DESCRIPTION OF CLAW HORN LESIONS AND ASSOCIATED RISK FACTORS IN DAIRY CATTLE IN THE LOWER FRASER VALLEY, BRITISH COLUMBIA

by

ERIN LEIGH BELL

B.Sc., The University of British Columbia, 1998
B.Ed., The University of British Columbia, 2003

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES

(Department of Animal Science; Animal Welfare Program)

We accept this thesis as conforming to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

August 2004

© Erin Leigh Bell, 2004
Library Authorization

In presenting this thesis in partial fulfillment of the requirements for an advanced degree at the University of British Columbia, I agree that the Library shall make it freely available for reference and study. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by the head of my department or by his or her representatives. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Erin Leigh Bell
Name of Author (please print)

18/08/2004
Date (dd/mm/yyyy)

Title of Thesis: Description of claw horn lesions and associated risk factors in dairy cattle in the lower Fraser Valley, British Columbia

Degree: MSc
Year: 2004

Department of Animal Science; Animal Welfare Program

The University of British Columbia
Vancouver, BC Canada
ABSTRACT

One of the principal causes of lameness in cattle is claw horn lesions on the hoof, but the extent of the problem within Canada is unknown. My aim was to describe the prevalence of claw horn lesions in dairy cattle from the Fraser Valley of British Columbia and identify farm management and environment factors that are most associated with claw horn lesions in this area. I recorded the number, severity, and location of lesions in the claws of 624 Holstein cows from 20 herds during hoof trimming. Lesions were found in cows from all herds. The mean (± S.D.) herd prevalence of cows with at least one claw horn lesion was 85.7 ± 13.8%. Within the individual cow, I found differences in the number of lesions observed on different claws, with the hind lateral claws containing the most lesions. Cows were at greater risk for claw horn lesions at the beginning of their lactation and older cows were more likely to develop severe lesions. Overall, cows with higher body condition scores were less likely to have claw horn lesions than those with lower scores. Farms with high steps, computer grain feeders, narrow free stalls, shallow bedding, automatic alley scrapers, and flooring imperfections had a higher herd prevalence of claw horn lesions. In conclusion, claw horn lesions affect the majority of dairy cows in the lower Fraser Valley, and the risk of development is related to individual cow factors and farm characteristics.
# Table of Contents

Abstract ........................................................................................................... ii

Table of Contents ......................................................................................... iii

List of Figures ............................................................................................... iv

List of Tables .................................................................................................. v

List of Abbreviations .................................................................................... vi

Acknowledgements ......................................................................................... vii

Chapter I  General Introduction 1 ................................................................. 1
  1.1 References ............................................................................................ 6

Chapter II  Description of Claw Horn Lesions in Fraser Valley Dairy Herds 8
  2.1 Introduction ............................................................................................ 8
  2.2 Materials and Methods .......................................................................... 9
  2.3 Results .................................................................................................... 12
  2.4 Discussion ............................................................................................. 17
  2.5 References ............................................................................................ 21

Chapter III  Risk Factors For Claw Horn Lesions in Fraser Valley Dairy Herds 24
  3.1 Introduction ............................................................................................ 24
  3.2 Materials and Methods .......................................................................... 25
  3.3 Results .................................................................................................... 28
    3.3.1 Cow factors ..................................................................................... 28
    3.3.2 Farm factors .................................................................................... 31
  3.4 Discussion ............................................................................................. 34
  3.5 References ............................................................................................ 40

Chapter IV  General Conclusions: Directions for the Future .............. 43
  4.1 References ............................................................................................ 47
LIST OF FIGURES

Figure 1.1 The laminae, papillae, and corium inside the cow's hoof .................................. 2

Figure 2.1 Zones of the sole, according to the recommendations of the VIth Symposium on Diseases of the Ruminant Digit, Liverpool, 1990 .......................................................... 10

Figure 2.2 Lesion prevalence by farm (n=624) ................................................................. 13

Figure 3.1 The relationship between the total number of lesions and body condition score (BCS) (n=624) ...................................................................................... 29
LIST OF TABLES

Table 2.1 Mean (± SED) within cow differences in number of lesions between hind (H) and front (F) claws and between lateral (L) and medial (M) claws (n = 624) ......................14

Table 2.2 Number of lesions on hind-lateral (HL), hind-medial (HM), front-lateral (FL) and front-medial (FM) claws and each of the 5 zones within the claw ........................................15

Table 2.3 Prevalence of other anomalies by farm (percent of cows examined) ..................16

Table 3.1 Range and mean for quantitative descriptors of farm management and environment ...........................................................................................................27

Table 3.2 Cow risk factors for lesions identified using a stepwise regression model (n=361) ...............................................................................................................30

Table 3.3 Farm risk factors for lesions identified using a stepwise regression model (n=20) ..............................................................................................................33
**LIST OF ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCS</td>
<td>Body condition score</td>
</tr>
<tr>
<td>DHIA</td>
<td>Dairy Herd Improvement Association</td>
</tr>
<tr>
<td>DIM</td>
<td>Days in milk</td>
</tr>
<tr>
<td>DM</td>
<td>Dry matter</td>
</tr>
<tr>
<td>FL</td>
<td>Front lateral</td>
</tr>
<tr>
<td>FM</td>
<td>Front medial</td>
</tr>
<tr>
<td>HM</td>
<td>Hind medial</td>
</tr>
<tr>
<td>HL</td>
<td>Hind lateral</td>
</tr>
<tr>
<td>S.D.</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>S.E.D.</td>
<td>Standard error of the difference</td>
</tr>
</tbody>
</table>
ACKNOWLEDGEMENTS

I am grateful to all the dairy producers who participated in this project. I would also like to thank Nelson Dinn and the staff of the UBC Dairy Education and Research Centre in Agassiz, BC; Case Bosch of Bosch Hoof Trimming, Patrick Giroux of Giroux Hoof Trimming, Daryl Emanse of Precision Hoof Trimming and Doug Johnstone of RP Hoof Trimming for their excellent work trimming the hooves of the cows involved in the study; and Frances Flower for her assistance with the on-farm data collection.

I would like to gratefully acknowledge the guidance and support of my committee members, Drs. Dan Weary, David Fraser, Marina von Keyserlingk, and the late Jim Shelford. Their insight, patience, and encouragement over this extended journey was invaluable. In addition, I would like to thank my colleagues in the Animal Welfare Program at UBC for their advice and assistance, especially Paulo Corti for his excellent drawing of the internal structures of the hoof.

Financial support to Erin Bell was provided through a University Graduate Fellowship and a Walter C. Koerner Fellowship. This research was also supported by the Natural Sciences and Engineering Research Council of Canada through the Industrial Research Chair in Animal Welfare, and by contributions from the Dairy Farmers of Canada, the Beef Cattle Industry Development Fund, the BC Dairy Foundation, the BC SPCA, and members of the BC Veterinary Medical Association.
CHAPTER I: GENERAL INTRODUCTION

Lameness is one of the most important welfare issues for dairy cattle. Cattle welfare can be severely affected due to pain experienced by the animal and its reduced ability to function within the herd (Whay, 2002). The relative effect of lameness on the dairy industry is illustrated by the fact that lameness is frequently ranked among the top three most common disorders in dairy cattle (Whitaker et al., 1983; Enting et al., 1997). Costs include treatment, decreased productivity, and increased culling. Guard (2004) has estimated the financial cost of lameness at over 260 $ US per cow.

