The Effect of Breed and Crossbreeding on the Incidence of Recorded Lameness in the New Zealand Dairy Cattle

A thesis presented in partial fulfilment of the requirements for the degree of Master of Science in Animal Science

at

Massey University

Palmerston North

New Zealand

Aluna Raphael Chawala

2011

DEDICATION

To my parents

ABSTRACT

The objective of this study was to estimate genetic parameters and breed and crossbreeding effects on incidence of recorded clinical lameness (RCL) in New Zealand dairy cattle. Herd records from 76,357 cows distributed in 155 herds dedicated to the Livestock Improvement Corporation (LIC) sire-proving scheme during the 2005 to 2009 milking seasons were used to estimate the phenotypic, additive genetic, permanent environmental and residual variances of RCL in Holstein-Friesian (F), Jersey (J) and crossbred (FxJ) dairy cattle. RCL incident was coded "1" for cows that had at least one event of RCL at any day during lactation and a "0" code was used for cows without a RCL incidence. Genetic parameters and crossbreeding effects on RCL were estimated with a repeatability animal model, across breeds using restricted maximum likelihood methodology. The predicted mean incidence of RCL per herd was 7.74% with a range of 2% to 34%. Heritability estimate for the incidence of RCL for all lactations (1st to 10th lactation) was 0.016 ± 0.003 (± SE) and the repeatability was 0.071 ± 0.005. The heritability estimate of RCL using only first lactation records was 0.05 ± 0.01 (± SE). Jersey cows had a lower mean (± SE) incidence of RCL than F cattle (J: 6.67 ± 1.50%; JxF: 6.89 ± 1.50%; F: 8.83 ± 1.50%). The effect of first cross (F1) FxJ crossbred was -1.2 ± 0.3% (± SE), representing a mean 16% reduction in RCL compared to an average of the parental pure breed J and F cattle. There was considerable variation (-5 to 8%) between sire estimated breeding values for RCL, suggesting that sire selection across breed and within breed could result in increased genetic resistance to RCL in New Zealand dairy cattle. A significant (P<0.05) reduction in mean (± SD) milk solid yield per day was found in cows with RCL (1.40± 0.0012 kg) compared to cows without RCL (1.43± 0.0045 kg). Cattle in first lactation had a significantly higher incidence of RCL than cows in subsequent lactations (7.6 ± 0.1%, 4.6 ± 0.1%, 5.2 ± 0.1%, 6.3 ± 0.1% and 7.0 ± 0.2% (± SE) for first, second, third, fourth and fifth lactations, respectively). These results suggest that selection for resistance to lameness will result in a low rate of genetic gain but using Jersey sires can be an alternative to increase genetic resistance to lameness in New Zealand dairy cattle.

Keywords: Recorded clinical lameness, crossbreeding, genetic evaluation, dairy cattle.

ACKNOWLEDGEMENTS

I would like to acknowledge the Ministry of Foreign Affairs of New Zealand under NZAID agency for the scholarship that made possible the completion of this thesis. I offer my thanks to many people who made the completion of this thesis possible. Massive thanks to the following people who have contributed to the completion of my research work:

Associate Professor Nicolas Lopez-Villalobos, my main supervisor, thank you very much for your guidance, encouragement, patience, unconditional help and unlimited co-operation during the time of my study and research. You have made a significant contribution to my thesis as you have a long experience in animal breeding and genetics and you gave me constructive comments. Dr. Nicolas you had many ideas that have challenged me to develop and I have learnt a lot from you about genetics and breeding, and statistical analysis but above all human values.

Dr. Jean Margerison, my co-supervisor, thank you for taking time to read my draft of this thesis and for making valuable comments from a very clear mind. I am grateful for all your comments, suggestions and corrections. Your interest in ruminant lameness has led to follow up studies in my thesis and you have been a major source of information during our discussions.

Thanks to Livestock Improvement Corporation Ltd., New Zealand for providing me with the data set. I express my great gratitude to the Institute of Veterinary, Animal and Biomedical Sciences (IVABS) for creating an environment conducive to study. I would like to give thanks to NZAID and International Student Office staff: S. Hooker, O. Pimentel, K. Heagney, N. Banquette, R. Diane among others for your advice, kindness and support. Thanks to Massey staff for your constant support: Prof. Kevin Stafford, Wendy Graham, Debbie Hill and Kathy Hamilton.

Thanks to my fellow post-graduates and friends: Javier Baudracco, Jose Ramirez-Solis, Alfredo Lepori for their active co-operation in my early academic career. I would like to thank Peter Moono for being such a great

flatmate. I extend my thanks to Ps Dale Meacheam, Nick Ling for their spiritual guidance.

Thanks to the government of the United Republic of Tanzania through the Director of National Livestock Research Institute – Mpwapwa for allowing me to pursue my studies.

Bertha my mother and the late Raphael Chawala, my father, for raising me in a good way, I appreciate your love and support. Thanks to my family back home for your love, care and encouragement during my studies. Tulalumba Kidava (Mtegeta), Pili Mahinda, Neema, Joseph, Debora, Yunesi, Anna, Grace, Bryson, Lucy, Andolelye, Tinno and Laban Msigwa.

Lastly, thanks go to everyone who in one way or another has contributed to this study.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

CHL	Claw horn lesion
F	Holstein Friesian
FREQ	Frequency
h^2	Heritability
J	Jersey
JxF	Jersey Friesian cross breed
LIC	Livestock Improvement Corporation
NZ	New Zealand
r	Repeatability
SAS	Statistical Analysis System
SD	Standard deviation
SE	Standard error
RCL	Recorded clinical lameness
UK	United Kingdom
USA	United States of America
WLD	White line disease

CHAPTER ONE

1. GENERAL INTRODUCTION

This chapter presents an overview of the structure of the New Zealand dairy industry and the incidence of lameness in dairy cow herds (section 1.1), which is followed by (1.2 and 1.3) the statement of the research problem and rationale for the study. The chapter concludes with the objectives for this study.

1.1 Structure of the New Zealand dairy industry

Table 1.1. Distribution of dairy cows in New Zealand (NZ) South and North Islands for the 2007 to 2008 milk production season.

	Number of Cows
NZ North Island	
Waikato	1,299,625
Taranaki	475,521
Lower North Island	406,111
Bay of Plenty	328,412
Northland	311,654
NZ South Island	
Marlborough-Canterbury	635,696
Otago-Southland	600,748
West-Coast –Tasman	195,114
Total	4,252,881

Source:(Dairy New Zealand Economic Survey, 2008/2009).

The overall New Zealand dairy cattle population in the 2007/2008 season was 4.252 million, with the majority (over 66%) of dairy herds located in the North Island and the remaining (34%) in the South Island (Dairy New Zealand Economic Survey, 2008/2009). The national herd is composed of a combination of mainly Holstein-Friesian (F) (42.8%), followed by Holstein-Friesian Jersey crossbreeds (F x J) (34.9%) and Jersey (J: 13.8%), with a small proportion (8.5%) of other breeds, which includes Ayrshire, Guernsey, Brown Swiss and their cross breeds. The regional distribution of cow population in the season 2007-2008 is shown in Table 1.1.

In 2007-2008, the mean herd size was 305 cows in North Island and 526 in the South Island. The milk production systems are based on rotational grazing of perennial ryegrass (PRG) and PRG-clover pastures as the main feed base of the diet. This limits production of milksolids production to the amount of pasture grown on the farm (Holmes *et al.*, 2002), but increasingly there are varying levels of feed supplements, which include ensiled forages such as pasture, maize and cereal silages, along with by-products such as palm kernel expeller, corn syrup, molasses, dark distillers grains and other feed waste or surplus materials. The NZ climate allows grass to grow almost all year round and as a result the mean stocking rate is high at 2.8 cows / ha (Dairy New Zealand Economic Survey, 2008). There are seasonal changes in pasture quantity and quality, which are mainly influenced by changes in the four seasons and consistency of rainfall throughout the year. This effect is less marked in areas where pasture irrigation is available. The seasonal changes typically affect the NZ national milk production per cow, which is reduced during dry summers due to decreased pasture and feed availability, lower feeding quality and reduced feed conversion efficiency. This decrease in pasture intake and feed quality is often accompanied by a reduction in the cow body condition score, which sometimes leads to subsequent reduced cow fertility or lower milk production as a result of shorter lactations due to early drying off of lactating cows. However, the effective management of surplus pasture in the spring has been reported to maintain the efficiency of pasture utilization and productivity per hectare (Holmes *et al.*, 2002) to some extent.

The animal breeding goal of the New Zealand dairy industry is to increase farm profit ($) per 4.5 tonnes of pasture dry matter. The amount of pasture dry matter of 4.5 tonnes is a measure of an economic efficiency calculated as genetic superiority or inferiority to convert feed into profit (Lopez-Villalobos and Garrick, 2005). The breeding objective has been described by a list of traits that influence this goal along with a relative economic value for each trait. The current breeding objective combines the genetic merit of each animal in terms of milk production (volume, fat and protein levels), live weight, fertility, somatic cell count score and longevity along with the corresponding economic values (Livestock Improvement Corporation, 2009). The traits included in the breeding objective are those that reflect the profit from milk and beef and the costs of reproduction, animal health and feed (Lopez-Villalobos and Garrick, 2005). However, the improvement of traits related to the economic performance of the dairy industry has brought many challenges, one of which is an increase in the incidence of lameness within the herds (Chesterton *et al.*, 2008; Dewes, 1978; Westwood *et al.*, 2003).

Lameness is described as a disorder in locomotion associated with hoof pathologies caused by genetic, environmental and infectious factors (Baird *et al.*, 2009; O'Callaghan, 2002) along with management of the animal and its environment (Chesterton *et al.,* 1989). The incidence of lameness is considered to be important in terms of the health and welfare of New Zealand dairy cows (Chesterton *et al.*, 2008). Clinical lameness has been associated with poor animal health, lower milk production, reduced fertility and has a negative impact on feed intake (Alban, 1995; Alban *et al.*, 1995). The economic losses associated with lameness include decreased milk production, liveweight loss and increased costs due to animal culling, replacement and treatment (Tranter and Morris, 1991; Warnick *et al.*, 2001).

Some studies completed overseas have assessed the incidence of clinical lameness in dairy cattle under housing management and reported an incidence of lameness ranging between 3 and 57 percent in commercial dairy farms (Espejo *et al.*, 2006; Warnick *et al.*, 2001). In such lactating dairy cattle

factors such as environmental moisture levels, the size of exercise areas and frequency of ration formulation have been associated with changes in the prevalence of lameness overseas (Espejo and Endres, 2007). Moreover, the effect of breed and cross breeding on the incidence of lameness in dairy cattle under housing management systems has shown that Holstein Friesian cows were more prone to lameness than Norwegian cows (Baird *et al.*, 2009), while differences in live weight and milk yield would be contributory factors, as genetic traits related to live weight and stature suggest that dairy breeds that are heavier and taller are more prone to lameness than shorter and lighter breeds (Van Dorp *et al.*, 2004).

Clinical lameness in pasture-fed New Zealand dairy cattle has been reported to be one of the most costly diseases affecting dairy cows (Chesterton *et al.*, 2008; Cue *et al.*, 1996; Diaz-Lira *et al.*, 2009). The disease has been related to nutritional, environmental, climatic and animal factors. While extensive studies on lameness that associated lameness with nutrition and environmental factors have been completed, little has been investigated and quantified regarding the relationship among breeds and crossbreeding on the prevalence of lameness in New Zealand dairy cattle (Lethbridge *et al.*, 2008).

1.2 Statement of the research problem

In New Zealand there have been reports of the differences between dairy cattle breeds and the effect of crossbreeding on milk yield, fat and protein, live weight, somatic cell score, longevity, fertility (Harris, 2005) and incidence of clinical mastitis (Jury *et al.*, 2010). However, there has been very little similar research completed reporting the genetic effect of the incidence of lameness (Chesterton *et al.*, 2008). Several extensive studies on the effect of breed and cross breeding on the incidence of lameness have been reported overseas (Baird *et al.*, 2009; Olmos *et al.*, 2008; Onyiro *et al.*, 2008). However, New Zealand dairy cattle production systems are based on the production of pasture throughout the year, which differs from overseas where pasture production is more seasonal and approximately 83% of dairy cattle

are housed for a significant proportion (0.45 to 0.50) of the year and are often managed in higher feed input milk output systems (Van Arendonk and Liinamo, 2003). Therefore, the current study aims to investigate the effect of breed and crossbreeding on the recorded incidence of lameness in New Zealand dairy cattle under pasture based production systems.

1.3 Rationale for the study

There is a need to reduce lameness in dairy cows for better locomotion, improved animal welfare and reduced environmental impact in order to maintain a good image for the NZ dairy industry. Due to the marketing methods adopted for New Zealand dairy products and increasing consumer awareness regarding animal welfare, consumers are more likely to expect dairy products to be produced by cows that are allowed to graze naturally and that are managed to good standards of animal welfare. Lameness of dairy cattle is one of the main welfare problems for the dairy industry and is one of the main reasons for culling dairy cattle (Tranter and Morris, 1991) following infertility and mastitis. It has been estimated that approximately 39% of the total number of cows that suffers lameness are culled in New Zealand dairy herds (Tranter and Morris, 1991). However, many lame cows are not culled and remain in the national dairy herd. This lameness causes pain, discomfort and behavioural changes in dairy cows, which in turn cause reduced farm profit due to decreased milk production, body condition score and increased infertility, along with increased culling, herd replacement requirements and treatment costs. These costs along with labour requirements for animal treatment make lameness one of the most costly diseases of dairy cattle.

There is a need to quantify the effect of breed and crossbreeding on the incidence of lameness, to identify and contribute to the knowledge regarding factors associated with the occurrence of lameness in New Zealand dairy cattle, and to facilitate the prevention and control of lameness. The information from this study will be used to select sires and design a

crossbreeding system that may reduce the incidence of lameness, increase farm profit, improve animal welfare and enhance the sustainability of New Zealand dairy production systems.

The beneficiaries of the outcomes are dairy farmers, traders, extension officers and researchers. The farmers are the target group as their role is to produce high quality products in larger quantities according to the global market demands. Other beneficiaries include traders who act as intermediaries between farmers and the processing industries. Scientists and extension officers are two groups that will use the knowledge from the findings for further research or take directly to farmers for end use. The findings of this study will lay the foundation for studies in breed and crossbreeding effect on the incidence of lameness in New Zealand dairy cattle.

1.4 Objectives

The general objective of this study was to estimate genetic parameters and evaluate the effects of breed and crossbreeding (heterosis effects in first crossbreed (F1) animals) on the incidence of RCL in New Zealand dairy cattle.

CHAPTER TWO

2. LITERATURE REVIEW

This chapter begins with a definition and general description of lameness, followed by a review of different risk factors associated with lameness. The review focuses on the effect of nutrition, management, production systems and genetic factors on the incidence of lameness. The main theories linking lameness with other risk factors are described and, moving from risk factors contributing to lameness, the chapter concludes by reviewing the direct and indirect economic costs of clinical lameness.

2.1 Lameness

2.1.1 Definition and general description of lameness

Lameness is a physical disablement of a limb that impairs freedom of movement, usually accompanied by pain (Whay *et al.*, 1998) and discomfort (Chesterton *et al.*, 2008; Telezhenko and Bergsten, 2005). Alban *et al.* (1996), explained lameness as the clinical sign of a disease or abnormality of the muscular-skeletal system which impairs normal locomotion. Other authors (Groehn *et al.*, 1992) described lameness as any abnormality in locomotion which was often associated with tissue damage, pain and discomfort. The inability to walk develops from voluntary and involuntary efforts of an animal to reduce the level of discomfort due to injury of the muscles, ligaments, nerves or skin (O'Callaghan, 2002).

