jubb and Malmo, 1991; Willyanto, pers. comm.). Various reasons have been suggested for this unequal distribution of lesions between digits. Pinsent (1981) has suggested that lameness is more common in the hind legs because hind legs are involved with propulsion, during which the hind digits are subject to extra pressure due to friction. Another reason is that hind feet have a smaller ground contact area than front feet, resulting in increased stresses on the bearing surface. Extra stresses during mounting activity associated with oestrous behaviour is a further possibility.

The high proportion of hind leg lameness involving the lateral digits can be partly explained by Scott's work (1988a) which shows that higher pressures during load bearing occur mainly on the lateral digits and mostly around the heel region and abaxial walls and toes. Toussaint Raven *et al.*, (1985) has suggested that weight bearing by the lateral claws varies widely with successive movements of the body, whereas the loads on the medial claws are far more even. The continual shift and alteration of forces is considered to be more detrimental than the absolute load. Ossent *et al.* (1987) recorded greater ranges for loads on the lateral hind claws of heifers than on medial claws using a force plate, adding support to this suggestion. Another consideration is that lateral hind claws are less concave than medial hind claws. This could make them more susceptible to bruising and traumatic injury when cows are forced to walk long distances along rough raceways.

The types of lesions causing lameness are somewhat different to those found in most other studies. With the exception of Willyanto's study in northern Queensland in which 17% of lesions were recorded as bruised sole (Willyanto, pers. comm.) the very high proportion of lesions involving severe bruising (42%) has not been a common finding. McLennan (1988) reported that bruising caused 5.6% of lameness in Queensland; Arkins (1981) reported 5% in Ireland; Choquette-Lévy (1985) found 8.5% in Quebec, and Russell *et al.* (1982) reported 1.9% had overworn sole in the U.K. In Victoria, Jubb and Malmo (1991) recorded 4.2% of lesions as excessive wear of the sole. However, in a survey involving farms with incidences of lameness of 6% or more in south-western Victoria, Harris *et al.* (1988) found that 77% of 31 farmers questioned regarded bruised sole as a common clinical sign of lameness.

The bruising observed in this study appeared as areas of haemorrhage in the solar horn, either localised or extending over a wide area of the bearing surface of the hoof. Sometimes the horn of the sole was soft and could easily be depressed with the thumb. Pain was evident when hoof testers were applied. When bruising was severe, extravasated blood and tissue fluid that had accumulated between the stratum germinativum and the horn would result in separation of the old horn away from the secretory corium. Parts of the sole horn were shed as a result, and in some cases the entire sole was lost.

Haemorrhages in the sole are frequently reported and are commonly regarded as part of the "aseptic laminitis" syndrome. The term "subclinical laminitis" is used to describe the condition where there is no abnormality of stance or gait, but where changes including varying degrees of haemorrhage can be seen in the weight-bearing surface of the claw (Greenough, 1982, 1985; Weaver, 1988; Greenough and Vermunt, 1990). It is regarded as a major predisposing cause of many of the lesions seen in the soles of cattle, and frequently occurs in conjunction with other lesions such as white line disease (Greenough and Vermunt, 1990). Subclinical laminitis has widely been regarded in the northern hemisphere as having a nutritional aetiology. Diets with a higher concentrate:silage ratio (Manson and Leaver, 1989), with a higher crude protein component (Manson and Leaver, 1988b) and with a lower fibre content (Livesey and Flemming, 1984) have all been associated with a higher incidence of laminitis. Diets based on pasture with low levels of concentrate, such as those fed to dairy cattle in New Zealand, are not normally associated with this laminitis syndrome. The protein content of New Zealand pasture is very high and the fibre content low at the time of year when

this condition occurs (Coop, 1961), so a nutritional aetiology cannot be discounted. However, the syndrome occurred in winter for cows calving in autumn, suggesting that the major causal factors are related more to the stage of the annual herd management cycle than to nutrition.

David (1990) has suggested that the contribution of nutrition to the aetiology of laminitis has been over-emphasised. Behavioural, management and genetic factors are increasingly being shown to be of considerable importance.

The sole haemorrhages and lameness seen in this study appear to result from traumatic injury and bruising of the solar corium. Frequently, minor haemorrhages were observed without lameness and often were associated with other lesions. It is possible that horn with blood incorporated in it may be softer, weaker or less resilient and less able to protect the corium from further concussive damage.

White line disease, the other commonly observed lesion, has frequently been reported as a major cause of lameness and is regarded as the most common cause of lameness in the U.K. dairy cattle population (Baggott, 1982). Eddy and Scott (1980) reported white line disease to be responsible for 35% of lameness in a study in southern England, Russell *et al.* (1982) 18%, and Choquette-Lévy (1985) 12% in Quebec. Similarly, in a recent study of 24 herds in northern Queensland, Willyanto (pers. comm.) found white line disease accounted for 32% of lesions. In contrast, in other Australian studies, McLennan (1988) and Jubb and Malmo (1991) reported white line disease to be much less common (less than 10% of lesions) and suggested that it is mostly a problem in the northern hemisphere where cattle are housed and fed more concentrates. Our finding that 39% of the lesions involved white line separation and the work of Willyanto (pers. comm.) demonstrate that it can be a major problem for pasture-fed cattle as well.

It is generally considered that the horn of the white line is softer than elsewhere (Edwards, 1980). Most lesions are observed in the region of the abaxial border of the lateral hind digits near the sole-heel junction. This is the zone of greatest pressures during load bearing (Scott, 1988a). Factors predisposing to white line disease include excessive walking on hard surfaces, wet conditions underfoot, claw deformities and laminitis (Edwards, 1980; Greenough and Vermunt,1990). Under New Zealand conditions, the long distances walked to pasture, wet weather conditions during early

lactation, and at times unsuitable materials used for race surface construction are likely to be the major factors involved. Haemorrhages in the sole associated with subclinical laminitis are thought to reduce the integrity and strength of the white line (Greenough, 1985). Haemorrhages associated with traumatic injury could be involved in weakening the white line tissue, predisposing to its disintegration. Our finding that sole haemorrhage and white line disease frequently occurred together adds support to this argument.

Other lesions, such as sandcracks, axial groove fissures and footrot, accounted for only a small proportion of the clinical lesions observed. Sole ulcers, which are common in housed cattle, and acute laminitis, which occasionally occurs where large amounts of concentrates are fed, were not seen in this study and are rare in pasture-fed cattle in Australia and New Zealand. The axial wall cracks that Jubb and Malmo (1991) report as being so common in East Gippsland , Victoria (22% of lesions) were seen in only three cases (2% of lesions).

The age distribution of lame cows depends to a large extent on the types of lesions involved and the herd management. Excessive hoof wear and bruising are often most common in younger cows, low in social ranking in the herd (Dewes, 1978; Malmo, 1985). This was the case in Herd 3 where all cows were run as one mob. Where the younger cows were run separately and did not have to walk as far to pasture, they had a much lower incidence of lameness than did their older herd mates. Laminitis with sole haemorrhages has been observed to occur more frequently and more severely in first calf heifers (Bazeley and Pinsent, 1984; Colam-Ainsworth, 1989; Vermunt and Greenough, 1990). This has been partly explained by changes to a higher concentrate diet or by changes in housing. Under New Zealand conditions, there are few, if any, changes in diet, and no cattle are housed. The major change on entry into the milking herd is the sudden requirement to travel to and from pasture with older herd mates. It is noteworthy that lameness was common in heifers in this study in a herd where heifers joined the main herd at first calving, but the heifer incidence was low in the second high-incidence herd in which heifers were managed separately and had less walking to do. However, the older cows in the second herd (which had to walk much further) had a very high incidence of lameness.

Association of the onset of lameness with the stage of lactation has been another common finding (Prentice and Neal, 1972; Dewes, 1978; Eddy and Scott, 1980; Arkins, 1981; Russell *et al.*, 1982; Whitaker, 1983; Rowlands *et al.*, 1985; Choquette-Lévy, 1985; Harris *et al.*, 1988; McLennan, 1988; Collick *et al.*, 1989; Jubb and Malmo, 1991). As in our study these workers have reported a high proportion of lameness in the first 3 months of lactation. Various reasons have been suggested for this, including changes in husbandry at calving, changes in nutrition at calving (which often involve a switch to high concentrate rations), metabolic stresses associated with calving disorders or high production that may result in soft or poor quality horn, seasonal calving patterns that coincide with wet weather, and an association with oestral mounting activity. In New Zealand all of these (apart from changes to a high concentrate diet) could be involved.

Assessing the significance of the findings on the association of lameness onset with the mounting activity during oestrus presents problems. Some 95% of all cases of lameness in this study involving the bearing surface of the hoof occurred during the period of peak oestrous activity for the herd. Over the entire lactation, 36% of cases occurred within 14 days of known or apparent oestrus, although this period comprised less than 20% of the total lactation. What is not known is the time taken for lameness to develop after hoof damage. Perhaps bruising of the corium can produce lameness within a day or two. Lameness associated with the build-up of pressure under the sole when pus accumulates, as in septic pododermatitis, may take 10 -14 days to develop (Dewes, 1979). Lameness following injury to the white line that allows the accumulation of grit and dirt could take days or weeks to develop after the initial insult. Not until the impacted material penetrates the sensitive corium and a septic pododermatitis develops will lameness result.

The positive correlation between wet weather conditions and onset of lameness is consistent with reports from both Europe (Eddy and Scott, 1980; Arkins, 1981; Rowlands *et al.*, 1983; Williams *et al.*, 1986) and Australasia (Dewes, 1978; Anderson, 1985; Harris *et al.*, 1988; Jubb and Malmo, 1991). This association may be related to the softening or the reduction in strength of hoof material when subjected to a continuously wet environment, or it may be related to a change in the interaction between the environment and the claw. Sustained heavy rain washes the soil and dung off races that cows walk on, exposing bare stones and sharper rock material.

The losses associated with lameness include the costs of lost production, poorer reproductive performance, premature culling and treatment. In a case-control study, Collick *et al.* (1989) found lame cows to have higher culling rates, longer calving to conception intervals and lower pregnancy rates to first service than control cows. Lucey *et al.* (1986) reported considerable reductions in reproductive performance in lame cows, especially when lesions developed between 36 and 70 days after calving. In seasonally calving herds, reproductive performance is measured to a large extent by in-calf rates at specified times following the mating start date. The poorer reproductive performance of lame cows compared with their non-lame herd mates (Table 5.5) represents a significant economic loss that includes increased culling of empty cows this season, and either reduced milk yields associated with shorter lactations or increased costs associated with extra calving inductions next season.

The technique of survival analysis would seem useful in studying the epidemiology of lameness. It is a statistical procedure for data analysis in which the outcome variable of interest is time until an event occurs (Lee, 1984). As used in this study the variable of interest was the time from calving until the onset of lameness for cows in the milking herd only. A problematical feature of such data is the presence of censored observations. These represent subjects which have not shown the measured attribute (onset of lameness) because they have survived past the end of the study or have been withdrawn for some other reason (dried off, culled or died). In a dairy herd, cows are usually present in the milking herd for varying lengths of time. Some are removed by being culled or dying; the others are dried off at varying intervals after calving. Survival analysis can be used to overcome the data analytical problems associated with having many cows not available for the full period of analysis. It enables the comparison of survival times of cohorts despite the withdrawal of individuals throughout the study.

Study of the survival functions of different cohorts also provides more information than the simple comparison of incidences. From the survivorship function curves in Figures 5.6 and 5.9, it can be concluded that the reason for the higher incidence of lameness in 2-year-olds in Herd 1 than Herd 3 was not because the degree of exposure to risk factors for lameness was greater, but because the risk persisted for almost twice the length of time. Moreover, within Herd 1, the period of exposure to risk and the type of risk were both similar for the two age groups (3-8 years and 8+ years) that were managed together

in the one herd, but the probability of developing lameness was much greater for the older cows (8+ years).

This study has shown that there is a distinctive epidemiological pattern to clinical lameness in three New Zealand dairy herds, and some risk factors can be identified from the work. There are clearly some factors which operate at the level of the individual cow, as found in this study. However, as in an earlier study of herds with high and low lameness incidence (Chesterton *et al.*, 1989) there are clearly herd factors operating to produce the large between-herd variation in incidence found in this study. It is noteworthy that the lesions did not vary in nature between the herds, only in the proportion of animals affected.

Acknowledgments

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CHAPTER 6

Hoof growth and wear in pasture-fed dairy cattle[4]

[4] In press as: Tranter WP, Morris RS. New Zealand Veterinary Journal, 1992.

Abstract

Hoof growth and wear studies were conducted on ten spring-calving cows over 12 months. The rates of hoof wall growth and wear were lower in autumn and winter than during spring and summer. Both were greater in 2-year-old than in mature cows ($p < 0.05$). Sole wear occurred most rapidly along the abaxial edge of the weight-bearing surface, less quickly in the toe and heel areas and most slowly in the mid-sole region. Both hoof wall wear and sole wear were greater in lateral digits than in medial digits ($p < 0.01$).

Sole concavity, measured using a profile gauge, was greater in 2-year-olds prior to calving than in mature-aged cows ($p < 0.05$) but this difference disappeared soon after calving as concavity was lost in the younger cows. Medial digits almost always had greater concavity than lateral digits.

Sole concavity measurements were also made on the hind digits of 24 autumn-calving cows on four occasions at 2-monthly intervals. Prior to calving, concavity was much greater in these cows than in the spring-calving cows. However, concavity was lost rapidly following calving and concavity measurements were soon similar to those found in the spring-calving cows.

Introduction

In Australian and New Zealand dairy-farming systems where most milk production is from cattle at pasture, excessive hoof wear is regarded as a common predisposing cause of lameness (Malmo, 1991). The lesions associated with excessive wear include aseptic traumatic pododermatitis (sole bruising), septic traumatic pododermatitis (sole abscess) and sole foreign body injuries (Baggott and Russell, 1981; Malmo,1991). These lesions have been found to be the most common causes of lameness in a variety of studies.

In one of the earliest published surveys, Dewes (1978) reported a high incidence of lameness associated with excessive wear in 2-year-old Friesian heifers soon after calving. Hoof wear resulted in a complete loss of sole concavity, with bruising and sole abscess formation. In a survey of lameness on farms in south-western Victoria, Harris *et al.* (1988) found that farmers regarded overworn and bruised soles as two of the most common clinical signs of lameness. Bruised soles were positively associated with overworn soles and were present in 94% of herds with overworn soles. These workers suggest that the incidence of lameness due to overworn soles or bruised soles is likely to increase with the current trend towards larger herds. In other more recent studies, Jubb and Malmo (1991) found that underrun sole (sole abscess) and punctured sole accounted for 28% of lameness lesions in east Gippsland; Willyanto (pers. comm.) found bruised sole, excessive wear of sole and foreign body in sole to be responsible for 30% of lameness-causing lesions in cattle on the Atherton Tablelands; and Tranter and Morris (1991) reported that bruising and sole abscess accounted for 51% of lameness-causing lesions in three Manawatu herds. Less severe hoof haemorrhage, which was thought to be a subclinical manifestation of bruised sole, was also observed in up to 40% of cows under study in a herd with an annual lameness incidence of only 2% (Tranter *et al.*, 1991).

Pasture-fed dairy cattle frequently have to walk long distances each day along gravel or sometimes concrete raceways. Several hours are also spent standing on concrete each day at milking time. With this system of husbandry, especially during wet weather, severe wear of the hooves is common. The worst affected are often 2-year-old heifers entering the milking herd for the first time for two reasons. First, they tend to be milked last and consequently have maximum exposure to concrete surfaces. Second, because they are

lowest in social ranking in the herd, they tend to be jostled more by older more dominant cows.

Although numerous studies of hoof growth and wear have been conducted in the northern hemisphere where hoof overgrowth predisposes to lameness, most have involved housed cattle (Prentice, 1973; Dietz and Prietz, 1980; Clark and Rakes, 1982; Hahn *et al.*, 1986; Murphy and Hannan, 1986; Schlichting, 1987; Manson and Leaver, 1988b, 1989; Vermunt, 1990). There is little published information on cattle grazed year-round on pasture.

In order to better understand the dynamics of hoof growth and wear during the annual production cycle of a pasture-based dairy system, hoof measurements were performed on both autumn- and spring-calving cows of selected ages at various times of the year.

Materials and methods

The spring-calving herd

In June 1990, about 2 months prior to calving, ten cows (five two-year-olds, entering the milking herd for the first time, and five six-year-olds) were randomly selected from a spring-calving herd of 260 cows for hoof growth and wear studies. The herd, one of the Massey University seasonal supply dairy herds, was grazed on a 140 hectare farm near Palmerston North. All ten cows remained with their herdmates and were subjected to standard herd management practices throughout the study period.

During the winter months (June-July 1990), the cows were non-lactating. During the non-lactation period, grazing was restricted and cows were walked once daily to the dairy yards for silage supplementation. They were then returned to pasture except on those days when the farm manager considered the soil conditions were too wet and the cows were likely to cause excessive treading damage. On such days, the herd was confined to a sawdust loafing pad. After calving, 24-hour grazing was resumed and the cows walked twice daily to the dairy yards for milking. The milking herd was rotationally grazed around a sequence of 30 paddocks with grazing intervals of 30 - 40 days. Following

drying off at the end of April 1991 all cows remained at pasture, without regular walking to the dairy, until the end of the 12-month study period in June 1991.

Paddocks grazed during the winter (the non-lactation period) were about 700 metres from the dairy yards so the average daily distance walked on concrete or gravel was about 1.4 km. During lactation all 30 paddocks were grazed by the milking herd. It is estimated that the average distance walked each day along raceways during lactation was 2.8 km.

Sole concavity

On four occasions (prior to calving in June, and after calving in September, December and February), measurements of sole concavity were made on the digits of both right legs of the study cows using the techniques described by Tranter *et al.* (1992a).

Hoof wall growth and wear

Hoof wall growth and wear were measured over a 12-month period by placing a mark in the hoof wall of the digits of both right legs and monitoring its displacement from the periople line toward the end of the claw as described by Tranter *et al.* (1992a) and others (Clark and Rakes, 1982; Hahn *et al.*, 1984a; Hahn *et al.*, 1986; Manson and Leaver, 1988b; Vermunt, 1990). Measurements were performed at approximately 3-monthly intervals (June, September, December, February and June).

Sole wear

Sole wear was monitored during four periods of the year using the method described by Tranter *et al.* (1992a). In June, September, December and February, three parallel grooves, running abaxially to axially, were placed in the sole, and the time taken for them to disappear was recorded.

Comparison with autumn-calving cows

Sole concavity was also measured on 24 autumn-calving cows using the method described above. Using a stratified random-sampling technique, eight cows from each of three age groups (youngest third, middle third and oldest third of the herd) were selected for study in March 1991 from a dairy herd consisting of 180 autumn-calving cows. This Massey University herd grazed on a 110 hectare farm near Palmerston North, which was divided

into 60 paddocks. The grazing interval was about 60 days, and the average distance walked each day along raceways was about 3 km.

Sole concavity was measured on the digits of the hind legs on four occasions - prior to calving in March, and after calving in May, July and September.

Daily precipitation data were obtained from an official meteorological recording station in Palmerston North, situated within 3 km of both dairy farms.

Statistical analysis

Findings were recorded and analysed using the statistical package PANACEA 2 (PAN Livestock Services, Reading, England). Analysis of variance was used to test differences in sole concavity, sole wear, hoof wall growth and hoof wall wear between groups, digits and time of year. Summary statistics are quoted as mean ± standard deviation.

Results

Sole concavity

On most digits minimum concavity was observed at sites 10 mm from the abaxial hoof margin. Changes over time and differences between groups were similar for measurements taken at 20 mm and 30 mm from the abaxial hoof margin, so only the results of the 30 mm measurements are reported here.

Spring-calving cows

The sole concavity measurements for the spring-calving cows are shown in Tables 6.1 and 6.2. On all occasions, sole concavity was greatest in the medial hind digits and least in the lateral hind digits. Medial digits almost always had greater concavity than lateral digits.

Only small changes were observed over time. In the 6-year-old cows, sole concavity was minimal and showed little variation through the year. In contrast, a small but statistically significant loss of concavity ($p < 0.05$) was observed in the 2-year-old cows after calving

(Table 6.1). In June, prior to calving, concavity was greater in the 2-year-old cows than in the mature cows ($p < 0.05$), but this difference disappeared by September, after the cows had calved.

Both age groups suffered a loss of concavity in the lateral hind digits after calving, whereas changes over time were not statistically significant for the other digits. The combined age group sole concavity measurements for each digit are shown in Table 6.2.

Table 6.1. Sole concavity (mm) measured at 30 mm from the abaxial hoof margin of all four digits of the right legs of ten spring-calving cows at different times of the year [a]

Age class	n	June 90	Sep 90	Dec 90	Feb 91
Digits of 2-year-olds	20	2.56 ± 1.38^{y}	2.19 ± 1.24^{y,z}	1.49 ± 1.19^{z}	1.79 ± 1.29^{y,z}
Digits of 6-year-olds	20	1.81 ± 0.93	1.78 ± 0.98	1.84 ± 0.93	1.68 ± 0.96

[a] Values within each row not sharing the same superscript are significantly different ($p < 0.05$)

Table 6.2. Sole concavity (mm) measured at 30 mm from the abaxial hoof margin of the digits of the right legs of ten spring-calving cows[a]

Digit[b]	n	Jun 90	Sep 90	Dec 90	Feb 91
Med. RF	10	2.18 ± 0.89	1.93 ± 0.91	1.59 ± 0.69	2.04 ± 0.58
Lat. RF	10	1.71 ± 0.56	1.82 ± 0.43	1.81 ± 0.87	1.43 ± 0.54
Med. RH	10	3.55 ± 1.32	3.20 ± 0.88	2.78 ± 0.62	3.06 ± 0.71
Lat. RH	10	1.29 ± 0.64^{y}	0.99 ± 0.93^{y,z}	0.47 ± 0.59^{z}	0.42 ± 0.48^{z}

[a] Values within each row not sharing the same superscript are significantly different ($p < 0.05$)
[b] Med. = medial; Lat. = lateral; RF = right front; RH = right hind

Autumn-calving cows

Prior to calving, sole concavity was much greater in the autumn-calving cows than in the spring-calving cows (double for medial hind digits and three times for lateral hind digits). However, sole concavity was lost rapidly after calving (Table 6.3). By mid-May (about

50 days after calving for most cows) the concavity of the lateral hind digits had decreased to levels similar to that observed in spring-calving cows after calving. By mid-July, concavity of the medial hind digits had also decreased to levels observed in the spring-calving group. Concavity was again always greater in the medial hind digits than in the lateral hind digits.

Differences between age groups were not great. Prior to calving, concavity was greater in the middle age group of cows (4, 5 and 6-year-olds) than in the older and younger cows ($p < 0.05$), but there were no significant differences at other times.

Table 6.3. Comparison of changes in the sole concavity of right hind digits (from prior to calving through lactation) between spring- and autumn-calving cows. Concavity was measured at 30 mm from the abaxial hoof margin[a].

	n	Prior to calving	Calving + 50 days	Calving + 120 days	Calving + 180 days
Medial Digits:					
Autumn calving	24	6.92 ± 2.54[s]	3.91 ± 1.76[t]	2.42 ± 1.37[u]	2.45 ± 1.04[u]
Spring calving	10	3.55 ± 1.32	3.20 ± 0.88	2.78 ± 0.62	3.06 ± 0.71
Lateral Digits:					
Autumn calving	24	3.99 ± 1.62[v]	0.95 ± 0.56[w]	0.30 ± 0.41[x]	0.27 ± 0.37[x]
Spring calving	10	1.29 ± 0.64[y]	0.99 ± 0.93[y,z]	0.47 ± 0.59[z]	0.42 ± 0.48[z]

[a] Values within each row not sharing the same superscript are significantly different ($p < 0.05$)

Hoof wall growth and wear

Some differences were observed in the rates of hoof wall growth and wear at different times of the year. Wear rates were greatest during the spring and early summer months (post-September and post-December), and lowest during the post-February period. During the June - September period, for part of which cows were walking only once daily to the dairy, rates of wear were lower than during spring but still greater than during the February - June period (Table 6.4).

Hoof growth rates were greatest during summer and lowest during winter, resulting in a net loss of hoof wall over the winter/early spring period and in net gains during autumn. Over the entire 12 months hoof wall growth equalled hoof wall wear.

Rates of hoof wall growth and wear over the 12-month study were always greater in the 2-year-old cows than in the mature cows ($p < 0.05$). The average rates of hoof wall growth and wear over the 12 months for both age groups are presented in Table 6.5.

Table 6.4. Rates of hoof wall growth and wear for the digits of the right legs of ten spring-calving cows over the 12-month study period[a]

	n	Post-June	Post-Sep	Post-Dec	Post-Feb
Growth (mm/week)	40	1.00 ± 0.24^{q}	1.34 ± 0.25^{r}	1.56 ± 0.4^{s}	1.20 ± 0.17^{t}
Wear (mm/week)	40	1.13 ± 0.21^{u}	1.34 ± 0.12^{v}	1.42 ± 0.24^{v}	0.98 ± 0.15^{w}
Growth - wear[b] (mm/week)	40	-0.13 ± 0.23^{x}	0 ± 0.19^{y}	0.13 ± 0.37^{z}	0.22 ± 0.12^{z}

[a] Values within each row not sharing the same superscript are significantly different ($p < 0.05$)
[b] Net hoof wall growth

Table 6.5. Comparison of mean rates of hoof wall growth and wear between age groups for the digits of the right legs of ten spring-calving cows

	2-year-olds n = 20	6-year-olds n = 20
Hoof wall growth (mm/week)	1.38 ± 0.16	1.10 ± 0.07
Hoof wall wear (mm/week)	1.30 ± 0.15	1.09 ± 0.07

Hoof wall wear over the 12 months was greater in lateral digits than in medial digits. Hoof wall growth also tended to be greater in lateral digits but differences were not significant (Table 6.6). There were no differences in hoof wall growth or wear between front and hind digits (Table 6.7).