Corium tissue damage, sometimes referred to as laminitis, is a disease condition of the claw that is a primary cause of lameness (Clarkson et al., 1996). It affects the tissues of the hoof, especially the laminae and papillae (Figure 1.1). The term laminitis means inflammation of the laminae and was first described in horses (Ossent et al., 1997). Although it is not clear what role inflammatory cell components play in the disease in cattle, the same term is still used by some authors to refer to the disturbance in the microvasculature of the corium that results in deterioration of the dermal-epidermal junction (Ossent et al., 1997). Other authors disagree with the use of the term laminitis, referring to the condition by several other names instead, such as disturbed claw health (Somers et al., 2003). As well, some authors make reference only to the resulting lesions, using several names including hemorrhagic lesions, sole hemorrhages, and claw horn lesions (e.g. Webster, 2002). In this thesis I will refer to the condition as corium tissue damage and the resulting lesions as claw horn lesions.
The corium is the highly vascularized dermal tissue found just inside the hoof casing. At the junction of the dermal and epidermal tissues, the tissue of the corium is arranged in folds. The regular folds of tissue next to the hoof wall are referred to as the laminae. The papillae are the irregular folds of the equivalent layer of tissue in the sole of the hoof. The tissues of the corium serve three important functions: they act to absorb the shock created when the hoof impacts the ground; they attach the third phalanx, or pedal bone, to the hoof wall and sole; and finally, they produce the horny tissue of the wall and sole. Anything that disrupts the flow of blood to the corium will result in damage to these tissues and impair their ability to absorb shock or produce high-quality horn. Under extreme conditions the integrity of these tissues may be compromised to such an extent that the pedal bone detaches from the hoof casing.

Figure 1.1. The laminae, papillae, and corium inside the cow's hoof.
There are three forms of corium tissue damage: acute, subclinical, and chronic (Nocek, 1997). The exact etiology and pathogenesis of the condition has yet to be described for cattle. Acute corium damage may occur as a result of ruminal acidosis or a systemic illness in the cow. It is postulated that a dramatic decrease in ruminal and systemic pH leads to increased total blood flow (Nocek, 1997). Endotoxins and histamines released into the blood stream are thought to cause constriction and dilation of the blood vessels as well as circulation of unoxygenated blood through the hoof, although the literature is not clear about the specific actions of these substances (Nocek, 1997). These events result in an increase in blood pressure and seepage through vessel walls, usually culminating in vessel wall damage. This leads to haemorrhage within the hoof and possible swelling of the laminae and papillae between the rigid pedal bone and horn capsule causing pain (Ossent et al., 1997; Nocek, 1997). This stage of corium damage is often associated with sudden lameness.

Subclinical corium damage is the most common form observed in dairy herds (Greenough, 1985; Bergsten, 1994). Unfortunately, this condition often goes undetected because affected animals may not show any obvious signs of lameness. This phase of the disease can result from physical injury to the hoof or from damage sustained during the acute phase (Nocek, 1997). During the subclinical stage there is mechanical damage to the tissues of the foot. Vascular edema leads to local anemia and fewer nutrients reaching the epidermal cells. This leads to a disruption of keratin metabolism in the epidermis with the concomitant production of poor quality horn (Hendry et al., 1997). The horn - the outer casing of the hoof - becomes softer making it more prone to wear and damage and there is seepage from the vessels into the solar corium (Nocek, 1997). At this stage
ulceration, horn erosion, and hemorrhaging can occur in the weight bearing surfaces (Hendry et al., 1997). The wall and sole of the hoof become softer and more prone to wear and damage, and the pedal bone may begin to separate from the wall and sole as the laminae and papillae degenerate (Nocek, 1997). Signs of subclinical corium damage include yellowish discoloration of the sole, haemorrhaging, and sole ulcers (Hendry et al., 1997).

If the disease remains untreated, subclinical corium damage can lead to chronic corium damage. During this stage progressive degeneration of the vascular system and internal structures of the foot occurs, the final outcome being irreversible damage and lameness (Nocek, 1997). The hoof of an animal affected by chronic corium damage is often deformed with characteristic horizontal grooves across the wall.

Many reports on the prevalence of claw horn lesions, mainly from European herds, show that well over 80% of animals are affected (e.g. Bradley et al., 1989; Bergsten, 1994; Bergsten and Herlin, 1996), signifying that this is a widespread problem. However, there is little epidemiological data from North American herds where the intensive nature of the dairy operations often includes different environment and management factors than are found on many European farms. As well, the available evidence related to the environment and management risk factors for claw horn lesions to date is not conclusive (Logue, 2002). For these reasons, there is a real need for more information about the prevalence of claw horn lesions for North American dairy cattle and possible risk factors related to the cows' environment and common management practices.
This thesis describes an epidemiological study with two objectives. The first goal was to determine to what extent claw horn lesions are a problem for dairy cattle in the lower Fraser Valley region of British Columbia, an area of intensive dairy production within North America. The findings related to this objective are covered in the first paper of this thesis (Chapter II). The second goal was to examine management and environmental differences on the farms visited and identify those that pose risks in the development of claw horn lesions. Material related to this second aim is described in the second paper of the thesis (Chapter III).
REFERENCES


CHAPTER II - DESCRIPTION OF CLAW HORN LESIONS IN LOWER FRASER VALLEY DAIRY HERDS

INTRODUCTION

Lameness is one of the most prevalent and costly maladies affecting dairy cattle today. It has been ranked as the third most common disorder, behind mastitis and reproductive failure (Whitaker et al., 1983; Enting et al., 1997). Costs include treatment, decreased milk production, decreased reproductive performance, and increased involuntary culling.

One of the leading causes of lameness is corium tissue damage leading to claw horn lesion development (Collick et al., 1989; Frankena et al., 1992; Bergsten and Herlin, 1996). Lameness estimates vary widely with recent incidence and prevalence rates of 22 to 69 percent reported (Whitaker et al., 2000; Whay et al., 2002; Cook, 2004; Vermunt, 2004). Since claw horn lesions may be present without showing obvious signs of lameness, estimates of the number of cows affected by claw horn lesions are therefore greater, with recent reports ranging from 62 to over 80 percent (Bergsten, 1994; Smilie et al., 1996; Somers et al., 2003). Many authors believe that corium tissue damage, and specifically subclinical corium tissue damage, is the most important affliction of dairy cattle claws (Peterse, 1985; Frankena et al., 1992; Vermunt and Greenough, 1994).

Much of the epidemiological work focusing on lameness in dairy cattle has not differentiated between disorders of the leg or the hoof, nor have they specified whether lameness due to hoof disorders was caused by infectious (e.g. foot rot, fungus) or non-infectious lesions (e.g. claw horn lesions). In general, there is a scarcity of
epidemiological information on claw horn lesions. To my knowledge there has been no research published on the prevalence of claw horn lesions in Canadian dairy operations. The aim of the current study was to measure the presence and severity of claw horn lesions in one North American area of intensive dairy production, British Columbia’s lower Fraser Valley. A secondary aim was to determine which claws and which regions within the claw are most affected by these lesions.

MATERIALS AND METHODS

Animals

This study was conducted on 20 dairy herds in the lower Fraser Valley of British Columbia, Canada, between September 1999 and March 2001. Criteria for herd selection included owner consent and cooperation, indoor housing of the milking herd, a regular hoof trimming routine, and Holstein as the predominant breed of cattle. Sixteen of the 20 study herds were Dairy Herd Improvement Association (DHIA) members. Milking herd sizes ranged from 40 to 340 cows (median 97.5).

The hooves of a total of 624 Holstein cows were trimmed and observed for lesions. On the first 10 farms, the numbers and identities of the animals selected for trimming were determined by the herdsman as part of the farm’s routine hoof trimming schedule. For farms 11 to 20, cows were either randomly chosen or (for 2 farms) the entire herd was trimmed and examined. Three farms from the initial 10 herds were also revisited and a random sample chosen. Where random selection was practised, a minimum of 15 cows was chosen for trimming from each herd. In two of the latter cases
not all selected cows were successfully sorted for trimming so the sample sizes were 14 and 11 cows. All trimming was performed by professional hoof trimmers.

**Hoof lesions**

Each claw was divided into 6 zones (Figure 2.1) according to the recommendations of the VIth Symposium on Diseases of the Ruminant Digit, Liverpool, 1990 (Greenough & Vermunt, 1991). As each claw was trimmed, the presence of any claw horn lesions in Zones 1-5 were observed, their location noted, and the severity scored on a 5-point scale: 1 = slight discoloration, 2 = moderate discoloration, 3 = severe discoloration, 4 = exposed corium (sole ulcer), and 5 = abscessed ulcer (Greenough & Vermunt, 1991). All eight claws were trimmed and examined for each cow. Lesion scoring was carried out by a single observer. The presence of other conditions affecting foot health, such as infectious diseases or imbedded foreign objects, was also recorded.