Lameness can be categorized as acute or chronic depending on the severity and duration of the healing period (Vermunt, 1992). A cow responds to lameness with different behavioural patterns in attempts to protect the painful limb. Locomotion scoring systems have been used as a subjective technique for monitoring the overall lame condition of individual animals and herds. Farmers and researchers commonly use a numeric scale of five points (1 to 5) to describe the intensity of lameness in dairy cattle (Manson and Leaver, 1988; Sprecher *et al.*, 1997; Tranter and Morris, 1991). Table 2.1 presents the locomotion scoring in dairy cows on a scale of one to five (1 to 5).

Table 2.1. Locomotion scoring criteria in dairy cows.

Locomotion score	Clinical description	Description
Score 1:	Normal	- The cow stands with a level back posture and no abnormality of gait - Makes long confident strides
Score 2:	Mildly lame	- The cow stands with flat back, but develops an arched back while walking - Locomotion is slightly abnormal
Score 3:	Moderately lame	- The cow stands and walks with an arched back and short strides with one or more legs. - Slightly sinking of dew-claws in limb opposite the affected limb may be evident
Score 4:	Lame	- Curved back standing and walking - Favouring one or more limbs but can still bear some weight on them - Sinking of the dew –claws is evident in the limb opposite the affected limb
Score 5:	Severely lame	- The cow shows pronounced arching of back and is reluctant to move, with almost complete weight transfer off the affected limb

Source: (Manson and Leaver, 1988; Sprecher *et al.*, 1997; Tranter and Morris, 1991; Whay, 2002)

These locomotion scoring systems are based on observation of the cow standing and walking (Manson and Leaver, 1988; Tranter and Morris, 1991), with special emphasis on back posture (Sprecher *et al.*, 1997). On these scales, levels 1 and 2 represent a non lame status, while 3 indicates some claw tenderness and four and five represent clinical lameness (Manson and

Leaver, 1988; Tranter and Morris, 1991). A regular scoring of locomotion is regarded as a valuable tool in assessing lameness on dairy farms and routine scoring of the locomotion of dairy herds allows the identification of lame cows, the provision of early diagnosis and treatment and provides information on overall levels of lameness. The awareness of the incidence of lameness on the farm can be used to set targets for the reduction of lameness in individual cows and identify times of seasonal risk (Whay, 2002).

The scoring system in Table 2.1 provides information regarding the development, severity and general description of lameness. However, in order for the collected information to be of greater value, the recording of lameness should include other factors such as types of lesion, the severity and treatment records (Whay, 2002) along with accurate cow identification, date, the claw and area of the claw affected. In more recent work (Lethbridge *et al.*, 2008), the need to apply the international foot map (IFM) when communicating the area and the claw affected has been clarified. The accuracy and reliability on the information regarding lameness depends on the accuracy of identifying and recording lameness type, perception by the observer of degree of severity, and efficient and accuracy of detection. Locomotion scoring can be time consuming to undertake in the whole herd and requires a large area for effective assessment of the animals. The locomotion scoring system allows the assessment of locomotion to be carried out by an observer; however the scoring system gives no indication of the causes of the problem (Sprecher *et al.*, 1997; Whay, 2002).

2.1.2 Clinical lameness

Clinical lameness has been associated with imperfect locomotion, pain and other diseases and was characterised by reduced fertility and milk production from lactating dairy cows (Green *et al.*, 2002). Koenig, *et al*. (2005) divided the cases of clinical lameness into four major types. These are digital dermatitis, sole (CHL) ulceration, wall disorder (white line disease) and inter-digital hyperplasia. Lethbridge *et al.* (2008) described zones 1 and 2 of IFM (Figure 2.1) as regions of high incidence for white line lesions and zones 4

and 5 as regions for sole lesions. However, the causes of clinical lameness can also be divided into infectious and non-infectious.

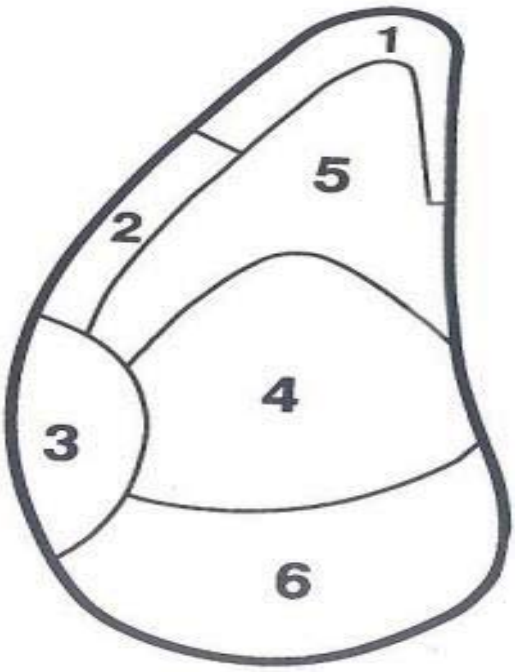

Figure 2.1. Zones of the sole Localization of claw and foot disorders.
Source: (Greenough and Vermunt, 1991).

The non-infectious types of lameness are white line disorder (WHD), claw horn lesions (CHL), heel horn erosion, strains of joints and tendons. WHD includes haemorrhage and separation, where the side wall of the claw separates from the sole, allowing the lamellae to come into contact with harmful substances in the environment, potentially resulting in a secondary infection. CHL are common in cows older than 3 years and have been associated with overgrowth of the claw leading to a frequently diagnosed lesion region in zone 4 and 5 (Greenough and Vermunt, 1991). Heel horn erosion is an uneven loss of horn tissue, that causes a wound deep in the hoof horn which results in lameness (Webster, 2001) related to the heel of the claw.

The most common infectious causes of clinical lameness in dairy cattle are foot rot and digital dermatitis. Dermatitis inter-digitalis is a form of digital dermatitis where the infection starts in the skin and progress in the tissue down to the coronary band. The infection occurs as result of bacteria infecting the soft tissue of the hoof or inter-digital cleft of the claw (Alban *et al.*, 1995). Dermatitis digitalis is another form of digital dermatitis characterised by inflammation and ulceration of the skin bordering the inter-digital spaces that are more or less circumscribed. The two forms of digital dermatitis have been associated with filamentous bacteria in the form of spirochetes (Borgmann *et al.*, 1996) and can cause tissue inflammation which can lead to foot disease such as foot rot (Alban *et al.*, 1995).

In general, clinical lameness of cattle has been considered as an extremely painful condition (Whay *et al.*, 1998) that causes a major welfare problem (Chesterton *et al.*, 2008; Galindo and Broom, 2000; Olmos *et al.*, 2009). The disease results from multi-factorial interactions of mechanical factors, farm management, nutrition and genetic factors (Olmos *et al.*, 2009). The disease results from a variety of lesions which makes it difficult to formulate a simple method for the prevention or control of lameness. This variety of pathogenesis and potential causes results in strategies for disease control that vary depending on incidence level and the type of risk factors associated with the specific type of disease (Chesterton *et al.*, 1989).

Webster (2001) classified the causes of lameness into three groups; claw horn lesions (CHL), skin lesions and non foot damage. The claw horn lesions are caused by disorders such as white line, sole bruising and ulceration and laminitis. The skin lesion causing lameness has been associated with foot rot disease, inter-digital growths and digital dermatitis. The non foot causes of lameness include bone, muscle and joint damage. The studies completed in New Zealand dairy herds have shown that the majority (90%) of all lameness involves the claw (Chesterton, 2006). The major causes of the claw-related lameness were associated with dietary

problems, physical damage, infection and poor foot conformation (Chesterton, 2006).

Studies on the cases of lameness in New Zealand have shown that CHL was responsible for 67 to 81% of lameness (Dewes, 1978; Tranter and Morris, 1991). Case-control studies of risk factors associated with CHL causing lameness in New Zealand dairy farms showed that the most frequently diagnosed types of lameness were white line disease (WLD) (38%), sole lesions (28%), axial cracks (14%), sole ulcer (1%), proximal leg (6%), foot rot (9%) and unknown factors (4%) (Chesterton, 2004; Chesterton, 2006). The WLD disease causing clinical lameness has been associated with long walking distance of larger herds (Gibbs, 2010). The high incidences of sole lesions in New Zealand herds were associated with management and animal factors (Chesterton *et al.*, 1989; Tranter and Morris, 1991; Tranter and Morris, 1992). However, overseas studies have shown parturition and the number of days *postpartum* or days in milk (DIM) to be a significant contributory factor. Current studies (Diaz-Lira *et al.*, 2009) on lameness frequency in the North Island shows that, sole bruising (52%), WLD (22%) and foot rot (13%) are major causes of lameness along with other causes, which included laminitis (8%) and tender feet with no diagnosis (5%). The majority of cases of lameness were reported in hind feet (84%) rather than fore feet (16%) (Diaz-Lira *et al.*, 2009). The most diagnosed causes of lameness in the South Island are WLD (57%), inter-digital and hoof lesion (13%), sole bruising and penetration (11%) and sole ulcer (3%) (Gibbs, 2010).

In the Northern hemisphere the most common lesions causing lameness in dairy cattle were sole ulcer (15.9%), WLD (15.5%), digital dermatitis (67%) and foot rot (62%%) (Laven and Lawrence, 2006). Although the risk factors of lesion are similar to those reported in New Zealand, sole ulcer and digital dermatitis were the most common causes of lameness found overseas. White line disease and sole lesions were the major causes of lameness in New Zealand. The differences in lesion causing lameness between New

Zealand and overseas was associated with differences in management systems. The high incidences of sole ulcer and digital dermatitis in Northern hemisphere have been associated with long periods of housing (Laven and Lawrence, 2006) on hard floor surfaces. This is contrary to New Zealand practice where the majority of animals are not confined in housing.

2.1.3 Incidence of lameness in dairy cattle

Globally, clinical lameness causes serious health and welfare problems in dairy herds (Chesterton *et al.*, 2008; O'Callaghan, 2002; Webster, 2001). A survey completed in New Zealand by Dewes (1978) and Tranter and Morris (1991) showed that the average incidence of lameness in New Zealand dairy herds was 14% with a range between 4 and 54%. The incidence rate varies between herds and locations depending on production systems, nutrition, infection and genetics (Vermunt and Parkinson, 2002). In the United Kingdom, the disease was ranked as the third most important affecting the dairy industry (O'Callaghan, 2002). In Irish pasture-based systems the annual incidence of lameness ranged between 2% and 54% (Olmos *et al.*, 2009). The range in annual lameness incidence rates has been found to be similar within partially housed and pasture-based milk production systems. However, the key factors contributing to lameness frequency in housed systems differs from pasture-based milk production systems (Clark *et al.*, 2007; Olmos *et al.*, 2009; Tranter and Morris, 1991).

In an investigation of lameness in New Zealand dairy herds, Dewes (1978) showed that heifers had a higher incidence of lameness compared to older cows. Similar findings on differences in lameness incidence rates between age groups have been reported (Chesterton *et al.*, 2008; Tranter and Morris, 1991). However, the two findings did not show the possible causes of the higher incidence rates of lameness in first lactation heifers compared to older cows in subsequent lactations. Greater proportions of lameness in New Zealand have been associated with white line disease (Chesterton *et al.*, 2008) sole injuries and foot rot (Tranter and Morris, 1991). Current studies have reported breed differences in the incidence of CHL resulting in

lameness (Chesterton *et al.*, 2008; Diaz-Lira *et al.*, 2009). Holstein Friesian cattle have appeared to be more susceptible to white line disorders than Jerseys (Chesterton *et al.*, 2008; Diaz-Lira *et al.*, 2009). In contrast, Jersey cows had higher levels of sole injuries, axial lesion and foot rot compared to Holstein Friesian cows (Chesterton *et al.*, 2008). Chesterton *et al.* (2008) also provided a broad overview of different lesions causing lameness and the associated factors, which affect the incidence of lameness. The previous findings suggest further research to identify risk factors associated with lameness and to quantify the impact of crossbreeding on the incidence rate of lameness in New Zealand dairy cattle (Chesterton *et al.*, 2008).

2.2 Factors affecting incidence of lameness

Lameness results from various predisposing factors, including environmental (Chesterton *et al.*, 1989; Dewes, 1978), management (Beusker, 2007; Chesterton *et al.*, 2008), nutrition (Westwood *et al.*, 2003; Westwood and Lean, 2001) and genetic factors (Alban, 1995; Baird *et al.*, 2009; Diaz-Lira *et al.*, 2009). These factors, separately or in combination with other factors, may result in complex and severe lameness (Sauter-Louis *et al.*, 2004). The potential factors causing lameness are discussed in detail in this section.

2.2.1 Management

Animal management is an important human factor that influences the well being of the cow and is considered to be an important factor in the occurrence of lameness in pastoral milk production systems (Chesterton, 2004). A lack of knowledge of the risk factors, incorrect diagnosis, clinical approach and treatments can lead to inadequate preventive measures being taken and play a major role in the occurrence of the disease (Greenough, 1997). In addition, education of farmers, stockpersons and technicians can play an important role in controlling the disease (Rama and Veterinarian, 2006).

Chesterton *et al*. (1989) investigated the environmental and behavioural factors influencing claw lameness in New Zealand dairy herds. In this case study, it was found that the major factors contributing to lameness were movement of animals to the milking shed (40%), characteristics of milking process (24%) and cow factors (9.5%). Some animal management characteristics such as patience of farmer behind cows and maintenance of the main track accounted for 21.7% and 13.5% of lameness prevalence levels, respectively. The risk factors associated with the milking process were space in the collecting yard, patience of farmer in the shed and ease of movement of cows in milking shed/parlour. The use of biting dogs, poor maintenance of the tracks and cow flow on track were other factors that influenced the high levels of prevalence of lameness in dairy cow herds (Chesterton *et al.*, 1989; Sauter-Louis *et al.*, 2004).

Hoof trimming is among the management practices used by farmers in housed cows to maintain hoof health, but in grazing systems this is not frequently practised. Vermunt and Greenough (1995) reported a mean monthly hoof growth rate of about 5 mm on the dorsal and abaxial surfaces of hind lateral claws in various age groups of dairy cattle. If the claw wear is less than the growth, the overgrown part can lead to incorrect weight bearing and hence increased pressure on the corium. Claw trimming is considered to be beneficial, as it can significantly reduce lameness by preventing disease development (Burgi, 2000). Correct claw length and angle allow a cow to have a good balance and equal distribution of weight between the claws. Hoof trimming is beneficial as it alleviates the injuries that might cause inflammation that can develop into sole ulcers which are the most common cause of lameness in dairy cows (Burgi, 2000; Greenough, 1997; Manske *et al.*, 2002).

The use of footbaths is being practiced as a technique for reducing reservoirs of organisms on the inter-digital skin of the claw. Traditionally, footbaths are placed at the door of the milking parlour to allow animals to clean their feet before or after entering the parlour (Chesterton *et al.*, 1989; Greenough,

1997). However, under New Zealand conditions the benefit of using footbaths as a means of controlling lameness is debatable (Vermunt and Parkinson, 2002).

2.2.2 Effect of herd size on incidence of lameness

Over the past decades, the herd size of the New Zealand dairy herds has been increasing while the herd number has decreased (Figure 2.2). The number of herds has declined from 17,000 in 1978/79 season to approximately 11,500 herds in 2008/2009 season. The average herd size has increased from 120 cows in 1978/1979 season to about 366 cows in 2008/2009 (Dairy New Zealand Economic Survey, 2008). The dairy cow production system has changed from a smaller herd size to larger, to meet the demand for higher management intensity. Larger herd sizes reduce the space requirement per cow and as a result competitive social behaviour is expressed by animals, which can lead to damage of the feet (Chesterton *et al.*, 1989).