Table 6.6. A comparison of mean rates of hoof wall wear, sole wear, hoof wall growth and sole concavity between lateral and medial digits for both right legs of ten spring-calving cows over 12 months

	Lateral digits n = 20	Medial digits n = 20	p
Hoof wall wear (mm/week)	1.28 ± 0.26	1.16 ± 0.25	< 0.01
Sole wear (weeks)	3.70 ± 1.28	4.30 ± 1.38	< 0.001
Hoof wall growth (mm/week)	1.33 ± 0.41	1.23 ± 0.25	> 0.05
Sole concavity (mm)	1.20 ± 0.80	2.50 ± 1.00	< 0.01

Table 6.7. A comparison of mean rates of hoof wall growth and wear between front and hind digits for the right legs of ten spring-calving cows over 12 months

	Front digits n = 20	Hind digits n = 20	p
Hoof wall growth (mm/week)	1.26 ± 0.23	1.26 ± 0.15	> 0.05
Hoof wall wear (mm/week)	1.18 ± 0.17	1.22 ± 0.16	> 0.05

Sole wear

The pattern of sole wear was similar on each of the four occasions that grooves were placed in the weight-bearing surface of the right leg digits of the ten spring-calving cows. Wear (as measured by the number of weeks taken for the grooves to disappear) occurred most rapidly along the abaxial edge, less quickly in the toe and heel areas and most slowly in the mid-sole region. The average times taken for grooves to disappear at each of the sites of observation for all four occasions are shown in Table 6.8 and the order of wear is shown in Figure 6.1.

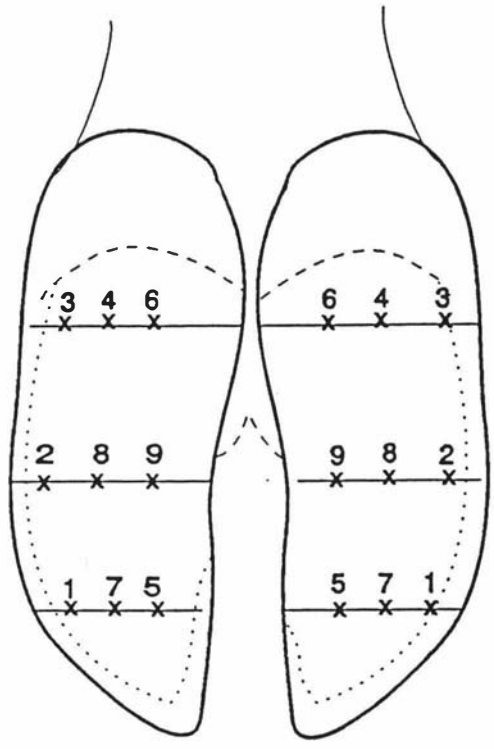

Figure 6.1. **The most typical order in which grooves disappeared over time from the soles of the digits of ten spring-calving cows. The dotted line represents the white line.**

Table 6.8. Pattern of sole wear (measured in weeks taken for 1.5 mm grooves to disappear) observed in the right leg digits of ten spring calving-cows on four measurement occasions

Order of wear	Site of measurement[a]	n	Wear in weeks[b]
1	Toe 10 mm	160	2.5 ± 0.9^{t}
2	Mid-sole 10 mm	160	2.9 ± 0.7^{s}
3	Heel 10 mm	160	3.2 ± 1.8^{u}
4	Heel 20 mm	160	4.3 ± 1.0^{v}
5	Toe 30 mm	160	4.4 ± 1.3^{v,w,x}
6	Heel 30 mm	160	4.5 ± 1.0^{w,x}
7	Toe 20 mm	160	4.6 ± 1.2^{x,y}
8	Mid-sole 20 mm	160	4.8 ± 1.3^{y}
9	Mid-sole 30 mm	160	5.0 ± 1.2^{z}

[a] Distance from the abaxial hoof margin
[b] Values for wear not sharing the same superscript are significantly different ($p < 0.05$)

Rates of sole wear showed a similar pattern to that observed for hoof wall wear, with rates being slowest following the February grooving. During winter prior to calving (June), when cows were walking only once daily to the milking yards, wear rates at sites away from the abaxial hoof margin were greater than those following the February grooving, but were slightly lower than during September and December (Table 6.9). The number of wet days and the daily average hours of sunshine during the first 4 weeks following each grooving are also presented in Table 6.9.

Table 6.9. Sole wear (measured in weeks taken for 1.5 mm deep grooves to wear out) for the digits of the right legs of ten spring-calving cows, and weather conditions recorded during the first 4 weeks following each grooving. Wet days refer to days with > 0.1 mm precipitation.

	Jun 90	Sep 90	Dec 90	Feb 91
Wet days	21	10	17	9
Av. daily sunshine hours	2.6	5.0	6.5	5.9
Av. daily maximum temperature	13.1°C	14.1°C	20.5°C	20.3°C
Sole wear[a]:				
10 mm from abaxial margin[b]	2.6 ± 0.8	2.9 ± 0.5	2.9 ± 0.4	3.0 ± 0.5
20 mm from abaxial margin[b]	4.7 ± 0.8^{w}	4.2 ± 0.5^{x}	4.1 ± 0.9^{x}	5.1 ± 1.2^{w}
30 mm from abaxial margin[b]	4.5 ± 0.8^{y}	4.3 ± 0.5^{y}	4.4 ± 0.9^{y}	5.3 ± 1.0^{z}

[a] Values are composites of toe, mid-sole and heel observations made at the specified distance from the abaxial hoof margin

[b] Values for wear within each row not sharing the same superscript are significantly different ($p < 0.05$)

For almost every site of measurement, the rates of wear were greater in lateral digits than in medial digits (Table 6.10).

Table 6.10. A comparison of sole wear (measured in weeks taken for 1.5 mm deep grooves to wear out) between lateral and medial digits for both right legs of 10 spring-calving cows on four measurement occasions

Site of measurement[a]	Lateral digits n = 80	Medial digits n = 80	p
Toe 10 mm	2.2 ± 0.7	2.8 ± 0.9	< 0.01
Toe 20 mm	4.2 ± 1.1	4.9 ± 1.3	< 0.01
Toe 30 mm	4.3 ± 1.3	4.3 ± 1.3	NS
Mid-sole 10 mm	2.7 ± 0.7	3.1 ± 0.7	< 0.01
Mid-sole 20 mm	4.3 ± 1.1	5.2 ± 1.2	< 0.01
Mid-sole 30 mm	4.7 ± 1.2	5.3 ± 1.2	< 0.01
Heel 10 mm	3.0 ± 0.8	3.5 ± 0.7	< 0.01
Heel 20 mm	3.9 ± 1.0	4.6 ± 0.9	< 0.01
Heel 30 mm	4.2 ± 0.9	4.7 ± 1.0	< 0.01

[a] Distance from abaxial hoof margin

The relationship between mid-sole hoof wear, as measured in weeks taken for grooves to disappear, and sole concavity was examined. Weeks taken for groove disappearance at 30 mm from the abaxial margin was positively correlated with sole concavity measured at the same site (r = + 0.27).

Discussion

Sole concavity

Information on changes in the depth of sole concavity in cattle has not previously been published. In the only report of measurements of sole concavity in cattle we have found (McDaniel, 1983), little information was provided on the method that was used but it appears that the area of the sole that failed to contact the ground was measured. Cows in comfort stalls had twice the area of concavity as those in free stalls.

Our method of using an engineer's profile gauge to reproduce the contour of the weight-bearing surface of the digit provided a useful method for objectively measuring sole concavity. Changes in sole concavity over time provide another measure of sole wear. Notable findings of this study included the large difference in sole concavity between autumn-calving and spring-calving cows prior to calving and the rapid loss of concavity that occurred after calving, especially in the autumn-calving cows.

It is a common practice for rising 2-year-old heifers to be mixed with the adult milking herd either at their first calving or, for spring-calving herds, a few months prior to calving at the beginning of winter. Before heifers join the herd, they remain at pasture and only rarely do their digits encounter abrasive surfaces. Under these conditions their hooves are almost always healthy and well-shaped. Lameness is uncommon. The soles of their hooves normally have a slightly concave profile which, in hind legs, is more pronounced on medial than lateral digits (Toussaint Raven *et al.*, 1985). On a smooth surface weight is borne by the distal border of the wall (largely abaxially) (Toussaint Raven *et al.*, 1985; Scott, 1988a, 1988b) and on part of the horn of the heel. Sole hoof is 5 - 7 mm thick (Toussaint Raven *et al.*, 1985) and provides good protection to the inner structures of the digit. The concave shape of the sole surface ensures that most force is exerted on the wall and abaxial areas of the sole. Shocks are progressively absorbed as the sole presses on to the ground, and spreading of the digits occurs to some extent. It is when housing conditions result in insufficient wear or when wear is excessive due to frequent yarding and walking for long distances on rough surfaces that the risk of lameness increases (Dewes, 1978; Toussaint Raven *et al.*, 1985; Malmo,1991).

The first measurements of concavity in this study were made in June on animals from the spring-calving herd prior to calving. The mature cows appeared to have regained little of their normal concavity since drying off at the end of April. Presumably this was due to the continual wear caused by once daily walking to the milking yards and the few hours spent on concrete each day while consuming silage. The 2-year-old heifers, having been in the herd for just 3 weeks, had greater sole concavity than their older herd mates.

By September, when all animals had calved and twice daily walking to the milking yards had commenced, sole concavity of the 2-year-old heifers was significantly less than in June. There was now little difference between the two age groups. Further losses of sole

concavity occurred between September and December, especially in the hind lateral digits. There was a tendency for medial digits to regain some sole concavity by February.

In contrast, cows from the autumn-calving herd had considerable sole concavity at the time calving commenced in March. The older cows had even greater sole concavity than the 2-year-old heifers entering the milking herd for the first time. Both heifers and cows from this herd had spent the dry summer months of January and February at pasture with almost no exposure of their hooves to gravel or concrete. It appears that under these conditions cows that had been previously lactating were able to fully regain the normal sole concavity that is observed in young heifers prior to calving. However, the loss of sole concavity was substantial and rapid as soon as twice daily exposure to gravel raceways and concrete yards recommenced. Within 50 days of calving, the soles of the lateral hind digits of most cows were visibly flat and sole concavity measurements were similar in magnitude to those observed in the spring-calving cows at the same stage of lactation. Considerable sole wear also occurred on the medial hind digits but again medial digits retained more concavity than lateral digits. This commonly observed difference between medial and lateral hind digits leads to the suspicion that lack of sole concavity of lateral digits may have a causal relationship with the higher incidence of lameness in lateral digits that has frequently been reported (Tranter and Morris, 1991).

While the exact mechanism by which sole concavity changes has not been investigated directly, the simultaneous measurement of sole concavity, hoof growth and hoof wear supports the suggestion that change in concavity is a direct consequence of wear exceeding growth. The effect of this is most marked in early lactation and it is hypothesised that this is due to the abrasive surfaces in yards and raceways with which hooves come in contact once twice daily walking to the dairy for milking resumes.

Hoof wall growth and wear

A number of factors such as age, season, nutrition and environment have been reported to have an effect on hoof wall growth and wear. The rates of hoof wall growth and wear recorded in this study are similar to those reported for cows kept on concrete (Clark and Rakes, 1982; Manson and Leaver, 1988b, 1989) and to those reported for young cows kept on pasture or dry lot (Hahn *et al.*, 1986) but lower than those for young cows confined to new abrasive concrete (Hahn *et al.*, 1986). The growth and wear rates

observed in this study are considerably higher than those reported by Prentice (1973) in mature Ayrshire cows kept on concrete during winter and at pasture during summer.

Hoof wall growth rates were higher in the younger cows. This was to be expected as similar differences between age groups have been previously reported. Prentice (1973) found higher hoof wall growth rates in calves and yearlings than in mature cows. Hahn *et al.* (1978) observed higher hoof wall growth rates in first rather than second lactation cows and Glicken and Kendrick (1977) also showed that the hooves of younger cows grow faster than those of older cows. However, Clark and Rakes (1982) found that rates of hoof wall growth were not related to age.

In our study, hoof wall growth rates were lowest in late autumn and winter and highest during summer (Table 6.4). Such a seasonal variation in hoof growth has been observed by other authors (Clark and Rakes, 1982; Hahn *et al.*, 1986; Vermunt, 1990). Hahn *et al.* (1986) suggested that the cyclical nature of hoof growth could be due to variation in environmental temperature, photoperiod, herd management or nutrition. Clark and Rakes (1982) demonstrated that periods of increased hoof horn growth coincided with increasing photoperiod length while decreased hoof growth coincided with decreasing photoperiod length. Periods of increasing hoof growth also coincided with increasing photoperiod length in Vermunt's studies (1990). However, Wheeler *et al.* (1972), working with sheep, concluded that hoof growth was not influenced by photoperiod but that it was greatly reduced by low temperatures. Close examination of our results (Table 6.4) reveals that hoof wall growth rates were higher during the warm summer months when photoperiod length was starting to decrease than they were during the late spring when photoperiod length was increasing. This suggests that total photoperiod length or environmental temperature have a greater influence than changes in photoperiod. Vermunt (1990) also raised the possibility of hoof growth being affected by changes to the circulation during extremely cold weather that involve constriction of arterioles and dilation of arteriovenous shunts. This process is unlikely to have occurred at the relatively mild temperatures that the cows in this study were exposed to. Mean daily temperatures during the months of low hoof growth rate (June - September) were 9 - 10 °C compared with the -10 to -20°C recorded during Vermunt's study.

The nature of the surface to which hooves are exposed has a large influence on the rate of hoof wear. Cows housed on abrasive concrete have been shown to have 35% greater

hoof wear than did cows at pasture Hahn *et al.*, (1986). In Vermunt's study (1990), hoof wall wear was lower in heifers housed outdoors on a dry feedlot than in heifers housed indoors on slats. Hoof growth and wear were both lower in calves reared on deep litter than in those housed on slatted flooring (Schlichting, 1987). Higher wear rates were also observed in steers kept on slats than in those kept on straw (Murphy and Hannan, 1986). For pastured cattle, environmental influences on hoof wear are likely to be largely mediated through raceway and yard surface conditions. During prolonged wet weather, soil and dung are washed off the race surface or, when drainage is inadequate, water lies on the surface turning superficial layers in to mud. In either case, stones and sharper rock material are exposed, making the surface more abrasive. In addition, wet concrete is more abrasive than dry concrete (Camara and Gravert, 1971).

Hoof growth is increased by hoof trimming (Manson and Leaver, 1988b, 1989) suggesting that some compensatory mechanism stimulates hoof horn growth. Consequently, factors that influence hoof wear at the different stages of the annual production cycle are also likely to affect the rate of hoof growth.

The lack of difference in hoof wall growth and wear rates between front and hind digits (Table 6.7) is consistent with the findings of Clark and Rakes (1982). However, Prentice (1973) reported that hoof wall of front digits grew faster than that of rear digits while Hahn *et al.* (1986) found that hoof wall of hind digits grew faster than that of front digits.

Sole growth and wear

In contrast to the large numbers of studies on hoof wall growth and wear, there is little published information on sole horn growth and wear since these are more difficult to measure. Greenough *et al.* (1990) calculated net growth rates for sole horn by comparing sole horn thickness of hooves of groups of beef steers after slaughter.

Although monitoring the rate at which grooves disappeared from the sole provided only a crude measure of sole wear, some valuable insights into the process of sole wear were gained from this study. The pattern of wear was similar during each of the four periods of observation. Wear occurred most rapidly along the abaxial margin of the bearing surface of the hoof, fastest cranially near the toe and slightly less quickly at the mid-sole

and heel sites of observation. Wear occurred more slowly at the other toe and heel sites and was almost always slowest in the axial mid-sole area.

The slower wear rates in June can be explained by the fact that cows were not lactating and were walking only once daily to the yards. However, the reason for the slower rates of wear in February is unclear. One possible reason is the drier condition of the surface of the raceways associated with a combination of fewer wet days, more hours of sunshine and higher daily maximum temperatures during the four weeks following placement of the grooves. The average daily hours of sunshine and average daily maximum temperature were similar in February to those in December but there was only about half as many wet days. September had a similar number of wet days to February but daily temperatures were considerably lower and daily hours of sunshine lower. Thus the effects may be seen as an interaction between various factors that influence the microclimate in the immediate vicinity of the hoof.

The differences in the rates of sole wear between lateral and medial digits was not surprising as sole wear was dependent on sole concavity and concavity was consistently greater in medial digits. In addition, previous work has shown that lateral digits experience higher pressures during load bearing, providing another possible explanation (Toussaint Raven, 1973; Ossent *et al.*, 1987; Scott, 1988a).

These results confirm that numerous factors influence hoof growth and wear. Wear is greatest in early lactation. Any sole concavity regained during the dry period is lost soon after calving when cows recommence twice daily walking to the dairy for milking.

Acknowledgments

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CHAPTER 7

Seasonal variation in the physical hoof characteristics of 10 cows over 12 months[5]

[5] Submitted as: Tranter WP, Morris RS, Horne DJ; Morgan DE. New Zealand Veterinary Journal, 1992.

Abstract

A longitudinal study of the physical hoof properties of 10 cows was conducted over a 12 month period to evaluate the influence of environmental moisture on the physical nature of bovine hoof. On four occasions during the year, hoof moisture was measured using a hand-held moisture meter, hoof hardness using a Shore Type D Durometer and the elastic modulus, compressive strength and resilience of hoof tissue were measured using compression tests on biopsy samples collected from the soles and hoof walls of the left leg digits of the 10 cows.

The wetness of the environment to which the cows' hooves were exposed during the two weeks prior to each occasion of hoof measurements was assessed using details of soil gravimetric moisture content, total rainfall, number of wet days and hours of sunshine. There was a clear tendency for hoof moisture to vary in concert with these various measures of environmental moisture. When the association was evaluated using regression analysis gravimetric soil moisture content was the most influential contributor in the regression relationship between hoof moisture and the measures of environmental wetness.

Sole hoof hardness and the elastic modulus, compressive strength and resilience of sole samples were negatively correlated with sole hoof moisture levels. Likewise, wall hardness and the elastic modulus of wall hoof samples were negatively correlated with wall moisture percentage. In contrast, the compressive strength of wall hoof seemed to be unaffected by moisture content while resilience of wall hoof increased with increasing hoof moisture levels.

Results of the study confirm that the physical properties of the external layers of hoof keratin are affected by environmental moisture conditions. The extent to which each of these properties is related to the various mechanical modes of failure that are involved in producing lameness lesions in dairy cattle is discussed.

Introduction

An association between wet weather conditions and the incidence of lameness in dairy cattle has been reported both in Europe (Eddy and Scott, 1980; Arkins, 1981; Rowlands *et al.*, 1983; Williams *et al.*, 1986) and in Australia and New Zealand (Dewes, 1978; Anderson, 1985; Harris *et al.*, 1988; Jubb and Malmo, 1991; Tranter and Morris, 1991). This association may be due to changes in the physical characteristics of hoof material when subjected to a continuously wet environment or it may be related to a change in the interaction between the environment and the claw.

Wet conditions are thought to soften the hoof, predisposing cows to lameness. Some have suggested that softening of the hoof horn results in greater surface wear and hence increased susceptibility to sole puncture (Jubb and Malmo, 1991). Baggott and Russell (Baggott and Russell, 1981) have suggested that the association between wet conditions and sole ulcers may be due to the poor resistance of the horn to concussion under such conditions. Wet conditions underfoot are thought to weaken the integrity of the white line, by both softening it and reducing its strength, thereby predisposing it to the disintegration observed in white line disease.

Although various studies have shown that hoof hardness falls as hoof moisture levels increase, little is known about changes to other physical properties of hooves when they are subject to unusually wet conditions. In their studies of the effects of dietary supplementation of biotin on pig hoof horn, Webb *et al.* (1984) have suggested other properties of hoof, such as its compressive strength, elasticity and resilience, are also important when considering modes of mechanical failure that result in foot lesions of livestock.

In this study a number of different hoof properties were measured at different times of the year. The objective was to determine to what extent these properties varied with changes in environmental moisture conditions.

Materials and methods

Preliminary study

In February 1990, 5 cows were randomly selected from a dairy herd of 260 Friesian cows for initial studies of hoof moisture levels using the method outlined below. Measurements were made on the digits of the left legs every second month until June 1990.

Main study

Five two-year-old cows, entering the milking herd for the first time, and five six-year-old cows were randomly selected from the same herd of 260 cows in June 1990 approximately two months prior to calving. All 10 cows remained with their herdmates and were subjected to normal herd management throughout the study period. The herd, one of the Massey University seasonal supply dairy herds, was grazed on 90 hectares near Palmerston North.

During the winter months (June/July 1990), all cows were non-lactating. During the non-lactating period, grazing was restricted and cows were walked once daily to the dairy yards for silage supplementation. They were then returned to pasture except on those days when the farm manager considered soil conditions were wet and cows were likely to cause excessive treading damage. On such days, the herd was confined to a sawdust loafing pad. After calving, 24 hour grazing was resumed and cows walked twice daily to the dairy yards for milking. The milking herd was rotationally grazed around a sequence of 30 paddocks.

Physical measurements

On four occasions (June, September, December and February), measurements of hoof hardness and hoof moisture were made on the digits of the left legs of the study cows using the methods previously described (Tranter *et al.*, 1992a). On each occasion, hoof samples were also collected from the ten cows for compression tests. From each digit of each left leg a toe clipping was taken with foot trimmers and a sole sample was collected from near the sole-heel margin.

Compression tests

From the samples collected, rectangular hoof blocks were prepared and compression tests were performed as previously described (Tranter *et al.*, 1992a). From the force deformation curves that were produced the elastic modulus, compressive strength and resilience of each sample were derived.

Weather conditions and soil moisture measurements

The soil on the farm where the cattle were grazed, Tokomaru silt loam, is fine textured and has very poor natural drainage. Although subsurface drainage has been installed over nearly all the farm, the surface soil is often wet during and after rainfall in winter and spring.

To determine the gravimetric water content in the top 3 cm of soil, six cores were removed from each of three typical paddocks on the farm. Soil sampling was carried out on alternate days over each two week period prior to hoof measurements.

Rainfall and sunshine hours were measured at a meteorological site three kilometres from the farm.

Statistical Analysis

Findings were recorded and analyzed by microcomputer using the statistical package PANACEA 2 (PAN Livestock Services, Reading, England). Differences in the physical characteristics of hoof at different times of the year were assessed by analysis of variance. Summary statistics are quoted as mean ± standard deviation. Regression analysis was used to test the linear relationship between hoof moisture and the other hoof characteristics measured and between hoof moisture and environmental conditions. Correlation coefficients are quoted for each analysis. The size of the coefficient of determination and the results of an analysis of variance (comparing the mean square due to regression with the residual mean square) were used to test the strength of regression relationships. Results are reported in the form of the number of residual degrees of freedom in the analysis of variance, the regression degrees of freedom in all cases being 1. The p value quoted is the probability that this degree of linear relationship could occur by chance.

Results

Preliminary study of hoof moisture

The results of the preliminary study of hoof moisture levels along with details of rainfall prior to each measurement date are shown in Table 7.1. Hoof moisture measurements were highest in the wet winter month and lowest in the dry months of February and April. Weather conditions during February 1990 were particularly dry and this is reflected in the low values obtained for hoof moisture on that occasion.

Table 7.1. Hoof moisture levels in the left leg digits of five cows on three different occasions

	FEBRUARY 90 n = 20	APRIL 90 n = 20	JUNE 90 n = 20
Sole Moisture %	28.2 ± 1.2^{s}	30.8 ± 1.3^{t}	34.5 ± 2.5^{u}
Heel Moisture %	33.0 ± 3.2^{v}	37.6 ± 4.1^{w}	40.3 ± 6.2^{w}
Wall Moisture %	24.3 ± 1.3^{x}	25.9 ± 1.2^{y}	28.4 ± 2.9^{z}
Wet days in week prior to measurement*	0	1	3
Rainfall in mm in week prior to measurement	0.5	6.5	29.1

Values in the same row not sharing the same superscript are significantly different ($p < 0.05$) when tested by analysis of variance
* Days with > 1.0 mm rainfall

Main study : physical hoof characteristics

The mean values for the various hoof properties as measured on the four different occasions are detailed in Tables 7.2, 7.3, and 7.4. Table 7.2 also includes details of the weather conditions prior to each day of measurement and the gravimetric soil moisture content on the day of measurement.

The hooves under study were subject to the wettest environmental conditions in June when regular rainfall and a small number of hours of sunshine resulted in high soil moisture levels. Hoof moisture content was highest at this time and hoof hardness lowest for all three sites of measurement. The elastic modulus, compressive strength and resilience for the sole were also lowest in June. Wall measurements for elastic modulus were low as well. However, the values for hoof wall for resilience and compressive strength were unexpectedly higher than at other times.

Rainfall for February, normally one of the driest months of the year, was unusually high. Consequently soil moisture content, usually very low over summer, was similar to values measured in September. Almost 10 mm of rain fell in the 24 hours prior to the February measurement. Hoof moisture levels were significantly lower than in June but tended to be higher than in September and December. There was little variation in hoof hardness between the February, December and September readings (Tables 7.2, 7.3, and 7.4).

Gravimetric soil moisture content was lowest in December. At this time, sole elastic modulus, resilience and compressive strength were higher than they were at other measurements.

Table 7.2. Sole hoof characteristics of the left leg digits of ten cows on four different occasions*

	JUN 90 n = 40	SEP 90 n = 40	DEC 90 n = 40	FEB 91 n = 40
Moisture %	33.8 ± 1.9[a]	30.3 ± 1.8[b]	31 ± 2.1[b]	32.6 ± 1.7[c]
Hardness (Shore units)	49.4 ± 2.2[d]	50.8 ± 3.2[e]	52.0 ± 3.2[e,f]	52.5 ± 2.9[f]
Elastic Modulus (MPa)	79 ± 23[g]	105 ± 26[h]	155 ± 45[i]	91 ± 28[j]
Compressive Strength (MPa)	7.0 ± 1.1[k]	8.0 ± 1.4[l]	11.2 ± 2.5[m]	8.7 ± 2.1[l]
Resilience (kJ/m^3)	124 ± 33[n]	147 ± 42[o]	206 ± 100[p]	175 ± 88[o,p]
Wet Days in week prior to measurement**	3	0	3	2
Rainfall in mm in week prior to measurement	15	0.3	13.3	11.2
Sunshine hours in week before measurement	14.3	35	44.1	48.3
Soil Moisture%	54.5%	45.7%	39.6%	43.1%

*Values in the same row not sharing the same superscript are significantly different ($p < 0.05$) when tested by analysis of variance

** Days with > 1.0 mm rainfall

Table 7.3. Heel hoof characteristics of the left leg digits of ten cows on four occasions*

	JUN 90 n = 40	SEP 90 n = 40	DEC 90 n = 40	FEB 91 n = 40
Moisture %	44.4 ± 6.5[a]	36.5 ± 4.5[b]	37.9 ± 5.0[b]	40.3 ± 4.2[c]
Hardness (Shore units)	34.8 ± 4.2[d]	40.1 ± 4.4[e]	40.8 ± 5.1[e]	39.4 ± 3.9[e]

*Values in the same row not sharing the same superscript are significantly different ($p < 0.05$) when tested by analysis of variance

Table 7.4. Wall hoof characteristics of the left leg digits of ten cows on four occasions*

	JUN 90 n = 40	SEP 90 n = 40	DEC 90 n = 40	FEB 91 n = 40
Moisture %	28.8 ± 1.5[a]	26.4 ± 1.0[b]	25.6 ± 1.2[c]	26.0 ± 1.3[b,c]
Hardness (Shore units)	66.9 ± 3.0[d]	74.4 ± 2.4[e]	74.8 ± 2.4[e]	74.1 ± 2.2[e]
Elastic Modulus (MPa)	289 ± 64[f]	369 ± 118[g]	381 ± 97[g]	367 ± 112[g]
Compressive Strength (MPa)	24.4 ± 3.2[h]	24.4 ± 3.3[h]	24.4 ± 3.5[h]	22.6 ± 3.0[i]
Resilience (kJ/m^3)	530 ± 219[j]	444 ± 198[j]	349 ± 157[k]	357 ± 145[k]

*Values in the same row not sharing the same superscript are significantly different ($p < 0.05$) when tested by analysis of variance

Association between hoof moisture and other hoof characteristics

The relationships between hoof moisture (independent variable) and the other physical properties of hoof that were measured (dependent variables) were evaluated using simple linear regression. These relationships were tested by ANOVA for significance and the results are shown in Tables 7.5 and 7.6.