**Legend**

1) white zone at the toe  
2) abaxial white zone  
3) abaxial wall-bulb junction  
4) sole-bulb junction  
5) apex of the sole  
6) bulb

**Figure 2.1.** Zones of the sole, according to the recommendations of the VIth Symposium on Diseases of the Ruminant Digit, Liverpool, 1990 (Greenough and Vermunt, 1991).
Statistical Analyses

Some previous studies utilising a similar lesion scoring system to that used here have analysed lesion severity by totalling the raw lesion scores (e.g. Bergsten, 1994) or by first adjusting scores geometrically (e.g. Greenough and Vermunt, 1991). These methods attempt to create a number representing an overall severity score that emphasises the more severe lesions. Yet, the biological importance of lesions with lower scores is not clear. In addition, severity scores are on an ordinal scale and thus poorly suited to these mathematical manipulations. For these reasons I chose to simply present the total number of lesions as well as the number of more severe lesions (lesion scores 3, 4, and 5), an approach similar to that of Smilie et al. (1999).

To determine if lesion prevalence differed between farms from which cows were and were not sampled randomly, the average farm lesion prevalences were compared using one-way analysis of variance. Three of the farms were sampled once as part of the farm’s routine hoof trimming schedule and once using my random sample. For these three farms I could perform a more sensitive within-farm test, using a general linear model that first removed the effect of farm (2 d.f.) and then tested the effects of the two sampling methods (1 d.f.) and the farm by sampling method interaction (2 d.f.) among cows. In situations where the same cow was scored on two occasions, only the scores from the second session were included in analyses.

I had no basis for predicting that claws on the left side of a cow should differ from those on the right, so the values from corresponding claws on the two sides of the cow were averaged. However, I did wish to compare how lesion prevalence varied among claws. For example, I wished to determine if hind lateral claws experienced a higher
prevalence of lesions than hind medial claws. To perform these comparisons, values of the various claws were subtracted from one another, resulting in within-cow differences. These differences could then be compared to null expectation (mean of 0) using a one-sample t-test, with an \( n \) of 624. To determine how the total number of lesions per cow compared with the number of severe lesions, these values were correlated using a Spearman’s rank correlation (\( n = 624 \)).

**RESULTS**

Preliminary analyses revealed no differences in overall lesion prevalence (\( F_{1,18}=2.58, P>0.1 \)) or severe lesion prevalence (\( F_{1,18}=0.17, P>0.1 \)) between those farms where cows were chosen randomly and those where the cows were chosen by the herdsman as part of the regular hoof trimming routine. Similarly, for the farms that were sampled twice I found no differences in total lesion prevalence (\( F_{1,118}=0.11, P>0.1 \)) or the prevalence of severe lesions (\( F_{1,118}=2.16, P>0.1 \)), and in neither case was the farm by sampling interaction significant, so these duplicate samples were combined.

Below, I describe a three-tiered approach to the analysis of the hoof lesions: first among cows, then among claws within cow, and finally among zones within the claw. Lesions were found in cows from all herds. Of the 624 cows examined, 84.3% had at least one lesion present in at least one zone. While there was some variation in lesion prevalence among herds (range 53.7 - 100.0%), in 17 of the 20 herds more than 70% of cows had at least one lesion (Figure 2.2). The mean (± S.D.) herd prevalence of cows with at least one lesion was 85.7 ± 13.8%. Severe haemorrhages or ulcers (lesion scores 3, 4, and 5) were found in 29.8% of the cows examined. Fourteen herds had greater than
30% of cows with at least one severe lesion (range 7.3 – 74.1%). The mean (± S.D.) herd prevalence of cows with at least one severe lesion was 34.9 ± 15.1%. Overall, the mean (± S.D.) number of lesions per cow was 3.39 ± 2.96 while on average each cow had 0.56 ± 1.07 severe lesions. The total number of lesions per cow was correlated with the number of severe lesions per cow ($r=0.44, P<0.001$).

<table>
<thead>
<tr>
<th>Farm</th>
<th>All lesion scores</th>
<th>Severe lesions (scores 3, 4, or 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Figure 2.2.** Lesion prevalence by farm ($n=624$) (i.e. percent of cows observed with at least one lesion at the time of hoof trimming).

Within cow, I found considerable differences among claws in the number and severity of lesions. As shown in Table 2.1, cows had more lesions on the hind claws than the front claws. This same pattern is evident considering only the lateral claws (i.e. more lesions on hind lateral claws (HL) than on the front lateral ones (FL)), but for medial claws I found no front-hind difference (i.e. HM and FM). Within the hind claws, cows
had more lesions on the lateral than on the medial surfaces. The reverse was true for the front claws. Overall, the HL claws contained 54.9 % of the lesions followed by the FM (17.7 %), the HM (16.4 %) and the FL (10.9 %).

Table 2.1. Mean (± SED) within cow differences in number of lesions between hind (H) and front (F) claws and between lateral (L) and medial (M) claws (n = 624). P-values are from t-tests ($H_0: \mu = 0$).

<table>
<thead>
<tr>
<th>Lesion Scores</th>
<th>Comparison</th>
<th>Mean difference ± SED</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>All lesion scores</td>
<td>[(HL + HM) – (FL + FM)]</td>
<td>0.72 ± 0.04</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td></td>
<td>(FL – HL)</td>
<td>- 0.75 ± 0.03</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td></td>
<td>(FM – HM)</td>
<td>0.03 ± 0.02</td>
<td>&gt; 0.1</td>
</tr>
<tr>
<td></td>
<td>(HL – HM)</td>
<td>0.65 ± 0.03</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td></td>
<td>(FL – HM)</td>
<td>- 0.12 ± 0.02</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

Severe lesions only
(i.e. score 3 - 5)

<table>
<thead>
<tr>
<th>Lesion Scores</th>
<th>Comparison</th>
<th>Mean difference ± SED</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>All lesion scores</td>
<td>[(HL + HM) – (FL + FM)]</td>
<td>0.14 ± 0.02</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>(FL – HL)</td>
<td>- 0.16 ± 0.02</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td></td>
<td>(FM – HM)</td>
<td>0.02 ± 0.01</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td></td>
<td>(HL – HM)</td>
<td>0.14 ± 0.02</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td></td>
<td>(FL – FM)</td>
<td>- 0.04 ± 0.01</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

I found a very similar pattern when considering only the most severe lesions (i.e. scores 3, 4, and 5), except that the difference between the FM and HM claws was significant in this analysis. Overall, the HL claws contained 62.9 % of the severe lesions followed by the FM (20.0 %), the HM (11.7 %) and the FL (5.4 %).

Table 2.2 illustrates the distribution of lesions among zones within the claw. Zones differed in the total number of lesions, with the greatest number occurring in Zone 4. Of the 2116 lesions observed, 47.1 % were located in Zone 4, 26.8 % in Zone 3, 14.2 % in Zone 5, 7.9 % in Zone 2, and 4.0 % in Zone 1. Once again, I found a very similar pattern of results for the most severe lesions. There were a total of 350 severe lesions
observed with 75.4% located in zone 4, 14.6% in zone 3, 6.6% in zone 5, 2.9% in zone 2 and 0.6% in zone 1.

Table 2.2. Number of lesions on hind-lateral (HL), hind-medial (HM), front-lateral (FL) and front-medial (FM) claws and each of the 5 zones within the claw. Severe lesions refer to lesions of severity scores 3, 4, and 5 only.