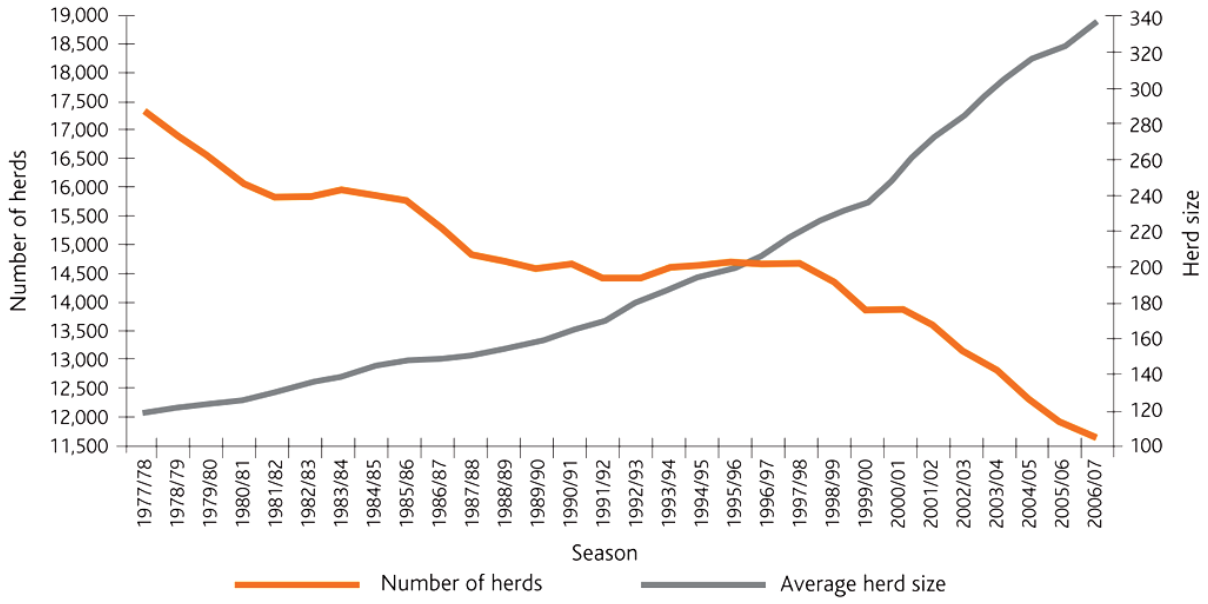

Figure 2.2. Trend in the number of herds and average herd size for the last 30 years.

Source: (Livestock Improvement Corporation, 2007).

The increase in average number of dairy cows per herd showed in figure 2.2 above explains the current incidence of lameness in New Zealand dairy farms. Studies shows that the mean incidences of lameness are higher (26%) in larger herds in South Island (Gibbs, 2010) compared to 14 -16 % incidences reported in smaller herds in North Island (Dews, 1978; Tranter and Morris, 1991). In addition, Chesterton *et al*., (1989) reported fewer incidences of lameness in smaller herd sizes compared to larger herds in New Zealand dairy farms. A similar observation was reported by Alban (1995) in Danish dairy herds. This illustrates the similarity of the effect of herd size in disease distribution in housed production systems in Denmark and in pasture-based systems in New Zealand. Under intensive housing conditions, higher incidence of lameness in larger herds was associated with more mechanised procedures in larger herds than in smaller herds. An increase in the degree of mechanization may reduce the time the farmer spent with each cow which may lead to poor detection of lameness in its early stages (Alban, 1995).

2.2.3 Effect of intensive systems versus grazing systems on lameness incidence

Several studies have reported higher incidence of lameness in confined cows compared to grazing cows (Arvai, 2009; Clarkson *et al.*, 1996; Olmos *et al.*, 2009). The effects of production systems on lameness incidence have been described by many authors (Alban, 1995; Chesterton *et al.*, 1989; Vermunt and Greenough, 1995). Olmos *et al*. (2009) noted a lower level of hoof disorders and higher incidence of lameness for pasture based dairy cows compared to cubicle/free stall housed dairy cows. The average total lying period was shorter for confined dairy cows relative to cows grazing on pasture. The lower prevalence in clinical lameness in grazing systems may be due to undisrupted lying down which improves cow welfare in terms of lameness compared to confined systems. It is believed that a longer lying time is a good indicator of animal welfare, because it increased cow comfort and that may lead to reduced hoof disorders (Olmos *et al.*, 2009).

The majority of the risk factors leading to lameness in housed systems are associated with housing (Boyle *et al.*, 2008; Olmos *et al.*, 2009). Among the pasture based cows in New Zealand the annual incidence of lameness ranging from 4 to 54 percent of the herd has been linked to poor roadway surfaces, long walking distances to milking parlour, poor herding skills and type of feed (Tranter and Morris, 1991). A study conducted in the South Ireland showed that the prevalence of clinical lameness was 17% in pasture based system and 61% in housed systems (Olmos *et al.*, 2009). The average duration of lameness was reported to be lower in pasture systems (35 days) against 40 days among confined cows (Olmos *et al.*, 2009). The prevalence of clinical lameness for pasture cows tends to vary from one location to another (Olmos *et al.*, 2009; Tranter and Morris, 1991), but studies indicate that clinical lameness is higher under confinement systems (Clarkson *et al.*, 1996; Olmos *et al.*, 2009).

2.2.4 Nutrition

Several authors (Manson and Leaver, 1988; Rama and Veterinarian, 2006; Vermunt, 1992) have described the relationship between nutrition and the problem of laminitis in dairy cows. Metabolic disorders such as rumen acidosis have been associated with laminitis and are a major risk for lameness. Theories linking rumen function and lameness depend on the milk production system and the type of feeds used. Studies show that dietary factors associated with lameness vary between pasture and cereal-based diets. This implies that the rumen function in pasture based diets differs from that of cattle offered cereals. There is a minimum level of fibre in the diet which is necessary for maintaining ruminal pH within the normal range (pH of 5.5 to 7.2) (Sutton *et al.*, 1986). Sub-acute ruminal acidosis usually occurs at pH of 5.5 while the pH of 5 cause acidosis and the animal is more susceptible to laminitis below these critical pH values. The incidence of ruminal acidosis in intensive production systems is highest in the first months after parturition and declines rapidly within three to four months post calving (Nocek, 1997). The high incidence of acidosis following calving has been related to poor adaptation to high levels of concentrate feeds for cows accustomed to higher fibre diets. In New Zealand, the incidence of acidosis in

cows grazing pasture has been associated with sugar-based diets such as molasses and, corn syrup which are highly fermentable in the rumen, thus lowering rumen pH. However, it is debatable if nutrition is a problem causing lameness in New Zealand due to lack of local research in this area (Gibbs and Laporte, 2006).

2.2.5 Crossbreeding and lameness

The Holstein-Friesian breed has been regarded as the most important breed for milk production for many years, but has been reported to have lower rates of fertility, survival and longevity (Harris, 2005). In order to maximize the traits of low heritability, crossbreeding to exploit heterosis has been regarded as an alternative for improving these traits (Montgomerie, 2005). Crossbreeding under specific production environments requires a long term strategy and a higher return is achieved when the production per cow is maximized (Lopez-Villalobos and Garrick, 2005).

Several New Zealand dairy farmers are using selection and crossbreeding as main strategies for genetic improvement. The history of the New Zealand national herd has changed from the predominantly Jersey cows in 1960s to Holstein- Friesian and the crossbred of the two pure breeds. Crossbreeding for increasing productivity occurred from the 1980s as New Zealand breeding companies introduced new Holstein genes from North America (Harris, 2005; Montgomerie, 2005). Since then, the genetic background of lameness has not been addressed fully, as genes coding for high milksolids production do not necessarily code for healthy feet. The current scoring systems have not yet concluded whether genetics plays a role in dairy cow lameness (Olmos *et al.*, 2009; Vermunt and Greenough, 1995).

For economic reasons, farmers have selected animals in favour of increased milk yield rather than animal welfare (Harris, 2005; Van Dorp *et al.*, 2004). Phenotypic cow characteristics related to lameness such as colour of the hoof sole, claw angle and inner structure of the claw have not been widely

used as criteria for selecting animals in the New Zealand dairy industry (Harris, 2005). Greenough (1997) reported a significant effect of claw traits in the prevalence of lameness and life time performance of dairy cows. Several authors have suggested the use of parents with good legs, feet and rump conformation such foot angle and rear legs width in breeding programmes (Boettcher *et al.*, 1998; Vermunt and Greenough, 1995). Worldwide, selection indices for cow health have focused on mastitis and fertility and little has been done on lameness (Harris, 2005; Miglior *et al.*, 2005).

2.3 Crossbreeding and heterosis

The main objective of crossbreeding is to utilize the advantage of hybrid vigour or heterosis. Hybrid vigour or heterosis is "the amount by which the phenotypic performance of the first generation cross (F1) exceeds the mid parent performance" (Dickerson, 1972; Gregory *et al.*, 1978). However the phenomenon does not necessary mean that the crossbred offspring will have increased performance over the most superior of the two straightbred parents (Cundiff, 1970).

The level of heterosis is highly marked when two breeds of different ancestry are crossed. This means that the greater the difference between two breeds, the greater is the hybrid vigour revealed by the crossbred animal (Dickerson, 1972; Simms *et al.*, 1990). It is believed that the extra performance of the crossbred animal is the improvement of the production losses that occurred through inbreeding in the parent breeds. The amount of heterosis that is maintained in a herd depends on the type of crossbreeding systems. Some mating plans have been found to reduce hybrid vigour while some do maintain high levels of hybrid vigour. Subsequent backcross of the crossbred animal to one of the parental breed will reduce the amount of heterosis expected due to increased level of inbreeding which reduces hybrid vigour. Inbreeding has been found to fix the homozygous gene pair which results in reduced performance in the newly born offspring. Often the high expression

of heterosis is found between the *Bos indicus* and *Bos taurus* as they do not share recent common ancestors (Dickerson, 1972; Gosey, 2005).

Traits of higher heritability can be passed to the offspring by applying selection pressure on a certain trait. This means, the traits of low heritability will respond poorly to selection and therefore improvement of the trait through selection is difficult. However, traits with low heritability mostly respond well through crossbreeding (Gosey, 2005). Therefore heterosis is more important for reproductive, survival and health traits which have low heritability and which respond poorly through selection (Handley *et al.*, 2001).

2.4 Estimation of crossbreeding parameters

Crossbreeding parameters can be estimated by least squares techniques (Searle, 1982). The general linear model in matrix notation for estimation of crossbreeding information can be represented as follows:

$$\mathbf{y} = \mathbf{X\beta} + \mathbf{e} \qquad [1]$$

where **y** is a vector of phenotypic observations, **β** is the vector of crossbreeding parameters, **X** is a matrix relating the observations in **y** with the parameters in **β** and **e** is the vector of residual error. The generalised least squares estimator of **β** can be calculated by solving the following least squares equations:

$$\boldsymbol{\beta} = (\mathbf{X}'\mathbf{X})^{-1}\mathbf{X}'\mathbf{y} \qquad [2]$$

where $\mathbf{X}'$ is a transpose matrix of **X** and $(\mathbf{X}'\mathbf{X})^{-1}$ is the inverse of the product of matrix **X** and its transpose.

2.4.1 Numerical example for estimating crossbreeding parameters

A numerical example for estimation of crossbreeding parameters is demonstrated below. In this example, five breed groups of cows are presented. The dependent variable is measured as one (1) representing

cows that presented clinical lameness and zero (0) for healthier cows. The breed composition of different breed groups is presented in Table 2.2. This example considers two pure breeds of F and J, where the mating of the two breeds produces the first crossbred group (F x J). The back-crossed breed groups are produced by mating first crossbred cows to F bulls (3/4F 1/4J) or J bulls (1/4F 3/4J).

Table 2.2. Example of lameness incidence[1] for five different breed groups[2] of dairy cows.

Breed group				
F	3/4F 1/4 J	1/2F 1/2J	1/4F 3/4J	J
1	1	1	1	1
1	1	0	1	0
1	1	0	0	0
1	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0
0	0	0	0	0

[1] "1" for cows that had at least one event of lameness at any day during lactation and "0" for cows without an event of lameness.

[2] F = Holstein-Friesian and J = Jersey.

A linear model representing the data from Table 2.2 can be as follow:

$$y = \beta_0 + \beta_1 g_J + \beta_2 h_{FxJ} + e \qquad [3]$$

where **y** is the response variable (1 or 0), β_0 is an intercept (or breed used a reference), β_1 is the regression coefficient of proportion of J on incidence of lameness (the estimate of breed effect, g_J), β_2 is the regression coefficient of breed heterozygosity (h_{FxJ}) on incidence of lameness (the estimate of first cross heterosis) and e is a residual error.

The coefficient of breed heterozygosity can be calculated using a simplified formula suggested by Dickerson (1973):

$h_{ií} = (\alpha^{s}_{i} \times \alpha^{d}_{i}) + (\alpha^{s}_{í} \times \alpha^{d}_{i})$

Where, $h_{ií}$ = breed heterozygosity between breed i and í

α^{s}_{i} = proportion of genes of breed i in the sire

$\alpha^{s}_{í}$ = proportion of genes of breed í in the sire

$\alpha^{d}_{í}$ = proportion of genes of breed í in the dam

α^{d}_{i}= proportion of genes of breed i in the dam

The data in Table 2.2 are presented in matrix form following model [3] as follows:

$$\begin{bmatrix} 1 \\ \vdots \\ 0 \\ \vdots \\ 1 \\ \vdots \\ 0 \\ \vdots \\ 1 \\ 0 \\ \vdots \\ 1 \\ \vdots \\ 0 \\ \vdots \\ 1 \\ \vdots \\ 0 \end{bmatrix} = \begin{bmatrix} 1 & 0 & 1 \\ \vdots & \vdots & \vdots \\ 1 & 0.25 & 05 \\ \vdots & \vdots & \vdots \\ 1 & 0.5 & 1 \\ \vdots & \vdots & \vdots \\ 1 & 0.75 & 0.5 \\ \vdots & \vdots & \vdots \\ 1 & 1 & 0 \end{bmatrix} \begin{bmatrix} \beta_0 \\ \beta_1 \\ \beta_2 \end{bmatrix} + \begin{bmatrix} e_1 \\ e_2 \\ e_3 \end{bmatrix}$$

y = **Xβ** + **e**

with solution

β= (X′X)$^{-1}$X′y

where

$$\mathbf{X'y} = \begin{bmatrix} 11 \\ 3.75 \\ 3.5 \end{bmatrix}; \mathbf{(X'X)} = \begin{bmatrix} 40 & 20 & 16 \\ 20 & 15 & 8 \\ 16 & 8 & 12 \end{bmatrix}; \mathbf{(X'X)^{-1}} = \begin{bmatrix} 0.10357 & -0.1 & -0.07143 \\ -0.1 & 0.2 & 0 \\ -0.07143 & 0 & 0.17857 \end{bmatrix}$$

$$\begin{bmatrix}\beta_0\\ \beta_1\\ \beta_2\end{bmatrix} = \begin{bmatrix}0.10357 & -0.1 & -0.07143\\ -0.1 & 0.2 & 0\\ -0.07143 & 0 & 0.17857\end{bmatrix}\begin{bmatrix}11\\ 3.75\\ 3.5\end{bmatrix}$$

$$\begin{bmatrix}\beta_0\\ \beta_1\\ \beta_2\end{bmatrix} = \begin{bmatrix}0.514\\ -0.35\\ -0.16\end{bmatrix}$$

The estimates of the crossbreeding parameters are therefore, 0.514 for the intercept, -0.35 the regression of proportion of J on incidence of lameness and -0.16 for the estimate of F1 heterosis of FxJ. This means that the incidence of clinical lameness in Jersey cows was 35% lower than F cows. The heterosis effect was -16% lower incidence of clinical lameness in crossbred cows compared to the average of the two purebreds.