Table 7.5. Regression relationships between sole hoof moisture (independent variable) and other sole hoof properties (dependent variables)

	Hardness	Elastic Modulus	Compressive Strength	Resilience
Correlation coefficient	-0.33	-0.39	-0.41	-0.35
Regression constant	66	336	22.4	535
Regression coefficient[a]	-0.5 ± 0.1	-7.2 ± 1.4	-0.43 ± 0.08	-11.7 ± 2.5
ANOVA:				
DF	156	153	152	153
F Ratio	19.2	27.0	30.3	21.6
p	< 0.001	< 0.001	< 0.001	< 0.001
Coefficient of Determination	0.11	0.15	0.17	0.12

[a] Values are quoted as mean ± standard error

Table 7.6. Regression relationships between wall hoof moisture (independent variable) and other wall hoof properties (dependent variables)

	Hardness	Elastic Modulus	Compressive Strength	Resilience
Correlation coefficient	-0.75	-0.34	0.05	0.32
Regression constant	119	888	21.4	513
Regression coefficient[a]	-1.7 ± 0.1	-20.1 ± 4.6	0.09 ± 0.15	35 ± 8.5
ANOVA:				
DF	157	151	151	151
F Ratio	200	19.4	0.38	17.0
p	< 0.001	< 0.001	0.539	< 0.001
Coefficient of Determination	0.56	0.11	0.003	0.10

[a] Values are quoted as mean ± standard error

Association between hoof moisture and environmental moisture conditions

Variables that could influence the wetness of the environment that the cow's hooves were exposed to were evaluated for their association with hoof moisture levels. These variables included "gravimetric soil moisture content on the day of hoof measurement", "rainfall in mm during the week prior to hoof measurement", number of wet days (days with > 1.0 mm rainfall) during the week prior to hoof measurement", and "hours of sunshine during the week prior to hoof measurement". Using backward stepwise multiple

regression analysis, "Hours of sunshine" was eliminated from the final equation. Table 7.7 gives details of the final regression equation for the three sites of hoof moisture measurement.

Table 7.7. Regression relationships between hoof moisture level (dependent variable) and "soil moisture", "rainfall in week prior to hoof measurement" and "number of wet days in week prior to hoof measurement" (independent variables)

	Sole	Heel	Wall
Beta coefficients:			
Soil moisture	9.99	31.9	22.1
Wet days in last week	-2.79	-3.5	0.63
Rainfall in last week	0.74	1.07	-0.1
ANOVA:			
DF	156	158	158
F Ratio	26.9	18.4	49.6
p	<0.001	<0.001	< 0.001
Coefficient of Determination	0.34	0.26	0.49

Discussion

White line disease, sole bruising and septic pododermatitis (sole abscess) are among the most common lameness-causing foot lesions observed in Australia and New Zealand (Jubb and Malmo, 1991; Tranter and Morris, 1991). All have been shown to be associated with rainfall and all are thought to be due, at least in part, to traumatic damage to the hooves. White line disease involves disintegration of the softer horn that forms the junction between the outer hoof wall and underlying sole, and impaction into this site of dirt, grit and small stones. Septic pododermatitis with underrunning of the sole is presumed in most cases to be due to entry of infection through small defects in the hoof. These defects are again most likely to occur along the horn of the white line.

Changes in the physical properties of the hoof that result in weakening of the horn and that make it more susceptible to breakdown are likely therefore to predispose cows to lameness. There is however no single ideal measure of hoof strength. Hardness of the hoof, which reflects its resistance to penetration, is usually regarded as an important property. The compressive strength is another item which has been measured by a few

earlier workers (Simmins and Brooks, 1980; Webb *et al.*, 1984). Webb *et al.* (1984) describe compressive strength as the amount of stress on the tissue which corresponds to the point at which cellular structures are ruptured. The elastic modulus is a measure of the rigidity or stiffness of a material and is yet another hoof property that is likely to contribute to the hoof's ability to resist traumatic damage. Resilience is a further characteristic which measures both the elasticity of a material and its strength.

Sole bruising involves traumatic damage and haemorrhage in the corium underlying the sole. Softening of the horn, thinning of the sole due to excessive wear or lowering the resilience of the horn (the amount of energy the hoof is capable of absorbing) are all likely to predispose cows to this form of injury.

Although changes in hoof moisture content have commonly been suggested as one of the reasons for the association between wet weather and lameness in dairy cattle, there are few documented reports of the degree to which hoof moisture changes. Our findings confirm that variation in hoof moisture is associated with the wetness of the soil environment. With only four times of the year at which observations were made, the nature of the link between measures of environmental moisture level and moisture content of the wall, sole and heel can only be considered in general terms. There is clearly a tendency for hoof moisture in Tables 7.1 to 7.4 to vary in concert with the various measures of environmental moisture content, with sole and wall showing stronger linkage than did heel moisture.

The relationship was then examined in more detail by multiple regression analysis, with backward elimination of variables. Of the variables examined that describe the wetness of the environment the gravimetric soil water content was clearly the most influential contributor in the regression relationship between hoof moisture and the various measures of environmental wetness described in Table 7.7. The other variables are causally linked to soil moisture, and although both wet days and total rainfall contributed to explaining the variation in hoof moisture, their influence varied between parts of the hoof and was much weaker than the most direct antecedent factor - soil moisture. Williams *et al.* (1986) associated lameness incidence with amount of rainfall during the two weeks prior to onset, and with potential soil moisture deficit. They suggested that it might be possible to obtain stronger correlations between lameness incidence and the wetness of cow's feet by using more refined measures of soil moisture. Our findings add

support for this suggestion by confirming that hoof moisture is significantly influenced by soil moisture content and by confirming that hoof moisture levels affect other physical hoof properties which are likely to modify the hoof's ability to resist traumatic damage.

Various workers have studied factors that may influence hoof hardness. Clark and Rakes (1982) found that hoof hardness in cows supplemented with methionine was lower than in unsupplemented cows. In their study hoof hardness was unrelated to hoof colour. Baggott *et al.* (1988) showed that hoof hardness is related to the chemical composition of the horn. A high concentrate/silage ratio in the diet reduced hoof hardness in Manson and Leaver's study (1989). In an earlier report (1988b), they had shown no effect of hoof trimming on horn hardness. Reilly and Brooks (1990) reported that biotin supplementation, previously shown to influence the physical properties of pig hoof (Webb *et al.*, 1984), produced significantly harder hoof tissue in cattle.

Hoof hardness also depends on the moisture content of the horn (Dietz and Prietz, 1980). Negative correlations between hoof hardness and hoof moisture have been reported by others (Martig *et al.*, 1980). Our results confirm these previous reports and show that the correlation between hardness and moisture is greater for wall horn than it is for the sole.

The horny capsule surrounding the claw is responsible for protecting the sensitive tissues of the foot from infection and traumatic injury. It has often been suggested that properties other than hoof moisture and hardness are likely to be important in providing the hoof with this ability to resist external damaging influences. Resistance to abrasive wear, viscoelasticity, fracture toughness, concussion resistance, elasticity, resilience, strength, stiffness, and mechanical hysteresis are all properties of hoof that have been mentioned in reports of lameness-related research as being of importance. Some have been studied in the hooves of dead animals but rarely have any been measured on hoof biopsy samples collected from live animals.

The techniques used in this study were successful in providing hoof samples of adequate size to perform compression tests, yet not causing any detriment to the cows from which they were collected. Some difficulties were encountered in preparing symmetrical blocks of tissue for testing and this may have contributed to some of the wide variation in the measured values (note the large standard deviations in Tables 7.2 and 7.4). Despite these

practical difficulties the results obtained provide useful insights into relationships among the various measures of response to compression.

The elastic modulus, compressive strength and resilience of the sole samples were all influenced by wet environmental conditions. It appears that they respond far more to wet conditions than do the simpler measures of moisture and hardness - variation in these two values do not explain the pattern of lameness since they show very limited variation. Likewise, the elastic modulus of wall hoof samples was negatively correlated with wall moisture percentage. In contrast, moisture content did not appear to influence wall compressive strength, and wall resilience increased at higher moisture levels.

On examination of results from this and related studies, a tentative hypothesis can be put forward, as a basis for further research, to explain the well recognised association between lameness onset and rainfall. The wall of the hoof forms a frame, within which the sole is attached in a manner which could be regarded as similar to the mat on a trampoline. As the animal walks, the wall is pressed outwards by contact with the ground, and the sole is stretched within the limits imposed by its connection to the wall through the white line. The tension is even greater on the abaxial digits of the hind feet, as the animal uses these to exert forward propulsive force for the next stride. It is likely that additional stresses are exerted during wet weather due to the reduction in the elastic modulus of both wall and sole which equates to a reduction in rigidity or stiffness of the hoof tissue. Although our results suggest there is little change in the compressive strength of wall hoof under wet external conditions, a reduction in elastic modulus may allow the wall hoof to bend away from the sole more readily and hence impose additional stresses.

The initiation of many hoof lesions is associated with a loss of integrity of the sole - wall junction. In order to fully understand what happens at this site, it would be desirable to conduct tests directly on hoof tissue from the white line. However far more refined techniques would be necessary to adequately study this physically complicated structure under realistic conditions. At this stage we can only assume that the physical hoof properties of tissue from the white line behave similarly to those of sole hoof. Under wet conditions, we would expect moisture content of white line hoof to rise and that this would be associated with a reduction in hardness, compressive strength, elastic modulus and resilience. In addition, under such conditions, it seems likely that extra stresses

would be placed on the junction because of the fall in elastic modulus of both sole and wall hoof as described above.

Lack of sole concavity may also contribute to the initiation of lesions. When cows calve in New Zealand, the sole of the foot is at its most concave and is able to progressively absorb force as the digits themselves spread apart and flatten to absorb the force. As the concavity of the sole rapidly disappears after calving (Tranter and Morris, 1992), much greater forces are exerted directly on the sole and the white line during each step.

Yet another probable contributing factor is the change during wet weather in the nature of the surface of the raceways that cows walk on. Continued movement of cattle along a race when surface water is present results in pugging of the surface layers and exposure of grit, bare stones and sharper rock material.

When the extra stresses resulting from changes in hoof shape after calving and changes in physical hoof properties during wet weather are compounded by changes in the nature of the surface that cows walk on, the risk of minor cracks and defects at the sites of greatest stress must rise substantially. Once the initial defect is present, the stage is set for lesion development as grit and dirt accumulate at the site and are slowly forced deeper. The continued disintegration of the sole - wall junction results eventually in white line disease. Minor defects that allow infection to enter and then become trapped are likely to be the initial cause of sole abscess development.

The reduction in resilience of the sole when soil moisture content was high as observed in this study also provides an explanation for the recognised association of hoof haemorrhage and lameness due to sole bruising with wet weather. It is logical to conclude that a lowering of horn resilience or the amount of energy the hoof is capable of absorbing would result in an increased likelihood of concussive damage to the soft tissues that underly the sole. Again the combined effects of exposure of sharper rock material for cows to walk on during wet weather and a lowering of the ability of the sole to protect the underlying sensitive tissues are likely to be two of the most important factors that contribute to sole bruising in pasture fed cattle.

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CHAPTER 8

A case-control study of lameness in dairy cows[6]

[6] Accepted for publication as: Tranter WP, Morris RS, Williamson NB. Preventive Veterinary Medicine, 1992.

Abstract

A case-control study was conducted to compare the physical hoof properties of digits responsible for clinical lameness with those of other digits on the same cows and with those of equivalent digits on non-lame control cows. The control cows were herd-mates matched by age, breed and stage of lactation. Hoof moisture, hoof hardness and sole concavity were measured on farm. The resilience, compressive strength and elastic modulus of both sole and wall hoof were measured on biopsy samples collected from both case and control digits.

White line disease, sole bruising and septic pododermatitis accounted for 92% of the clinical lameness lesions in the case digits examined. Less severe forms of white line disease and hoof haemorrhage were also observed frequently in the non-lame digits of both the case and control cows.

Variables were screened for association using univariate procedures (Student's paired t-test and Wilcoxon's matched pairs test). Conditional logistic regression analysis was finally used to identify which risk factors were associated with lameness.

When digits on the matched cows were used as controls, sole and heel moisture, sole hardness, sole concavity and wall colour were selected for inclusion in the model derived to explain differences between the physical properties of lame and control digits. Values for each of these properties were lower in the lame digits than in the controls. The control digits had a higher percentage of black coloration than the lame digits.

Sole hardness and sole concavity (both lower in the lame digits) were also selected for inclusion in a conditional logistic regression model derived when the adjoining digits on the same legs of the lame cows were used as controls.

None of the physical hoof properties measured were associated with lameness when attempts were made to fit a model using the equivalent digits on the opposite legs of the lame cows as controls.

Production of the lame cows was also compared with that of matched herd-mates. Total lactation yields of milk, milk fat and milk protein were lower for the lame cows than for the control cows ($p < 0.05$).

Introduction

In a recent case-control study on 62 dairy farms in Taranaki, New Zealand, Chesterton *et al.* (1989) examined differences between herds with a high incidence of lameness and those with a low incidence. The average maintenance of the farm track (less well maintained in case herds) and the degree of patience shown by the farmer in bringing the cows to milking (lower in case herds) were the most influential variables explaining variation between case and control herds. As well as these differences between herds, variation in lameness susceptibility is also known to exist between cows and between digits within cows.

Previous studies of the lesions causing lameness in New Zealand herds (Dewes, 1978,79; Tranter and Morris, 1991) support the belief that most problems arise from traumatic injury to the digits. Such injury may cause bruising, white line disintegration or septic pododermatitis. However, rather than the injury being a chance event where individual hooves are damaged at random throughout the year by a cow accidentally stepping on a prominent object, epidemiological studies have shown that lameness and hoof lesions occur with predictable and repeatable patterns and that certain animal factors predispose to lameness (Tranter and Morris, 1991; Tranter *et al.*, 1991). Most lameness, for example, occurs in the hind legs and 80% of hind leg lameness occurs in the lateral claws. Eighty percent of all lameness occurs in the first 4 months of lactation and lameness onset is associated with wet weather conditions. Even in herds that experience very minor lameness problems, lesions can be observed in non-lame cows. Some of these subclinical lesions appear to be less severe cases of the same types of lesions that produce clinical lameness. However, again they occur with specific distribution in the cow and at specific times of year and breeding season, with waves of each type of lesion passing through the herd at a particular time.

Methods have been developed to measure various physical properties of hoof (Tranter *et al.*, 1992a). The primary objective of this study was to determine whether variation in any of these properties is associated with lameness in individual cows or digits.

A second objective was to compare milk production performance of lame cows with matched herd-mates. In an earlier study of lameness in three herds differences in production between case and control cows which were considered to have been caused by lameness were observed (Tranter and Morris, 1991). Since in the current study production information was available

for lame cows from a greater number of herds, it was considered it would be useful to repeat such an investigation.

Materials and Methods

Eight dairy herds in the Manawatu district of New Zealand were selected for participation in a case-control study of lameness in March 1990. Inclusion of herds was based on a history of some lameness in previous years, willingness of managers to cooperate and the availability of adequate facilities for examination of lame cows. All herds comprised almost entirely Friesian cattle and crossbreds with predominantly Friesian genotype. Any animals of other breeds in the herds were excluded from the study. The average herd size was 210. Most cows in these herds which became lame due to a hoof lesion over a twelve month period were used as cases in the study. Control cows, matched on age and proximity of calving date (within one month) were selected from within the same herds. When comparisons were being made between cows (eg lactation production), lame cows were treated as cases. However, when digits were being compared, a case was defined as the lame digit on the lame cow and the control could be the equivalent digit on the control cow, the equivalent digit on the opposite leg of the lame cow or the adjoining digit on the same leg of the lame cow, according to the particular comparison being conducted.

All cows were examined by the same veterinarian within three days of the onset of lameness as identified by the herd managers. Each control cow was examined (by the same veterinarian) on the same day as the case animal. Both cows were observed while walking and the lame limb was identified.

The lame limb was examined and the lame digit(s) identified by using hoof testers. On the lame cow, both digits from the lame leg and both digits from the opposite leg were examined and measurements taken. On the control cow, only the digits of the leg equivalent to the lame leg of the lame cow were examined. Digits were classified according to their relationship with the lame digit:

Lame cow:

Class 1 : lame digit (e.g. left hind lateral)

Class 2 : equivalent digit on opposite leg (e.g. right hind lateral)

Class 3 : adjoining digit on lame leg (e.g. left hind medial)

Control cow:

Class 1 : digit equivalent to lame digit on lame cow (e.g. left hind lateral)

Where cows were lame in a second leg, both lame digits were considered as separate cases and digits were classified in each instance as above.

The following details were measured and recorded :

lesion type(s): both lesions considered to be causing lameness and other (subclinical) hoof lesions

lesion severity

sole and wall colour

sole concavity

hoof moisture (sole, heel and posterior wall)

hoof hardness (sole, heel and posterior wall)

hoof elastic modulus (sole and wall)

hoof compressive strength (sole and wall)

hoof resilience (sole and wall)

Lesions were classified as outlined by Tranter *et al.* (1991) and following the system described by Mills *et al.* (1986). Lesion severity was scored from 1 to 3 as defined in Table 8.1. In the final analysis, these lesion scores were converted to a weighted severity score using a doubling scale in a way similar to that used by Greenough and Vermunt (1990) (Table 8.1).

Table 8.1 Description of system used to score severity of hoof lesions

Observed Score	Weighted Severity	Description
1	1	Minor superficial lesion (< 3 mm deep) or faint diffuse haemorrhages; no lameness or pain reaction with hoof testers.
2	2	More extensive lesion (> 3 mm deep) but not extending to the sensitive corium or more severe patchy haemorrhage; no lameness or pain reaction with hoof testers.
3	4	Extensive lesion or bruising with dark red horn discoloration extending to the sensitive corium; obvious pain reaction to hoof testers over the lesion.

Sole and wall colour were recorded as percentage of black coloration but only 0%, 25%, 50%, 75%, and 100% figures were used. As the pigment in the hoof wall of Friesian cows is often deposited only in the superficial layers, this percentage referred only to the colour of the surface layer of hoof.

Sole concavity at 20 and 30 mm from the abaxial hoof margin was measured using a profile gauge, hoof moisture was measured using a Delmhorst Type BD-7 Moisture Meter, and hoof hardness using a Shore Type D Durometer as described elsewhere (Tranter *et al.*, 1992a).

Using the methods described by Tranter *et al.* (1992a), hoof samples were collected from the toe and wall from 2 digits on the lame cow (class 1 and 2) and from one digit on the control cow (class 1). From these samples, rectangular blocks of hoof were prepared and compression tests performed using an Instron Universal Testing machine. Force deformation curves were charted on a synchronously-driven recorder. From these curves, the elastic modulus, compressive strength and resilience of each sample were derived.

All available production records of both case and control cows were collected at the end of their lactations.

Statistical analysis

The data were analysed using the programs PANACEA 2 (PAN Livestock Services, Reading, England), SOLO (BMDP Statistical Software Inc., Los Angeles, California) and EGRET (Statistics and Epidemiology Research Corporation, Seattle, Washington). In a preliminary analysis, differences between case and control digits for each variable were tested for statistical significance. For this purpose, Student's paired t-test was used where the assumptions of the test could be met. For other variables that were not normally distributed (e.g. hoof colour and sole concavity), data were analysed using the Wilcoxon matched pairs test. Variables associated with lameness ($p < 0.1$) were offered in a multivariable analysis. Forward stepwise conditional logistic regression was used to identify the most important risk factors as those having p values for Wald's test of < 0.03. Factors having marginal significance ($p = 0.03 - 0.07$) were tested using the likelihood ratio chi square test.

Differences in lactation production between lame and non-lame cows were tested for significance using Student's paired t-test.

Results

Hoof lesions

94 lame cows were examined, 7 of which were simultaneously lame in more than one leg. Some cows and digits chosen as controls became lame later. White line disease, sole bruising and septic pododermatitis (sole abscess) accounted for most of the unweighted severity score-3 lesions that were recorded in the lame digits (Figure 8.1). The digit distribution of these 3 most common lesions is shown in Figure 8.2. Many lesions were observed also on the digits examined on the control cows, and on the non-lame digits of the lame cows. In most instances these were less severe forms of bruising and white line disease. Table 8.2 shows the frequency of lesions and the total weighted lesion severity scores for the lame digits and for the various control digits for the 101 cases examined.

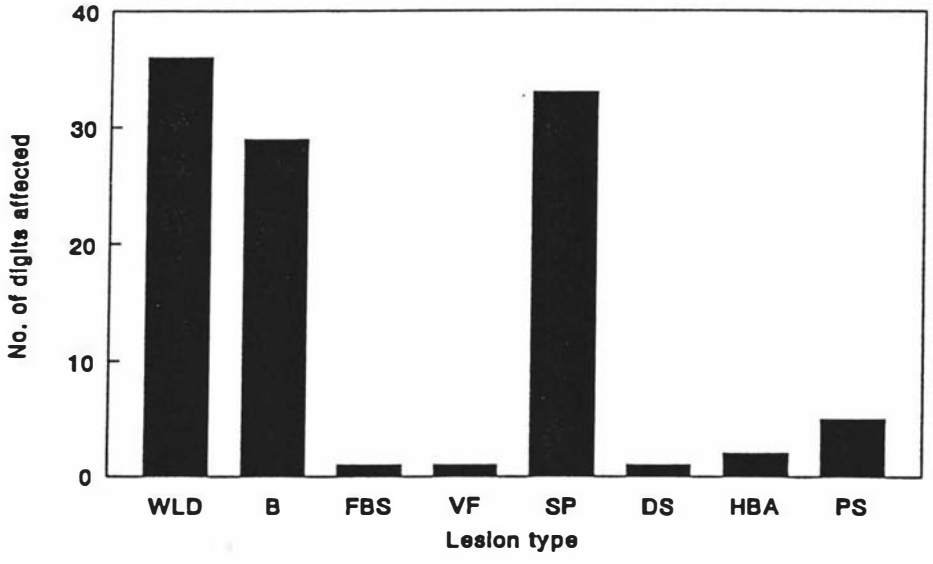

Figure 8.1. Severity score-3 lesions observed in lame digits of lame cows

WLD	=	White line disease
B	=	Bruising
FBS	=	Foreign body in sole
VF	=	Vertical Fissure
SP	=	Septic pododermatitis
DS	=	Deep sepsis
HBA	=	Heel bulb abscess
PS	=	Punctured sole

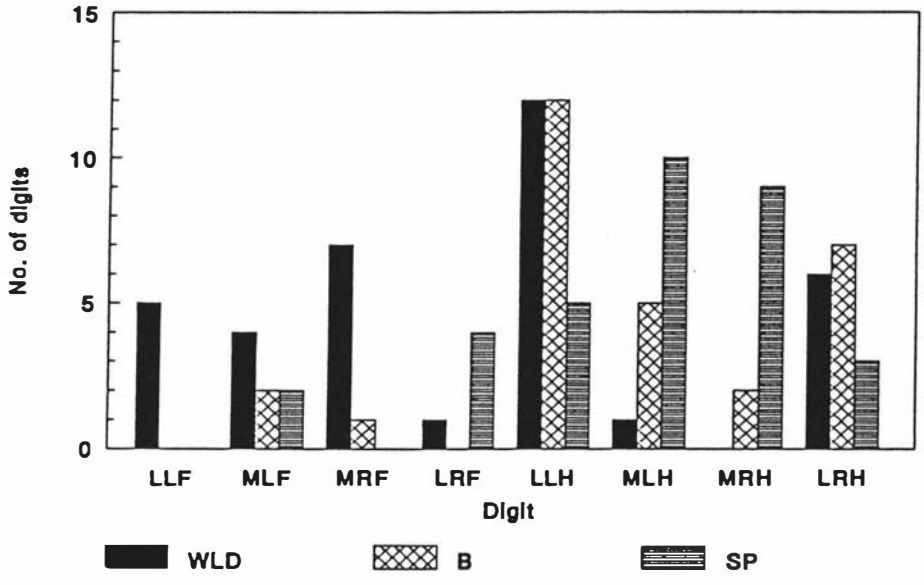

Figure 8.2. Digit distribution of severity score-3 lesions in the lame digits of the lame cows. WLD = White line disease; B = Bruising; SP = Septic pododermatitis.

Table 8.2. Lesion frequencies and totals of weighted severity scores on lame and control digits

	Lame Digit (Class 1)	Control Leg (Class 2)	Control Digit (Class 3)	Control Cow (Class 1)
WHITE LINE DISEASE:				
Severity 1	9	20	23	21
Severity 2	14	24	10	18
Severity 3	36	5	0	2
Total weighted score	**181**	**88**	**43**	**65**
BRUISING:				
Severity 1	17	18	16	29
Severity 2	12	20	10	11
Severity 3	29	14	6	1
Total weighted score	**157**	**114**	**60**	**55**
SEPTIC PODODERMATITIS:				
Severity 1	0	0	0	1
Severity 2	0	1	1	0
Severity 3	33	1	0	0
Total weighted score	**132**	**5**	**2**	**1**

Physical hoof characteristics

Lame cow vs control cow

Observations for affected digits on the lame cows were compared with those for the equivalent digits on the control cows. Wall colour, sole concavity, sole and heel moisture, sole hardness, and sole and wall resilience were lower in the lame digits than in the controls while wall elastic modulus was higher in the lame digits than in the controls ($p < 0.1$) (Table 8.3).