<table>
<thead>
<tr>
<th>ZONE</th>
<th>HL</th>
<th></th>
<th>HM</th>
<th></th>
<th>FL</th>
<th></th>
<th>FM</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>Severe</td>
<td>All</td>
<td>Severe</td>
<td>All</td>
<td>Severe</td>
<td>All</td>
<td>Severe</td>
</tr>
<tr>
<td>1</td>
<td>40</td>
<td>1</td>
<td>22</td>
<td>0</td>
<td>16</td>
<td>1</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>74</td>
<td>5</td>
<td>39</td>
<td>1</td>
<td>26</td>
<td>3</td>
<td>28</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>288</td>
<td>32</td>
<td>100</td>
<td>8</td>
<td>73</td>
<td>3</td>
<td>107</td>
<td>8</td>
</tr>
<tr>
<td>4</td>
<td>557</td>
<td>164</td>
<td>159</td>
<td>32</td>
<td>81</td>
<td>9</td>
<td>199</td>
<td>59</td>
</tr>
<tr>
<td>5</td>
<td>203</td>
<td>18</td>
<td>26</td>
<td>0</td>
<td>34</td>
<td>3</td>
<td>38</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>1162</td>
<td>220</td>
<td>346</td>
<td>41</td>
<td>230</td>
<td>19</td>
<td>378</td>
<td>70</td>
</tr>
</tbody>
</table>

In addition to claw horn lesions, I observed several other foot disorders during hoof trimming (Table 2.3). The most common of these was digital dermatitis. Fourteen of the 20 herds were affected, with the prevalence in each herd ranging from 1.8% to 53.3% of the cows examined. Double sole (under-run sole) was also observed in 14 herds with a prevalence range for affected herds of 1.8% to 30.0%. The remainder of the conditions listed occurred in no more than half of the herds visited. Deformed claws were seen on cows from 10 herds. Among affected herds the prevalence ranged from 2.4% to 20.0%. I found rocks imbedded in the hooves of cows from seven herds. Cows in four herds had broken pedal bones in at least one claw. Corns (interdigital hyperplasia) were found on cows from four herds. The least common ailment was foot rot (interdigital necrobacillosis) - this was found in cows from only three herds.
### Table 2.3. Prevalence of other anomalies by farm (percent of cows examined). ('-' denotes that the condition was not present)

<table>
<thead>
<tr>
<th>Farm</th>
<th>n</th>
<th>Digital dermatitis</th>
<th>Double sole</th>
<th>Deformed claw¹</th>
<th>Rock²</th>
<th>Broken pedal bone</th>
<th>Corn³</th>
<th>Foot Rot⁴</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>51</td>
<td>33.3</td>
<td>2.0</td>
<td>3.9</td>
<td>-</td>
<td>-</td>
<td>2.0</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>1.8</td>
<td>3.6</td>
<td>10.9</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1.8</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>-</td>
<td>12.5</td>
<td>-</td>
<td>4.2</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>4.2</td>
<td>20.8</td>
<td>-</td>
<td>8.3</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>-</td>
<td>30.0</td>
<td>-</td>
<td>-</td>
<td>30.0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>29</td>
<td>17.2</td>
<td>-</td>
<td>3.4</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>34</td>
<td>32.4</td>
<td>5.9</td>
<td>-</td>
<td>2.9</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>39</td>
<td>23.1</td>
<td>15.4</td>
<td>2.6</td>
<td>2.6</td>
<td>7.7</td>
<td>-</td>
<td>2.6</td>
</tr>
<tr>
<td>9</td>
<td>56</td>
<td>-</td>
<td>1.8</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>33</td>
<td>3.0</td>
<td>21.2</td>
<td>9.1</td>
<td>-</td>
<td>3.0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>27</td>
<td>29.6</td>
<td>11.1</td>
<td>-</td>
<td>3.7</td>
<td>3.7</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>21</td>
<td>-</td>
<td>-</td>
<td>4.8</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>15</td>
<td>33.3</td>
<td>6.7</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>14</td>
<td>41</td>
<td>19.5</td>
<td>-</td>
<td>2.4</td>
<td>4.9</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>15</td>
<td>95</td>
<td>32.6</td>
<td>19.0</td>
<td>12.6</td>
<td>-</td>
<td>3.2</td>
<td>35.3</td>
<td>5.3</td>
</tr>
<tr>
<td>16</td>
<td>15</td>
<td>6.7</td>
<td>13.3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>17</td>
<td>14</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>18</td>
<td>11</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>9.1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>19</td>
<td>15</td>
<td>13.3</td>
<td>-</td>
<td>13.3</td>
<td>-</td>
<td>6.7</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>20</td>
<td>15</td>
<td>53.3</td>
<td>13.3</td>
<td>6.7</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

All herds: 15.2 ± 16.1 (Digital dermatitis), 8.8 ± 9.0 (Double sole), 3.8 ± 5.5 (Deformed claw¹), 2.0 ± 3.5 (Rock²), 0.9 ± 2.0 (Broken pedal bone), 2.4 ± 6.9 (Corn³), 0.5 ± 1.3 (Foot Rot⁴)

Mean (± S.D.)

Affected herds: 21.7 ± 15.0 (Digital dermatitis), 12.6 ± 8.3 (Double sole), 7.6 ± 7.8 (Deformed claw¹), 5.7 ± 3.8 (Rock²), 4.4 ± 2.2 (Broken pedal bone), 11.9 ± 12.4 (Corn³), 3.2 ± 1.8 (Foot Rot⁴)

Mean (± S.D.)

---

¹Characterised by horizontal ridges on the wall, elongated toe and weight bearing transferred to the heel (also known as 'slipper foot')
²Rock – rock(s) imbedded in one or more claws
³Corn - interdigital hyperplasia
⁴Foot rot - interdigital necrobacillosis
**DISCUSSION**

Hemorrhages of the claw sole can be used as retrospective indicators of claw health (Bergsten, 1994), especially if the severity of the lesions are measured (Frankena et al., 1992; Greenough and Vermunt, 1991). To my knowledge, there has been only one previous North American study using standardised scoring methods to compare a number of herds for claw horn lesions (Smilie et al., 1999).

The mean number of cows per herd with signs of claw horn lesions in the current study was 85.7%, with 84.3% of all animals affected. These results from British Columbia are somewhat higher than the overall prevalence of 62.1% for 13 herds in Ohio, USA (Smilie et al., 1999). European reports show a prevalence of between 75% (Smits et al., 1992) and 85% (Bradley et al., 1989). Few previous epidemiological studies reported the prevalence of cows affected by claw horn lesions. Many focused instead on the prevalence of lameness and the proportion of cases that can be attributed to hoof lesions (e.g. Murray et al., 1996; Russell et al., 1982). Since claw horn lesions can be present without overt signs of lameness, the numbers of animals reported in such studies likely underestimates the true prevalence of claw horn lesions within those populations.

Although the total number of lesions per cow was correlated with the number of severe lesions per cow, the coefficient of correlation was less than 0.5. It is therefore not surprising that herds with a high prevalence of lesions overall did not always have a correspondingly high prevalence of severe lesions. For example, two farms (farms 3 and 9) that were among the most affected for overall lesion prevalence were among the least affected for severe claw horn lesions. Smilie et al. (1999) reported a similar lack of consistency among herds with respect to the prevalence of total and more severe lesions.
These results underscore the multifactorial etiology of claw horn lesions and emphasise the importance of establishing how different lesion severities affect the cow and contribute to lameness.

In the current study I scored the number and severity of lesions on all eight claws for each animal, but other authors have scored lesions on only one front and one rear foot (e.g. Smilie et al., 1996; Smilie et al., 1999) or on both rear feet (e.g. Bradley et al., 1989; Greenough and Vermunt, 1991; Smits et al., 1992). My results can therefore be used to judge the partial samples used in previous work. I found that the hind lateral claws are most at risk for lesions. Other work examining all claws has also found that foot lesions are more common in the hind claws (Bergsten, 1994), particularly the hind lateral claws (e.g. Russell et al., 1982; Murray et al., 1996; Leach et al., 1998). Russell et al. (1982) and Murray et al. (1996) only considered lame animals and also included additional types of lesions other than those involving corium tissue damage, but the greatest proportion of lesions they reported were the result of damage to the corium. Therefore their results still emphasise the vulnerability of the hind feet to these types of lesions. Since the large majority of total and severe lesions in my study (71.3% and 74.6%, respectively) were found in the hind claws it would seem that inspection of these claws can be utilised as a reliable indicator of a cow’s overall hoof health, at least under the free-stall conditions I examined.