2.5 Estimates of genetic parameters of lameness

Much of the variability of incidence of lameness seems to be associated with environmental and nutritional factors. However, several studies have related the incidence of this disease with cow traits like breed, live weight and milk production (Boettcher *et al.*, 1998; Koenig *et al.*, 2005). Worldwide, several authors have reported the heritability and correlations of the disease with different production traits and conformation traits (Koenig *et al.*, 2005).

2.5.1 Heritability estimates of incidence of lameness

Phenotypic variation among individuals may be due to genetic or environmental causes. Heritability is defined as the proportions of phenotypic variation in a population that is due to the genetic variations among individuals. Heritability is expressed on a scale of 0 to 1 or 0 to 100% and is often generalised such as low, medium and high. The heritability of 0-0.1 (0-10%) is generalised as low, 0.1- 0.3 (10 – 30%) as medium and above 0.3 (30%) as high or strong (Dalton, 1980). If the heritability is high, the genetic progress from one generation to another can be fast and can be achieved by selection. In contrast, if the heritability is low, the genetic change through

selection can be quite low. However, it has been reported that traits with low heritability mostly respond well through crossbreeding (Dickerson, 1972).

The heritability of incidence of lameness appears to be influenced by other factors that are related to claw and foot disorders, breed, live weight and milk production. Worldwide, numerous studies have been carried out to determine the heritability of incidence of lameness and associated risk factors in dairy herds (Cue *et al.*, 1996; Fatehi *et al.*, 2003; Koenig *et al.*, 2005; Van Dorp *et al.*, 2004). Several authors have reported the heritability for incidence of foot and leg problems evaluated in different environments as shown in Table 2.3 below.

Table 2.3. Estimate of heritabilities for incidence of feet and legs problems for different breeds in different management systems.

Trait	Breed	Production systems	Heritability (h^2)	References
Feet and legs	Canadian-HF	Intensive	0.052- 0.170	(Boettcher *et al.*, 2003; Fatehi *et al.*, 2003)
Feet and legs	Germany-HF	Intensive	0.073– 0.140	(Koenig *et al.*, 2005)
Feet and legs	NZ-HF	Grazing	0.076	(Cue *et al.*, 1996)
	NZ-Jersey	Grazing	0.068	(Cue *et al.*, 1996)
	NZ-Ayrshire	Grazing	0.121	(Cue *et al.*, 1996)
	NZ- HF X Jersey	Grazing	0.074	(Cue *et al.*, 1996)

The information in Table 2.3 above was reported from different studies overseas and in New Zealand purebred and crossbred dairy cows. In general, the heritability estimates in intensive production systems tended to be higher than those on grazing systems. High values of heritability estimates in intensive production systems show some indication of genetic and environmental interaction (GxE) for incidence of feet and leg problems. However, some studies in Canada indicated less evidence of GxE interaction for conformation traits such as feet and legs (Boettcher *et al.*, 2003). Researchers have related the higher incidences of lameness in intensive production systems with longer periods of movements in concentrate floors allowing more leg and foot injuries (Fatehi *et al.*, 2003). These results indicate that intensive housing systems have a detrimental effect on foot and leg injury than grazing systems. In contrast, the lower heritabilities for different breeds in New Zealand grazing systems were due to reduced leg injuries compared to intensive production systems that use housing (Berry *et al.*, 2005; Fatehi *et al.*, 2003).

Table 2.4. Estimates of heritabilities of incidence of clinical lameness and other diseases associated with lameness in Holstein-Friesian cows**.**

Trait	Heritability	Country	References
Clinical lameness	0.09 – 0.16	Canada	(Van Dorp *et al.*, 1998; Zwald *et al.*, 2004)
Digital dermatitis	0.073 – 0.13	Germany	(Koenig *et al.*, 2005; McDaniel, 1997)
Sole ulcer	0.086 – 0.39	Germany	(Koenig *et al.*, 2005; McDaniel, 1997)
Wall disorder	0.104	Germany	(Koenig *et al.*, 2005)
Inter-digital hyperplasia	0.115	Germany	(Koenig *et al.*, 2005)

Estimates of heritability for incidence of clinical lameness are presented in Table 2.4 and range from 0.09 to 0.16. The estimates of heritability for incidence of hoof disorders associated with lameness in Holstein-Friesian cows reported by different authors ranged from 0.073 to 0.13 for digital dermatitis, 0.086 to 0.39 for sole ulcers, 0.104 for inter-digital hyperplasia and 0.115 for wall disorders (Boelling *et al.*, 2001; Boelling and Pollott, 1998; Zwald *et al.*, 2004). However, the estimates of heritability for lameness incidence in dairy cows varied across lactations (Boelling and Pollott, 1998; Zwald *et al.*, 2004).

Alban (1995) reported higher incidences of lameness in Danish Black and Red Danish cows than in Danish Jersey cows. The higher incidence of lameness in red and black Danish cows was associated with higher live weight while Danish Jersey cows have a lower live weight. Several studies have evaluated the genetic relationship between lameness and other disorders associated with lameness (Koenig *et al.*, 2005). Lyons *et al.* (1991) found the heritability of leg problems was 0.09 and that of foot problems was 0.2. However, several cases of leg and foot problems seem to be caused by environmental and nutritional factors (Chesterton *et al.*, 2008). Some studies reported estimates of heritability of incidence of clinical lameness and other diseases associated with claw and hoof problems (Table 2.4).

2.5.2 Genetic correlations between incidence of lameness and hoof disorders and production traits

The theory of genetic correlation suggested that two traits are genetically associated when a group of genes affects two or more traits. This is a measure of the strength of the relationship between two traits. The genetic correlations are indicated by values from +1 to -1 and show how one variable change affect another. The positive correlations show that the increase in value of one trait is associated with a positive change in the other trait. In contrast, the negative correlation show that the increase in values of one trait is associated with lower values of the other trait while a correlation of “0” value means that there is no associations between two traits (Dalton, 1980).

Koenig, *et al.* (2005) reported the genetic correlation between incidence of lameness and incidence of claw and foot disorders. The genetic correlations showed that cows susceptible to claw and foot disorders were likely to be susceptible to lameness and other diseases. The genetic correlation of lameness and other feet conformation traits are shown in Table 2.5.

Table 2.5. Estimated genetic correlations between the incidence of lameness and leg conformation traits in Holstein-Friesian dairy cows.

Leg conformation trait	Genetic correlation	Reference
Rear legs side view	0.13	(Boettcher *et al.*, 1998)
Foot angle	-0.34 to 0.76	(Boelling and Pollott, 1998; Boettcher *et al.*, 1998)
Rear legs, rear view	-0.68	(Boettcher *et al.*, 1998)

The leg conformation traits reported on Table 2.5 above showed a moderate genetic correlation with incidence of lameness. However, the decrease in foot angle was highly correlated with an increased risk of lameness. Huang *et al.* (1995) reported breed differences in claw score for certain foot conditions in USA dairy breeds. Jersey and Ayrshire breeds had better claw scores than Holstein-Friesian and Guernsey. In New Zealand, Jersey cows have been reported to have harder claw horn and there are indicators that they have fewer predispositions to lameness than Holstein-Friesian cows (Chesterton *et al.*, 1989). A heavier live weight (Boettcher *et al.,* 1998) and white claw colour (Chesterton *et al.*, 1989) of Holstein-Friesian cows have been positively associated with incidence of lameness. The effect of breed was ranked as the second factor that contributes to leg conformation in United States of America (USA) (Huang *et al.*, 1995), while laminitis and heel erosion were

highly correlated to other claw traits and varied among breeds (Huang and Shanks, 1995).

2.6 Economics of lameness

This final section of the literature review focuses on the economic impact of clinical lameness in the dairy industry. Clinical lameness has a direct and indirect impact on economic losses in dairy cows. The direct impact of lameness includes the cost in terms of money and labour required for treatment of sick animals. However, the indirect costs take in reduced milk yield, fertility, culling, loss of body condition and time spent for treating and management of affected cows (Alban *et al.*, 1995; Boettcher *et al.*, 1998; Vermunt and Greenough, 1995).

Worldwide, several studies have been carried out to determine the cost of lameness in dairy herds (Alban, 1995; Harris *et al.*, 1988; Koenig *et al.*, 2005). The involuntary culling and replacement costs of 7% to 15% because of lameness have been reported (Esslemont and Kossaibati, 1997; Koenig *et al.*, 2005). Cows that present lameness during the lactation period have a higher risk of being culled in a particular period than a healthy cow due to reduced milk production and fertility (Rajala-Schultz and Gröhn, 1999). In addition, cows that show lameness during lactation are likely to be lame in the next lactation and this appears as significant risk factor for culling throughout lactation (Calavas *et al.*, 1996).

A number of studies have shown that the occurrence of lameness significantly lowers the milk yield in an affected cow (Onyiro *et al.*, 2008; Warnick *et al.*, 2001). In one study involving two New York dairy farms in United States of America, losses of milk production between 0.5 to 5.5 kg per day per lame cow were reported (Warnick *et al.*, 2001). Another study in Scotland, estimated daily milk losses of 0.78 to 5 kg of milk per lame cow (Onyiro *et al.*, 2008). Hernandez *et al.* (2002), reported lower milk production in lame cows than healthy Holstein-Friesian cows in Frolida, USA. However,

the difference was not significant. Warnick, *et al*. (1995) reported a cumulative reduction of 320 kg milk in 305 days in milk from lame cows, compared with non-lame cows, in New York Holstein-Friesian cows. Another study reported a total lactation loss in milk production from a lame cow of about 295 kg in 100 days, compared to a normal cow (Warnick *et al.*, 2001). The decrease in milk yield was associated with reduced lactation length and lactation number. The trend in decrease in milk production was higher for cows in second lactation and successive lactations and for cows in a more severe condition of lameness. Booth *et al*. (2004) reported an increase in risk of culling from early to mid lactation (between 61 and 121 days in milk) and towards the end of lactation. In general, lameness was found to have the greatest effect in mid lactation and less effect in late lactation (Booth *et al.*, 2004).

Reduced fertility related to lameness may have a significant contribution to financial losses in dairy herds. Collick *et al.* (1989) reported calving intervals up to 14 days longer due to sub-optimal oestrous expression for lame cows compared with normal cows. In addition, the pregnancy rate was 10% lower in lame cows, 2.14 services were required per conception in lame cows compared to healthy cows (1.72) and the difference of services between the two groups were significant. A reduction in conception rates in lame cows was associated with decreased food intake and loss in body condition which had also a negative effect on fertility. The mean costs of lameness per lame cow per lactation in different countries are compared in Table 2.6.

Table 2.6. The estimated cost in $US (local currency) of lameness per cow lactation in dairy herds in Australia, New Zealand, United Kingdom (UK), United States of America (USA) and Netherlands.

Country	Cost of lameness $US/cow/lactation	Reference
UK	415 (UK£273)	(Kossaibati and Esslemont, 1997)
USA	200	(Amstutz, 1985)
Netherlands	128 (NLG 230)	(Enting *et al.*, 1997)
New Zealand	65 to 173 ($NZ 94 to 250)	(Lambourne, 2010; Tranter and Morris, 1991)
Australia	1.7 to 170 ($AU 2 to 200)	(Harris *et al.*, 1988; Malmo and Vermunt, 1998)

Table 2.6 describes the economic importance of lameness worldwide. All these studies show that there is a large variation in lameness cost between countries. A relatively higher cost of lameness was reported in housing production systems than in grazing systems. This reflects the fewer cases of lameness in grazing systems in New Zealand and Australia than in intensive production in UK and USA (Clarkson *et al.*, 1996; Olmos *et al.*, 2009). In UK, lameness was ranked as the most important disease contributing to financial loss of about £30 million each year (Greenough, 1997). However, the cost of lameness may be extended beyond financial losses to animal welfare implications. This implies that pain associated with chronic leg and foot lesions causing lameness impairs the ability of the cow to perform normal behaviours.

2.7 Summary of the literature review

This review of the literature indicates that lameness is a complex health problem, and is considered to be an important worldwide disease in intensive and pastoral dairy farming systems. The disease results in economic losses and painful conditions that lead to poor cow welfare. The risk factors causing lameness in dairy cow production can be categorised into environmental, genetic, physiological, management, nutritional and human factors. Globally, the impact of lameness has been quantified in terms of reduced milk production, fertility and feed intake. In pastoral production systems in New Zealand and Australia, the major factors associated with lameness include poor design and level of maintenance of raceways, herd management, nutrition and genetic factors. On the other hand, in intensive production systems, nutrition and housing systems are major factors contributing to lameness. This review shows the need for research and strategies of reducing the incidence of lameness in pastoral and intensive dairy production systems. Therefore, research on bovine lameness should focus on a wide range of factors leading to lameness in different environments. Indeed, it is important that research should keep providing answers for lameness problems raised in New Zealand and overseas.

CHAPTER THREE

3. MATERIALS AND METHODS

3.1 Data and descriptive statistics

The Livestock Improvement Corporation (LIC) provided records on clinical lameness collected during the seasons 2005/2006 to 2008/2009 in herds dedicated to the sire-proving scheme. Included was information on farm location, calving date, parity number, breed composition, date of treatment and type of treatment for individual cows. Clinical lameness was coded "1" for cows that presented at least one event of clinical lameness on any day at risk in the season and "0" for healthy cows. Cows that may have experienced a low degree of lameness were not recorded because they did not receive veterinary treatment. The progeny test dairy sires were evaluated in sire proving scheme herds and daughters were scored to provide sire evaluation for production, disease and conformation traits. All records collected by farmers were entered onto LIC progeny testing scheme database and were examined by the LIC's livestock selection team. Herd milk records of milk, fat and protein yields for all cows in each of the herds were provided. The records corresponding to parities of more than 10 were removed from the data because their number was too small. Any farms with less than 100 dairy cows were omitted from the analysis. Following editing, 111,565 cow records, 76,357 number of cows, 155 dairy herds and 261 contemporary groups of herd-years were available for further analysis. Contemporary group was defined as a group of cows calving in the same herd and year. Some herds did not participate in all four seasons (years) of the study because some herds drop out after one season and new herds entered into the proving scheme. The total number of sires of F, J and FxJ participating in the progeny-testing scheme was 4299. The records for clinical lameness and milk yield traits of different breeds including Holstein-Friesian (F), Jersey (J) and crossbred (FxJ) were used for data analyses.

The age structure of the 111,565 lactations was the following: 23, 19, 15, 12, 10, 8, 5, 4, 2, and 1% for lactation number from 1 to 10 respectively. The percentage of cows with 1, 2, 3 and 4 records were 66.1, 23.2, 9.1 and 1.56%, respectively, resulting in an average of 1.5 records per cow.