The 8 variables associated with lameness on screening were examined by forward stepwise conditional logistic regression and of these 5 were selected for inclusion in a final model (Table 8.4, in order of entry). Sole and wall resilience and wall elastic modulus were excluded.

Lame leg vs control leg on lame cow

The physical hoof characteristics from the affected digit on the lame cow were compared with those from the equivalent digit on the opposite leg of the lame cow. Wall colour and wall moisture were lower in the lame digits than in the controls while wall hardness and wall compressive strength were higher ($p < 0.1$) (Table 8.3). When these variables were offered for inclusion in a conditional logistic regression model none were selected.

Lame digit vs control digit

Comparisons made between the physical characteristics of the affected digit of the lame cows and the non-affected digit on the same leg are also shown in Table 3. The concavity of the soles of the affected digits was lower than that of the control digits as were sole, heel and wall hardness ($p < 0.1$). Sole hardness and sole concavity 20 mm were selected for inclusion in a final model (Table 8.3).

The differences in sole concavity between digits of the lame leg were compared with the differences between digits on the equivalent leg of the control cow. The differences were greater between digits on the lame cow's leg than between digits on the control cow's leg ($p < 0.1$).

Production differences between lame and non-lame cows

Production records were available for 90 of the lame cows. The differences in production between these cows and matched herd-mates of similar age and calving date appear in Table 8.5. Yields of the lame cows were significantly less than those for the controls ($p < 0.05$) to 2 weeks after their last herd test for the lactation. The average lactation length of lame cows was shorter, due mostly to premature culling of some cows because of their poorer production. Even when lactation-to-date production levels of the two groups were compared as at the last herd test prior to either one of each matched pair leaving the herd, differences were noted (Table 8.5). The average time from calving to the onset of lameness was 114 ± 64 days.

Table 8.3. **Comparison of the physical characteristics of the hoof of 101 lame digits on 94 cows with those of the equivalent digits on 94 control cows (control cow - class 1); with those of the equivalent digits on the opposite legs of the lame cows (lame cow - class 2); and with those of the adjoining digits on the same legs of the lame cows (lame cow - class 3).**

Risk Factor	Lame Cow Class 1 n = 101	Control Cow Class 1 n = 101	Lame Cow Class 2 n = 101	Lame Cow Class 3 n = 101
Sole Colour (% black)	18 ± 15	20 ± 16	18 ± 14	19 ± 16
Wall Colour (% black)	25 ± 18	29 ± 18*	27 ± 18*	26 ± 18
Sole concavity at 20 mm (mm)	0.6 ± 0.7	0.8 ± 0.8*	0.6 ± 0.6	1.0 ± 1.0*
Sole concavity at 30 mm (mm)	1.3 ± 1.3	1.5 ± 1.3	1.2 ± 1.1	1.9 ± 1.7*
Sole moisture %	30.7 ± 2.6	31.5 ± 2.3*	31.2 ± 2.5	30.6 ± 2.4
Heel moisture %	35.8 ± 4.9	38.6 ± 5.0*	36.7 ± 4.9	36.3 ± 4.9
Wall moisture %	27.0 ± 2.0	27.1 ± 1.8	27.4 ± 2.1*	26.7 ± 1.5
Sole Hardness (Shore units)	47.6 ± 6.3	49.2 ± 3.8*	46.9 ± 6.5	50.6 ± 5.0*
Heel Hardness (Shore units)	36.4 ± 6.0	35.8 ± 4.9	35.8 ± 5.9	37.8 ± 5.4*
Wall Hardness (Shore units)	70.0 ± 5.0	69.9 ± 4.4	69.4 ± 4.7*	71.1 ± 4.2*
Sole elastic modulus (MPa)	68 ± 35	62 ± 24	64 ± 32	
Sole compressive strength (MPa)	6.8 ± 2.0	6.6 ± 1.7	6.5 ± 1.9	
Sole resilience (kJ/m^3)	144 ± 62	160 ± 89*	138 ± 63	
Wall elastic modulus (MPa)	336 ± 106	305 ± 97*	321 ± 93	
Wall compressive strength (MPa)	22.3 ± 4.7	22.2 ± 4.2	21.7 ± 4.5*	
Wall resilience (kJ/m^3)	405 ± 180	470 ± 213*	412 ± 189	

* Values are significantly different from those of the lame digits (class 1) on the lame cows ($p < 0.1$)

Table 8.4. **Conditional logistic regression models derived to explain differences between 101 lame digits on 94 lame cows and equivalent digits on 94 control cows (model 1); between 101 lame digits and equivalent digits on the opposite legs of the lame cows (model 2); and between 101 lame digits and adjoining digits on the same legs of the lame cows (model 3).**

Risk Factor	Beta	SE (beta)	p value	Odds Ratio
MODEL 1[a]:				
Heel Moisture	-0.18	0.05	< 0.001	0.84
Sole hardness	-0.19	0.06	0.002	0.83
Sole Moisture	-0.23	0.11	0.04	0.79
Sole concavity (20 mm)	-0.74	0.31	0.03	0.47
Wall colour	-0.23	0.12	0.06	0.79
MODEL 2:				
No variables selected				
MODEL 3[b]:				
Sole Hardness	-0.20	0.04	< 0.001	0.82
Sole Concavity (20 mm)	-0.54	0.20	0.006	0.58

[a] Likelihood chi-square = 43.13, df = 5, $p < 0.001$
[b] Likelihood chi-square = 32.57, df = 2, $p < 0.001$

Table 8.5. **Production in 90 lame cows and 90 matched herd-mates[a]**

	Total lactation yields		Lactation-to-date yields (for similar days in milk)	
	Lame cows	Controls	Lame cows	Controls
Milk (l)	3729 ± 1034*	3986 ± 971	3750 ± 1030*	3952 ± 939
Fat (kg)	166 ± 43**	181 ± 39	167 ± 43*	176 ± 41
Protein (kg)	126 ± 35**	139 ± 29	127 ± 34**	135 ± 31
Days in milk	242 ± 41**	252 ± 31	242 ± 41	243 ± 40

[a] Values are quoted as mean ± standard deviation
Student's paired t-test * $p < 0.05$, ** $p < 0.01$

Discussion

Variation in the physical properties of hoof with different environmental moisture conditions has been observed in an earlier study (Tranter *et al.*, 1992c) providing some possible explanations for the well recognised association between high incidence of lameness and wet weather. A case-control study design was chosen to determine which, if any, of these various physical hoof properties, for which measurement methods were available, might be associated with an individual cow's susceptibility to lameness. The advantages of using a case-control study were its low cost and the relative ease of getting an adequate number of cases to study in a relatively short time period. Hoof properties associated with lameness in this study could then be examined further using other study techniques. Since cows were examined as soon as possible after lameness onset, measurements should reflect the state of the hooves at the time cows became clinically lame. However it is recognised that, for some properties, differences detected between case and control digits may be either causes or consequences of lameness. Ideally, measurements would be taken before cows became lame but such a cohort approach would create impossible examination workloads.

The pattern of lameness seen in cows in this study (Figure 8.1) is similar to that previously reported (Tranter and Morris, 1991), with white line disease, sole bruising and sole abscess being the 3 most common lesion types recorded. Digit distribution was also reasonably similar with white line disease occurring most frequently in the medial front and lateral hind digits, bruising most frequently in the lateral hind digits and septic pododermatitis most frequently in the medial hind digits (Figure 8.2).

The finding that many of the non-lame digits examined on both the lame and non-lame cows also had subclinical white line lesions and sole haemorrhages was expected since this had been noted in earlier work (Tranter *et al.*, 1991). Use of a weighted lesion-severity scoring system as described by Greenough and Vermunt (1990) was useful in that it gave recognition to the greater clinical importance of the more severe lesions. The notable finding when the total weighted lesion severity scores for the different classes of control digits were studied was that the control digits of the opposite legs on the lame cows had the highest scores for these two lesions. Fewer and less severe lesions were observed on the control digits of the control cows than there were on the control digits of the opposite legs of the lame cows. This would suggest that variation in susceptibility to these 2 lesions is greater between cows than it is between like-positioned digits within cows.

In contrast, few of the control digits of either case or control cows examined showed any evidence of septic pododermatitis with underrunning of the sole. This lesion is presumed to be due to entry of infection through minor defects in the hoof (probably along the white line). These lesions are usually small and unimportant and pass unnoticed on routine examination. Occasionally, infection reaches the sensitive corium, becomes trapped, and an inflammatory process follows with buildup of pressure that results in severe pain and lameness. This then appears to be an acute process (without subclinical manifestation) that develops over a 1 to 2 week period.

Lame digits had softer soles than control digits on the control cows and than adjoining digits on the same legs of the lame cows. However, this difference is in part an effect of the lameness lesions. Bruised soles where blood is both incorporated in the horn material and present underneath the horny sole are clinically softer than healthy ones. Lower hardness readings also were observed in many cases of septic pododermatitis where the sole was underrun with pus and tissue fluids. However, hoof hardness may still be important in determining susceptibility to traumatic hoof injury. It is possible that all digits (both cases and controls) were softer due to wet conditions at the time lameness occurred. This is supported by comparison of the results of this study with earlier work (Tranter *et al.*, 1992c). Hoof hardness of control digits with low lesion severity scores (lame cows - class 3 and control cows - class 1) was similar to that observed in healthy hooves that had been subjected to very wet conditions, but lower than that observed in the same hooves during drier weather. Variation in hoof hardness, therefore, appears to depend more on environmental moisture than on intrinsic differences between digits in the same or different cows.

Some useful insights were gained in this study in relation to the importance of sole concavity. Lateral hind claws are less concave than medial hind claws (Tranter and Morris, 1992). This could partly explain why more lameness is observed in lateral hind claws than medial ones as worn flat soles may be more susceptible to bruising and traumatic damage. In this study, the lame digits had less concavity than did the adjoining digits on the same legs and than the equivalent digits on the control cows. Sole concavity, as measured at 20 mm from the abaxial hoof margin, was selected for inclusion in both conditional logistic regression models that were derived. Furthermore, the differences in concavity between digits on the lame leg of the lame cow were greater than the differences in concavity between digits on the control leg of the control cow. This can be explained by the lack of concavity in the lame digits and hence the greater difference between lame and control digits of the lame legs. These findings

suggest then that lack of sole concavity is probably causally associated with occurrence of lameness, and is not merely an incidental finding caused by both lameness and lack of concavity being more common in lateral claws.

Since most lameness lesions occur on the bearing surface of the hooves, the physical properties of the sole would be expected in principle to be causally more important than those of the heel and wall. However, for those properties measured by compression tests (elastic modulus, compressive strength and resilience) on sole samples, there were few differences between case and control digits. As was the case for hoof hardness, values measured (for both cases and controls) were similar or lower than those observed in hooves during wet weather in a previous study (Tranter *et al.*, 1992c). Perhaps the low values observed for these properties in this study in both case and control digits can be explained by the wet environmental conditions that usually coincided with lameness onset. Like hardness, the three variables, elastic modulus, compressive strength and resilience, appear to be more influenced by environmental moisture than by cow or digit factors.

In analyses where the lame digits were compared with control digits on different cows, sole and heel moisture were negatively associated with lameness. This contrasts with what was expected. In previous work sole moisture content was negatively correlated with sole hardness, elastic modulus, compressive strength and resilience (Tranter *et al.*, 1992c). Lower sole moisture could therefore be expected to be associated with harder, stronger and more resilient soles which should protect against rather than predispose to lameness. Consequently, to find lower sole and heel moisture in lame cows than in control cows was unexpected and there is no immediately apparent biological explanation.

Inclusion of wall colour in the conditional logistic regression models derived to explain differences between lame digits and equivalent digits on control cows supports the belief held by many that black hooves are less susceptible to lameness than white hooves. It also supports the findings of Chesterton *et al.* (1989) who reported that in low lameness incidence herds it was more likely that the claws of cows were more pigmented.

To further evaluate the importance of these physical hoof properties as lameness risk factors a cohort study comparing hooves with different properties is required. Treatment methods that influence the physical nature of hooves will need to be used to make this possible.

The differences in production between lame and control cows are very similar to those observed previously (Tranter and Morris, 1991). However, the results of this study have greater validity for two reasons. Firstly in this study controls were chosen at the time of lameness onset and prospective comparisons were made whereas previously comparisons were made retrospectively after controls were selected at the completion of their lactations. Secondly, lame cows in this study were selected from 8 herds as opposed to 3 herds in the previous one.

It is not possible to be certain whether the lower production is a cause or an effect of lameness but it is more likely to be the latter. If level of production was causally associated with lameness, then it is probable that high production, not low production, would predispose to a case. This would reduce the likelihood of observing lower production in lame cows. The findings of both studies similarly suggest that considerable production losses are associated with lameness in pasture-based grazing systems.

Acknowledgements

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CHAPTER 9

Chemical treatment of hooves of cattle collected after slaughter[7]

[7] Submitted as: Tranter WP, Morris RS. New Zealand Veterinary Journal, 1992.

Abstract

Hooves harvested from cattle after slaughter were subjected either to soaking in water for 24 hours or exposure to the drying effects of the atmosphere for a similar period. Changes in hoof hardness and moisture content were monitored. The ability of various available topical hoof treatments to influence these changes in both situations was evaluated. Moisture percentage fell and hoof hardness increased over 24 hours in both treated and untreated hooves that were exposed to atmospheric drying. The effects of treatment were minimal. In hooves soaked in water for 24 hours, sole moisture changed little, heel moisture decreased while wall moisture increased. Hoof hardness at all sites was reduced. Changes in moisture and hardness in hooves subjected to treatment prior to soaking were in most cases similar to those for hooves soaked without treatment.

Introduction

Regular footbathing has been widely recommended for the prevention of lameness in dairy cattle. A variety of chemical agents have been promoted on the basis of their known antibacterial action or on their perceived ability to harden and strengthen claw horn. There is reasonable evidence that regular footbathing with solutions such as formalin or copper sulphate can effectively control or prevent interdigital lesions that are mediated by bacterial invasion. However, there is little documented information available on the ability of such treatments to alter the physical properties of hoof tissue in a way that would modify its susceptibility to traumatic injury.

The association between lameness incidence and wet weather conditions is well documented and is frequently considered to be due, in part, to a reduction in the hardness and strength of hoof tissue when it is subjected to a continuously wet environment (Eddy and Scott, 1980; Arkins, 1981; Rowlands *et al.*, 1983; Williams *et al.*, 1986; Jubb and Malmo, 1991). Changes in hoof moisture and other physical hoof properties have been observed in earlier studies in which cows' feet were exposed to varying soil moisture conditions (Tranter *et al.*, 1992c). More information on the way in which hoof properties respond to environmental influences is required in an effort to develop further lameness control strategies.

In these experiments hooves harvested from dead cattle were subjected to either soaking in water for 24 hours or exposure to the drying effects of the atmosphere for a similar period. Changes in hoof hardness and moisture content were monitored. The ability of various available topical hoof treatments to influence these changes in both situations was evaluated.

Methods

Study 1

Hooves were collected in batches from 21 cows after slaughter. Each batch consisted of 24 hooves (3 cows). One or two batches were processed in any one 24 hour period as follows.

Within two hours of collection, the hooves were washed and dried with paper towelling. At three sites on each hoof, as shown in Figure 9.1 (sole, heel and abaxial wall), the hoof surface was lightly sanded to prepare a flat surface. At each site, hoof moisture was measured using a Delmhorst moisture meter and hoof hardness was measured using a Shore Type D Durometer as has been described elsewhere (Tranter *et al.*, 1992a).

Hooves were then subjected to one of the following treatments:

No treatment (NT)
Water (W)
10% copper sulphate (CS)
10% zinc sulphate (ZS)
10% formalin (F)
10% glutaraldehyde (G)

Treatment was applied by soaking the hooves in the appropriate solution for 5 minutes. Following treatment, they were dried and then subjected to hardness and moisture measurement at the same three sites (sole, heel and abaxial wall) at 30 minutes, 90 minutes and 24 hours after removal from the treatment solution.

Seven batches of hooves were treated. This resulted in 168 hooves being treated in total, 28 in each treatment group).

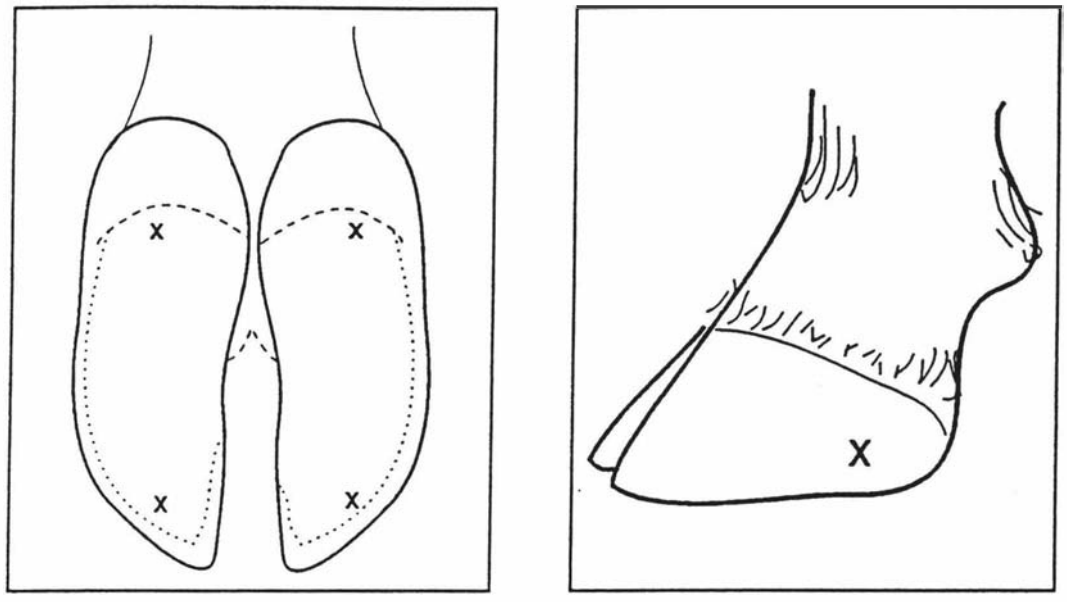

Figure 9.1. Sites of hardness and moisture measurements

Study 2

Hooves were collected from 25 cows after slaughter, again in batches, but this time each batch consisted of 28 hooves (3½ cows). One or two batches were processed in any one 24 hour period.

The hooves were prepared for chemical treatment as described for study 1. Hardness and moisture were again measured at the three sites (sole, heel and wall) prior to treatment. Hooves were subjected to one of the following treatments:

No treatment (NT)

Water for 5 minutes (W)

10% copper sulphate for 5 minutes (CS)

10% zinc sulphate for 5 minutes (ZS)

10% formalin for 5 minutes (F)

10% glutaraldeyhde for 5 minutes (G)

Acrylic acid and methylacrylic ester (Hoof Bond[8]) (HB) applied by sponge. For this treatment hooves were then exposed to UV light using a hand held lamp for five minutes.

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[8] Hoof Bond, Inc., 131 Old Route 132, Hyannis, MA 02601, USA

Following treatment the hooves were then washed under running water and soaked in water. At 1 hour, 6 hours, and 24 hours after treatment the hooves were removed from the water, dried with paper towelling and then left to air dry further for 30 minutes. Hardness and moisture were then measured as in study 1 and the hooves were returned for further soaking in water.

Seven batches (196 hooves) were treated in total which resulted in 28 hooves in each treatment group.

Statistical analysis

Data were recorded and analysed using the statistical package SOLO (BMDP Statistical Software Inc., Los Angeles, California). Differences in the changes in hoof hardness and moisture over time on the same experimental units (ie. hooves) were evaluated using repeated measures analysis of variance. On all tables, p values are quoted separately for differences between treatments (treatment), for differences between times of measurement (time), and for differences between treatments over time (treatment x time).

Results

Study 1

Moisture percentage fell and hoof hardness increased over the 24 hour study period at all three sites of measurement for hooves in all treatment groups. These differences in moisture and hardness between times of measurement were highly significant (Time: $p < 0.001$) (Tables 9.1 and 9.2).

Hooves receiving treatment behaved slightly differently to the untreated controls. Sole and heel moisture content in the treated hooves (all groups) were marginally higher 30 minutes after treatment than at the pretreatment reading, whereas moisture content had already begun to fall after 30 minutes in the untreated hooves (Table 9.1). In addition, hoof hardness in the treated hooves changed little between the pretreatment and 30 minutes post treatment measurements whereas the controls were already slightly harder by this time (Table 9.2). In all other respects hooves of all treatment groups behaved

similarly. When tested by repeated measures analysis of variance no significant differences were detected between treatment groups, including the untreated controls, for changes in both sole and wall moisture and for changes in hardness at all sites over time. Differences between treatments were significant for changes in heel moisture over time but hooves of all treatment groups showed the same pattern of increased moisture 30 minutes after treatment (untreated controls excepted) with a substantial reduction in hoof moisture percentage over the remainder of the 24 hour study period. Differences were largely due to the fall in moisture at 30 minutes in the untreated controls and a slightly greater increase in moisture percentage at 30 minutes in the copper sulphate treated hooves than was recorded in hooves receiving one of the other treatments.

Since there were few differences between changes in hoof moisture and hardness between treatments over time, the results for all treatments have been pooled and these are presented as bar graphs in Figures 9.2 and 9.3.

Table 9.1. Hoof moisture percentage for hooves in six different treatment groups at four different times of measurement during a 24 hour study period

Sole Moisture:

	Pre-treatment	30 minutes	90 minutes	24 hours
NT	30.6 ± 3.2	29.8 ± 2.9	28.8 ± 2.5	25.6 ± 1.6
W	30.1 ± 3.2	30.6 ± 3.7	28.9 ± 2.3	25.3 ± 1.2
CS	30.7 ± 3.1	31.6 ± 3.6	30.1 ± 3.0	25.9 ± 1.0
F	30.8 ± 3.0	31.1 ± 3.0	29.5 ± 2.0	25.7 ± 1.0
G	30.6 ± 3.0	30.8 ± 3.4	29.4 ± 2.3	25.5 ± 0.9
ZS	31.3 ± 4.2	31.6 ± 5.6	30.0 ± 4.0	25.6 ± 1.3

Treatment: $p = 0.63$; Time : $p < 0.001$; Treatment x time : $p = 0.43$.

Heel Moisture:

	Pre-treatment	30 minutes	90 minutes	24 hours
NT	33.9 ± 4.0	33.0 ± 4.5	30.9 ± 4.0	26.5 ± 2.4
W	33.3 ± 4.1	34.1 ± 5.1	31.0 ± 3.4	26.3 ± 1.6
CS	33.9 ± 4.9	35.8 ± 4.8	32.4 ± 3.6	26.5 ± 1.6
F	33.8 ± 3.4	33.3 ± 3.5	31.2 ± 2.4	26.5 ± 1.2
G	33.2 ± 3.6	33.4 ± 3.9	31.5 ± 2.9	26.2 ± 1.8
ZS	33.5 ± 4.2	34.4 ± 4.3	32.1 ± 4.1	26.4 ± 1.9

Treatment: $p = 0.76$; Time : $p < 0.001$; Treatment x time : $p < 0.001$.

Wall Moisture:

	Pre-treatment	30 minutes	90 minutes	24 hours
NT	25.2 ± 1.4	25.0 ± 1.3	24.8 ± 1.2	24.1 ± 1.2
W	25.2 ± 1.2	25.2 ± 1.2	24.8 ± 1.2	24.1 ± 0.9
CS	25.0 ± 0.9	24.9 ± 1.1	24.8 ± 1.2	24.1 ± 1.2
F	25.4 ± 7.3	25.1 ± 1.2	24.9 ± 1.4	24.3 ± 1.0
G	25.2 ± 1.1	25.2 ± 1.5	25.0 ± 1.4	24.3 ± 1.0
ZS	25.0 ± 1.2	25.1 ± 1.3	24.7 ± 1.4	24.1 ± 1.1

Treatment: $p = 0.96$; Time : $p < 0.001$; Treatment x time : $p = 0.96$.

Table 9.2. Hoof hardness (in Shore hardness units) for hooves in six different treatment groups at four different times of measurement during the 24 hour study period

Sole Hardness:

	Pre-treatment	30 minutes	90 minutes	24 hours
NT	52.6 ± 5.2	54.6 ± 5.6	55.4 ± 5.3	66.5 ± 7.0
W	52.4 ± 4.3	52.3 ± 5.0	54.5 ± 4.8	67.2 ± 4.7
CS	54.3 ± 5.9	54.2 ± 4.7	55.5 ± 4.5	66.5 ± 5.5
F	53.6 ± 4.9	53.9 ± 4.2	56.0 ± 4.8	66.3 ± 3.7
G	54.5 ± 4.1	54.7 ± 3.6	57.2 ± 5.1	67.5 ± 4.2
ZS	55.0 ± 5.7	55.0 ± 4.8	56.8 ± 6.5	67.3 ± 4.1

Treatment: $p = 0.56$; Time : $p < 0.001$; Treatment x time : $p = 0.13$.

Heel Hardness:

	Pre-treatment	30 minutes	90 minutes	24 hours
NT	41.1 ± 5.4	43.2 ± 6.3	44.9 ± 5.6	58.3 ± 8.5
W	41.7 ± 5.4	42.2 ± 5.9	44.0 ± 6.9	57.7 ± 8.2
CS	42.1 ± 5.2	43.4 ± 5.7	45.5 ± 5.8	58.8 ± 7.7
F	42.3 ± 5.3	41.9 ± 4.0	44.8 ± 6.0	57.6 ± 6.6
G	42.9 ± 6.1	42.9 ± 5.7	45.6 ± 5.9	58.2 ± 7.0
ZS	42.2 ± 6.3	43.0 ± 4.9	47 ± 5.9	59.5 ± 6.6

Treatment: $p = 0.92$; Time : $p < 0.001$; Treatment x time : $p = 0.44$.