The majority of all observed lesions in my study were found in the zones of the sole (Zones 3, 4, and 5). The same was true when only the more severe lesions were considered. Some studies have found more lesions in the zones of the white line (Zones 1 and 2) (e.g. Leach et al., 1998), but others have found results similar to mine (e.g.
Frankena et al., 1992). It is not unexpected that cows housed on concrete should develop more lesions in Zones 3, 4, and 5 because the sole surface of the hooves of these cows is flatter than the concave sole surface of cows kept on pasture. Thus, more weight is likely transferred to the sole in Zones 3-6, as has been found in cows housed on slatted concrete floors (van der Tol et al., 2002). Since the horn of the sole in Zones 3-6 is softer than that of the wall in Zones 1 and 2 (Vermunt and Greenough, 1995), the sole horn may sustain compressive pressure injuries more easily than the wall horn, resulting in more lesions in Zones 3-6. The frequency and method with which the hooves of indoor-housed cows are trimmed will also likely have an impact on the location of pressure points and the subsequent development of lesions.

Digital dermatitis is a relatively common problem in intensive dairy operations (Smits et al., 1992), and is often cited by producers as being one of their top concerns. I found that 70% of herds were affected by this ailment, but the prevalence was lower than the prevalence of claw horn lesions on each farm visited. Even when considering only the more severe lesions there were only three herds where the proportion of cows affected by digital dermatitis was greater than that affected by severe sole hemorrhage or ulcer. The greater visibility, and therefore easier detection, of digital dermatitis compared to claw horn lesions might partly explain the greater awareness by producers.

Broken pedal bones were rare, but cows suffering from this problem were found in 20% of the herds visited. Although the causes of these injuries could not be determined in this study, the welfare implications of this condition may warrant further investigation of possible risk factors. Likewise, rocks in the claw horn, double sole, corns and foot rot
were all relatively rare, with prevalence rates consistent with previous studies (Russell et al., 1982; Enevoldsen et al., 1991; Smits et al., 1992; Nocek et al., 2000).

In conclusion, claw horn lesions affect the majority of cows in lower Fraser Valley free-stall dairy operations. Lesions were concentrated on the sole (Zones 3, 4, and 5) and on the hind lateral claws. The number of animals affected by claw horn lesions far outweighs the number affected by any of the other foot conditions observed.
REFERENCES


22


CHAPTER III - RISK FACTORS FOR CLAW HORN LESIONS IN LOWER FRASER VALLEY DAIRY HERDS

INTRODUCTION

Lameness is one of the three most common disorders affecting dairy cattle (Whitaker et al., 1983; Enting et al., 1997), resulting in increased costs to the producer and decreased welfare for the cow (Whay et al., 1997, 1998). A primary cause of lameness is corium tissue damage leading to the development of claw horn lesions. While estimates of the prevalence of claw horn lesions among dairy cattle vary, there is agreement that the majority of animals are affected (e.g. Bradley et al., 1989; Smilie et al., 1996; Chapter II).

The development of claw horn lesions in dairy cattle can be due to a number of factors (Bell and Weary, 2000). Nutritional aspects, such as high ratios of easily fermentable carbohydrates and protein to fibrous components in the diet, have long been recognised as contributing to the development of claw horn lesions (e.g. Livesey and Fleming, 1984; Peterse et al., 1984; Manson and Leaver, 1989). Yet, cows fed identical diets vary in prevalence of claw horn lesions, indicating that environmental factors also play an important role. Likewise, cows kept in similar environments and fed similar diets can vary in the prevalence and severity of lesions, indicating that management also plays a role in the development (Greenough and Vermunt, 1991; Bergsten and Frank, 1996a, 1996b; Vaarst et al., 1998).
To the author's knowledge there has been only one previous epidemiological study investigating the risk factors for claw horn lesions on North American dairy farms (Smilie et al., 1996). Much of the research examining the factors involved has been conducted in Europe (e.g. Barker et al., 2004), where housing and management systems differ from those commonly used in North America. The aim of this study was to examine cattle in one area of intensive dairy production (the lower Fraser Valley of British Columbia), to determine individual cow characteristics, farm management, and environmental factors that are most associated with claw horn lesions.

**MATERIALS AND METHODS**

**Animals**

This study was conducted on 20 dairy herds in the lower Fraser Valley of British Columbia, Canada, between September 1999 and March 2001. The farm and animal selection was as described in Chapter II. The hooves of a total of 624 Holstein cows were trimmed and scored for lesions. Each cow was also given a body condition score (BCS) on a scale of 1-5 as she left the trimming chute (Ontario Ministry of Agriculture and Food, 1992). Additional data collected for each individual cow included parity and days in milk (DIM). For those herds with Dairy Herd Improvement Association records I also collected the projected milk production at 305 DIM for each cow.

**Hoof lesions**

Each claw was divided into 6 zones (see Chapter II) according to the recommendations of the VIth Symposium on Diseases of the Ruminant Digit, Liverpool,
1990 (Greenough & Vermunt, 1991). As each claw was trimmed, the presence of any claw horn lesions in Zones 1-5 were noted, and the severity scored on a 5-point scale: 1 = slight discoloration, 2 = moderate discoloration, 3 = severe discoloration, 4 = exposed corium (sole ulcer), and 5 = abscessed ulcer (Greenough & Vermunt, 1991). All eight claws were trimmed and examined for each cow. Lesion scoring was carried out by a single observer.

Environment and management

Features of the cows' environment that were investigated on each farm include flooring characteristics and the height of steps within the areas accessed by the cows (Table 3.1). Additionally, I noted if there were any flooring imperfections where cows might injure themselves, such as in large cracks or holes in the concrete (greater than 1 cm deep, 2 cm wide, and 5 cm long). Free stall width and length and the height of the neck rail were also recorded. The bedding in the stalls was classified deep bedded if the depth exceeded 5 cm. All farms had concrete flooring that differed only in the surface treatment (e.g. grooves, slats, or smooth or brushed surface).

Information pertaining to management practices thought to influence hoof health (Bell and Weary, 2000) was gathered for each farm. Specifically, I recorded alley cleaning methods (automatic alley scraper or tractor scraper) and the frequency of routine hoof-trimming (1 or 2 times/year).

Components of the diet and feeding practices thought to influence the development of claw horn lesions were also examined (Bell and Weary, 2000; Table 3.1). I recorded the feeding system (total mixed ration or use of computer grain feeder) and the
number of feedings per day. In addition, I noted the percentage of concentrate and forage in the diet (on a dry matter basis) using farm records.

Table 3.1. Range and mean for quantitative descriptors of farm management and environment.

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Range</th>
<th>Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutrition factors:*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Concentrate in diet (%)**</td>
<td>19</td>
<td>30.20 - 67.40</td>
<td>49.17</td>
<td>9.35</td>
</tr>
<tr>
<td>Environment factors:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average height of steps (cm)</td>
<td>20</td>
<td>0.00-12.30</td>
<td>3.77</td>
<td>4.85</td>
</tr>
<tr>
<td>Number of areas of flooring imperfections</td>
<td>20</td>
<td>0-3</td>
<td>0.65</td>
<td>0.95</td>
</tr>
<tr>
<td>Freestall width (cm)</td>
<td>20</td>
<td>110.00-128.00</td>
<td>118.26</td>
<td>3.82</td>
</tr>
<tr>
<td>Bedding depth (cm)**</td>
<td>20</td>
<td>0.50-15.00</td>
<td>4.36</td>
<td>3.76</td>
</tr>
</tbody>
</table>

*Some nutrition factor values were not available for all farms.
** Eleven farms (55%) used computerised grain feeding systems where cows were allotted their total grain ration for the day in 4-8 separate feedings. Those farms without computer grain feeders fed their cows grain mixed with the forage as a total mixed ration 1-2 times per day.
***Bedding depth for deep-bedded stalls was measured to a maximum depth of 15cm.