The types of lameness that were found were classified into white line disease, foot rot, hoof crack, sole bruise, sole penetration and others for unknown causes of lameness and were analysed with the FREQ procedure of SAS (2002). The descriptive statistics of incidence of lameness and milk production traits were analysed using SAS Version 9.2 (SAS, 2002). The structured query language was used to sort, summarise, merge, create new variables and to combine datasets. The analysis included the fixed effect of contemporary group (herd – year), breed groups (F, J and FxJ), lactation number and month of calving.

Production traits included average daily yields of milk, fat and protein. Means and standard deviations of daily yields of milk, fat and protein for healthy and affected cows were obtained using the MEANS procedure of SAS (2002).

3.2 Calculations of breed proportions and coefficients of breed heterozygosity

The breed composition of each cow was described in terms of proportions of F and J. All cows with breed composition of more than 87.5% or less than 12.5% HF or J were described as pure breed and the rest were described as crossbred. The breed composition of each cow was described in terms of proportions of F and J. The proportions of genes from each breed were calculated for each animal using the following formula (Jury *et al.*, 2010; Penasa *et al.*, 2010):

$$\alpha^{p}_{i} = (\alpha^{s}_{i} + \alpha^{d}_{i})/2$$

where: α^{p}_{i} = proportion of genes from breed *i* in the progeny,

α^{s}_{i} =proportion of breed i in the sire and

α^{d}_{i} = proportion of breed *i* in the dam.

The coefficient of FxJ breed heterozygosity (h_{FxJ}) for each cow was calculated using the following formula (Jury *et al.*, 2010; Penasa *et al.*, 2010):

$$h_{FxJ} = \alpha^{s}_{F}\,\alpha^{d}_{J} + \alpha^{s}_{J}\alpha^{d}_{F}$$

where h_{FxJ} is a coefficient of expected breed heterozygosity between fractions of F and J in the progeny, α^{s}_{F} is a proportions of F in the sire, α^{d}_{J} is a proportion of J in the dam, α^{s}_{J} is a proportion of J in the sire and α^{d}_{F} is a proportion of F in the dam.

3.3 Model for estimation of genetic and crossbreeding parameters

The variance components and crossbreeding parameters for incidence of clinical lameness in dairy cows were estimated using the restricted maximum likelihood procedure by fitting an animal model in ASReml software program (Glimour *et al.*, 2002). A univariate repeatability model (Henderson, 1973; Mrode, 2005) was used considering fixed and random effects:

$$\mathbf{Y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wp} + \mathbf{e}$$

where,

y is the vector of phenotypic observations

b is the vector of fixed crossbreeding effects

X is an incidence matrix relating records to fixed effects

Z is an incidence matrix relating records to animal effects

W is an incidence matrix relating the records to permanent environmental effects

a is a vector of additive random animal effects

p is a vector of random permanent environmental effects

e is the vector of random residual effects

The **Z** matrix allows inclusion of sire and dams related to cows with records and **W** is an identity matrix. The fixed effects included in **b** were contemporary group of herd-year, month of calving, lactation number and the covariables of proportion of Jersey and coefficient of breed heterozygosity of FxJ.

The following assumptions were made: It was assumed the following expectations E(**y**) = **Xb,** E(**a**) = **0** and E(**e**) = **0** and variances **var**(**a**) = **A**σ^2_a = **G,** var(**p**) = **I**σ^2_p and var(**e**) = **I**σ^2_e = **R** and hence, var(**y**) = **ZAZ′**σ^2_a + **WI**σ^2_p**W′** **+ R** where σ^2_a, σ^2_p and σ^2_e are animal, permanent environment of cow and residual variances, respectively.

A is the numerator relationship matrix between all animals considered in the data set. The numerator relationship matrix was based on the knowledge of the pedigree relationship of parents and offspring suggested by Mrode (2005). The pedigree file included parents and grandparents of a cow with records.

The mixed model equations used for the estimation of fixed effects, prediction of breeding values and the effect of permanent environmental effects are presented below:

$$\begin{bmatrix} \mathbf{X'X} & \mathbf{X'Z} & \mathbf{X'W} \\ \mathbf{Z'X} & \mathbf{Z'Z} + \mathbf{A^{-1}}\alpha_1 & \mathbf{Z'W} \\ \mathbf{W'X} & \mathbf{W'Z} & \mathbf{W'W} + \mathbf{I}\alpha_2 \end{bmatrix} \begin{bmatrix} \mathbf{b} \\ \mathbf{a} \\ \mathbf{p} \end{bmatrix} \begin{bmatrix} \mathbf{X'y} \\ \mathbf{Z'y} \\ \mathbf{W'y} \end{bmatrix}$$

with $\alpha_1=\sigma^2_e/\sigma^2_a$ and $\alpha_2=\sigma^2_e/\sigma^2_p$ (Mrode, 2005) and A^{-1} is the inverse of numerator relationship matrix. Therefore, the mixed model equations for the best linear unbiased estimator (BLUE) of estimable function of b and the best linear unbiased prediction of additive random animal effects (a) and cow effects (p) were obtained as follows:

$$\begin{bmatrix} \mathbf{b} \\ \mathbf{a} \\ \mathbf{p} \end{bmatrix} = \begin{bmatrix} \mathbf{X'X} & \mathbf{X'Z} & \mathbf{X'W} \\ \mathbf{Z'X} & \mathbf{Z'Z} + \mathbf{A^{-1}}\alpha_1 & \mathbf{Z'W} \\ \mathbf{W'X} & \mathbf{W'Z} & \mathbf{W'W} + \mathbf{I}\alpha_2 \end{bmatrix}^{-1} \begin{bmatrix} \mathbf{X'y} \\ \mathbf{Z'y} \\ \mathbf{W'y} \end{bmatrix}$$

The matrices **X′X, X′Z, X′W, X′y, Z′X, W′X, W′Z** and **Z′Z** were obtained by using matrix multiplication principles. The numerator relationship matrix was based on the knowledge of the pedigree relationship of parents and offspring

with $\alpha_1=\sigma^2_e/\sigma^2_a$ and $\alpha_2=\sigma^2_e/\sigma^2_p$ (Mrode, 2005). Means and standard errors of incidence of RCL for each lactation and for purebred 100% HF, 100% J and first cross 50% HF – 50% J including heterosis, were estimated using the capabilities of ASReml (Glimour *et al.*, 2002) with the command 'predict'.

3.3.1 Estimation of heritability and repeatability

Heritability and repeatability of clinical lameness were calculated from the variance estimates obtained from the ASReml analysis.

Heritability (h^2) of the incidence of RCL was calculated as:

$$h^2 = \sigma^2_a / (\sigma^2_a + \sigma^2_p + \sigma^2_e)$$

Similarly, the repeatability (r) of the incidence of RCL was calculated as:

$$r = (\sigma^2_a + \sigma^2_p) / (\sigma^2_a + \sigma^2_p + \sigma^2_e)$$

Breed effects and variance components (residual and animal) were estimated again using records from only first lactation cows with the same animal model but not including the fixed effect of lactation number and the random permanent of cow.

CHAPTER FOUR

4. RESULTS

4.1 Mean milk production and overall incidence of RCL

There were 111,565 lactation records that were used to assess the incidence of RCL while 111405 lactation records were used to assess the milk production traits (Table 4.1). The overall predicted incidence of RCL per herd was 7.74% with a range of 2% to 34%.

Table 4.1. Mean (± SD) of daily milk yield (kg), fat (kg), protein (kg) and milksolids production (kg fat + protein) of dairy cattle from Livestock Improvement Corporation (LIC) dedicated sire improvement scheme herds in New Zealand.

Mean	No. obs.	Mean (± SD)
Milk yield (kg/cow/day)	111405	16.91 (± 4.91)
Milk fat yield (kg/cow/day)	111405	0.80 (± 0.21)
Milk protein yield (kg/cow/day)	111405	0.63 (± 0.17)
Mean milksolids (kg/cow/day)	111405	1.43 (± 0.38)

4.2 Frequency count of clinical lameness in 305 days in milk

The frequency count of RCL (Figure 4.1) tended to vary with number of days in milk (DIM). The general trend shows that most of the cases of RCL occured between 0 and up to 90 DIM. The number of cases of clinical lameness started to decrease after 90 DIM with a sharp reduction in the incidence of RCL after 150 DIM.

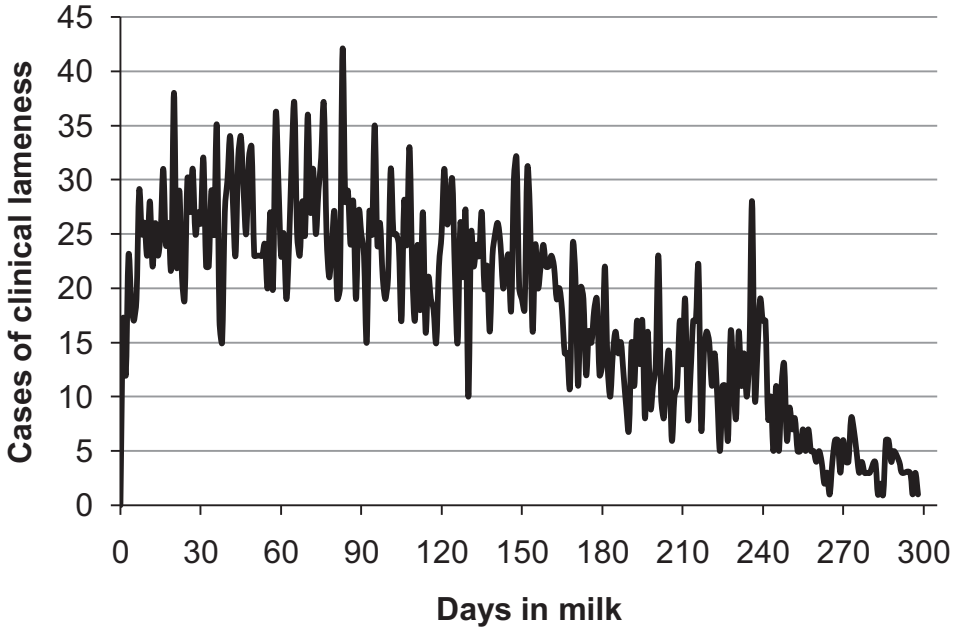

Figure 4.1. Distributions of clinical lameness cases according to days in milk (DIM).

4.3 Incidence of RCL in different lactations

There was a tendency for the incidence of RCL to increase from the second to tenth lactation. The first lactation cows had a significantly ($P<0.05$) higher incidence of RCL compared with second, third, fourth, fifth and sixth lactation cows (Figure 4.2). While cows in the seventh, eighth, ninth and tenth lactation had significantly ($P<0.05$) higher RCL than cows in first, second, third, fourth, fifth and sixth lactation. The lowest incidence of RCL occurred in the second lactation which was different ($P<0.05$) to the average of incidence in all lactations except the average incidence in the third lactation (4.1%). The bar graph presented in figure 4.2 includes 95% confidence interval of the incidence of RCL in different lactations.

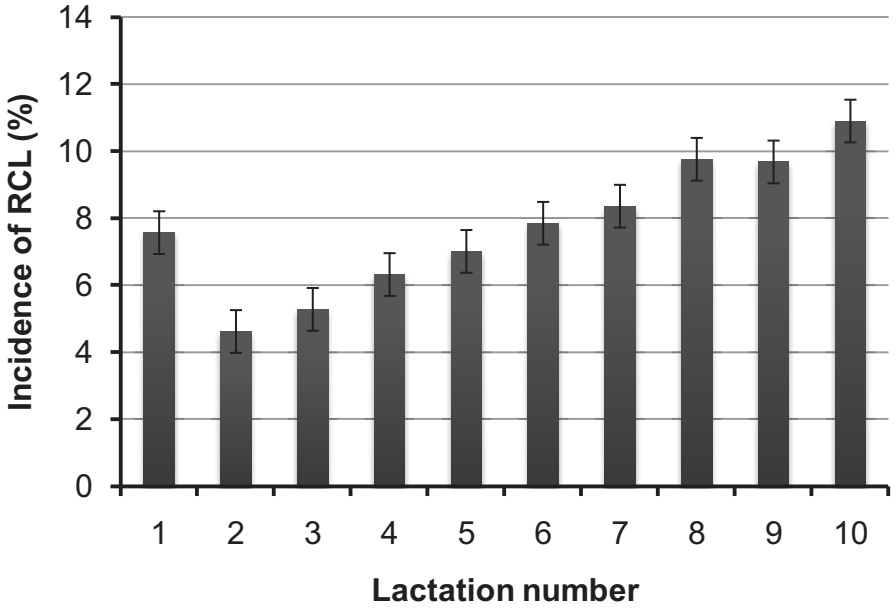

Figure 4.2. Incidence of RCL in different lactation numbers.

4.4 Incidence of RCL in different production seasons

The cows lactating in the season 2005/2006 had a significantly higher incidence of RCL than cows in the 2006/2007, 2007/2008 and 2008/2009 seasons (Table 4.2). The individual cow risk for RCL was reduced by 7.6%, 2.7% and 26.7% for years 2005/2006, 2006/2007 and 2007/2008, respectively.

Table 4.2. Mean (± SE) estimated incidence of RCL during the 2005, 06, 07 and 08 milk production seasons.

Season	Incidence of RCL
2005/2006	0.079 (± 0.0018)
2006/2007	0.073 (± 0.0016)
2007/2008	0.071 (± 0.0015)
2008/2009	0.052 (± 0.0027)

4.5 Comparison of daily milk production from cows with and without a recorded incidence of clinical lameness

The mean daily milk yield of cows (Table 4.3) without a RCL were higher for F cows followed by the FxJ crossbred cows, while J cows had the lowest milk yield. The lactating cows of all breeds with no recorded incidence of clinical lameness had higher mean daily milk yields, which were 2.3% for J, 2% for F and 1.96% FxJ - higher than cows of the same breed that had a recorded incidence of clinical lameness. RCL had a similar effect on milk fat and protein yields for healthy cows with no RCL, which were highest in FxJ, followed by F and lowest for J cows kg/cow/day. The reduction in milk fat yield due to clinical lameness compared to healthy cows was greatest (2.4%) for FxJ, followed by (1.4%) J and least (1.25%) for F cows. The mean protein yields for healthy cows were greatest for F cows, followed by FxJ and then J cows, which was equivalent to a decrease in protein yield for cows with RCL compared to health cows that was greatest (3.7%) for J cows, followed by (1.6 %) FxJ cows and least for (1.5%) for F cows. The incidence of RCL significantly ($P<0.05$) reduced milk volume, fat and protein yields, consistently in F and FxJ cows.

Table 4.3. Means (±SD) of daily milk production traits of Holstein Friesian (F), Jersey (J) and crossbred (FxJ) cows from New Zealand LIC designated dairy herds that had (lame) or did not have (Normal) a recorded incidence of clinical lameness.

Breed	No. of records	Health status	Mean (± SD) milk production yield trait kg /cow/day		
			Milk yield (kg)	Milk fat (kg)	Milk protein (kg)
Holstein	37367	Normal	18.3 ± 5.14	0.80 ± 0.220	0.65 ± 0.180
Friesian	2713	Lame	18.0 ± 5.07	0.79± 0.220	0.64 ± 0.170
Jersey	13896	Normal	13.4 ± 3.55	0.73 ± 0.190	0.54 ± 0.130
	883	Lame	13.1 ± 3.50	0.72 ± 0.180	0.52 ± 0.130
Crossbred	53159	Normal	16.8 ± 4.59	0.82 ± 0.210	0.63 ± 0.160
FxJ	3437	Lame	16.5 ± 4.54	0.80±0.210	0.62 ± 0.160

4.6 Types of RCL

The majority of RCL of lameness (Table 4.4) did not have the pathology involved recorded. Of the RCL that had the pathogenesis recorded the most frequently diagnosis was foot rot, followed by claw horn disorders of which white line disease was the most frequent, followed by sole haemorrhage and penetration other causes of lameness recorded included hoof cracking.