Wall Hardness:

	Pre-treatment	30 minutes	90 minutes	24 hours
NT	75.7 ± 3.6	76.5 ± 3.5	76.9 ± 4.1	78.8 ± 2.6
W	75.3 ± 3.9	75.6 ± 2.9	76.5 ± 3.3	78.8 ± 3.1
CS	75.2 ± 3.9	75.7 ± 3.5	76.8 ± 2.9	78.1 ± 2.7
F	75.7 ± 3.3	75.4 ± 3.5	76.3 ± 3.7	77.4 ± 3.1
G	75.1 ± 4.1	73.8 ± 4.0	75.8 ± 3.9	77.6 ± 3.0
ZS	74.6 ± 4.4	75.3 ± 3.6	76.7 ± 4.1	78.1 ± 3.3

Treatment: $p = 0.61$; Time : $p < 0.001$; Treatment x time : $p = 0.29$.

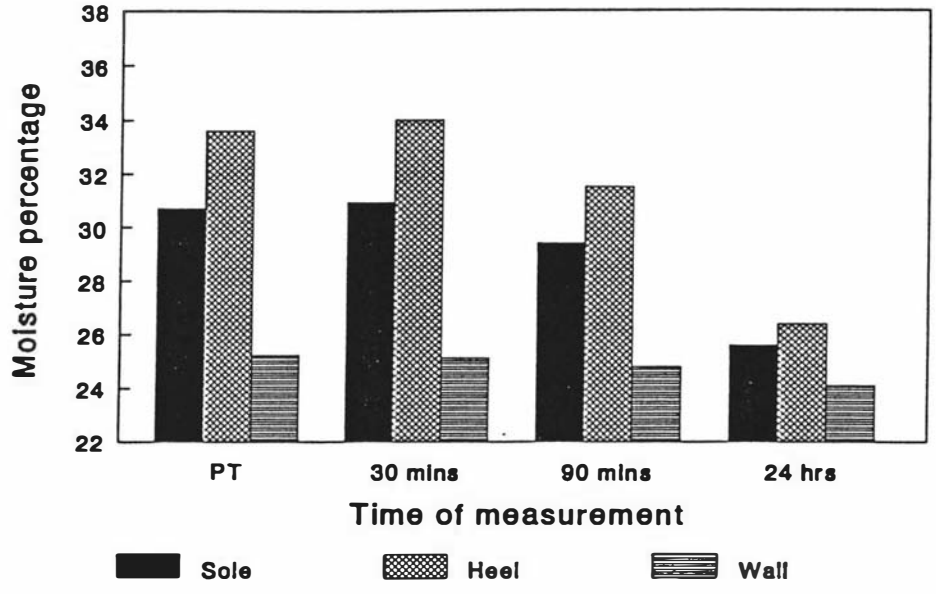

Figure 9.2. Changes in hoof moisture percentage when hooves were subjected to chemical treatment and left exposed to atmospheric drying over a 24 hour period. Results of measurements for hooves of all treatment groups have been pooled.

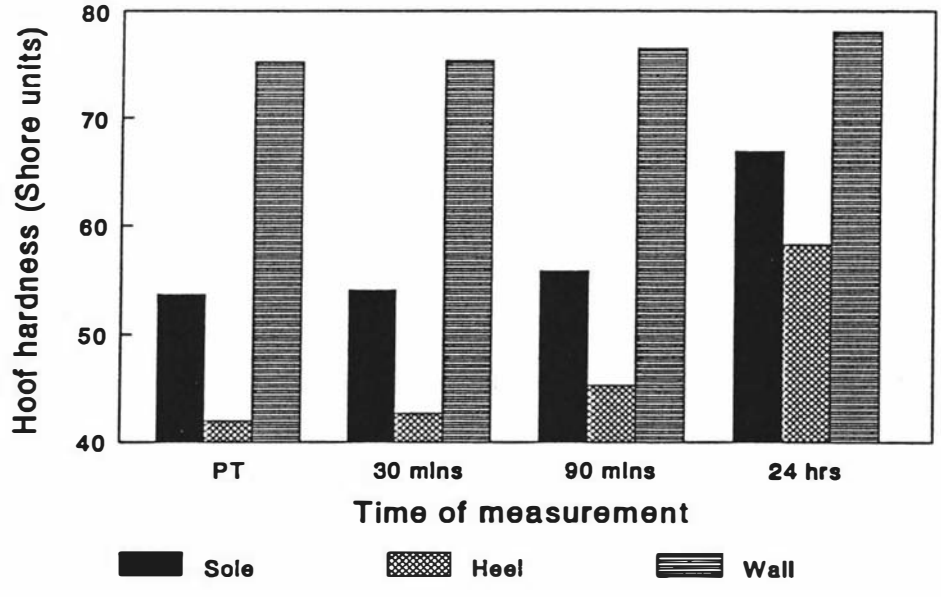

Figure 9.3. Changes in hoof hardness when hooves were subjected to chemical treatment and left exposed to atmospheric drying over a 24 hour period. Results of measurements for hooves of all treatment groups have been pooled.

Study 2

In this study hooves receiving no treatment behaved similarly to that described for study 1. Hoof moisture fell and hoof hardness increased over the 24 hour period. For hooves receiving any of the other treatments, changes in hoof moisture were affected by site of measurement with sole moisture remaining remarkably constant (Table 9.3), heel moisture falling slightly (Table 9.4) and wall moisture increasing (Table 9.5). Hoof hardness at all sites in the treated hooves decreased over time (Time : $p < 0.001$) (Tables 9.3, 9.4, and 9.5).

With the exception of heel moisture for Hoof Bond-treated hooves, there was little difference in the changes observed over time between treatment groups for both moisture and hardness. Hooves receiving any of the chemical treatments behaved similarly to those receiving water soaking only. As can be seen in Tables 9.3, 9.4, and 9.5, in most instances there was little significant difference (Treatment x time: $p > 0.05$) between changes in moisture and hardness over time when results from hooves that were not subjected to any treatment (NT) were excluded from the analyses.

In the Hoof Bond treatment group, heel moisture fell more quickly and to lower levels than did hooves receiving any of the other chemical treatments following the pattern observed in untreated hooves left exposed to the atmosphere (Table 9.4 and Figure 9.6). When data from both the untreated hooves and those treated with Hoof Bond were excluded from the analysis there were no significant differences between treatments (Treatment x time: $p = 0.4$). The only other significant difference detected between treatment groups over time using repeated measures analysis of variance was with heel hardness. Glutaraldehyde treated hooves experienced a greater fall in hoof hardness than did hooves receiving any of the other treatments (Table 9.4).

In Figures 9.4 to 9.9 which have been included to graphically portray the changes that occurred in hoof moisture and hardness over time results from treatment groups that were not significantly different have been pooled.

Table 9.3. Sole moisture percentage and hardness (in Shore hardness units) for hooves in seven different treatment groups at four different times of measurement during a 24 hour study period

Sole Moisture:

	Pre-treatment	1 hour	6 hours	24 hours
NT	32.4 ± 2.6	31.3 ± 2.4	31.1 ± 2.4	30.1 ± 2.5
W	33.3 ± 2.6	33.9 ± 2.9	34.5 ± 3.3	33.8 ± 2.8
CS	32.9 ± 2.7	32.5 ± 2.5	32.6 ± 2.6	32.4 ± 2.5
F	33.0 ± 2.9	32.4 ± 2.2	33.2 ± 2.9	32.7 ± 2.6
G	31.9 ± 2.5	32.2 ± 3.0	32.5 ± 2.7	31.6 ± 1.9
ZS	32.1 ± 3.5	32.6 ± 3.7	32.5 ± 2.9	31.8 ± 1.8
HB	32.0 ± 2.9	31.5 ± 2.7	31.7 ± 2.4	32.0 ± 3.0

Treatment : $p = 0.003$; Time : $p = 0.003$; Treatment x time : $p < 0.001$.

With NT excluded: Treatment: $p = 0.03$; Time : $p = 0.052$; Treatment x time : $p = 0.14$

Sole Hardness:

	Pre-treatment	1 hour	6 hours	24 hours
NT	52.9 ± 4.5	54.0 ± 4.8	54.2 ± 4.7	56.5 ± 4.6
W	50.4 ± 5.6	49.2 ± 5.7	47.2 ± 6.0	46.3 ± 5.8
CS	53.9 ± 2.5	52.4 ± 2.1	51.1 ± 3.1	50.6 ± 3.6
F	52.4 ± 3.3	51.3 ± 3.1	49.2 ± 3.0	49.0 ± 4.1
G	52.9 ± 3.5	52.4 ± 3.4	49.6 ± 3.3	48.6 ± 3.2
ZS	52.9 ± 2.8	52.1 ± 3.1	50.9 ± 2.8	49.4 ± 2.9
HB	52.3 ± 4.4	51.4 ± 4.2	49.6 ± 4.0	48.3 ± 4.8

Treatment : $p < 0.001$; Time : $p < 0.001$; Treatment x time : $p < 0.001$.

With NT excluded: Treatment : $p = 0.005$; Time : $p < 0.001$; Treatment x time: $p = 0.22$.

Table 9.4. Heel moisture percentage and hardness (in Shore hardness units) for hooves in seven different treatment groups at four different times of measurement during a 24 hour study period

Heel Moisture:

	Pre-treatment	1 hour	6 hours	24 hours
NT	36.6 ± 5.0	33.4 ± 3.9	33.8 ± 4.6	33.4 ± 4.3
W	37.9 ± 5.9	38.0 ± 5.5	38.0 ± 5.8	37.1 ± 4.8
CS	38.4 ± 5.8	37.7 ± 5.7	37.4 ± 5.2	36.2 ± 5.4
F	38.6 ± 6.4	37.3 ± 4.3	37.0 ± 4.4	36.0 ± 4.1
G	37.2 ± 6.7	37.4 ± 6.2	36.9 ± 5.2	35.8 ± 5.2
ZS	37.4 ± 6.0	36.6 ± 5.7	36.3 ± 4.6	35.9 ± 4.6
HB	36.4 ± 4.5	34.3 ± 3.2	33.8 ± 3.2	33.3 ± 2.6

Treatment : $p = 0.02$; Time : $p < 0.001$; Treatment x time : $p < 0.001$.

With NT excluded: Treatment : $p = 0.14$; Time : $p < 0.001$; Treatment x time : $p = 0.02$.

With NT and HB excluded: Treatment : $p = 0.91$; Time: $p < 0.001$; Treat x time : $p = 0.4$.

Heel Hardness:

	Pre-treatment	1 hour	6 hours	24 hours
NT	40.0 ± 4.6	41.1 ± 4.7	41.9 ± 5.6	42.2 ± 5.7
W	39.6 ± 3.6	38.6 ± 3.5	37.3 ± 2.5	36.3 ± 2.9
CS	38.2 ± 4.7	38.1 ± 4.3	36.8 ± 4.1	36.1 ± 4.9
F	38.4 ± 5.3	38.3 ± 5.6	38.1 ± 5.2	35.3 ± 5.0
G	40.2 ± 6.3	39.3 ± 6.4	37.5 ± 5.4	35.9 ± 5.2
ZS	39.2 ± 6.5	39.8 ± 6.0	38.4 ± 6.2	36.9 ± 5.6
HB	40.5 ± 5.7	40.4 ± 5.8	39.1 ± 5.6	37.4 ± 5.3

Treatment : $p = 0.03$; Time : $p < 0.001$; Treatment x time : $p < 0.001$.

With NT excluded: Treatment : $p = 0.55$; Time : $p < 0.001$; Treatment x time : $p = 0.01$.

With NT and G excluded: Treatment x time : $p = 0.12$

Table 9.5. Hoof wall moisture percentage and hardness (in Shore hardness units) for hooves in seven different treatment groups at four different times of measurement during a 24 hour study period

Wall Moisture:

	Pre-treatment	1 hour	6 hours	24 hours
NT	25.3 ± 1.0	24.8 ± 1.2	24.4 ± 1.1	24.3 ± 1.1
W	25.5 ± 1.3	26.1 ± 2.2	25.9 ± 1.4	26.6 ± 1.3
CS	25.4 ± 1.2	25.4 ± 1.4	25.7 ± 1.4	26.5 ± 1.4
F	25.8 ± 2.4	25.5 ± 1.2	25.7 ± 1.0	26.4 ± 1.0
G	25.4 ± 1.2	25.4 ± 1.2	25.8 ± 1.2	26.4 ± 1.3
ZS	25.5 ± 1.1	25.5 ± 1.3	25.8 ± 1.4	26.4 ± 1.4
HB	25.4 ± 1.2	24.9 ± 1.1	25.4 ± 1.2	26.0 ± 1.3

Treatment : $p < 0.001$; Time : $p < 0.001$; Treatment x time : $p < 0.001$.

With NT excluded: Treatment: $p = 0.5$; Time : $p < 0.001$; Treatment x time : $p = 0.57$.

Wall Hardness:

	Pre-treatment	1 hour	6 hours	24 hours
NT	75.8 ± 2.3	77.0 ± 2.2	78.0 ± 1.9	79.2 ± 1.9
W	74.0 ± 3.7	72.6 ± 3.5	71.1 ± 3.3	69.7 ± 4.1
CS	74.0 ± 2.8	73.0 ± 3.0	71.6 ± 3.9	69.7 ± 3.8
F	74.4 ± 3.1	73.5 ± 2.9	71.8 ± 3.8	70.0 ± 3.1
G	74.3 ± 3.3	73.3 ± 3.2	71.1 ± 4.1	69.0 ± 3.8
ZS	74.3 ± 3.7	73.4 ± 3.5	72.2 ± 3.9	70.4 ± 4.4
HB	75.0 ± 3.5	73.6 ± 3.2	72.1 ± 3.2	70.6 ± 3.3

Treatment : $p < 0.001$; Time : $p < 0.001$; Treatment x time : $p < 0.001$

With NT excluded: Treatment : $p = 0.86$; Time : $p < 0.001$; Treatment x time : $p = 0.49$.

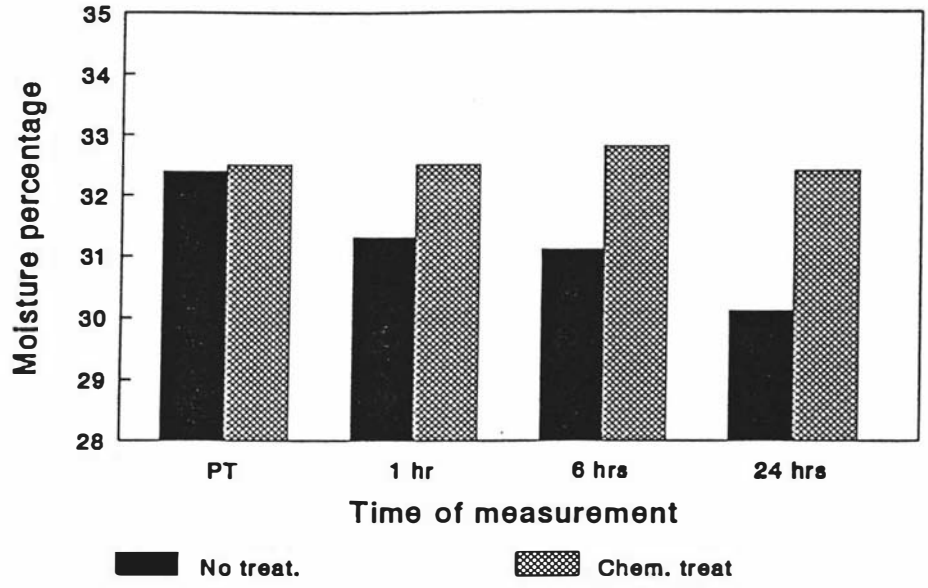

Figure 9.4. Changes in sole moisture over a 24 hour period for hooves receiving either no treatment or chemical treatment plus water soaking

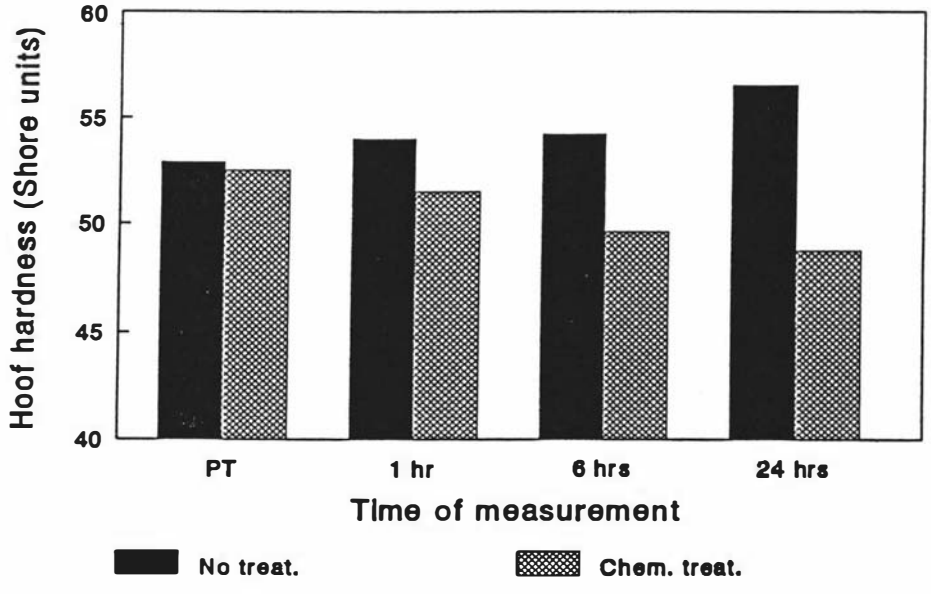

Figure 9.5. Changes in sole hardness over a 24 hour period for hooves receiving either no treatment or chemical treatment plus water soaking

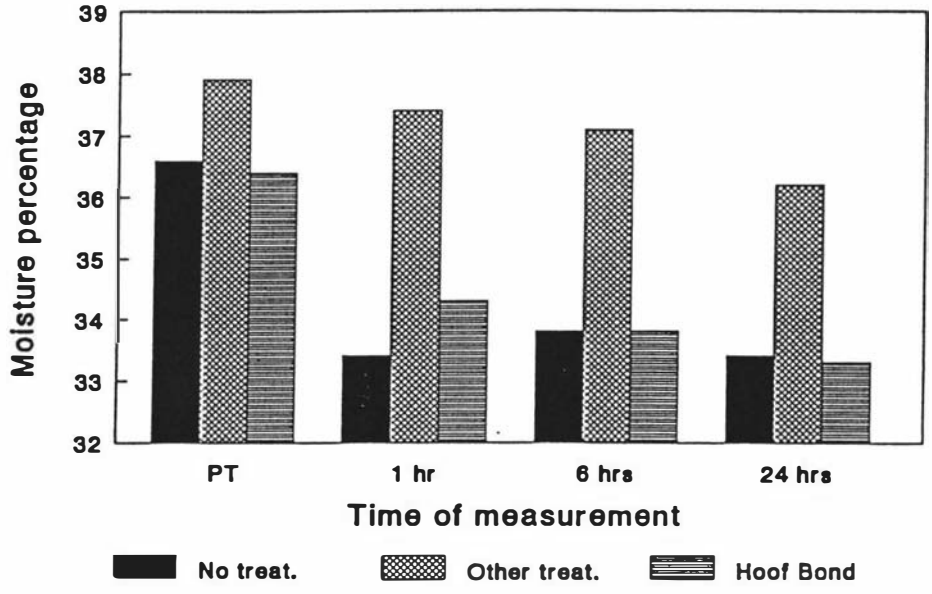

Figure 9.6. Changes in heel moisture over 24 hours for hooves receiving either no treatment, Hoof Bond plus water soaking or one of the other treatments plus water soaking

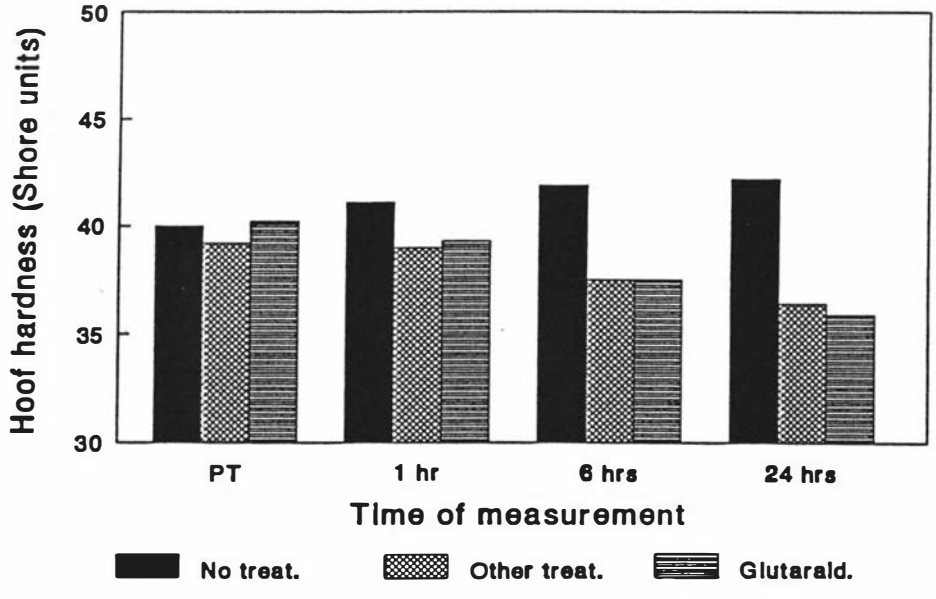

Figure 9.7. Changes in heel hardness over 24 hours for hooves receiving either no treatment, glutaraldehyde plus water soaking or one of the other chemical treatments plus water soaking

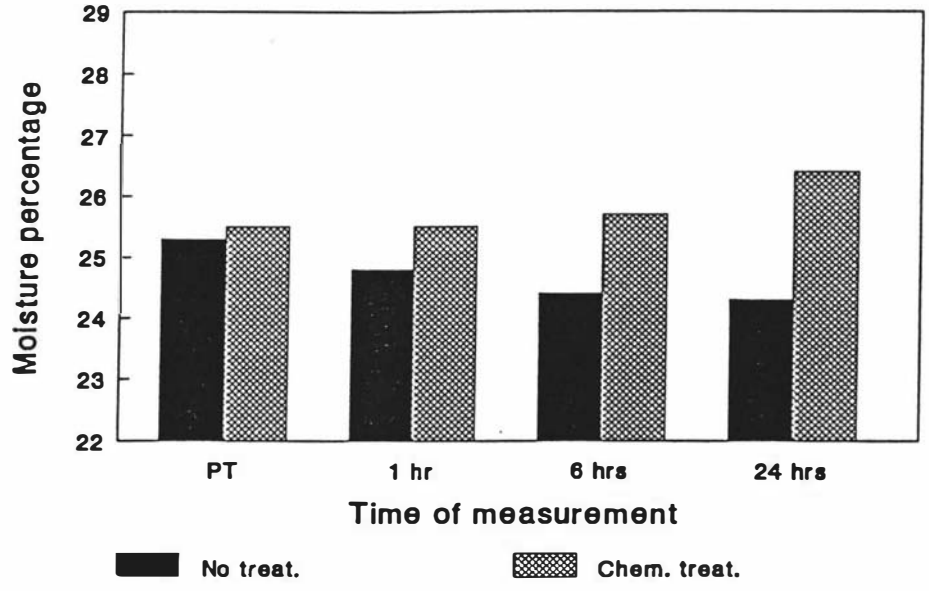

Figure 9.8. Changes in wall moisture over a 24 hour period for hooves receiving either no treatment or chemical treatment plus water soaking

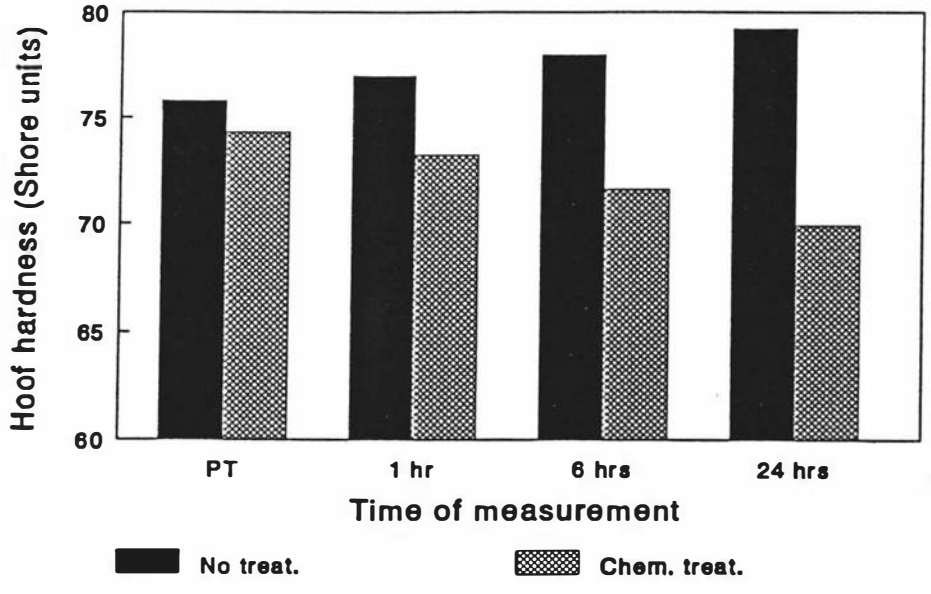

Figure 9.9. Changes in wall hardness over a 24 hour period for hooves receiving either no treatment or chemical treatment plus water soaking

Discussion

Hoof treatments in sheep and cattle have largely been used as surface antibacterial agents. However, several recent studies have indicated that most lameness in dairy herds in the pasture-based farming systems of Australia and New Zealand results from excessive wear of the hooves and traumatic injury to the hooves and sensitive corium of the foot (Dewes, 1978; Harris *et al.*, 1988; Malmo, 1991; Tranter and Morris, 1991). To be effective as a means of lameness prevention, topical hoof treatments need to be capable of hardening or strengthening hoof keratin or modifying it in a way that will reduce its susceptibility to external damaging influences. In these two studies some of the more common and readily available hoof treatments were applied to hooves collected from dead animals and their effect on hoof hardness and moisture content evaluated.

Formalin was chosen as footbaths containing formalin have often been advocated to control foot disease in cattle (Weaver, 1974; Greenough *et al.*, 1981; Baggott and Russell, 1981; McDaniel, 1983; Toussaint Raven *et al.*, 1985). It is relatively inexpensive and universally available. Its antibacterial properties are well recognised and make it an effective agent for the treatment and control of footrot in sheep and interdigital disease in cattle (Arkins, 1981; Skerman *et al.*, 1983a, 1983b; Arkins *et al.*, 1986; Roztocil *et al.*, 1988). However, its ability to modify claw horn and render it more resistant to external damage are less well proven. There is some evidence that, with repeated application, it will reduce hoof moisture content (Dietz and Koch, 1972; Arkins *et al.*, 1986). However, there is only limited documented evidence to support the commonly held belief that it has the potential to harden hoof and increase the resistance of hoof to abrasive wear (Camara and Gravert, 1971; Pietzsch and Schauer, 1973). Glutaraldehyde has similar properties to formalin with well recognised antibacterial activity, plus the possible ability to modify the characteristics of structural protein in the hoof. However, it is less irritant than formalin.