Statistical analyses

Lesion prevalence was analysed using both the total number of lesions and the number of severe lesions only (lesion scores 3, 4, and 5), rather than creating an overall lesion severity score for the animals (see Chapter II). Lesion prevalence on farms where cows were chosen randomly and those where the cows were chosen by the producer was compared during preliminary analyses using ANOVA. No differences were found so all farms were included in the analyses reported below. Similarly, for the three farms that
were sampled twice, preliminary analyses compared prevalence during the two sampling sessions. Again, no significant differences were found between the two samples so these were combined for the analyses reported below. In situations where the same cow was scored on both occasions, only the scores from the second session were included in analyses.

I examined prevalence of lesions among cows in relation to the individual cow factors (parity, DIM, and BCS) using stepwise multiple regression, with cow as the observational unit and $P=0.05$ as the criterion for entry or exit from the model. Next, I examined the relationship between herd prevalence measures and the management and environment factors for that herd, again using stepwise multiple regression, with farm as the observational unit and $P=0.05$ as the criterion for entry or exit from the model. In both cases, the risks posed by cow or farm factors were analysed both for the total number of lesions and the number of severe lesions only.

**RESULTS**

**Cow factors**

Of the cows examined, nearly 85% had at least one claw horn lesion while 34% of cows had at least one severe lesion (see Chapter II). The risk of lesions varied with cow parity, days in milk, and body condition, although these effects combined only accounted for a small proportion of the variation in both total number of lesions as well as the number of severe lesions (Table 3.2). The total number of lesions was primarily affected by two cow factors: the stage of lactation (DIM) and the body condition (BCS) of the cow (Figure 3.1). When all claws were considered together, cows later in lactation and
with higher body condition had fewer lesions (0.007 lesions fewer per day in milk and 1.34 lesions fewer per half body condition score).

![Lesions vs Body Condition Score](image)

**Figure 3.1.** The relationship between the total number of lesions and body condition score (BCS) \( (n=624) \).

Examining the claws separately, cows in earlier stages of lactation were more likely to have lesions on their hind lateral, hind medial, front lateral, and front medial claws. Thinner cows were also at risk for lesions on their hind medial and front medial claws. Cows in later parities were more likely to have lesions on their front medial claws than younger cows.
Table 3.2. Cow risk factors for lesions identified using a stepwise regression model (n=361\textsuperscript{a}). The cumulative R\textsuperscript{2} for the model is included after the addition of each significant item (*=P<0.05, **=P<0.01, ***=P<0.001).

<table>
<thead>
<tr>
<th>Lesion Scores</th>
<th>Claw</th>
<th>Risk Factor</th>
<th>Slope</th>
<th>Partial R\textsuperscript{2}</th>
<th>Cumulative R\textsuperscript{2}</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Lesions</td>
<td>All claws</td>
<td>DIM</td>
<td>-0.007</td>
<td>0.08</td>
<td>0.08***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BCS</td>
<td>-1.340</td>
<td>0.03</td>
<td>0.11***</td>
</tr>
<tr>
<td></td>
<td>Hind lateral</td>
<td>DIM</td>
<td>-0.002</td>
<td>0.07</td>
<td>0.07***</td>
</tr>
<tr>
<td></td>
<td>Hind medial</td>
<td>BCS</td>
<td>-0.213</td>
<td>0.04</td>
<td>0.04***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DIM</td>
<td>-0.001</td>
<td>0.02</td>
<td>0.06*</td>
</tr>
<tr>
<td></td>
<td>Front lateral</td>
<td>DIM</td>
<td>-0.001</td>
<td>0.02</td>
<td>0.02**</td>
</tr>
<tr>
<td></td>
<td>Front medial</td>
<td>BCS</td>
<td>-0.184</td>
<td>0.04</td>
<td>0.04***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PARITY</td>
<td>0.043</td>
<td>0.03</td>
<td>0.07**</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DIM</td>
<td>-0.001</td>
<td>0.01</td>
<td>0.08*</td>
</tr>
<tr>
<td>Severe</td>
<td>All claws</td>
<td>PARITY</td>
<td>0.137</td>
<td>0.05</td>
<td>0.05***</td>
</tr>
<tr>
<td>lesions</td>
<td></td>
<td>BCS</td>
<td>-0.312</td>
<td>0.01</td>
<td>0.06*</td>
</tr>
<tr>
<td></td>
<td>Hind lateral</td>
<td>PARITY</td>
<td>0.043</td>
<td>0.03</td>
<td>0.03***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>DIM</td>
<td>-0.001</td>
<td>0.02</td>
<td>0.05*</td>
</tr>
<tr>
<td></td>
<td>Hind medial</td>
<td>PARITY</td>
<td>0.011</td>
<td>0.02</td>
<td>0.02*</td>
</tr>
<tr>
<td></td>
<td>Front lateral</td>
<td>BCS</td>
<td>-0.041</td>
<td>0.02</td>
<td>0.02*</td>
</tr>
<tr>
<td></td>
<td>Front medial</td>
<td>PARITY</td>
<td>0.014</td>
<td>0.02</td>
<td>0.02*</td>
</tr>
</tbody>
</table>

\textsuperscript{a} This sample size is less than the total sample due to missing values for factors on some farms.
The number of severe lesions varied with parity, body condition, and stage of lactation. Overall, the risk of severe lesions increased with increasing cow parity (0.137 lesions per parity) and with decreasing body condition (0.312 lesions per half body condition score). Parity was again a positive risk factor in the development of severe lesions in the hind lateral, hind medial, and front medial claws. Cows early in lactation were also at greater risk for injuries of this type in their hind lateral claws. The front lateral claws were most at risk for severe lesions in cows with lower body condition.

Farm factors

Several farm management and environment factors were associated with a higher prevalence of claw lesions within a herd (Table 3.3). On average, farms with higher steps, narrower free stalls, less bedding material in the free stalls, computer grain feeders, automatic alley scrapers, or more flooring imperfections (large cracks or holes in the concrete) were more likely to experience a higher prevalence of claw horn lesions.

The total number of lesions varied with several farm factors. Looking at the number of lesions on all claws, the factor that posed a significant risk was higher steps in the barn (0.12 lesions per cow for each cm increase in step height). For the hind lateral claws, the risk of lesions increased with step height (0.03 lesions per claw for each cm increase in step height). The risk of lesions on these claws also increased in narrower free stalls (0.05 lesions per claw for each cm narrower), with imperfections in the concrete flooring (0.27 lesions per claw for each large hole or cracked area), and less bedding in the stalls (0.38 lesions per claw for cows on farms with less than 5 cm of bedding). Lesion numbers in the hind medial claws were greater in cows from farms with higher...
steps in the barn. There were no significant risk factors for lesions in the front lateral claws. Lesion numbers in the front medial claws were greater on farms using computer grain feeders and those with higher barn steps.

Fewer farm factors affected the number of severe claw horn lesions. When all claws were considered together, cows from farms that used computer grain feeders and automatic alley scrapers had more severe lesions (0.32 and 0.38 severe lesions per cow, respectively) than those from farms without. When the analysis was limited to the hind lateral claws, the number of severe lesions was again higher on farms using computer grain feeders as well as on those with imperfections in the concrete flooring. There were no significant risk factors for the hind medial claws when considering only severe lesion numbers. The risk of severe lesions in the front lateral claws was influenced by the presence of imperfections in the concrete flooring and computer grain feeders, while the number of severe lesions in the front medial claws varied with the use of computer grain feeders and the depth of bedding in the stalls.
Table 3.3. Farm risk factors for lesions identified using a stepwise regression model (n=20). The cumulative $R^2$ for the model is included after the addition of each significant item (*=$P<0.05$, **=$P<0.01$, ***=$P<0.001$).