Table 4.4. Pathology of lameness involved in recorded incidence of lameness (%) in LIC designated dairy cattle in New Zealand dairy herds.

Pathology	Percentage (%)	No. of cases
Not diagnosed	63.0	3572
Diagnosed - Inter-digital foot rot	20.9	1183
- White line disease	7.8	441
- Sole haemorrhage	4.1	233
- Sole penetration	3.0	170
- Hoof cracking	1.2	68
Total	100.0	5667

4.7 Effect of breed on incidence of RCL

The overall predicted incidence of RCL in the dataset was 7.74 percent and 7.7% for first lactation cows (Table 4.5). The Holstein-Friesian cows had the highest incidence, followed by FxJ cows while J cows had the lowest incidence of RCL. The significant difference ($P<0.05$) on incidence of RCL was found between F and J, F and FxJ, however there was no significance between J and FxJ cows.

Table 4.5. Predicted means (± SE) of recorded incidence of clinical lameness in New Zealand dairy cows according to differing breeds of dairy cattle.

Breed	Incidence of RCL (%) using first lactation records	Incidence of RCL (%) using all lactation records
Holstein Friesian (F)	8.58 (± 0.2)[a]	8.83 (± 1.50)[a]
Jersey (J)	6.50 (± 0.2)[b]	6.67 (± 1.50)[b]
F1 - crossbred (FxJ)	6.89 (± 0.3)[b]	6.89 (± 1.50)[b]

Means within columns with different superscript are statistically different at 5% level of significance.

4.8 Estimation of genetic parameters for incidence of RCL

The estimates of genetic parameters for the incidence of RCL are presented in Table 4.6. The heritability for incidence of RCL for first lactation cows was 0.053 ± 0.014 and 0.016 ± 0.003 for all lactations while the repeatability was 0.071 ± 0.005.

Table 4.6. Estimates (± SE) of variance components, heritability and repeatability for incidence of RCL in New Zealand dairy cows.

Parameter	Estimate using first lactation records	Estimate using all lactation records
Genetic variance (σ^2_a)	0.003	0.0009
Permanent environmental variance (σ^2_p)	-	0.004
Residual variance (σ^2_e)	0.060	0.053
Phenotypic variance	0.063	0.057
Heritability (h^2)	0.053 ± 0.014	0.016 ± 0.003
Repeatability (r)	-	0.071 ± 0.005

4.9 Distribution of sires breeding values for incidence of RCL

The distribution of sires' breeding values for incidence of RCL by breed is shown in Figure 4.3. The estimated breeding values for RCL vary from -5 to +8%. The actual number of sires by breed was 2,182 Holstein Friesian, 1,517 Jersey and 600 FxJ crossbred sires. Holstein Friesian bulls had higher breeding values for RCL than J bulls and the FxJ bulls had intermediate breeding values.

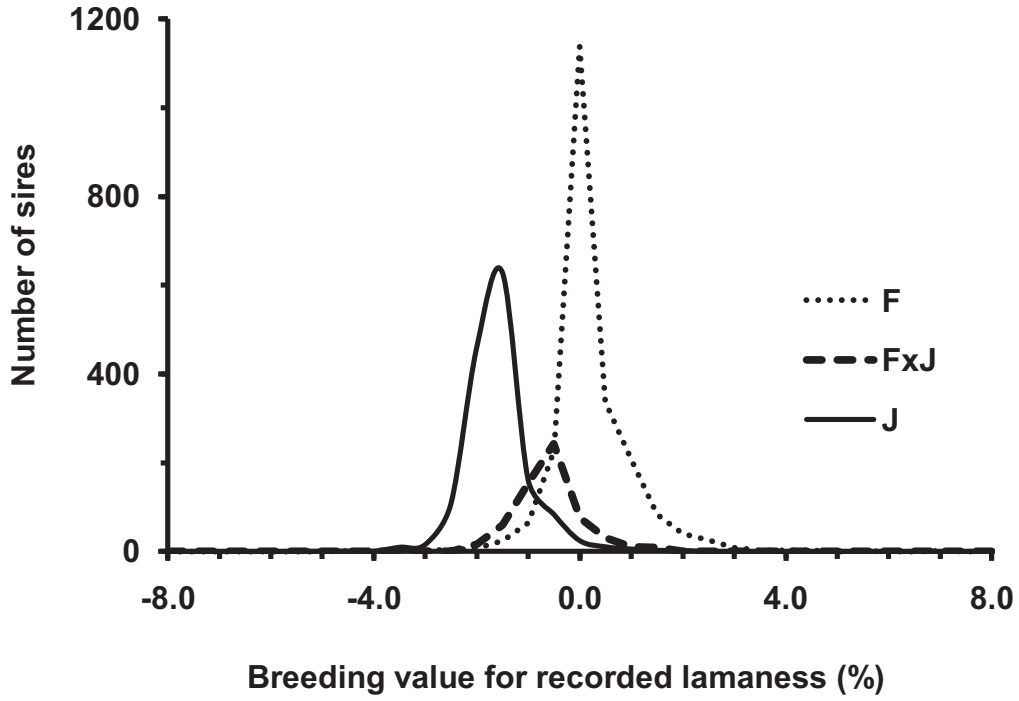

Figure 4.3. Distribution of sires according to estimated breeding values for RCL and breed (F-Holstein-Friesian, J=Jersey and FxJ =Crossbred).

4.10 Heterosis effects on incidence of RCL

The results from ASReml show that the heterosis effect (Table 4.7) of First cross FxJ crossbred was -1.2%. The breed effect measured as difference of incidence of RCL in Holstein-Friesian cows minus the incidence in Jersey cows was -1.96%. Overall, crossbred cows had a lower incidence of RCL compared to the average of the purebred and J cows had a lower incidence of RCL than F cows.

Table 4.7. Estimates (± SE) of heterosis effect of RCL in New Zealand dairy cows.

Parameter	Estimate of RCL (%)
Breed effect (F - J)	-1.96 ± 0.4
First cross heterosis (F x J)	-1.2 ± 0.3

CHAPTER FIVE

5. DISCUSSION

5.1 Overview

The overall level of recorded incidence of RCL was (7.74%) with a range from 2% to 34%. The mean incidence of RCL (7.74%) in this study was lower compared to previous multi herd studies of reported mean incidence of clinical lameness of 14 -16% from three herds in North Island (Tranter and Morris, 1991) and 26.6% from 43 herds in South Island (Gibbs, 2010). However, the mean level of RCL was within the range of 2% to 38% (Tranter and Morris, 1991) and 4.3% to 64.4% (Gibbs, 2010), of the New Zealand reported incidence of lameness. The lower incidence of clinical lameness may be due to the following reasons; firstly, the subjective and time consuming nature of the assessment of the locomotion score, secondly, potential lack of adequate observation, and thirdly, lack of recording of cows that suffer some level of lameness, that were not subsequently recorded as clinically lame. Therefore, the reported mean incidence of RCL could be the tiny tip of the large iceberg, which might not represent the actual incidence of lameness in most of the New Zealand dairy farms due to natural underestimation caused by personnel, reliance on records and frequency of recording.

Harris *et al.* (1988) reported a mean incidence of 7% in 73 herds in Australia, which is comparable to the current (7.74%) incidence of RCL in New Zealand. However, this was lower compared to some reported incidence rates of 46% in USA (Warnick *et al.*, 2001), 38% in UK (Kossaibati and Esslemont, 1997) and 25% in Scotland (Whitaker *et al.*, 1983). On the other hand, the subjective nature of the observation for clinical lameness may have an effect on recorded milk yield due to underestimation, as cows that were considered health probably had some level of lameness. In the majority of RCL the pathogenesis was not recorded which potentially indicates that the record does not provide enough information to reach the conclusion for the main causes of RCL.

From this study it is well appreciated that reducing clinical lameness requires attention to both genetics and management practices. Farmers have been motivated to conduct a routine recording of clinical lameness to improve the welfare of their animals, and if clinical lameness recording is done frequently, there is a potential that genetic evaluation for clinical lameness can be regularly performed. Routine data recording, genetic evaluation and selection of cattle based on resistance to clinical lameness will reduce the prevalence of clinical lameness in dairy cattle.

5.2 Distribution of RCL in 305 days in milk

The majority of recorded cases of RCL were detected in early lactation, with fewer incidences occurring through mid to late lactation. The results from this study are consistent with previous studies by Dewes (1978) who reported higher incidences of lameness between 14 and 84 days in lactation in New Zealand dairy cows. Similar cases of lameness in early lactation were reported in pasture-fed dairy cows in Australia (Jubb and Malmo, 1991). Early lactation (0-120 days after calving) has been associated with the occurrence of hoof lesions and this could be one of the reasons why the highest levels of lameness occur a few weeks after calving (Greenough and Vermunt, 1991; Leach *et al.*, 1997). The severity of sole ulcers and white line diseases in dairy cows at first calving have been associated with biomechanical and histopathological changes in the connective tissues of the hooves leading to increased incidence of clinical lameness (Tarlton *et al.*, 2002). There is some evidence from overseas studies that diet (Logue *et al.*, 1994; Webster, 2001) and metabolic stress in early lactation (Baggott *et al.*, 1988) appear to be associated with weakening of the hoof horn tissue and predispose cows to clinical lameness. In another study, Barkema *et al.* (1994) reported higher incidence of lameness in early lactation in Dutch dairy farms. An initial peak in the occurrence of clinical lameness in early lactation in this study could be associated with the highest increase in milk yield in the first three months of lactation. Dohoo *et al*. (1982) also reported greater incidence of feet and leg problem after parturition, in Canadian Holstein

Friesian cows. Furthermore, Zwald, *et al*. (2004) reported greater incidence of metabolic diseases around calving within 50 days after calving. Studies in New York showed that higher incidences of lameness in mid lactation and towards the end of lactation was responsible for higher culling rate during the lactation (Booth *et al.*, 2004). The current and the previous studies suggested that the overall clinical lameness resistance in early lactation can be used as selection criteria for disease resistance in breeding programs.

5.3 The effect of RCL on milk production

Lactating cows with a recorded incidence of lameness had lower milk production compared to those with no recorded lameness. A detrimental effect of lameness on milk production has been reported in other previous studies (Green *et al.*, 2002; Onyiro *et al.*, 2008; Warnick *et al.*, 2001). This significant reduction in daily mean milk production due to clinical lameness may be explained by delay in identifying lame cows, or delay in treatment of the lame cows. Severely lame cows could have lower milk yield because lame cows were highly likely to be put on once a day milking or were dried off. The subjective nature of the detection of clinical lameness by farmers and lack of accurate diagnosis (63%) of cases may be among of the factors that contribute to poor detection and appropriate treatment of clinical lameness and subsequent reduction in milk yield. This reduction in milk volume and solids yield was slightly higher in Jersey cows than F and FxJ cows. Warnick *et al.* (2001) reported a significant association of weekly decrease in milk production with lameness in United States. In another study, conducted in a herd of 500 cows located in Florida (USA), showed that cows with lameness produced less milk than healthy cows, but the difference was not significant (Hernandez *et al.*, 2002) .

5.4 Pathology of recorded incidence of lameness

The pathogenesis of lameness was not recorded in the majority of cases, which could indicate poor recording methods or lack of diagnosis. This needs further study, possibly using farmer interviews to consider how this situation

could be improved to reduce the impact of lameness and to gain better records for research into the prevention of lameness in dairy cattle. Of the cases of RCL that were diagnosed and recorded, the main causes were inter-digital foot rot and claw horn disorders and these were the most frequent cases of RCL in this study. These findings show that the majority of the cows did not have the pathology recorded, but of those recorded the lameness was due inter-digital foot rot and claw horn related disorders. Chesterton *et al*. (1989) related that traumatic lesions causing lameness correlate with poorly maintained tracks and poor handling of cows. Interestingly, foot rot was regarded as one of the infectious causes of lameness in this study causing up to (20.9%) of all incidence of RCL in New Zealand herds, which differs from previous studies in other countries (Laven and Lawrence, 2006) and warrants further research on the timing and potential causes and prevention of this type of disease.

5.5 Effect of lactation number on incidence of RCL

Cows in first lactation were more likely to be clinically lame than cows in second to sixth lactation. This higher incidence of lameness on first lactation compared to following lactations has been reported among the Danish dairy cows (Alban, 1995). The relatively high incidence of lameness in first lactation was associated with sudden environmental and metabolic changes associated with first calving (Alban, 1995). Overseas claw horn disorders (CHD) are the main cause of lameness in dairy cattle, and haemorrhaging of bovine claw horn (BCH) of the sole (S) and white line (WL) areas has been found to increase *postpartum* (Offer *et al.*, 2000). This is, in part, directly related to hormonal changes at parturition that reduce the support capability of collagen fibres that suspend the pedal bone within the claw capsule, thus increasing the pressure upon the corium particularly in the sole area (Tarlton *et al.*, 2002). The higher estimates of RCL in first lactation compared to cows in second up to fifth lactation may be associated with selection bias, as only those cows that were relatively healthy in previous lactation would have an opportunity to be selected in the succeeding lactation. The impact of lameness on feed intake (Bareille *et al.*, 2003) and fertility, animal selection

and culling for clinical lameness practised by farmers may explain the reduction of incidence of clinical lameness seen in second lactation, but may indicate the loss of genetic potential from the dairy herd, should lameness in first lactation dairy cattle be preventable using management and feeding practices. The previous study of lameness incidence in New Zealand herds showed that a high proportion of two year old milking cows were lame compared to cows in other lactations (Dewes, 1978). The previous study associated longer periods of time spent milking first lactation cows in the concrete yard as the main risk factor for lameness in first lactation cows compared with other cow categories. However, this could be prevented through animal management and feeding practices (Chesterton *et al.*, 1989).

On the other hand, Bielfeldt *et al.* (2005) associated the increased risks of lameness in older cows with a previous history of foot lesions, inflammation of joints and damage to ligaments. In addition, studies of grazing cows in Australia have shown that older cows (6 to 10 years old) tend to have more white line problems as well as sole ulcers compared to young cows (Jubb and Malmo, 1991). Similarly, Offer *et al.* (2000) reported less cases of white line problems and sole lesion scores for cows in first lactation than cows in subsequent lactations. In contrast, Sogstad *et al*. (2005) found lower frequency of lameness in young cows than older cows among Norwegian dairy cattle. The association of lactation number with clinical lameness in this study was consistent with studies in the USA (Warnick *et al.*, 2001) and with Finnish Ayrshire dairy cows (Rajala-Schultz and Gröhn, 1999). In both USA and Finland studies showed that lameness had a negative effect on milk yield and cows in first lactation were the most affected compared to other lactations.

5.6 Incidence of RCL in different production seasons

The occurrence of lameness was significantly higher in 2005/2006 (7.9%) decreasing up to 5.2% in 2008/2009. The results of the incidence of clinical lameness in different seasons show that, there is a reduction in risks for

clinical lameness from year 2005/2006 to year 2008/2009. An explanation for the results here could be that the current recording systems allow farmers to cull some of their cows that have higher risks for clinical lameness. In the New Zealand context, more lameness has been associated with seasons of high rainfall and prolonged periods of wet weather (Diaz-Lira *et al.*, 2009), which is when 'standing cows off' from pasture and the use of feed pads is likely to increase.