Like formalin, copper sulphate is widely used as a footbathing medication. It has been shown to penetrate hoof faster than formalin does (Malecki and McCausland, 1982) but any effects it has on physical hoof properties are not documented.

Zinc sulphate has been shown to have significant curative effects on footrot infection in sheep (Cross and Parker, 1981; Skerman *et al.*, 1983a; Malecki and Coffey, 1987). Cross

(1978) has suggested this action may be mediated by an ability to inactivate the proteolytic enzymes produced by *Bacteroides nodosus*. It penetrates hoof relatively quickly (as compared with other hoof treatments) (Malecki and McCausland, 1982), and is well retained by hoof tissue. However, no information is available as to its effect on physical hoof properties.

Hoof Bond has recently been marketed for prevention of hoof disorders in cattle, sheep, pigs and horses. This product, applied as a liquid, is stated to "bond to hoof keratin on exposure to ultraviolet light forming a hard but flexible shield". It is suggested, in marketing literature for the product, that this process strengthens the hoof, seals minor cracks and defects and prevents excessive drying or entry of moisture. However, no evidence could be found in the scientific literature to support this product claim.

In these two studies the duration of exposure to the chemicals under investigation was restricted to five minutes to reflect the agent contact time that might normally occur with routine field footbathing practice. Our results indicate that such brief treatment has little effect on the response of hoof to external hydrating or dehydrating conditions. To be effective, surface treatment may need to be applied for longer periods which is largely impractical on commercial farms. Alternatively, treatments may need to be applied on numerous occasions. To evaluate this latter option studies need to be conducted on live animals.

A notable finding of the first of the two studies conducted was the extent of the reduction in moisture content and of the increase in hardness, regardless of treatment applied, in hooves left exposed to normal atmospheric conditions for a period of 24 hours. Greatest changes occurred in heel hoof, normally the softest horn with highest moisture content, and smallest changes occurred in wall hoof which is the hardest and driest horn of the bovine digit. It would be expected that fluids would be lost through the external hoof surface in live animals in a similar way except when feet are exposed to wet environmental conditions. The difference in live animals is that hydrating fluids and nutrients are continually diffusing from the vascularised corium underlying the epidermal wall to the avascular keratin layers that form the horny covering of the claw, thereby maintaining hoof moisture and hardness at fairly constant levels.

In the second study where hooves were soaked in water following treatment, treated hooves, with the exception of those treated with Hoof Bond, behaved similarly to untreated hooves that were also soaked. Sole hoof moisture changed little over the 24 hours, heel hoof moisture decreased slightly, while water was absorbed by the drier wall hoof. The different pattern of change in moisture content observed in Hoof Bond treated hooves was confined to the heel region. In other respects the Hoof Bond treated hooves behaved similarly to those treated with any of the other treatments.

It is not surprising that hoof hardness increased as hoof moisture decreased in study one, and that it decreased as hoof moisture increased with water soaking, since the rigidity of keratin is believed to be inversely related to its water content (Fraser *et al.*, 1972). Hoof keratin is a protein composite consisting of two phases: a fibre phase mainly constructed from long slender α-helical microfibrils, which is crosslinked to an amorphous protein phase (Fraser and MacCrae, 1980). Cells responsible for keratin synthesis die in the final stages of their differentiation when disulphide cross-links are established in the keratin proteins within their cytoplasm. In the absence of water extensive hydrogen bonding exists between polymers of the matrix phase. This is thought to decrease the mobility of the matrix polymers (Fraser and MacCrae, 1980). When keratin is highly hydrated the matrix lacks much of the secondary bonding it possesses in the dry state. Longer distances occur between cross link positions giving the matrix polymers greater freedom of movement. This translates into lower stiffness since the secondary cross links are not available to carry load, and greater extensibility due to the ability of the polymers to rearrange under load (Bertram and Gosline, 1987).

Acknowledgements

The authors wish to thank the company and staff at AFFCO, Feilding, for assistance with the collection of abattoir material. They are also grateful to Hoof Bond Inc. and its New Zealand distributors, W. Wiggins Ltd., for supplying all the Hoof Bond used in the study. The technical assistance provided by Miss V. Tilson and Mrs. J. King was appreciated.

CHAPTER 10

Failure of topical hoof treatment to prevent lameness or alter physical hoof properties in dairy cattle[9]

[9] In press: Tranter WP, Morris RS. Veterinary Record, 1992.

Abstract

Two hoof treatments were evaluated for their effects on the incidence of clinical lameness and subclinical hoof lesions and for their effect on hoof moisture, hoof hardness and sole concavity in a 185 cow dairy herd. Digits of the right legs only were exposed to daily footbathing with 5 - 7.5% formalin solution (F) using a divided footbath. A mixture of acrylic acid and methylacrylic ester (HB) was applied to the hooves of one hind leg of 94 cows on three occasions at two monthly intervals. No significant differences were observed in the incidence of lameness between F treated, HB treated , F + HB treated and untreated digits. Detailed measurements were performed on the hind leg digits of 24 cows from the herd on four occasions during the study. None of the treatments had any effect on the incidence of subclinical hoof lesions or on changes in sole concavity. Minor reductions in hoof moisture levels were observed in formalin-treated hooves, but differences were significant only for heel moisture and developed only towards the end of the study. Feet in the F + Hb treatment group showed higher wall hardness at each evaluation but this was not exhibited by either treatment alone.

Introduction

Lameness in dairy cattle is a major cause of economic loss and inconvenience to farmers. The economic and welfare aspects are considerable and include reduced milk yield, a significant reduction in fertility and increased culling. Some lameness lesions are extremely painful and result in cows being lame for up to three months (Tranter and Morris, 1991). Most cases of lameness are associated with foot lesions with the majority of these involving disorders of the claw.

Recommendations for the prevention of lameness in predominantly pasture-fed herds emphasise the need to adequately maintain the surface condition of the farm raceways along which cattle are brought for milking and the need for patient handling of stock before and during milking (Dewes 1978; Harris *et al.*, 1988; Chesterton, 1989). However, despite their best efforts, farmers are sometimes faced with situations where prevention of lameness remains difficult. As the incidence of lameness is substantially higher in wet weather, lameness is more difficult to prevent in unusually wet seasons or where dairying is conducted in very wet climates. Lameness tends also to be a bigger problem in autumn-calving herds where cows are milked through wet winter months. Road building materials that are suitable for construction of good raceways are not always available. In situations where a new milking shed has been installed and the concrete of the yards has been left too rough, the risk of lameness increases dramatically. Hoof treatments capable of modifying the susceptibility of cows' hooves to traumatic injury and excessive wear would offer a means of lameness prevention.

Regular footbathing using formalin or copper sulphate has been widely recommended for the prevention of lameness in dairy cattle. However, as there have been so few clinical trials performed to objectively assess the benefits, there is still much confusion and debate on its effectiveness. Davies (1982) reported a reduction in lameness rates of about 12% in 27 U.K. herds using 1% formalin footbaths four times per week. In two experiments using 5% formalin four times per week, Arkins *et al.* (1986) concluded that treated claws had a lower incidence and severity of heel erosions, a lower moisture content and a reduced severity of haemorrhage of the sole at some sites in the claw compared with untreated controls. However, treatment had no significant effect on the incidence of clinical lesions of the claw.

The lesions causing lameness in dairy cattle in New Zealand and Australia are somewhat different to those reported from the northern hemisphere (McLennan, 1988, Jubb and Malmo, 1991; Tranter and Morris, 1991). A clinical trial was conducted using two available hoof treatments, to objectively measure their effects on physical hoof properties and to assess their ability to prevent claw disease.

Materials and methods

The herd chosen for this study was an autumn-calving herd in which the annual incidence of clinical lameness had been over 10% in the two years prior to commencement of the study. All 183 cows calved between early March and mid May 1991 and were grazed on 110 hectares near Palmerston North, divided into 60 paddocks.

In this trial the hooves of cows were subjected to four different treatments: daily formalin footbathing (F), bimonthly hoof bonding with a mixture of acrylic acid and methylacrylic ester (HB)[10], a combination of both F and HB (F + HB) or no treatment (NT). Treatment HB is described by the manufacturers as a non-toxic resinous liquid which forms a molecular bond with an animal's hoof on exposure to sunlight or UV light.

Hoof treatment

Treatment F was applied daily to the right feet only of all cows after the evening milking from calving through till the end of October 1991. A divided footbath (2.5 metres long) was used, the right side being filled to a depth of 3 cm with a formalin solution and the left side being continuously flushed with fresh running water. The treatment solutions were prepared using formalin concentrate (Formalin[11]) which was supplied in 20 litre containers as 37 percent formaldehyde. The footbath was placed in the exit race in such a way that the left feet were placed in the water and the right feet in the formalin solution. Use of the bath was trialled prior to the commencement of the study. Samples collected from the footbath after each batch of 25 cows had passed through were analysed to determine active formalin concentration using the Hantsch reaction (Nash,

[10]Hoof Bond, Inc., 131 Old Route 132, Hyannis, MA 02601, USA

[11] Pitman Moore (NZ) Ltd, Private Bag, Upper Hutt, New Zealand

1953). This work revealed that the concentration of formalin fell rapidly with use (Figure 10.1). To ensure that all feet treated were exposed daily to a minimum concentration of 5% formalin, a 7.5% solution was freshly prepared prior to use of the footbath each evening and additional concentrate was added after approximately 100 cows had passed through. Occasional checks performed during the 6 months of the trial confirmed that this protocol was successful in maintaining the concentration above 5%.

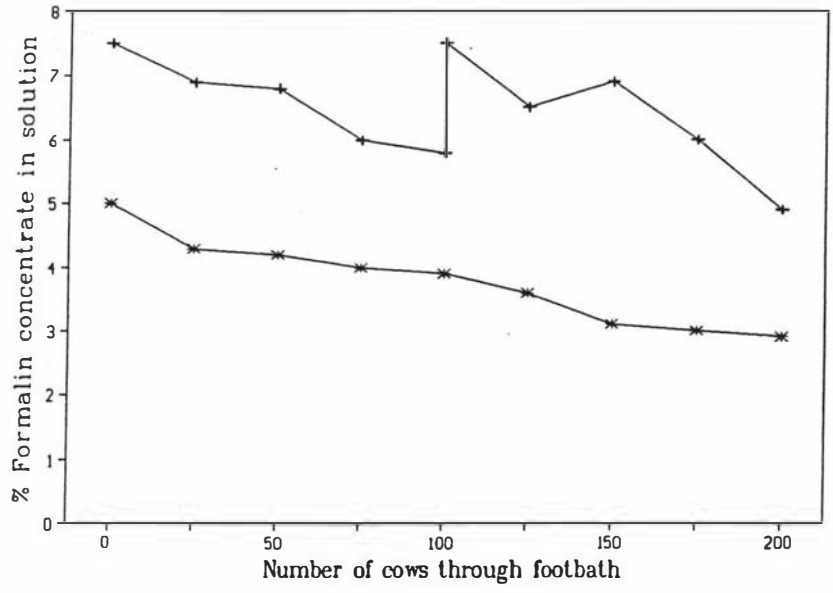

Figure 10.1. **Change in formalin concentration with passage of cows through footbath. + = 7.5% starting concentration with additional concentrate added after 100 cows; * = 5% starting concentration without recharging.**

Treatment HB was applied to one hind foot (left for half the animals and right for the remainder) of 94 randomly selected cows on three occasions (March prior to calving, mid-May and mid-July). The appropriate leg was lifted and the hooves were washed thoroughly, rinsed with alcohol and dried using paper towelling. HB was applied by sponge to the sole and walls of the hoof of both digits and the hooves were then exposed to UV light for 30 seconds using a hand held lamp. To treat one hind foot on each cow on each occasion took approximately eight minutes. The numbers of feet receiving each treatment are shown in Table 10.1.

Table 10.1. **Numbers of feet receiving each treatment. HB = bimonthly Hoof Bond application (acrylic acid and methylacrylic ester); F = daily formalin footbathing; HB + F = both HB and F; NT = no treatment.**

	HB	F	HB + F	NT
Fore legs	0	183	0	183
Hind legs	47	136	47	136
TOTAL	47	319	47	319

Clinical lameness

All cows, identified by the herdsman as being lame, were examined within three days of lameness onset. Details of lameness severity, lesion type, lesion severity and site of lesion were recorded using the classification and scoring systems described by Tranter *et al.* (1991).

Subclinical hoof lesions and physical hoof characteristics

12 HB-treated cows and 12 non-HB treated cows were selected for additional measurement. These were randomly selected from within three age groups (youngest third, middle third and oldest third of cows in the herd). Measurements were performed on both hind legs (4 hind digits per cow - 96 digits in total) on four occasions: in March prior to treatment, in May and July two months after HB treatment and prior to HB retreatment and in September two months after the last HB retreatment. The design is summarised in Table 10.2.

Table 10.2. **Digits of the hind legs of 24 cows that were examined bi-monthly for subclinical hoof lesions and on which physical measurements were performed**

YOUNGEST THIRD (32 Digits)		MIDDLE THIRD (32 Digits)		OLDEST THIRD (32 digits)	
NT (8)	F + HB (8)	NT (8)	F + HB (8)	NT (8)	F + HB (8)
HB (8)	F (8)	HB (8)	F (8)	HB (8)	F (8)

On each occasion, each hind foot was lifted, washed and examined. Type, severity and site of all observed lesions were recorded. Sole concavity at 30 mm from the abaxial hoof margin was measured using a profile gauge; sole, heel and wall moisture were measured using a Delmhorst moisture meter and sole, heel and wall hardness were measured using a Shore Type D durometer. The methods used have been previously described (Tranter *et al.*, 1992a).

Statistical analysis

Findings were recorded and analysed using the statistical programs PANACEA 2 (PAN Livestock Services, Reading, England) and SOLO (BMDP Statistical Software Inc., Los Angeles, California). The significance of differences in lameness incidence and numbers of subclinical lesions between treatment groups was tested using chi-squared analysis. Differences in hoof moisture, hoof hardness and sole concavity were assessed using analysis of variance. Sole concavity results are presented in Table 10.5 as untransformed values, but were log transformed prior to analysis to normalise the data.

Results

Clinical lameness

49 lesions, of severity score 3, were observed in 47 cases of clinical lameness during the course of the study. White line disease and septic pododermatitis (sole abscess) were the most common lameness-causing lesions (Figure 10.2). Two cases that involved pastern infections appeared to result from initial skin scalding and subsequent secondary bacterial invasion, caused by formalin in the first few weeks following introduction of footbathing. Because of this the depth of formalin in the bath was lowered to 3 cm to minimise skin exposure to formalin. No more of these clinical lesions occurred. However, minor skin scabbing was observed on the pasterns of the right hind legs of almost all cows throughout the study. These lesions were rarely observed in the right fore legs, presumably because of the more upright nature of the fore pasterns.

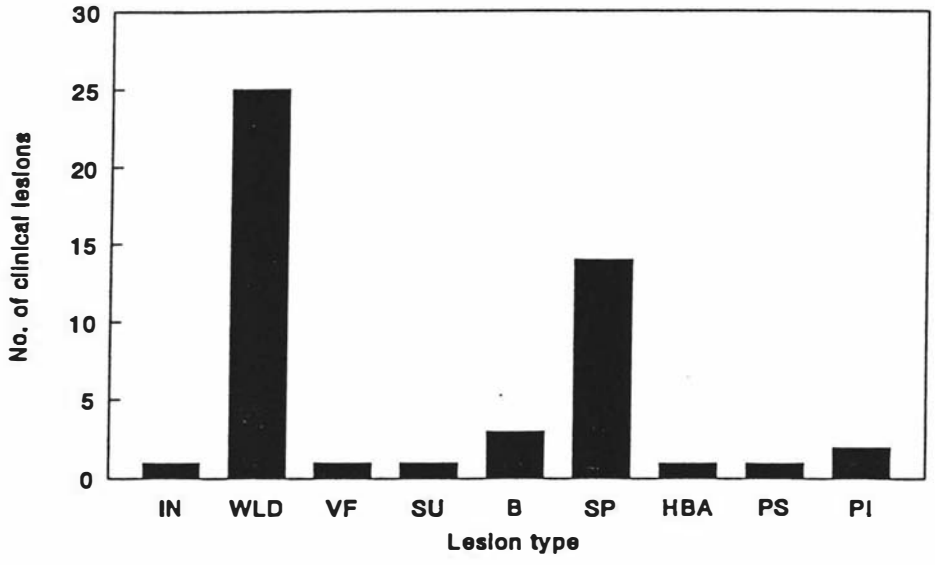

Figure 10.2. **Lesions causing clinical lameness during the 7 month study period.**

IN	=	Interdigital Necrobacillosis
WLD	=	White Line Disease
VF	=	Vertical Fissure
SU	=	Sole Ulcer
B	=	Bruising
SP	=	Septic Pododermatitis
HBA	=	Heel Bulb Abscess
PS	=	Punctured Sole
PI	=	Pastern Infection

The incidences of clinical lameness in the different treatment groups are shown in Table 10.3. There were no significant differences between treatments ($\chi^2 = 3.36.$; $p > 0.05$) in the total incidence of lameness, and no notable difference in the lesions seen on the different feet according to treatment given.

Table 10.3. **Lameness incidence in different treatment groups**

	HB	F	HB + F	NT
Fore legs	-	3.3%	-	3.3%
Hind legs	10.6%	11.0%	8.5%	6.6%

Subclinical lesions

The number of hind leg digits with subclinical hoof lesions in the 24 cows selected for regular hoof examination are shown in Table 10.4. Few hoof lesions were observed in March prior to calving. White line lesions were observed in approximately one third of the digits at the May examination and in almost half in July and September. Little sole haemorrhage was observed in May but again almost half the digits had some degree of haemorrhage in July and September. Most lesions occurred on abaxial claws. There were no significant differences between treatment groups in the number of digits with either lesion at any of the examinations ($p > 0.05$).

Table 10.4. Subclinical hoof lesions observed in hind leg digits of the 24 cows selected for bi-monthly hoof examination

	HB	F	HB + F	NT
White Line Separation:				
March	2	1	0	3
May	10	9	3	8
July	9	8	8	10
September	10	10	10	11
Haemorrhage:				
March	1	0	0	0
May	0	3	3	0
July	10	9	8	8
September	11	10	10	10

Hoof moisture, hoof hardness and sole concavity

The results of hoof moisture, hoof hardness and sole concavity measurements for hooves in each treatment group at each of the four observations are shown in Table 10.5. Few differences were observed between treatment groups. In March, prior to initial treatment, some differences existed in sole and heel hardness between feet selected for subsequent treatment. These must be ascribed to chance. Feet treated with both formalin and Hoof Bond showed higher wall hardness at each evaluation, but this was not exhibited by feet receiving either treatment alone. While the same trend was found

for sole hardness, the differences were never significant. Minor reductions in hoof moisture levels were observed in formalin-treated hooves, but differences were significant only for heel moisture and developed only towards the end of the study.

Table 10.5. **Physical properties of the hooves of the hind digits of 24 selected cows at the four different times of measurement. Values in the same row with different superscripts are significantly different ($p < 0.05$).**

March - prior to treatment:

	F	HB	F + HB	NT
Sole moisture %	33.0 ± 3.1	33.0 ± 3.7	34.0 ± 2.1	33.7 ± 2.4
Heel moisture %	40.5 ± 4.9	40.9 ± 5.2	42.1 ± 4.3	42.8 ± 3.9
Wall moisture %	25.2 ± 1.2	25.0 ± 1.5	25.0 ± 1.0	24.8 ± 1.2
Sole Hardness (Shore units)	52.1 ± 3.3^{w}	52.2 ± 3.7^{w}	49.3 ± 4.4^{x}	49.3 ± 5.1^{x}
Heel Hardness (Shore units)	37.9 ± 3.0^{y}	40.3 ± 3.0^{z}	37.3 ± 2.6^{y}	37.3 ± 3.4^{y}
Wall Hardness (Shore units)	73.2 ± 2.6	73.1 ± 3.1	73.6 ± 2.7	73.0 ± 2.4
Sole concavity (mm)	5.0 ± 2.3	5.1 ± 2.6	6.0 ± 2.4	5.8 ± 3.0

May:

	F	HB	F + HB	NT
Sole moisture %	31.9 ± 2.2	31.6 ± 2.2	31.2 ± 1.7	31.6 ± 1.8
Heel moisture %	37.6 ± 4.3	38.0 ± 4.8	38.3 ± 3.7	39.4 ± 4.1
Wall moisture %	26.1 ± 1.0	26.3 ± 1.2	25.6 ± 1.1	25.9 ± 0.9
Sole Hardness (Shore units)	52.1 ± 2.3	52.1 ± 3.0	52.8 ± 2.4	51.8 ± 2.3
Heel Hardness (Shore units)	38.0 ± 4.0	40.3 ± 4.3	38.1 ± 3.6	38.1 ± 2.6
Wall Hardness (Shore units)	71.6 ± 2.3^{y}	70.5 ± 2.1^{y}	73.0 ± 2.3^{z}	71.4 ± 2.4^{y}
Sole concavity (mm)	2.6 ± 2.0	2.3 ± 1.8	2.7 ± 2.3	2.1 ± 1.9

July:

	F	HB	F + HB	NT
Sole moisture %	29.8 ± 1.7	30.3 ± 1.9	29.8 ± 1.3	30.2 ± 1.8
Heel moisture %	34.0 ± 2.1	34.0 ± 3.0	34.0 ± 2.5	35.5 ± 3.3
Wall moisture %	26.5 ± 1.7	26.8 ± 1.8	26.3 ± 1.9	26.0 ± 1.2
Sole Hardness (Shore units)	51.3 ± 4.4	50.4 ± 3.9	51.5 ± 4.5	50.3 ± 3.6
Heel Hardness (Shore units)	39.3 ± 5.1	39.9 ± 6.1	39.6 ± 5.4	37.4 ± 6.9
Wall Hardness (Shore units)	70.2 ± 3.4^{y}	70.0 ± 2.9^{y}	72.5 ± 3.0^{z}	71.1 ± 2.4^{y,z}
Sole concavity (mm)	1.6 ± 1.7	1.2 ± 1.1	1.5 ± 1.4	1.2 ± 1.6

September:

	F	HB	F + HB	NT
Sole moisture %	28.4 ± 1.1	29.2 ± 1.7	28.2 ± 1.4	28.7 ± 1.2
Heel moisture %	32.5 ± 2.2^{y}	34.4 ± 3.0^{z}	32.9 ± 2.6^{y,z}	34.2 ± 2.9^{z}
Wall moisture %	25.0 ± 1.3	25.9 ± 1.3	25.2 ± 1.2	25.2 ± 1.2
Sole Hardness (Shore units)	51.7 ± 3.9	51.1 ± 4.1	52.1 ± 2.6	50.5 ± 3.5
Heel Hardness (Shore units)	40.2 ± 3.9	39.9 ± 4.2	39.3 ± 3.9	38.0 ± 3.6
Wall Hardness (Shore units)	72.6 ± 3.2^{y,z}	71.3 ± 3.3^{y}	73.8 ± 2.1^{z}	73.1 ± 3.5^{y,z}
Sole concavity (mm)	1.5 ± 1.3	1.3 ± 1.4	1.4 ± 1.4	1.2 ± 1.4

Discussion

Footbaths have been widely recommended to control and prevent lameness in dairy herds (Toussaint-Raven and Cornelisse, 1971; Weaver, 1974; Greenough *et al.*, 1981; McDaniel, 1983). Formalin and copper sulphate solutions are the most commonly used materials. They are used to remove erosive materials from the foot, to reduce bacterial contamination and to harden the hoof horn.

Footbaths have been advocated for the control of many different foot lesions including both interdigital conditions and disorders of the claws. Cornelisse *et al.* (1982) describe the use of formalin for prevention of interdigital dermatitis. Amstutz (1985) cites Bayley's recommendation of 5% formalin once or twice per week as a prevention for infectious pododermatitis (footrot). Baggott and Russell (1981) recommend footbaths for both interdigital infections and for diseases, injuries or excessive wear of the claw horn. They also recommend their use for lesions associated with horn softening, such as white line disease. Toussaint Raven *et al.* (1985) recommends formalin bathing for the control of heel horn erosion. This is supported by Gibson (1984/85) who states that footbaths are very useful for control of both heel horn erosion and foul of the foot (interdigital necrobacillosis). In a discussion of white line disease, Edwards (1980) suggests that the regular use of footbaths during the housed period can be beneficial for the prevention of this condition. Davies (1982) encourages regular footbathing in herds with a high incidence of interdigital disease, heel erosion and sole ulceration. However he states it is less likely to benefit herds where the predominant problem is white line disease or sole puncture. In a review of special procedures used for treatment and prevention of foot disease, Nelson and Petersen (1984) suggest that the effectiveness of footbaths in the prevention of lameness has not been thoroughly examined. They do reduce lameness from diseases involving the interdigital skin but are ineffective in treating diseases of the hoof horn.

Recommendations on frequency of application, concentration of active ingredient and contact time required for effect have also varied widely. Consequently a wide range of footbathing strategies have been adopted (Sumner and Davies, 1984). It is common knowledge that formalin is an irritant solution and that repeated applications with solutions that are too concentrated will have a necrotising effect. Arkins *et al.* (1986) reported twice daily bathing produced hyperaemia and skin necrosis. Baggott and Russell

(1981) recommended that formalin not be used more than once or twice per week or the hoof horn will become too brittle. In studying the harmful effects of formalin in work with sheep, Littlejohn (1972) found too frequent application was more damaging than concentration as weekly applications of 10 % solutions were well tolerated whereas the same exposure twice weekly produced hyperaemia, hyperkeratinisation and necrosis of the interdigital skin.