<table>
<thead>
<tr>
<th>Lesion Scores</th>
<th>Claw</th>
<th>Risk Factor</th>
<th>Slope</th>
<th>Cumulative $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Lesions</td>
<td>All claws</td>
<td>Height of steps in barn</td>
<td>0.12</td>
<td>0.34**</td>
</tr>
<tr>
<td></td>
<td>Hind lateral</td>
<td>Height of steps in barn</td>
<td>0.03</td>
<td>0.24*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Freestall width</td>
<td>-0.05</td>
<td>0.42*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Imperfections in concrete flooring</td>
<td>0.27</td>
<td>0.57*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bedding depth</td>
<td>-0.38</td>
<td>0.68*</td>
</tr>
<tr>
<td></td>
<td>Hind medial</td>
<td>Height of steps in barn</td>
<td>0.01</td>
<td>0.29*</td>
</tr>
<tr>
<td></td>
<td>Front lateral</td>
<td>None significant</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Front medial</td>
<td>Use of computer grain feeder</td>
<td>0.15</td>
<td>0.54***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Height of steps in barn</td>
<td>0.01</td>
<td>0.64*</td>
</tr>
<tr>
<td>Severe lesions only</td>
<td>All claws</td>
<td>Use of computer grain feeder</td>
<td>0.32</td>
<td>0.40**</td>
</tr>
<tr>
<td>(ie. Scores 3, 4, or 5)</td>
<td></td>
<td>Use of automatic alley scrapers</td>
<td>0.38</td>
<td>0.56*</td>
</tr>
<tr>
<td></td>
<td>Hind lateral</td>
<td>Use of computer grain feeder</td>
<td>0.09</td>
<td>0.31*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Imperfections in concrete flooring</td>
<td>0.09</td>
<td>0.51*</td>
</tr>
<tr>
<td></td>
<td>Hind medial</td>
<td>None significant</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Front lateral</td>
<td>Imperfections in concrete flooring</td>
<td>0.02</td>
<td>0.39**</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Use of computer grain feeder</td>
<td>0.01</td>
<td>0.52*</td>
</tr>
<tr>
<td></td>
<td>Front medial</td>
<td>Use of computer grain feeder</td>
<td>0.03</td>
<td>0.43**</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bedding depth</td>
<td>-0.05</td>
<td>0.63**</td>
</tr>
</tbody>
</table>
DISCUSSION

The prevalence of claw horn lesions varied in relation to features of both the cow and the farm. The multifactorial etiology of claw horn lesions was reflected in my results, although differences among cows explained less of the variation in lesion numbers than farm factors. In general, cows were more likely to develop lesions in the earlier stages of lactation while cows in later parities were more likely to have severe lesions than younger cows. Days in milk was a significant factor in the development of lesions of all severities, but when considering only severe lesions this factor appeared to play a less substantial role. Conversely, parity was a significant factor in the development of severe lesions, but it played a small role in the development of lesions of all severities. Since the prevalence of less severe lesions is greater than the prevalence of severe lesions (see Chapter II), when lesions of all severities are considered together this relationship is primarily driven by the numbers of less severe lesions. Therefore, my results on risk factors for lesions might indicate that all cows are at risk for the development of claw horn lesions at the start of their lactations, and these lesions are more likely to become more severe in older cows during other stages of lactation.

Earlier studies following lesion development in dairy cows have shown similar results. Under experimental conditions, Webster (2001) scored lesions four weeks before calving and four, eight, sixteen and twenty-four weeks after calving, and found that the number of lesions for primiparous cows peaked in early lactation (week 8, 56 DIM). Other studies have also found that lesion scores peak in early lactation for primiparous cows (Leach et al., 1997; Offer et al., 2000), although the exact timing of the peak varies between studies. Few studies have described the development of claw horn lesions in
multiparous cows, but Offer et al. (2000) demonstrated that peak lesion scores occurred at later days in milk for older cows than for primiparous cows, although these peaks still occurred in the earlier stages of lactation. Offer et al. (2000) also showed that the severity of the lesions increased with parity, much as my results suggest. These results support the growing consensus (Manske, 2002; Drendel et al., 2004) that the transition period plays an important role in the hoof health of heifers, and that damage sustained during each lactation is cumulative, leading to the development of more severe lesions during successive lactations.

Some previous work has addressed the relationship between body condition and lameness (e.g. Manson and Leaver, 1988; Manson and Leaver, 1989), but there is little data on the relationship between body condition and claw horn lesions (Webster, 2001). My data shows a relationship between BCS and both the total number of lesions and the number of severe lesions, with thinner animals more likely to have claw horn lesions than fatter cows. My data does not allow me to address cause; low BCS cows may be more likely to develop lesions, or cows with lesions may be more likely to lose body condition. Either way, these effects suggest that producers should be aware that thin cows are more likely to require treatment for claw horn lesions.

Flooring features in dairy barns are thought to influence the development of claw horn lesions in dairy cattle (Bergsten & Frank, 1996; Fankena et al., 1992). My analysis showed that the height of steps in barns contributes to the risk of lesions in all claws, especially the HL, HM, and FM claws, which was similar to that reported by Philipot et al. (1994). It is not clear why steps pose as a risk factor. It may be because they are a potential injury point where cows might slip and bump their hooves, or cows might spend
time standing with two feet on the raised area of the step and two feet on the lower flooring surface, perhaps creating an abnormal pressure distribution among the claws. Whatever the reason, steps appear to increase the risk of lesions and should be avoided when designing cattle housing facilities.

The only other flooring feature I found to be a risk factor for claw horn lesions was flooring imperfections, such as large holes or cracks in the concrete, which are likely sources of injury to the hoof. All farms had concrete flooring, and earlier epidemiological and experimental work has shown that cows experience a higher risk of claw horn lesions on concrete than other flooring (e.g. Bergsten, 1994; Bergsten and Frank, 1996a; Greenough and Vermunt, 1991). It is likely that the presence of flooring imperfections only increases the possibilities for hoof injury on concrete floors. Care should be taken to repair damaged flooring areas in order to prevent injuries possibly leading to lesion development.

Automatic alley scrapers were found to be a risk factor for severe lesions in all claws. The most likely explanation for this is that these manure-cleaning systems are likely sources of injury to the hooves. Cows might injure their hooves on the scraper in their attempts to step over the scraper as it is moving down the alley. For systems with a cable that lies on the concrete alley surface, cows can also injure their hooves on the cable. Automatic scrapers might also benefit hoof health by reducing the likelihood of cows standing in manure (Bergsten and Pettersson, 1992; Fitzgerald et al., 2000) because these systems clean the alleys more often than tractor-scaper systems. Time spent standing in wet conditions is known to reduce hoof hardness (Borderas et al., 2004),
likely making the hooves more susceptible to injury. However, I found no evidence of a positive effect of alley scrapers on sole lesions.

Two stall characteristics were found to be risk factors for the development of claw horn lesions: stall width and bedding depth. Cows on farms with narrower free stalls tended to have more lesions in the HL claws than cows from farms with wider stalls. The link between claw horn lesions and stall width is likely through the animals’ standing and lying behaviour as it relates to stall width. Tucker et al. (2004) found that cows with access to wider stalls spent longer lying down and also increased the amount of time spent standing with all four hooves in the stall. Both of these behaviours might be beneficial for claw health, the first because the cows spend less time standing and the second because more of their standing time is spent on a softer surface (the bedding surface) rather than on the concrete flooring in the alley. As well, this study found that cows increased the amount of time spent standing with only the front hooves in the stall when they had access only to narrower stalls. This standing behaviour has been associated with an increased number of claw horn lesions (Colam-Ainsworth et al., 1989; Galindo and Broom, 2000), and may have contributed to the increased number of lesions seen in my study in cows from farms with narrower stalls. I also found that cows on farms with shallower bedding were more likely to have claw horn lesions in the HL and more severe lesions in the FM claws. Again, claw health may be linked to bedding depth through the cows’ behaviour. In a study on the amount of bedding in free stalls, Tucker and Weary (in press) found that cows spent longer lying down in stalls with deeper bedding, which may help to reduce the number of claw horn lesions. These authors also found that the amount of time spent standing with only the two front hooves in the stall
increased with decreased bedding, a behaviour which could have contributed to the increased number of lesions found in cows from farms with shallower bedding in my study.