5.7 Breed differences in RCL

This study shows that incidence of RCL in F cows was higher than in FxJ and J cows for both first lactation and across all lactations. These breed differences agree with previous studies (Chesterton *et al.*, 2008; Chesterton *et al.*, 1989; Diaz-Lira *et al.*, 2009). This higher incidence of lameness in F than J and FxJ may be associated with poor horn puncture resistance (Lethbridge *et al.*, 2008) and higher milk yield (Diaz-Lira *et al.*, 2009). Jersey cows are lighter than F cows, and therefore the weight differences may be one reason for lower risk of lameness in J cows. Chesterton *et al*. (1989) associated the white colour of F cow claw horn to be more susceptible to lameness than black claw horn of J. The lower recorded incidence of lameness for Jersey cows in this study was consistent with previous overseas studies (Alban, 1995).

5.8 Genetic parameters

5.8.1 Variance components and heritability

The estimates of genetic variance in incidence of RCL were low, resulting in low heritability. These results indicate that selection of cows based on resistance to clinical lameness would result in a low rate of genetic gain for this disease. The variation of incidence of RCL is therefore explained by variation in environmental factors rather than genetic variation between individual cows. Incidence of lameness can be more related to environmental conditions, farm and herd size, management practices and the pathogens to which animals are exposed (Chesterton *et al.*, 1989) . The previous studies

have shown that cows in the South Island are more susceptible to lameness than cows in the North Island mainly due to growing herd size, high production and longer walking distances from the milking shed to the grazing paddocks (Gibbs, 2010).

5.8.2 Heritability

The estimate of heritability across ten lactations for incidence of RCL in this study (h^2 = 0.016) was lower compared to estimates of heritability in indoor feeding systems h^2=0.09 (Zwald *et al.*, 2004) and h^2 =0.16 (Van Dorp *et al.*, 1998). This lower heritability value reported in this study is mainly due to larger environmental variations that are difficult to control by farm management practices. It has been shown that traits with low heritability poorly respond to selection but can respond well through crossbreeding (Gosey, 2005). It is based on these findings that farmers should aim to use crossbreeding to improve animal resistance to clinical lameness. Despite the low heritability of RCL, results from this study suggest breed differences and crossbreeding can be an alternative to reduce incidence of RCL in New Zealand dairy cows.

The estimate of heritability for first lactation cows (h^2 = 0.05) was closer to the value reported by Berry et al. (2010) in Irish dairy cattle under grazing (h^2=0.04). No major differences in the heritability for RCL in first lactation (h^2 = 0.05) cows and across ten locations (h^2 = 0.016) were detected. However, the heritability estimate for first lactation was higher compared to all lactations (1st to 10th lactation). The analysis of RCL using all lactation data may have underestimated the heritability value of RCL because some herds drop out after one season and new herds entered into the proving scheme leading to decrease number of cows with first lactation records in subsequent lactations. There is evidence that young first calving heifers appears to be marked with reduction in horn growth in late pregnancy leading to softer horn formation and therefore predisposing to mechanical damage of the underlying horn tissue (Choqette-Levi et al., 1985).

5.8.3 Repeatability

The estimate of repeatability (0.07) for incidence of clinical lameness can be considered as low. The low estimate of repeatability for incidence of RCL obtained in this study was similar to the value reported by Berry *et al*. (2010) (r = 0.07) in Irish dairy cattle under grazing conditions. The estimates of repeatability of RCL was higher than the estimate of heritability, indicating the existence of permanent or non-additive genetic effects common to all lactations. However, the estimate of repeatability from this study must be considered with caution because the number of lactations per cow was only 1.5 and some herds dropped out recording events of lameness during the four milking seasons considered in this study. Warnick *et al.* (2001) associated culling bias with the increased risk of lameness of the same animals in different lactations. The cows with higher milk production potential were significantly more likely to remain in the herd regardless of clinical lameness problems compared to cows with low production. This selection and culling bias was considered as a major problem contributing to repeated incidence of lameness in different lactations (Calavas *et al.,* 1996). In New Zealand herds, the decision to culling is based on whether a cow is empty and lame cows are more likely to be empty (Tranter and Morris, 1991).

5.9 Breed and heterosis effects

The absolute value of heterosis for incidence of RCL found in this study was -1.2%, which represents minus 16% of the average of the purebreds. The effect of breed was -1.96% lower incidence of RCL in Jersey cows. The F cows have been previously reported to be more prone to lameness than J cows (Chesterton *et al.*, 1989). The genetic differences of claw traits such as white line and softer feet of the F are major reasons that make the breed more susceptible to lameness compared to J and FxJ. The conformation of the hind legs and claws affect the occurrence of lameness particularly during the first lactation (Capion *et al.*, 2008) and animals with a shallow leg and claw angle tended to bare a greater proportion of weight on the hind legs claws, which increases the risk of developing CHLs and subsequent

lameness. Additionally, Boettcher *et al*. (1998) related the higher incidence of clinical lameness in F with higher live weight of the breed compared to J cows.

5.10 Distribution of RCL by F, J and FxJ sires

There was a considerable variation between sires' estimated breeding values for RCL with values ranging from -5 to +8%. This suggests that selection of specific sires across and within breeds used in a crossbreeding program can result in the increase of genetic resistance to RCL in New Zealand dairy cattle. Although the heritability value is low, selecting bulls based on lower breeding values for clinical lameness may improve resistance to lameness.

CHAPTER SIX

6. CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusion

This study has answered some questions regarding the effect of breed and crossbreeding on lameness incidence in the New Zealand dairy cattle. Jersey cows had in average 1.9% less incidence of RCL than Holstein-Friesian cows and the first-cross heterosis FxJ was minus 16% of the average purebreds. The estimates of heritability for incidence of RCL in dairy cows were low (h^2 = 0.016 across 10 lactations and h^2 = 0.05 for first lactation cows) and therefore selection for resistance to clinical lameness diseases will result in a low genetic gain. However, a significant variation in breeding values existed between dairy sires in their daughter's susceptibility to clinical lameness suggesting that selection of specific sires of different breeds used in a crossbreeding program can result in some increased genetic resistance to lameness. The breed differences for RCL suggest that Jersey sires can be a genetic alternative to reduce incidence of RCL in New Zealand dairy herds. In addition, incorporating clinical lameness into the overall selection program will need quantification of the economic values for clinical lameness. Indeed, the financial losses arising from loss of milk production, reduced fertility, treatment costs and animal welfare issues justifies including the disease in the breeding objective.

6.2 Recommendations and Future considerations

- Improvements should be made in recording systems for lameness and the identification of the pathology of lameness.
- Bulls with lower breeding values for RCL should be chosen for breeding purposes to increase herd resistance for clinical lameness.
- The current consumer's awareness of food production based on animal welfare standards suggests that the control of lameness may increase the market opportunities for dairy products.

- Research should be carried out to evaluate the effect of subclinical lameness on milk yield and animal welfare.
- Further investigations are required to quantify the effect of lameness on culling rate and fertility of the cows.

References

Alban, L. 1995. Lameness in Danish dairy cows: frequency and possible risk factors. *Preventive Veterinary Medicine,* 22(3): 213-225.

Alban, L.; Agger, J. & Lawson, L. 1996. Lameness in tied Danish dairy cattle: the possible influence of housing systems, management, milk yield, and prior incidents of lameness. *Preventive Veterinary Medicine,* 29(2): 135-149.

Alban, L.; Lawson, L. & Agger, J. 1995. Foul in the foot (interdigital necrobacillosis) in Danish dairy cows - frequency and possible risk factors. *Preventive Veterinary Medicine,* 24(2): 73-82.

Amstutz, H. 1985. Prevention and control of lameness in dairy cattle. *The Veterinary Clinics of North America. Food Animal Practice,* 1(1): 25-37.

Arvai, V. 2009. Literature Review: A Comparison of Dairy Production Systems. *Department of CARRS, Michigan State University*: 1-31.

Baggott, D.; Bunch, K. & Gill, K. 1988. Variations in some inorganic components and physical properties of claw keratin associated with claw disease in the British Friesian cow. *British Veterinary Journal,* 144(6): 534-542.

Baird, L.; O'connell, N.; Mccoy, M.; Keady, T. & Kilpatrick, D. 2009. Effects of breed and production system on lameness parameters in dairy cattle. *Journal of Dairy Science,* 92(5): 2174-2182.

Bareille, N.; Beaudeau, F.; Billon, S.; Robert, A. & Faverdin, P. 2003. Effects of health disorders on feed intake and milk production in dairy cows. *Livestock Production Science,* 83(1): 53-62.

Barkema, H. W.; Westrik, J. D.; Van Keulen, K. A. S.; Schukken, Y. H. & Brand, A. 1994. The effects of lameness on reproductive performance, milk production and culling in Dutch dairy farms. *Preventive Veterinary Medicine,* 20(4): 249-259.

Berry, D. P.; Harris, B. L.; Winkelman, A. M. & Montgomerie, W. 2005. Phenotypic associations between traits other than production and longevity in New Zealand dairy cattle. *Journal of Dairy Science,* 88(8): 2962-2974.

Berry, D.P.; Kearney, J.F.; Twomey, K.; Cromie, A.R & Evans, R.D. 2010. Revision of the fertility and health genetic evaluations for Irish Holstein-Friesian dairy cattle. *Proceedings of the 9th World Congress on Genetics Applied to Livestock Production*, Leipzig, Germany, Communication No 0642.

Beusker, N. 2007. Welfare of Dairy Cows: Lameness in Cattle–A Literature Review. *Inaugural Dissertation: From the Institute of Animal Hygiene, Animal Welfare and Animal Ethology School of Veterinary Medicine.* Hannover.

Bielfeldt, J. C.; Badertscher, R.; Tölle, K. H. & Krieter, J. 2005. Risk factors influencing lameness and claw disorders in dairy cows. *Livestock Production Science,* 95(3): 265-271.

Boelling, D.; Madsen, P. & Jensen, J. 2001. Genetic Parameters of Foot and Leg Traits in Future AI Bulls: II. Correlation to Body Conformation Traits in Daughters. *Acta Agriculturae Scandinavica, Section A-Animal Science,* 51(2): 122-128.

Boelling, D. & Pollott, G. 1998. Locomotion, lameness, hoof and leg traits in cattle I: Phenotypic influences and relationships. *Livestock Production Science,* 54(3): 193-203.

Boettcher, P.; Dekkers, J.; Warnick, L. & Wells, S. 1998. Genetic analysis of clinical lameness in dairy cattle. *Journal of Dairy Science,* 81(4): 1148-1156.

Boettcher, P.; Fatehi, J. & Schutz, M. 2003. Genotype x environment interactions in conventional versus pasture-based dairies in Canada. *Journal of Dairy Science,* 86(1): 383-389.

Booth, C.; Warnick, L.; Grohn, Y.; Maizon, D.; Guard, C. & Janssen, D. 2004. Effect of lameness on culling in dairy cows. *Journal of Dairy Science,* 87(12): 4115-4122.

Borgmann, I. E.; Bailey, J. & Clark, E. G. 1996. Spirochete-associated bovine digital dermatitis. *The Canadian Veterinary Journal,* 37(1): 35-37.

Boyle, L.; Olmos, G.; Llamas Moya, S.; Palmer, M.; Gleeson, D.; O'brien, B.; Horan, B.; Berry, D.; Arkins, S. & Alonso Gómez, M. 2008. Cow Welfare in Grass Based Milk Production Systems. *Project 5403: Ireland*: 1-38.

Burgi, K. 2000. Maintenance of Hoof Trimming: Why, When and How. *Dairyland Hoof Care Institute Inc. Presented at the 2000 North American Veterinary Conference, Orlando, Fla.USA (www.dairylandhoofcare.com).* 1-3.

Calavas, D.; Faye, B.; Bugnard, F.; Ducrot, C. & Raymond, F. 1996. Analysis of associations among diseases in French dairy cows in two consecutive lactations. *Preventive Veterinary Medicine,* 27(1-2): 43-55.

Capion, N.; Thamsborg, S. M. & Enevoldsen, C. 2008. Conformation of hind legs and lameness in Danish Holstein heifers. *Journal of Dairy Science,* 91(5): 2089-2097.

Chesterton, R. N. 2004. Lameness-your questions answered. *Proceedings of the Society of Dairy Cattle Veterinarians of the NZVA*: 35-39.

Chesterton, R. N. 2006. Technology - the answer to lameness. *Proceedings of the Society of Dairy Cattle Veterinarians of the NZVA*: 23-27.

Chesterton, R. N.; Lawrence, K. & Laven, R. 2008. A descriptive analysis of the foot lesions identified during veterinary treatment for lameness on dairy farms in North Taranaki. *New Zealand Veterinary Journal,* 56(3): 130-138.

Chesterton, R. N.; Pfeiffer, D.; Morris, R. & Tanner, C. 1989. Environmental and behavioural factors affecting the prevalence of foot lameness in New Zealand dairy herds - a case-control study. *New Zealand Veterinary Journal,* 37(4): 135-142.

Choquette-Levy, L; Baril J.; Levy M.; St-Pierre H. 1985: A study of foot disease of dairy cattle in Quebec. Canadian Veterinary Journal 26(9): 278-281.

Clark, D.; Caradus, J.; Monaghan, R.; Sharp, P. & Thorrold, B. 2007. Issues and options for future dairy farming in New Zealand. *New Zealand Journal of Agricultural Research,* 50(2): 203-221.

Clarkson, M.; Faull, W.; Hughes, J.; Manson, F.; Merritt, J.; Murray, R.; Sutherst, J.; Ward, W.; Downham, D. & Russell, W. 1996. Incidence and prevalence of lameness in dairy cattle. *The Veterinary Record,* 138(23): 563-567.

Collick, D.; Ward, W. & Dobson, H. 1989. Associations between types of lameness and fertility. *The Veterinary Record,* 125(5): 103-106.

Cue, R. I.; Harris, B. L. & Rendel, J. M. 1996. Genetic parameters for traits other than production in purebred and crossbred New Zealand dairy cattle. *Livestock Production Science,* 45: 123-135.

Cundiff, L. V. 1970. Experimental results on crossbreeding cattle for beef production. *Annual Meeting of the American Science Society of Animal Science*: 694 - 705.

Dairy New Zealand Economic Survey 2008/2009. DairyNZ Economic Survey 2007-2008 (http://www.dairynz.co.nz). Retrieved on 10th October 2010.

Dalton, D. C. 1980. Population Genetics, Selection and Breeding IN DALTON, D. C. (Ed.) *An Introduction to Practical Animal Breeding.* London, Granada Publishing.

Dewes, H. 1978. Some aspects of lameness in dairy herds. *New Zealand Veterinary Journal,* 26(6): 147-157.

Diaz-Lira, C. M.; Margerison, J. K.; Lopez-Villalobos, N. & Gibbs, S. J. 2009. BREEF COMMUNICATION: Factors associated with frequency of lameness in dairy cattle managed in pasture based systems with the addition of supplementary feeds. *Proceeding of the New Zealand Society of Animal Production,* 69: 51-53.

Dickerson, G. E. 1972. Inbreeding and heterosis. *Animal Breeding and Genetics Symposium*: 54-76.

Dickerson, G. E. 1973. In breeding and heterosis in animals. *Proceedings of the Animal Breeding and Genetics Smposium in honor of Dr. J. L. Lush, American Society of Animal Science and American Dairy Science Assocaition,* Champaign, IL, pp. 54-77.

Dohoo, I.; Martin, S.; Meek, A. & Sandals, W. 1982. Disease, production and culling in Holstein-Friesian cows I: The data. *Preventive Veterinary Medicine,* 1(5): 321-334.