The wide variation in recommendations for use of protective hoof treatments and the debate over which lesions can be controlled by footbathing is almost certainly due to the sparsity of documented information on their effectiveness. In two different studies comparing treated and untreated cows, Arkins (1981) and Arkins *et al.* (1986) found that formalin footbathing decreased the incidence of lameness due to disease of the interdigital skin but had no effect on the incidence of claw disease. When he compared treated and untreated claws on the same cows in a trial using a divided footbath, he found formalin treatment had no significant effect on the overall incidence of foot disease or on the incidence of any individual clinical lesion. However, he did observe a lower incidence and severity of heel horn erosions in treated claws and a reduction in sole haemorrhages at some sites. In a similarly designed study using a herd of about 100 cows, Russell (1984/85) found that formalin-treated left feet had about one third fewer lesions than non-treated controls, with the greatest reduction being in lesions involving erosion and invasion of the horn or skin. Roztocil *et al.* (1988) reported that formalin footbathing was effective in the treatment of cattle suffering from digital dermatitis.

There are no published reports of controlled trials conducted in New Zealand or Australia using protective hoof treatments. However, in a herd case-control study Chesterton *et al.* (1989) did observe that herds with a high lameness incidence were twice as likely to be using a formalin footbath than were low lameness incidence herds. This positive association between the presence of a footbath and lameness was suggested by these authors to be more probably an effect of high lameness incidence than a cause. They proposed that farms encountering more problems with lameness tried to solve them by using a footbath but this did not solve the underlying causes of the lameness problem.

A notable finding from our study was the rapid reduction in formalin concentration with use. After the passage of only 100 cows (right feet only) the concentration had fallen from an initial 5% to a little over 3%. This would suggest that common footbathing

practice on farms (where footbath solutions are routinely not replaced for several days despite the passage of 600-800 cows through the bath) have little chance of providing effective treatment to the majority of animals.

Application of formalin more frequently (daily) and in a more concentrated form (5 - 7.5%) than is routinely practised necessitated restricting the depth of solution in the footbath to three centimetres so as to reduce skin contact. Since the main objective of the hoof treatment was not to prevent interdigital disease but to harden and strengthen the claw horn, this depth was considered adequate. However, even with the precaution of restricting the depth, most treated hind feet developed hyperkeratotic lesions, which if removed left raw granulating areas. Except for two cows that developed iatrogenic lameness early in the study (when the depth of solution was five centimetres) these lesions appeared to cause little harm or discomfort.

The failure of formalin to influence hoof wear (as measured by loss of sole concavity in cows after they rejoined the milking herd at calving) or substantially alter hoof hardness contrasts with the results of previous *in vitro* studies. Camara and Gravert (1971) observed that hoof tissue was hardened when treated with formalin and wore less than untreated tissue. Pietzsch and Schauer (1973) also recorded an increase in resistance to abrasion in claw horn treated with formalin for 15 seconds four times daily. In an analysis of data collected from 81 Dutch herds, Smit *et al.* (1986a) found the use of formalin was associated with longer claws and suggested this may have been caused by hoof hardening which in turn reduced rates of wear.

Another property of formalin that has been described is its ability to reduce hoof moisture content. It is absorbed very poorly by hoof horn but since it forms cross linking methylene bridges within proteins it may make hoof tissue more impervious (Malecki and McCausland, 1982). Dietz and Koch (1972) recorded a reduction in water content of 3-5% in treated claws while Arkins *et al.* (1986) found a reduction of 1.8% which represented an actual moisture loss of 0.5%. In our study formalin-treated hooves tended to have lower sole and heel moisture levels of the order observed by Arkins, but except for heel moisture in September these differences were not significant. In addition any differences observed were small in comparison with the more substantial changes in hoof moisture content that occur with changes in environmental moisture conditions (Tranter *et al.*, 1992c). Previous workers including Arkins have measured hoof moisture

by collecting hoof shavings from the sole surface and oven drying the samples until they reached a constant weight. Our method using a hand held moisture meter measured moisture to a deeper level of three millimetres.

A product containing acrylic acid and methacrylic esters (Hoof Bond) has recently been marketed for prevention of hoof disorders by topical application to the hoof. This product, applied as a liquid, bonds to hoof keratin on exposure to ultraviolet light forming a hard but flexible protective shield. It is suggested that this process strengthens the hoof, seals minor cracks and defects and prevents excessive drying or entry of moisture. Since some of the common forms of lameness involve gradual disintegration of soft hoof tissue (white line disease) or entry of infection through minor hoof defects (septic pododermatitis) a product with such features could potentially be of value. However, the failure of Hoof Bond to influence the incidence of lameness or any of the other claw quality variables measured indicates that further research and development of this technology will be required if it is to have commercial application in dairy herds. Hooves treated with both Hoof Bond and Formalin showed increased wall hardness but this did not have any detectable effect on hoof wear or occurrence of hoof lesions in the experimental herd.

The complete failure of formalin footbathing to prevent lameness, reduce the incidence of subclinical hoof lesions or substantially alter hoof moisture, hardness or sole concavity changes adds to the accumulating evidence that it is ineffective as a method of preventing claw-related lameness in dairy cattle under pasture-fed systems.

It can be concluded from this and earlier related studies (Chesterton *et al.*, 1989; Tranter and Morris, 1991) that lameness in New Zealand dairy herds results primarily from adverse environmental conditions, exacerbated considerably by management actions taken by farmers which increase the risk of animals being exposed to injury-causing circumstances. However, each of the various types of lameness lesions are not chance events, but develop in characteristic patterns both between different digits and between various phases of the milking season (Tranter and Morris, 1991; Tranter *et al.*, 1991). At present the best approach for veterinarians to adopt for lameness prevention is to investigate herds as described by Chesterton (1989), and to recommend changes in management procedures and improvements in race construction to reduce the risk factors identified by Chesterton *et al.* (1989).

It does not appear that application of either formalin or Hoof Bond can be recommended on the basis of findings from this study. However, given the association found in earlier studies between wet environmental conditions and incidence of lameness (Tranter and Morris, 1991), there remains scope for exploring methods which would either reduce the susceptibility of hoof horn to the effects of moisture, or would block one of the sequence of steps which leads to development of clinical lameness. These steps are not fully clear, and may, for example, involve bacterial action as well as mechanical damage. Further research to develop lameness prevention methods should therefore be targeted at identifying steps in the initiation of hoof lesions which could be prevented by some form of intervention. One such candidate approach would be dietary supplementation with biotin which has been shown to benefit hoof structure and integrity (Webb *et al.*, 1984; Simmins and Brooks, 1988; Reilly and Brooks, 1990) and may have protective value for hooves akin to that of fluoride in the prevention of dental caries. However, the use of biotin would require careful field evaluation before it could be recommended.

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CHAPTER 11

General Discussion

Introduction

Lameness varies in incidence between herds to an extent which demonstrates that herd-level risk factors must influence the disease; within herds the occurrence of the disease is also far from random. In order to better understand the epidemiology of lameness, variation both between and within herds must be examined. In an earlier study of lameness in pasture-fed dairy cattle which preceded this project, Chesterton *et al.* (1989) examined differences in the occurrence of risk factors between herds with high and low incidence rates of lameness. To complement this earlier work, the objective of this investigation was to determine which risk factors predispose individual cows to lameness. Although large differences in lameness incidence rates between herds were observed, it was not possible to examine between-herd factors that might be associated with lameness because of the within-herd study approach used.

The findings of each individual study in the series that is reported here have already been discussed at the conclusion of each chapter. This general discussion will consider risk factors and control measures for lameness that were mentioned in the literature review in Chapter 2 and will discuss the contribution that this work has made to the current state of knowledge of the subject.

Clinical lameness

Although the case study described in Chapter 5 involved only three herds it represented 134 cases of lameness and was an important part of the investigation since it is only the second such study to have been reported from New Zealand. Further, it is the first New Zealand lameness study to describe lesions using the now standard terminology for digital disease that has been recommended by the international working group on Disorders of the Ruminant Digit (Espinasse *et al.*, 1984) and others (Mills *et al.*, 1986). Use of this terminology is essential if results of different studies are to be compared. Since in this study details of all cases of lameness observed by the farm managers were recorded, the results provide a more complete picture of the pattern of lameness in these herds than would have been possible if a survey of cases treated by veterinarians only had been conducted. Many previously reported studies have used veterinary practice records to gather epidemiological information on lameness. Although this approach has usually

allowed a greater number of farms to be surveyed, estimates of incidence rates have, in some instances, been unreliable and details of lesion types have been incomplete since details of cases not attended by veterinarians could not be included.

Few studies have reported details of the duration of lameness. This study clearly shows that some lesion types, because they cause lameness for longer periods, are more serious and are likely to be responsible for greater production losses than others. Although interdigital necrobacillosis causes severe lameness at onset it readily responds to antibiotic therapy and cows are lame for just a few days. Consequently, it is of little concern to most farmers and the need for development of more effective control strategies is not as great for this lesion type as it is for lesions involving the bearing surface of the hoof. Even if an effective vaccine was developed for interdigital necrobacillosis it is unlikely to be used widely because of the less serious nature of the lameness that results from this lesion type.

It is useful to compare the types of lesions observed in this investigation with those reported from surveys that have been conducted in different farming systems. These differences by themselves provide insights into the importance of the various factors considered to predispose cattle to lameness. In our pasture-based farming systems sole ulcers are rarely seen, yet they are one of the more common conditions that affect the claws of cattle kept under confined conditions. This demonstrates that continual exposure of hooves to unyielding concrete surfaces must be strongly influential on development of this lesion. Similarly, acute and chronic laminitis are very rarely seen in Australia and New Zealand, at least in part because the level of concentrate feeding is lower than in most other dairying countries.

White line disease, which was commonly observed in our studies, has frequently been reported as being one of the most common forms of lameness in the U.K. dairy cattle population. It was also the most common lesion observed in Willyanto's study of 24 herds in northern Queensland, accounting for 32% of 1373 lameness lesions recorded (Willyanto, pers. comm.). In contrast, in other Australian studies, McLennan (1988) and Jubb and Malmo (1991) found white line disease to be much less common and suggested that it is mostly a problem in the northern hemisphere where cows are housed and fed large amounts of concentrates. Clearly it is not restricted to such systems. Risk factors that the herds in our studies are most likely to have in common with northern

Queensland herds include long distances walked each day to pasture, very wet weather conditions and use of unsuitable materials for race construction.

These same factors also appear to predispose cows to sole bruising. In both the case study in this series and the study of Dewes (1978) sole bruising was one of the most commonly observed lesions resulting in clinical lameness. Similarly, Willyanto (pers. comm.) found that bruised sole was common in northern Queensland herds and accounted for 17% of lesions in his study. In most other studies, both in Australia and the northern hemisphere, it has accounted for only a small proportion of lesions.

Yet another difference is that of axial wall cracks as reported by Jubb and Malmo (1991). This lesion is common in cattle in the Macalister Irrigation area of Gippsland, Victoria yet has accounted for a very small proportion of lesions in other studies. This difference prompted Jubb and Malmo to suggest that interdigital trauma and repeated wetting and drying of hooves associated with irrigation practices may be risk factors for this lesion.

Subclinical hoof lesions

The observations made of subclinical hoof lesions throughout these studies provided some useful insights into the epidemiology of lameness in pasture-fed herds. It seems likely that the white line separation and sole haemorrhages that were commonly observed in non-lame digits were minor manifestations or early stages of processes that may result in clinical disease. They occurred with a definite epidemiological pattern. In the longitudinal study described in chapter 4, lesions were observed with specific claw distributions in the cow and at specific times of the year with waves of each type of lesion passing through the herd at a particular time.

Given the markedly non-random distribution of these lesions in time and between digits, it seems likely that certain risk factors are operating to initiate these lesions but a second separate stage is required to convert the subclinical lesions into clinical lesions. This second stage may involve either more intense exposure to the same risk factors, or exposure to a second additional set of risk factors. From experience in working with two herds which were under the same ownership and located within 2 km of each other, the fact that one herd had severe lameness problems while the other herd (used for the

subclinical lameness study) had numerous lesions but virtually no clinical lameness, suggests that the difference arises principally from the intervention of additional risk factors, rather than simply an increase in the intensity of the same factors that produced the subclinical lesions. The hypothesis is therefore suggested that the pathogenesis of lameness is a two-stage process which should be evaluated as such.

Considerable emphasis has been placed on the study of sole haemorrhages in cattle in recent times. Throughout these studies sole haemorrhages were frequently observed. They appeared as red speckles, streaks or minor blotches and were concentrated in the mid-sole, abaxial sole and heel regions. These lesions have many characteristics in common with descriptions of subclinical laminitis. They were more prevalent in hind feet than front feet and more prevalent in the lateral claws. Such an uneven distribution of haemorrhages between lateral and medial claws is similar to that described for subclinical laminitis in the hind feet of young cattle (Bradley *et al.*, 1989; Greenough and Vermunt, 1991). At some examinations haemorrhages were observed in several zones on the one digit. Greenough and Vermunt (1991) consider that the presence of haemorrhages in several zones of the sole is indicative of subclinical laminitis. However one feature often mentioned in descriptions of subclinical laminitis and which was rarely observed in our studies is the presence of soft waxy horn with yellow discoloration of the sole.

One of the major difficulties in associating the haemorrhages observed in this work with subclinical laminitis is the lack in New Zealand herds of most of the nutritional factors commonly considered to predispose to the syndrome. It is more likely that the haemorrhagic lesions that are commonly observed in New Zealand are caused by traumatic injury and bruising of the solar corium. Mechanical overloading and traumatic injury of the solar corium have previously been implicated in the aetiology of solar haemorrhages and the subclinical laminitis syndrome, but they have received far less attention than nutrition in the veterinary literature. This raises a number of questions.

Firstly, are the lesions involving solar haemorrhages that are observed in New Zealand cattle different from those described as subclinical laminitis? As mentioned above the lesions have many characteristics in common but detailed examinations using histologic and electron microscopic techniques would be required to determine if they are in fact the same syndrome.

Secondly, if the lesions are similar is it likely that traumatic injury does play a more significant role in the aetiology of the subclinical laminitis syndrome? In most recent discussions of subclinical laminitis the role of nutrition has been emphasised. Clearly feeding high energy/low fibre rations and rapidly increasing the amount of concentrate after calving can predispose cows to subclinical hoof lesions and/or lameness (Peterse, 1979; Livesey and Flemming, 1984; Moser and Divers, 1987; Manson and Leaver, 1988a; Greenough *et al.*, 1990). However, perhaps as David (1990) has suggested, there has been a tendency to overemphasise the contribution of nutrition to the exclusion of other contributing factors. Close scrutiny of many of the published reports of laminitis (Livesey and Flemming, 1984; Bazely and Pinsent, 1984; Mgassa *et al.*, 1984; David, 1984/85; Colam-Ainsworth, 1989; Greenough and Vermunt, 1991) reveals that management and behavioural factors as well as nutritional influences were likely to have been involved in the aetiology of lesions described.

One possible explanation that would be consistent with most reported observations is that haemorrhages do occur as a result of mechanical trauma. Nutritional factors could exert their major influence by mechanisms that result in the production of physically softer and less resilient horn which is less capable of protecting the underlying corium from compressive stress and mechanical injury. Similarly wet environmental conditions could predispose to haemorrhage by increasing hoof moisture content which, in turn, alters other physical hoof properties that influence the ability of the external hoof layers to resist damaging impacts. The finding that the prevalence and severity of haemorrhages are almost always greater in the lateral digits of the hind legs is also consistent with this explanation. Lateral digits experience higher pressures during load bearing (Scott, 1988), experience greater variation of loads (Ossent *et al.*, 1987) and have less sole concavity. Hence, it would be expected that haemorrhages would be more common in these areas where higher pressures occur during weight bearing.

A third question raised is, if the solar haemorrhages observed in New Zealand cattle are similar to those described for subclinical laminitis, are there other nutritional factors that could be involved in their aetiology in pasture-fed cattle? As mentioned previously, most of the nutritional factors that have been implicated as predisposing to subclinical laminitis in cattle are not present in the diet of predominantly pasture-fed cattle. Concentrates are rarely fed and, when they are, levels are usually restricted to 1-2 kg per cow per day. The possible role of high protein and low crude fibre cannot be discounted but, at the

same time, there is little epidemiological evidence available to implicate these factors. Certainly it could not account for the large difference in prevalence of haemorrhage between digits. Nor could it account for the higher prevalence of haemorrhage in the 2-year-old cows in the longitudinal study described in Chapter 4 since cows of both age-groups grazed the same pastures throughout the study. It is more likely that haemorrhages were more common in the younger cows due to trauma resulting from unplanned foot placement during behavioural interactions with older more dominant herd mates.

In recent times the term "subclinical laminitis" seems to have become synonymous with sole haemorrhage. David (1990) has suggested the term laminitis be used with more precision. Traditionally, laminitis has implied a nutritional aetiology and to extend use of the term to cover any inflammation of the solar corium and any lesion involving sole haemorrhage is likely to continue to add confusion to discussion of the subject. It would be preferable to restrict use of the term laminitis to cover the clinical manifestations of acute and chronic laminitis and to use more specific terminology when describing subclinical hoof lesions and hoof quality characteristics.

Claw quality

Although most workers have emphasised the importance of claw quality in protecting cows from lameness, there is little available information to indicate which claw properties provide resistance to damaging influences. Terms such as "poor quality hoof" have frequently been used in discussions on subclinical laminitis, without definition.

In this work, techniques have been developed to measure a number of physical hoof properties that may be important in providing hoof with the ability to resist external damaging influences. In some cases methods were adapted from those described by previous workers, while in other cases totally new techniques have been developed. For some properties, such as hoof moisture content, it was possible to validate the technique used so as to be confident that it was accurately measuring the property concerned. For other properties, it was not possible to validate the technique used but results of measurements appeared to make biological sense. The techniques developed were

performed on live animals under commercial farm conditions and provided some useful insights into the mechanisms considered to be involved in development of hoof lesions.

The process of collecting hoof biopsy samples and preparing them for compression tests was time consuming and labour intensive. However, undertaking this work meant that hoof characteristics distinctly different from hardness could be measured. In studies evaluating the effects of wet weather on hoof tissue, measuring these extra properties produced a more comprehensive set of results than would otherwise have been possible, and enabled conclusions to be drawn with more confidence. However, it is doubtful whether the additional benefit gained by performing these tests, over and above that derived from using the simpler techniques, could be justified except for those involved in research.

Changes in hoof moisture content have commonly been suggested as the medium by which wet weather conditions predispose cows to lameness. Our work clearly confirms that hoof moisture is affected by environmental moisture conditions. Results from the longitudinal study described in chapter 7 show that hoof moisture was highest when regular rainfall and a small number of hours of sunshine resulted in high soil moisture levels. There is no single variable that can be used to describe the wetness of the environment to which hooves are exposed. However, the various measures of environmental moisture used in this study were successful in explaining a substantial proportion of the variation observed in hoof moisture content.

Our studies on hooves harvested from cattle after slaughter show that hoof moisture content is largely dependent on the continual diffusion of hydrating fluids from the vascularised corium to the avascular keratin layers that form the horn covering the claw. Hoof moisture levels fell considerably when hooves were left exposed to normal atmospheric conditions for a period of 24 hours after slaughter. Presumably fluids are lost through the external hoof surface in live animals in a similar way but diffusion of fluids from underlying tissues maintains hoof moisture at a constant level. Under conditions where hooves are subjected to a continuously wet external environment hoof moisture increases. Perhaps the presence of high surface moisture inhibits the outward diffusion of water, and this constant outward diffusion of water molecules has a protective action. If this were so, treating the hoof surface to make it resistant to water transfer may even be counter-productive.

Our work also confirms that most of the physical properties that one would logically consider to be important in providing hoof with the ability to withstand injury are affected by hoof moisture content. The hardness, elastic modulus, compressive strength and resilience of sole hoof tissue were all negatively correlated with sole moisture levels. Similarly for wall hoof, hardness and elastic modulus were negatively correlated with wall moisture content. However, moisture content did not appear to influence wall compressive strength, and wall resilience increased at high moisture levels.

It is not surprising that hoof hardness and elastic modulus were influenced by hoof moisture content. Hoof keratin is a protein composite consisting of two phases: a fibre phase mainly constructed from long slender α-helical microfibrils, which is cross-linked to an amorphous protein phase (Fraser and MacCrae, 1980). The molecular cross-linking within the keratin produces a stable composite of long, thin fibres embedded in the surrounding matrix. In the absence of water, extensive hydrogen bonding exists between polymers of the matrix phase. This is thought to decrease the mobility of the matrix polymers (Fraser and MacCrae, 1980). When keratin is highly hydrated the matrix lacks much of the secondary bonding it possesses in the dry state. Longer distances occur between cross link positions giving the matrix polymers greater freedom of movement. This results in a reduction in elastic modulus since the secondary cross links are not available to carry load, and greater extensibility due to the ability of the polymers to rearrange under load (Bertram and Gosline, 1987).

In chapter 7 a tentative hypothesis has been proposed to explain how changes in these physical hoof properties during wet weather might render hoof more susceptible to mechanical breakdown. It is likely that additional stresses are exerted during wet weather due to a reduction in the elastic modulus of both wall and sole which must allow the wall hoof to bend away from the sole more readily. Defects in the white line that result from these stresses could then be responsible for the initiation of white line separation.

It was also suggested that the loss of sole resilience that is associated with increased sole moisture levels may increase susceptibility to concussive damage to soft tissues under the sole, thereby increasing the likelihood of sole bruising. MacLean (1971) concluded that it was increased hoof moisture content that was responsible for physically softer horn in animals affected by laminitis. Perhaps a lowering of sole resilience occurs in hooves affected by subclinical laminitis, again due to a higher moisture content, rendering the

underlying tissues more susceptible to concussive damage and to the development of sole ulcers.

It is likely that other physical hoof properties, that may be important when considering modes of mechanical failure that result in foot lesions, are also influenced by hoof moisture content. Bertram and Gosline (1987) demonstrated that the fracture toughness of horse hoof was greater at intermediate levels of hydration than in fully hydrated and dehydrated hoof material. Maximum fracture toughness occurred at a hydration level which is within the range that has been found *in vivo* in equine hoof wall. These authors mentioned that increased stiffness following a reduction in hydration can adversely affect the fracture properties of a material making it brittle.

To protect hoof from changes in physical hoof properties during wet weather, one of the possible approaches would be to reduce water absorption through the external surface of the horn. If this was to be done by means of a surface hoof treatment, consideration would need to be given to the rate of sole wear since the surface hoof layer is continually removed. Our observations of the rate at which grooves that were placed in the hoof bearing surface disappeared in cattle walking twice daily to pasture indicate that sole wear occurs at a rate of 0.3 - 0.6 mm per week. To have any chance of success an external hoof treatment would need to either be applied at frequent intervals (at least once weekly) or be able to penetrate deeply. As indicated above, some forms of surface treatment may even be contra-indicated, if they were to reduce the outward diffusion of water molecules from the corium to the hoof surface.

An alternative approach worthy of future investigation could be some oral or systemic treatment capable of influencing physical hoof properties by altering the structure of keratin protein as it is produced in the corium. Reilly and Brooks (1990) have already reported that biotin supplementation, previously shown to influence the physical properties of pig hoof (Webb *et al.*, 1984), produced significantly harder hoof tissue in cattle. Baggott *et al.* (1988) have also reported that some inorganic elements are related to hoof hardness and variation in some of these appeared to be associated with lameness. It is important that adequate levels of these elements be available for the production of physically sound keratin by providing sufficient levels in the diet. Further, factors that may adversely affect the supply of nutrients to the corium by interfering with normal blood supply need further investigation.

A simple method of measuring hoof moisture in live cows, such as the one used in this work, may be valuable in future investigations of factors that influence physical hoof characteristics. Since many of the physical properties of hoof are correlated with hoof moisture it should receive the major focus of attention. Hoof hardness can also be readily measured but to focus on this property alone would be unwise as clearly a number of different properties are likely to be important.

Unfortunately, resistance to abrasive wear is not simply measured in the live animal. However, it seems logical to assume that hooves which had greater resistance to abrasive wear would be better able to retain concavity of the sole. Our work has shown that sole concavity can be simply and reliably measured and hence may also be worthy of study in future work.

Grossly abnormal claw shape and hoof overgrowth are uncommon in New Zealand dairy cattle except as a result of a previous disorder such as septic pedal arthritis. Presumably this is because regular long-distance walking results in sufficient rates of hoof wear to maintain normal hoof shape. Consequently there is little need for regular hoof trimming and it is rarely practised.

Hoof growth and wear

The influence of age and season on hoof growth and wear was similar in this work to that observed in most previous studies. Rates of hoof growth and wear were greater in 2-year-old than in mature cows and were greater in summer than in winter. Both wall wear and sole wear were greater in lateral digits than in medial digits. A possible reason for this difference between digits is that greater loads tend to be borne by lateral digits (Scott, 1988) and they are subject to greater ranges of loads than are medial digits (Ossent *et al.*, 1987).

Use of an engineer's profile gauge to reproduce the contour of the weight-bearing surface of the digit provided a useful method for objectively measuring sole concavity. Medial digits almost always had greater concavity than lateral digits. Further, lame digits tended to have less sole concavity than non-lame digits providing some evidence that lack of sole concavity is causally associated with occurrence of lameness.

Information on changes in the depth of sole concavity has not previously been available. An unexpected finding of the hoof wear studies described in chapter 6 was the rapid and substantial loss of sole concavity in hind digits following calving in both spring- and autumn-calving cows. This loss of concavity within a short period following calving demonstrates that horn is rapidly removed from the abaxial margins of the hoof bearing surface when hooves are regularly exposed to gravel raceways and concrete yards. It can be concluded that, even if it were possible to modify surface hoof tissue by external application of chemicals, such a measure is unlikely to be successful in preventing lameness purely because the treated horn would be quickly abraded away.

The results of the sole concavity measurements performed on mature autumn-calving cows suggest that during the non-lactating period, if hooves are not exposed to gravel or concrete, they are able to fully regain the normal concavity that is usually observed in young heifers prior to calving. However, even if there is limited exposure to gravel and concrete, as occurred during the non-lactating period in the spring-calving study cows when they were being supplemented with silage, this does not happen. Again this demonstrates that there is most likely little opportunity to reduce wear rates and prevent loss of sole concavity whenever cows are required to walk even moderate distances to pasture.

Risk factors associated with lameness

Risk factors that predispose individual cows to lameness have been the focus of this investigation. Those most likely to be present in pasture-based dairying systems are discussed below.

Cow factors

i) Digit distribution

Our finding that the majority of lameness occurred in the hind legs and that clinical lameness lesions as well as subclinical sole haemorrhage lesions occurred most commonly in the lateral digits of the hind legs is consistent with that reported in previous studies (Russell *et al.*, 1982; McLennan, 1988; Jubb and Malmo, 1991). The most logical explanation for this digit distribution is that hind legs are involved with propulsion and that the lateral digits are subject to greater variation in loads borne

than are medial digits. The relative lack of sole concavity of lateral digits compared with medial digits may also be causally associated with lesion development. It is arguable that digits with flat soles would be less capable of absorbing the stresses of impacts than those with some degree of concavity.