The use of a computer grain feeding system was a risk factor for severe lesions on all claws in the current study. However, I failed to find an effect of concentrate level in the diet on lesion number or severity. Though previous studies (e.g. Livesey and Flemming, 1984; Peterse et al., 1984; Manson and Leaver, 1989) have shown that the proportion of dietary concentrates can affect lameness and claw horn lesions, another recent study (Offer et al., 2004) examining the effects of over feeding concentrate on hoof health failed to find an effect on lesion development. In the case of the Offer et al. study, the concentrate was fed as part of a total mixed ration, while the concentrates in the earlier studies were often fed separately from the forage components of the diet, much like the situation on farms using computer grain feeders. Although the current study was not designed to investigate nutritional factors in detail, my results and those of Offer et al. (2004) suggest that the effects of concentrate levels on hoof health may not be as clear-cut as once thought. These results underscore the multifactorial etiology of claw horn lesions and also suggest that the method of feeding grain concentrates might be an important factor in the risk of claw horn lesion development. It may be that feeding concentrates mixed with forages could help to mitigate any negative effects of concentrate proportion within the diet of dairy cows.

In conclusion, the risk of lesions in intensive free stall dairy operations is related to individual cow factors as well as farm environment and management factors. Stage of lactation, body condition, and parity can be used to identify individuals within a herd that
are most at risk of developing claw horn lesions. Farms with high steps, flooring
imperfections, narrow stalls, little stall bedding, computer grain feeders, and automatic
alley scrapers are likely to experience more lesions and should consider changes to these
housing features. Although these conclusions apply only to the sample of farms
considered, I believe that these risk factors are also likely to be important for other free
stall dairy farms with similar environments and management practices to those in the
current study. The type and relative importance of risk factors for those farms using
practices more common in Europe, such as seasonal calving, access to pasture, tie stalls,
and straw yards would likely differ from those of my study. As well, other possible risk
factors, such as flooring type, could not be investigated in this study due to the relatively
low farm sample size and resulting lack of variability, but could prove to be important in
future studies.
REFERENCES


CHAPTER IV: GENERAL DISCUSSION - Directions for the future

My research demonstrates that the prevalence of lesions in British Columbia’s lower Fraser Valley dairy herds is comparable to rates reported in Europe (e.g. Somers et al., 2003) and somewhat higher than that reported in a US study (Smilie et al., 1996). Assuming that the average prevalence for the survey herds (about 85%) applies to herds elsewhere in the province, this implies that lesions likely affect approximately 60,000 dairy cows at any one time in British Columbia (Agriculture and Agri-Food Canada, 2003). It is probable that prevalence is similar elsewhere in North America, at least for cows housed in free-stall conditions. This thesis therefore underlines the importance of finding preventive solutions to the problem of corium tissue damage and the resulting claw horn lesions in North American dairy cattle.

In examining the prevalence of claw horn lesions in the study herds my results showed that at any one time the majority of cows had at least one lesion. Since all of the farms surveyed had concrete flooring that varied only in the surface treatment (e.g. grooves, slats, or smooth or brushed surface), I could not determine the relative lesion risk of concrete compared to other flooring substrates. However, the relatively high prevalence of lesions in cows from my study may, in part, be a confirmation of the overall negative consequence of concrete flooring on hoof health. The idea that concrete flooring might be detrimental to claw health no matter what treatment the surface is given is supported by the recent work of Somers et al. (2003) who found that greater than 80% of cows housed on concrete floors in their study had at least one claw disorder when examined, which was 20-25% more cows than those housed in straw yards. These
authors also found that there was little difference in claw health in cows housed on two different types of concrete flooring – solid floors and slatted floors. However, another recent study (Vokey et al., 2001) found that rubber mats covering concrete alleyways afforded little benefit in terms of preventing claw horn lesions or clinical lameness when compared to grooved concrete, suggesting a need for future research on alternative flooring surfaces for indoor-housed cattle.

In research on the effects of claw trimming, Manske (2002) found that trimming helped prevent lameness and decrease the prevalence of hind-claw lesions in cows housed in tie-stall barns with concrete or rubber, or in free-stall barns with concrete alleys. The cows in the present study were routinely trimmed 1-2 times per year (Chapter II) and yet the large majority of cows had at least one claw horn lesion when examined. This finding points to the need to determine the optimum timing and frequency of trimming for cows. There are many questions that still need to be answered such as, how often should cows be trimmed to gain the greatest preventive effects for claw lesions on specific flooring surfaces? At what age should cattle begin to be trimmed? Are there periods within the lactation cycle when trimming is most effective in protecting claw health? How much difference is there between hoof trimmers in the effectiveness of trimming, and can improved training overcome some of these differences?

This thesis did not investigate the prevalence of claw horn lesions in the heel (Zone 6). Following the example of previous research on claw horn lesions (e.g. Greenough and Vermunt, 1991), only the regions of the white line (Zones 1 and 2) and sole (Zones 3-5) were examined for lesions. However, Blowey et al. (2000) describe finding heel ulcers in Zone 6 that were associated with lameness, but which presented
themselves on the surface of the claw as small (2-3mm) lesions at the junction of Zone 4 and Zone 6. Only after following the track of the lesion to its origin did the researchers discover the existence of heel ulcers in Zone 6 that were resulting in lameness in the affected animals. I therefore recommend that future work also include lesions in Zone 6 in order to create a more complete picture of the hoof health of the animals being investigated. Heel horn erosion is another ailment affecting the heel region that might be of interest in future studies. Although many cattle are affected by heel horn erosion, lameness does not often result from this ailment alone (Weaver, 2000). However, there may be a relationship between heel horn erosion and the subsequent development of claw horn lesions (Weaver, 2000), and the nature and etiology of this relationship warrants further investigation.

The results of this thesis are consistent with other studies (e.g. Offer et al., 2000; Manske, 2002) showing a multifactorial etiology of claw horn lesions. Research on various risk factors such as nutrition, management, and environment has been reviewed (Bell and Weary, 2000; Logue, 2002; Vermunt, 2004). Nutritional factors have long been recognised as contributing to claw horn lesion development (e.g. Livesey and Fleming, 1984; Manson and Leaver, 1989; Offer et al., 2003). Chapter III of this thesis also showed that individual cow traits as well as farm factors such as step height, flooring imperfections, free-stall width, bedding depth, use of computer grain feeders, and the use of automatic alley scrapers can play a role in the development of claw horn lesions. However, the information provided by this thesis, together with other work in this area (Webster, 2001), suggests that individual cow factors such as body condition, parity, and stage of lactation play a smaller role in the risk of lesion development than management.
and environment factors. At this point, it is still not fully understood how nutrition and
the different environment and management features interact with each other to result in
corium tissue damage and claw horn lesions. There is a need for further experimental
work to better understand the pathogenesis of risk factors and their relative importance in
the development of claw horn lesions in dairy cattle.

In summary, I found in my study that prevalence rates for claw horn lesions and
other foot disorders for lower Fraser Valley dairy cattle were similar to those in European
herds, despite some differences in housing and management practices between the two
geographic regions. The claw horn lesions that I examined were concentrated in the zones
of the sole, especially in the hind lateral claws. These lesions were far more prevalent
than any of the other foot conditions that I observed. Producers should be aware that
cows in earlier stages of lactation, those with lower body condition, and older cows are at
greater risk for claw horn lesions. As well, farms with high steps, computer grain feeders,
flooring imperfections, narrow stalls, shallow bedding, and automatic alley scrapers are
likely to experience more claw horn lesions in their herds.

Based on my findings, I recommend that researchers help to educate producers
about the high prevalence of lesions and the possible risk factors. Future research is
needed to find alternatives to the identified environment and management risk factors. In
order to find better alternatives, more experimental work is required to better understand
the relative importance of risk factors and their causal relationship with claw horn
lesions. Although broken pedal bones were rare in my study, the negative impact that this
condition has on affected animals warrants investigation of possible risk factors and
preventive strategies.
REFERENCES