Factors, therefore, that increase stresses on the lateral digits or that are likely to increase hoof wear rates and result in loss of sole concavity are likely to predispose to lameness. These include management factors, such as impatient handling of stock and sudden introduction of new animals into an established dominance hierarchy of other animals, that result in continuous jostling and unplanned foot placement.

In both the case study and the case-control study reported here the distribution of sole abscess lesions fitted a different pattern to that described above, with the medial hind digits being most commonly affected. Unfortunately few conclusions can be drawn because the number of lesions observed was not great. This lesion is presumed to be due to entry of infection through minor defects in the hoof, probably along the white line. One would expect, therefore, a digit distribution similar to that shown by white line lesions. The fact that it appears to have a different digit distribution indicates that different risk factors may be involved in its aetiology. Since it is a commonly observed lesion that results in serious lameness in pasture-fed cattle, this consideration warrants future investigation.

ii) Stage of lactation.

In the case study described in chapter 5, 80% of all lameness occurred in the first four months after calving. This close association between the onset of lameness and time since calving, that occurred regardless of season of calving, has been previously reported (Dewes, 1978; Rowlands *et al.*, 1985; Collick *et al.*, 1989). A number of factors are likely to be involved.

In pasture-fed cattle lameness is uncommon during the non-lactating period as with a minimal requirement for walking along gravel raceways, little time spent in concrete yards and minimal behavioural interaction between animals, few stresses are placed on the feet. At calving, cows are suddenly reintroduced into the milking herd and resume long distance walking to pasture. In herds with concentrated calving patterns calving is usually spread over a two to three month period during which freshly calved

cows are continually added to the milking herd. Early lactation often coincides with wet weather. It also coincides with resumption of oestrous behaviour which involves mounting activity that almost certainly results in extra stresses being placed on the digits of the hind legs.

Later in lactation when the breeding season is over, there is less interference with the normal daily routine of the herd. New cows are rarely introduced, bulls are removed from the herd, and there is less requirement for drafting cows for such management procedures as artificial insemination. In most seasonal supply herds where calving and early lactation coincide with wetter months to take advantage of rapid pasture growth rates, late lactation often coincides with drier weather. Hence many of the factors considered to predispose cows to lameness are no longer present.

iii) Age

A noteworthy finding from the case study described was that lameness was common in two-year-old heifers in a herd where heifers joined the main herd at calving, but the heifer incidence of lameness was low in a second high incidence herd in which heifers were managed separately and had less walking to do. Almost certainly the incidence of lameness would have been higher in the younger cows in the second herd if all cows had been run together as one large mob of 180 cows. This is an example of how segregating the animals that were likely to be low in social ranking in the herd in order to reduce competitive social interactions and to reduce the amount of long distance walking, was most probably successful in reducing the level of damaging stresses to their hooves.

iv) Genetics

The role of genetics in influencing susceptibility to foot lameness in pasture-fed cattle is unknown. A genetic component to lameness susceptibility could be manifest through aspects of claw quality such as claw shape, hoof colour, or physical, chemical or histological characteristics of the horn. However, at present, there are no systems routinely available for objectively measuring any of these traits and, for most of them, their association with claw disorders has not been evaluated. Hence, it remains difficult to assess the influence of genetics on lameness.

Results of the case-control study confirmed that an association does exist between colour of the hoof wall and lameness. Lame digits were more likely to have claws that were less pigmented than equivalent digits on control cows. However, the influence of hoof colour on lameness susceptibility is likely to be small when compared with that of most of the other risk factors discussed here.

Nutrition

Greenough (1986) has suggested that high protein intake could be a risk factor for lameness in New Zealand herds. Such a nutritional aetiology cannot be discounted as the protein content of New Zealand pasture is often very high and the fibre content quite low at the times of the year when lameness is common. However, as explained earlier, there is little epidemiological evidence to implicate these factors. Given the huge variation in lameness incidence that is known to exist between farms and between seasons, it is unlikely that the slight variation that does occur in pasture protein and fibre levels between farms and between seasons has much influence on lameness susceptibility. Other management and environmental factors appear to be more important.

Housing

Since cattle are very rarely housed in Australia or New Zealand the influence of housing on lameness does not need to be considered in this discussion. However, this work does suggest that the nature of yards used to hold cattle during winter may influence claw health. In cows whose feet were examined monthly, heel erosion was most prevalent during winter which was the period when they were heavily stocked on small areas of pasture and frequently kept for long periods in wet, boggy, sawdust yards. This condition is generally regarded as being associated with unhygienic conditions when feet are kept wet, in stalled rather than pastured cattle. Our finding suggests that the prevalence of this lesion is likely to be high in any system where hooves are continuously exposed to wet unhygienic conditions.

Raceway maintenance and herd management

Since with the investigational approach adopted in these studies between herd differences could not be examined, the influence on lameness incidence of raceway design and maintenance and herd management could not be evaluated. However, it is clear from

other studies that these are among the most important factors that influence a herd's, and consequently an individual cow's, susceptibility to lameness in pasture-based dairying systems (Harris *et al.*, 1988; Chesterton *et al.*, 1989; Clarkson and Ward, 1991). These earlier studies demonstrated that variation between herds was associated with environmental and management factors. This study has shown that, while there is variation between cows within herds which is associated with the causal mechanisms for lameness, the risk factors are not susceptible to simple manipulation by currently available control measures at the level of individual animals. The herd factors identified by Chesterton *et al.* (1989) therefore seem a more promising area for immediate progress in lameness control.

Season and rainfall

Our work has given some valuable insights into the effects of wet weather on physical hoof characteristics. As discussed in the findings of the longitudinal study described in chapter 7, there was a clear tendency for hoof moisture to vary in concert with the various measures that were used to assess the wetness of the environment to which cows' hooves were exposed. In addition, most, but not all, of the other physical properties of hoof that were measured were influenced by wet environmental conditions.

The exact mechanisms by which wet weather predisposes cows to lameness have still to be elucidated but a tentative hypothesis has been proposed. It is likely that increased susceptibility to lameness during wet weather is due both to an alteration in physical hoof properties as has been demonstrated to occur in these studies and to a change in the nature of the surface of raceways that cows walk on.

Prevention of lameness

At present the best approach for veterinarians to adopt for lameness prevention in pasture-based dairying systems is to investigate herds as described by Chesterton (1989), and to recommend changes in management procedures and improvements in race construction to reduce the risk factors identified by Chesterton *et al.* (1989). When designing new races or attending to the maintenance of existing ones attention should also be paid to the basic principles of good road construction that have been described

by Bridges (1985a). Aspects of road maintenance that should receive particular emphasis include elimination of congestion points to facilitate "cow flow", use of a non-abrasive material for the race surface, ensuring the width of races is wide enough to allow the herd to move contentedly, and crowning of the road surface to achieve good drainage.

When budgeting for race maintenance, those involved should compare the costs of making improvements with the potential savings that may be achieved by reducing the incidence of lameness. It should be possible to use the production losses and treatment costs associated with lameness that were observed in these studies to assist with estimation of savings that could result from reduced lameness.

Patient animal handling, especially during periods of prolonged wet weather or where resources to upgrade raceways are limited, is likely to remain one of the most effective measures that farmers can adopt to prevent lameness. Segregating cows low in social ranking in the herd and adopting management strategies to minimise the amount of time cows spend on concrete will also be of value in many situations. However, just as farmers often need to be reminded of the importance of teat dipping in the control of mastitis, it is also likely that some will need to be periodically reminded of the benefits of careful stock handling in the prevention of foot lameness. Control strategies for lameness are more likely to be adopted by farmers if they understand the mechanisms of lesion initiation and development. As our understanding of these processes improves it is important that this information be passed on to them.

The complete failure of formalin footbathing in these studies to prevent lameness, reduce the incidence of subclinical hoof lesions or substantially alter hoof moisture, hardness or sole concavity changes has added to the accumulating evidence that it is ineffective as a method of preventing claw-related lameness in dairy cattle under pasture-fed systems. Further, none of the other available external hoof treatments offer promise at this stage.

There is no evidence at present to suggest that altering the diet of New Zealand dairy cattle will influence susceptibility to lameness. However, there has been a recent trend in some Australian herds to feed increasing amounts of concentrates usually in two feeds each day at milking time. In such herds, should the concentrate portion of the diet approach 50% of dry matter intake, nutritional management practices to prevent the

ruminal acidosis syndrome as have been outlined by Weaver (1979, 1988) should be adopted.

Other aspects of lameness control that have received attention in the veterinary literature appear to have little relevance to most dairying systems in Australia and New Zealand. Routine hoof trimming is unnecessary as hoof overgrowth is uncommon; vaccination against interdigital necrobacillosis is unlikely to be widely adopted, even if an effective vaccine was available, as this condition responds so readily to antibiotic treatment, and considerably more information would need to be available on the heritability of claw disorders before any programme of selective breeding to prevent lameness could be considered.

Proposal for pathogenetic mechanisms in foot lameness

Dewes (1978) provided the first description in the veterinary literature of mechanisms involved in the development of foot lameness in pasture-fed dairy cattle in New Zealand. Lameness incidence was highest in 2-year-old heifers and frequently involved excessive wear of the sole. Wear began soon after animals joined the milking herd and lameness resulted when animals walked long distances in wet conditions and after abrasive materials had accumulated on concrete holding yards. He described how time spent in yards, and the amount of movement of animals on abrasive surfaces at each yarding was influenced by the animal's social ranking in the herd and her sequence in the milking order.

In their case-control study of environmental and behavioural factors influencing foot lameness Chesterton *et al.* (1989) compared herds with high and low incidences of lameness. The average maintenance of the farm track and the degree of patience demonstrated by the farmer when handling his cows were the two most influential factors in explaining variation between case and control herds. Use of a biting dog and the percentage of feet that were white in colour were also found to be influential risk factors.

In another study of predominantly pasture-fed dairy cattle, Harris *et al.* (1988) also concluded that features of the farm track were associated with lameness. These included its length, the presence of steep slopes, the type of surface material, presence and

treatment of broken sections and maintenance including rolling history. The feeding of small amounts of concentrates during milking was also associated with a lower incidence of lameness. The authors concluded that this effect was probably mediated by providing a positive incentive for cows to travel contentedly from paddocks to the milking yards and from the yards into the dairy at milking time.

The results of this series of studies has further added to our understanding of the risk factors and mechanisms involved in lameness in pasture-fed cattle. Subclinical hoof lesions were observed with specific claw distributions and at specific times of the year, indicating that factors capable of influencing claw health operate even in herds with a low incidence of lameness. The majority of lameness in herds studied was caused by claw lesions involving white line disease, sole bruising and sole abscess. Studies of physical hoof properties that appeared likely to be important in providing hoof with the ability to protect the sensitive tissues of the foot from infection and traumatic injury were conducted. It was clearly shown that these properties are influenced by wet environmental conditions and suggestions were made as to why changes in these properties are likely to predispose to lameness. Observations of differences in sole concavity between lame and non-lame digits indicate that lack of sole concavity is probably causally associated with occurrence of lameness.

It is very clear, both from the studies described in this thesis and from the evidence in the published literature, that multiple factors interact to produce lameness in pasture-fed dairy cows in Australia and New Zealand. Moreover, these factors and their interactions differ among the various types of lameness. There appear to be risk factors which are present in most herds but operate to varying degrees in different animals because of the variation in the intensity of the influence of each risk factor in various animals. There are also risk factors which act at herd level to produce variation in lameness occurrence between herds. The evidence suggests that at least some of these herd factors are different in nature from the risk factors which operate at the level of individual cows. Thirdly, wet weather appears to exert considerable influence on the clinical manifestation of lameness.

In order to summarise the findings in this thesis and interpret the results in a way which aims to clarify the pathogenesis of clinical lameness, two path diagrams of proposed pathogenetic mechanisms are described in Figures 11.1 and 11.2. The aim of the

diagrams is to put forward proposals as a basis for discussion concerning the various mechanisms. They can also encourage testing of the hypotheses which are implicit in the diagrams. Other researchers can refute or refine the proposals put forward.

Figure 11.1 covers white line disease, plus sole abscess (which is hypothesised to arise from similar processes to those that produce white line disease). It is suggested that all animals are exposed to structural stresses on the white line, which can weaken it and make it susceptible to subclinical white line disease. The extent to which this occurs will be affected by higher than normal levels of activity (oestrus, long distances to grazing etc.) and unusually abrasive ground surfaces. There will also be other risk factors which are intrinsic to the animals (breed, hoof colour, parity etc.) which will also affect the incidence, but are not as easy to manipulate as the extrinsic factors mentioned earlier.

The expression of white line disease will become more likely when wet weather occurs, by increasing hoof moisture content (which adversely affects the physical properties of the hoof) and by exposing abrasive and damaging materials on the surface of races and yards.

Thirdly, poor maintenance of races and impatient handling of stock both create additional stress on the white line as animals move to and from milking, and thus produce much of the variation in lameness incidence found between herds, by exacerbating the incipient lesions caused by the other more universal factors.

Bruising is considered to have the same three causal components, but different specific elements in its causation, as shown in Figure 11.2.

The same group of factors present in most herds as for white line disease operate in bruising, but in this case the effect is structural stress on the sole surface. When these and other factors operate at a mild level then subclinical hoof haemorrhage may result, whereas if they operate more strongly then severe bruising may result and cause clinical lameness. In contrast to subclinical and clinical white line disease, these two are seen as alternative outcomes, not as sequential stages in the pathogenesis of the condition. Whether repeated trauma to a hoof with mild haemorrhage can precipitate severe bruising remains to be determined.

Wet weather exerts similar initial influences to those described for white line disease but in this case the effect is exerted on the moisture content of the hoof and thence on hardness and resilience of the sole, which makes mild or severe solar haemorrhage more likely. Rain also has an indirect effect by making ground surfaces more damaging.

In high lameness herds poor race maintenance and impatient handling put excessive physical stress on the sole when the animal moves on risky surfaces, thus placing the animal far more at risk of suffering bruising severe enough to create clinical lameness.

These proposed mechanisms are consistent with much of the data available for Australia and New Zealand, and are plausible explanations of the epidemiological patterns of lameness seen in the management system used in these environments. Whether or not they are accurate will only be found through future research, which can test their validity.

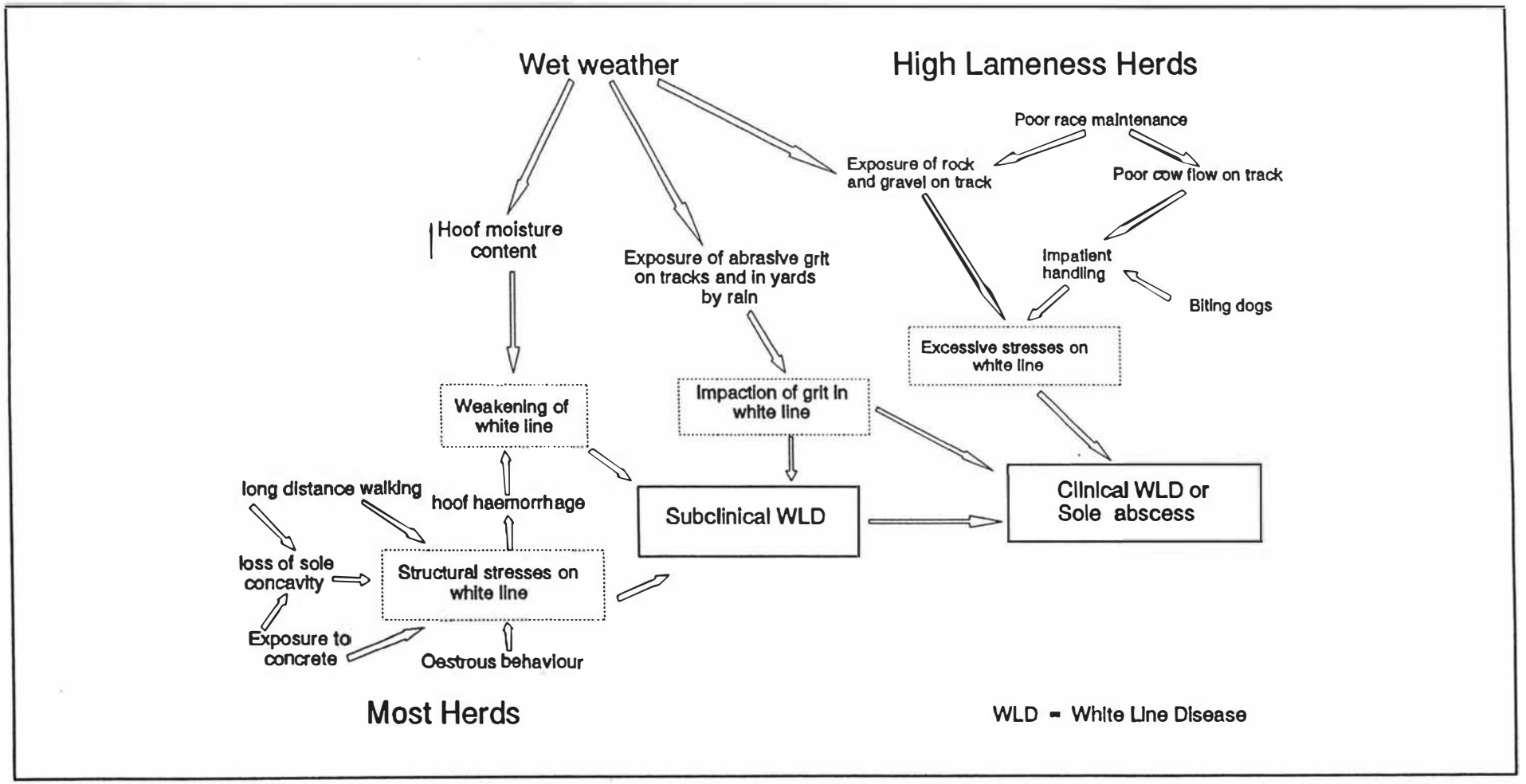

Figure 11.1. A path diagram for white line lesions that shows likely causal links between risk factors that predispose to both subclinical and clinical disease

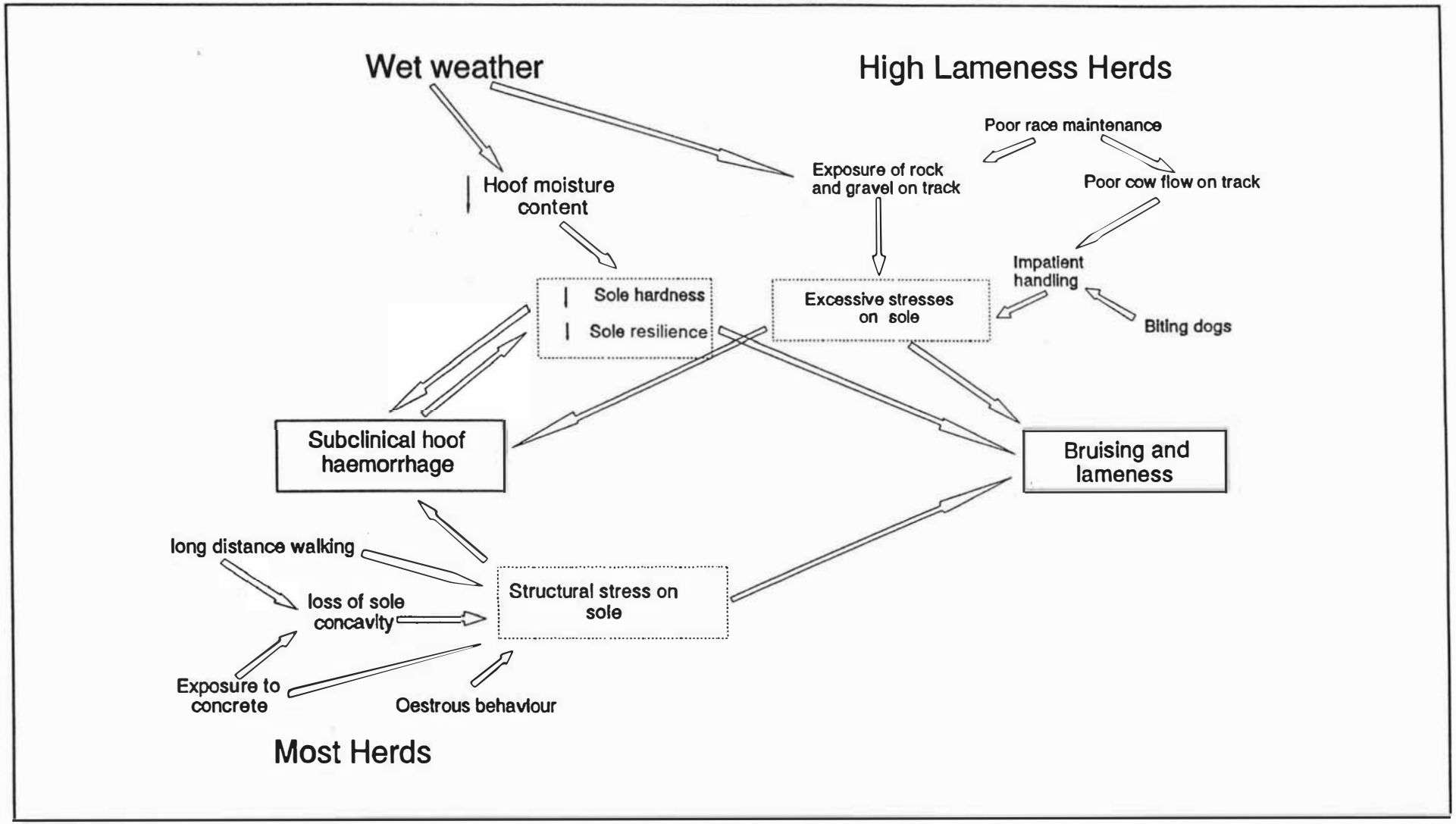

Figure 11.2. A path diagram that shows likely causal links between risk factors that predispose to both subclinical sole haemorrhage and lameness caused by bruised sole

Recommendations for future research

It can be concluded that lameness in New Zealand herds results primarily from adverse environmental conditions, exacerbated by management actions taken by farmers which increase the risk of animals being exposed to injury-causing circumstances. However, each of the various types of lesions are not chance events, but develop in characteristic patterns both between different digits and between various phases of the milking season. There remains scope for exploring methods which would either reduce the susceptibility of hoof horn to the effects of moisture, or would block one of the sequence of steps which leads to the development of clinical lameness. Some areas that should be addressed include:

a) Race construction

Since several studies have now concluded that aspects of race design and maintenance can influence the incidence of lameness, these should receive emphasis in future research. Further work should be undertaken to enhance understanding of the mechanisms by which changes in the nature of the race surface during wet weather predispose cows to lameness. This will help determine which materials are most suitable for use in the surface layers of farm races. There is a continuing requirement for extension activities to encourage farmers to use sound road construction methods when undertaking race maintenance, as has been previously emphasised by Bridges (1985a, 1985b).

b) Yard surfaces

Alternative methods for constructing surfaces for holding yards need to be studied. A number of methods for finishing concrete to provide non-slip surfaces are available (Furphy, 1991) but there is scope for exploring the potential benefits of less abrasive surfaces. One such approach would be to compare sole concavity changes, hoof wear and incidence of claw lesions in cows using yards with and without carpet or rubber matting placed on the surface.

c) Biotin supplementation

The effects of biotin supplementation on hoof integrity in pasture-fed cattle need to be carefully evaluated. Administration should not be difficult since the biotin could be mixed with preventative bloat treatments that cows in the majority of herds receive by

drenching for considerable periods each year over about the same time period as the lameness risk is high.

d) Management change

The effect of modifying management actions of farmers to reduce the specific stresses suffered by cows in high lameness herds could be evaluated through a field trial.

e) Hoof surface treatment

Results of hoof treatments in this research were disappointing, and give little optimism that refinement of currently available hoof treatments would achieve much. However it is possible that some different approach to surface treatment could produce results.

One tentative hypothesis, which arises from this project and which deserves further consideration is that an essential requirement for maintenance of healthy hoof is continuous outward diffusion of moisture through the hoof material from the corium, and that the moisture then evaporates from the hoof surface. This outward flow may be inhibited greatly in wet weather, and this in turn may lower resistance of the hoof to damage. If this is true then current hoof protectants may be of limited value, and new substances which allow this diffusion but protect the hoof from inward flow of water from outside may have to be identified and tested. Whether such an approach is feasible remains to be determined, especially since the high rate of hoof wear found in this study would make it difficult to maintain a surface coating on the hoof sufficient to provide protection.

Conclusions

Earlier epidemiological research on lameness in New Zealand had shown that variation in lameness between herds could be explained by a combination of factors, prominent among them being the maintenance and design of raceways, the degree of patience shown in moving animals along raceways and during milking, and special characteristics of the cows in particular herds. However, this earlier work was not designed to examine this cow-level variation critically.

This project set out to undertake a complementary examination of risk factor variation within herds, in order to build up an understanding of the pathogenetic mechanisms

which led to the development of clinical lameness in particular cows. It was hoped that this would lead to the development of preventive procedures which could be applied to cows in some form, and thus reduce the incidence of lameness.

The various studies reported here have demonstrated the nature of both clinical and subclinical foot lesions in dairy cows, and the impact of clinical lameness on productivity under New Zealand conditions. The scale of the total effects at herd level amply justify the development of better control measures.

Evidence has also been obtained in these studies to show that lameness and foot lesions are predictable in their occurrence among digits and over the course of a year. These findings demonstrate that lameness is not a chance event inflicted on some unfortunate cows, but a consequence principally of the operation of various risk factors which precipitate lesions mainly in cows which in epidemiological terms are at high risk.

Some of the links between these risk factors and the initiation of lesions have been explored by examining the physical characteristics of hooves, and this work helps to explain how risk factors may operate in physiological terms.

Traditional treatment and control methods for lameness have been based more on suppositions about their expected effects than on objective scientific data. A study of two recommended control methods for lameness has failed to demonstrate any effect either on lameness or on hoof characteristics, and likely reasons for the failure can be found in the detailed results of the study.

Of all the major diseases of dairy cattle, lameness is the one for which least preventive action can be taken. Having demonstrated that existing methods are ineffective, the next challenge lies in finding a control approach which modifies risk factors and reduces the occurrence of lameness. On the basis of this research, control by modification of raceway design and animal handling would appear to be the most rewarding option in the short term, although methods for reducing the susceptibility of individual cows remains worthy of further research.

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