Enting, H.; Kooij, D.; Dijkhuizen, A. A.; Huirne, R. B. M. & Noordhuizen-Stassen, E. N. 1997. Economic losses due to clinical lameness in dairy cattle. *Livestock Production Science,* 49(3): 259-267.

Espejo, L. & Endres, M. 2007. Herd-level risk factors for lameness in high-producing Holstein cows housed in freestall barns. *Journal of Dairy Science,* 90(1): 306-314.

Espejo, L. A.; Endres, M. I. & Salfer, J. A. 2006. Prevalence of lameness in high-producing Holstein cows housed in freestall barns in Minnesota. *Journal of Dairy Science,* 89(8): 3052-3058.

Esslemont, R. & Kossaibati, M. 1997. Culling in 50 dairy herds in England. *The Veterinary Record,* 140(2): 36-39.

Fatehi, J.; Stella, A.; Shannon, J. & Boettcher, P. 2003. Genetic parameters for feet and leg traits evaluated in different environments. *Journal of Dairy Science,* 86(2): 661-666.

Galindo, F. & Broom, D. 2000. The relationships between social behaviour of dairy cows and the occurrence of lameness in three herds. *Research in Veterinary Science,* 69(1): 75-79.

Gibbs, J. 2010. Dairy lameness in the South Island. *Proceedings of the 4th Australasian Dairy Science Symposium*: 424-427.

Gibbs, J. & Laporte, J. 2006. Rumen function and lameness in dairy cattle in the South Island of New Zealand: *Proceedings of the New Zealand Veterinary Association Dairy Cattle Veterinarians Conference.* 26th - 28th June, Napier, New Zealand.

Glimour, A. R.; Gogel, B. J.; Cullis, B. R.; Wellham, S. J. & Thompson, R. 2002. ASReml User Guide. *Release 1.0 VSN International Ltd., Hemel Hempstead, Hertfordshire, UK.*

Gosey, J. 2005. Crossbreeding in beef cattle: Evaluation of systems. *Journal of Animal Science,* 51: 1-35.

Green, L.; Hedges, V.; Schukken, Y.; Blowey, R. & Packington, A. 2002. The impact of clinical lameness on the milk yield of dairy cows. *Journal of Dairy Science,* 85(9): 2250-2256.

Greenough, P. 1997. Understanding herd lameness. *Proceedings of the 3rd Western Dairy Management Conference,* (Las Vegas, Nevada - March 13-15, 1997): 97-106.

Greenough, P. R. & Vermunt, J. J. 1991. Evaluation of subclinical laminitis in a dairy-herd and observations on associated nutritional and management factors. *Veterinary Record,* 128: 11-17.

Gregory, K. E.; Cundiff, L. V.; Koch, R. M.; Laster, D. B. & Smith, G. M. 1978. Heterosis and breed marternal and transmitted effects in beef cattle I: Pre-weaning traits. *Journal of Animal Science,* 47(5): 1031-1041.

Groehn, J.; Kaneene, J. & Foster, D. 1992. Risk factors associated with lameness in lactating dairy cattle in Michigan. *Preventive Veterinary Medicine,* 14(1-2): 77-85.

Handley, J.; Hamilton, T. & Miller, S. P. 2001. Crossbreeding Systems for Beef Production. *Ministry of Agriculture, Food and Rural Affairs. Ontario Fact Sheet,* 1-11.

Harris, B. 2005. Breeding dairy cows for the future in New Zealand. *New Zealand Veterinary Journal,* 53(6): 384-389.

Harris, D.; Hibburt, C.; Anderson, G.; Younis, P.; Fitspatrick, D.; Dunn, A.; Parsons, I. & Mcbeath, N. 1988. The incidence, cost and factors associated with foot lameness in dairy cattle in south-western Victoria. *Australian Veterinary Journal,* 65(6): 171-176.

Henderson, C. 1973. Sire evaluation and genetic trends. *Journal of Animal Science,* (Symposium): 10-41.

Hernandez, J.; Shearer, J. & Webb, D. 2002. Effect of lameness on milk yield in dairy cows. *Journal of the American Veterinary Medical Association,* 220(5): 640-644.

Holmes, C. W.; Brookes, I. M.; Garrick, D. J.; Mackenzie, D. D. S.; Parkinson, T. J. & Wilson, G. F. 2002. *Milk Production from Pasture,* Palmerston North, New Zealand, Massey University.

Huang, Y. C. & Shanks, R. D. 1995. Within herd estimates of heritabilities for six hoof characteristics and impact of dispersion of discrete severity scores on estimates. *Livestock Production Science,* 44: 107 - 114.

Huang, Y. C.; Shanks, R. D. & Mccoy, G. C. 1995. Evaluation of fixed factors affecting hoof health. *Livestock Production Science,* 44(2): 115-124.

Jubb, T. & Malmo, J. 1991. Lesions causing lameness requiring veterinary treatment in pasture-fed dairy cows in East Gippsland. *Australian Veterinary Journal,* 68(1): 21-24.

Jury, K.; Lopez-Villalobos, N.; Spelman, R. J.; Arias, J. & Heuer, C. 2010. Genetic analysis of incidences of clinical mastitis in New Zealand dairy cattle. *Proceeding of the New Zealand Society of Animal Production,* 70: 246-249.

Koenig, S.; Sharifi, A.; Wentrot, H.; Landmann, D.; Eise, M. & Simianer, H. 2005. Genetic parameters of claw and foot disorders estimated with logistic models. *Journal of Dairy Science,* 88(9): 3316-3325.

Kossaibati, M. A. & Esslemont, R. J. 1997. The costs of production diseases in dairy herds in England. *The Veterinary Journal,* 154(1): 41-51.

Lambourne, A. 2010. Focus, effort mean health hooves. *New Zealand Dairy Exporter May 2010 (Unpublished Data) NZX Agri Global HQ. P.O Box 529, Fielding 4740 - pp 112 - 113,* (www.dairyexporter.co.nz).

Laven, R. A. & Lawrence, K. R. 2006. An Evaluation of the seasonality of veterinary treatments for lameness in UK dairy cattle. *Journal of Dairy Science,* 89(10): 3858-3865.

Leach, K.; Logue, D.; Kempson, S.; Offer, J.; Ternent, H. & Randalls, J. 1997. Claw lesions in dairy cattle: Development of Sole and white line haemorrhages during the first lactation. *The Veterinary Journal,* 154(3): 215-225.

Lethbridge, L.; Margerison, J.; Reynolds, G.; Laven, R. & Brennan, C. 2008. Comparison of lameness and hoof horn puncture resistance of New Zealand Friesian and Jersey cross Friesian dairy cattle. *Proceeding of the New Zealand Society of Animal Production,* 68: 128-129.

Livestock Improvement Corporation 2007. New Zealand Dairy Statistics 2006-2007. *Livestock Improvement Corporation (www.lic.co.nz) Retrieved on 10th October 2010*.

Livestock Improvement Corporation 2009. Service Catalogue. *www.lic.co.nz : Retrieved on 10th October 2010.*

Logue, D.; Offer, J. & Hyslop, J. 1994. Relationship of diet, hoof type and locomotion score with lesions of the sole and white line in dairy cattle. *Animal Science,* 59(02): 173-181.

Lopez-Villalobos, N. & Garrick, D. J. 2005. Methodology for the design and enhancement of genetic improvement programs illustrated in the context of the New Zealand dairy industry. *Agrociencia,* IX: 553 - 568.

Lyons, D.; Freeman, A. & Kuck, A. 1991. Genetics of health traits in Holstein cattle. *Journal of Dairy Science,* 74(3): 1092-10100.

Malmo, J. & Vermunt, J. 1998. Lameness in dairy cattle-A review (Part 1). *Journal of the Australian Association of Cattle Veterinarians,* 9: 15-18.

Manske, T.; Hultgren, J. & Bergsten, C. 2002. The effect of claw trimming on the hoof health of Swedish dairy cattle. *Preventive Veterinary Medicine,* 54(2): 113-129.

Manson, F. & Leaver, J. 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. *Animal Production,* 47: 185-190.

McDaniel, B. 1997. Breeding programs to reduce foot and leg problems. *Interbull Bull,* 15: 115-122.

Miglior, F.; Muir, B. & Van Doormaal, B. 2005. Selection indices in Holstein cattle of various countries. *Journal of Dairy Science,* 88(3): 1255-1263.

Montgomerie, W. 2005. Ten years experience of multi-breed evaluations and crossbreeding in New Zealand. *Proceedings of the Interbull Annual Meeting, Uppsalla, Sweden,* 33: 98–101.

Mrode, R. A. 2005. *Linear Models for the Prediction of Animal Breeding Values,* UK, CABI Publishing pp 83-119.

Nocek, J. 1997. Bovine acidosis: implications on laminitis. *Journal of Dairy Science,* 80(5): 1005-1028.

O'callaghan, K. 2002. Lameness and associated pain in cattle-challenging traditional perceptions. *In Practice,* 24(4): 212-219.

Offer, J.; Logue, D. & Mcnulty, D. 2000. Observations of lameness, hoof conformation and development of lesions in dairy cattle over four lactations. *The Veterinary Record,* 147(4): 105-109.

Olmos, G.; Boyle, L.; Hanlon, A.; Patton, J.; Murphy, J. & Mee, J. 2009. Hoof disorders, locomotion ability and lying times of cubicle-housed compared to pasture-based dairy cows. *Livestock Science,* 125(2-3): 199-207.

Olmos, G.; Boyle, L.; Horan, B.; Berry, D.; O'connor, P.; Mee, J. & Hanlon, A. 2008. Effect of genetic group and feed system on locomotion score, clinical lameness and hoof disorders of pasture-based Holstein–Friesian cows. *The Animal Consortium,* 3(01): 96-107.

Onyiro, O.; Offer, J. & Brotherstone, S. 2008. Risk factors and milk yield losses associated with lameness in Holstein-Friesian dairy cattle. *The Animal Consortium,* 2(08): 1230-1237.

Penasa, M.; López-Villalobos, N.; Evans, R. D.; Cromie, A. R.; Dal Zotto, R. & Cassandro, M. 2010. Crossbreeding effects on milk yield traits and calving interval in spring-calving dairy cows. *Journal of Animal Breeding and Genetics,* 127(4): 300-307.

Rajala-Schultz, P. J. & Gröhn, Y. T. 1999. Culling of dairy cows. Part II. Effects of diseases and reproductive performance on culling in Finnish Ayrshire cows. *Preventive Veterinary Medicine,* 41(4): 279-294.

Rama, J. & Veterinarian, H. 2006. Risk factors of lameness in dairy cattle and its interaction with the grazing ecosystem of milk production. *The 15th International Symposium& Conference on Lameness in Ruminants, Kuopio, Finland.*: 69-73.

Rowlands, G. J. & Lucey, S. 1986. Changes in milk yield in dairy cows associated with metabolic and reproductive disease and lameness. *Preventive Veterinary Medicine,* 4(3): 205-221.

SAS 2002. TSAS/STAT software. Release 9.2. . *SAS Institute, Cary, North Carolina, USA*.

Sauter-Louis, C.; Chesterton, R. & Pfeiffer, D. 2004. Behavioural characteristics of dairy cows with lameness in Taranaki, New Zealand. *New Zealand Veterinary Journal,* 52(3): 103-108.

Searle, S. R. 1982. *Matrix Algebra Useful for Statistics,* New York, John Wiley and Sons.

Simms, D. D.; Zoellner, K. O. & Schalles, R. R. 1990. Crossbreeding Beef Cattle. *Cooperative Extension Service: Kansas University:Manhatan*: 1-8.

Sogstad, A. M.; Fjeldaas, T. & Osteras, O. 2005. Lameness and claw lesions of the Norwegian red dairy cattle housed in free stalls in relation to environment, parity and stage of lactation. *Acta Veterinaria Scandinavica,* 46(4): 203-218.

Sprecher, D.; Hostetler, D. & Kaneene, J. 1997. A lameness scoring system that uses posture and gait to predict dairy cattle reproductive performance. *Theriogenology,* 47(6): 1179-1187.

Sutton, J.; Hart, I.; Brosters, W.; Elliott, R. & Schuller, E. 1986. Feeding frequency for lactating cows: effects on rumen fermentation and blood metabolites and hormones. *British Journal of Nutrition,* 56(01): 181-192.

Tarlton, J. F.; Holah, D. E.; Evans, K. M.; Jones, S.; Pearson, G. R. & Webster, A. J. F. 2002. Biomechanical and histopathological changes

in the support structures of bovine hooves around the time of first calving. *The Veterinary Journal,* 163(2): 196-204.

Telezhenko, E. & Bergsten, C. 2005. Influence of floor type on the locomotion of dairy cows. *Applied Animal Behaviour Science,* 93(3-4): 183-197.

Tranter, W. & Morris, R. 1991. A case study of lameness in three dairy herds. *New Zealand Veterinary Journal,* 39(3): 88-96.

Tranter, W. & Morris, R. 1992. Hoof growth and wear in pasture-fed dairy cattle. *New Zealand Veterinary Journal,* 40(3): 89-96.

Van Arendonk, J. A. M. & Liinamo, A. E. 2003. Dairy cattle production in Europe. *Theriogenology,* 59(2): 563-569.

Van Dorp, T.; Boettcher, P. & Schaeffer, L. 2004. Genetics of locomotion. *Livestock Production Science,* 90(2-3): 247-253.

Van Dorp, T. E.; Dekkers, J. C. M.; Martin, S. W. & Noordhuizen, J. P. T. M. 1998. Genetic parameters of health disorders, and relationships with 305-Day milk yield and conformation traits of registered Holstein cows. *Journal of Dairy Science,* 81(8): 2264-2270.

Vermunt, J. 1992. Subclinical laminitis in dairy cattle. *New Zealand Veterinary Journal,* 40(4): 133-138.

Vermunt, J. & Greenough, P. 1995. Structural characteristics of the bovine claw: horn growth and wear, horn hardness and claw conformation. *British Veterinary Journal,* 151(2): 157-180.

Vermunt, J. & Parkinson, T. 2002. Claw lameness in dairy cattle: New Zealand-based research. *New Zealand Veterinary Journal,* 50(3): 88-89.

Warnick, L.; Janssen, D.; Guard, C. & Grohn, Y. 2001. The effect of lameness on milk production in dairy cows. *Journal of Dairy Science,* 84(9): 1988-1997.

Warnick, L.; Pelzer, K.; Meadows, A.; Dilorenzo, K. & Whittier, W. 1995. The relationship of clinical lameness with days in milk, lactation number, and milk production in a sample of Virginia dairy herds. *Journal of Dairy Science,* 78(suppl.1): 69 (Abstract).

Webster, A. 2001. Effects of housing and two forage diets on the development of claw horn lesions in dairy cows at first calving and in first lactation. *The Veterinary Journal,* 162(1): 56-65.

Westwood, C.; Bramley, E. & Lean, I. 2003. Review of the relationship between nutrition and lameness in pasture-fed dairy cattle. *New Zealand Veterinary Journal,* 51(5): 208-218.

Westwood, C. & Lean, I. 2001. Nutrition and lameness in pasture-fed dairy cattle. *Proceedings of the New Zealand Society of Animal Production,* 61: 128-134.

Whay, H. 2002. Locomotion scoring and lameness detection in dairy cattle. *In Practice,* 24(8): 444-449.

Whay, H.; Waterman, A.; Webster, A. & O'brien, J. 1998. The influence of lesion type on the duration of hyperalgesia associated with hindlimb lameness in dairy cattle. *The Veterinary Journal,* 156(1): 23-29.

Whitaker, D. A.; Kelly, J. M. & Smith, E. J. 1983. Incidence of lameness in dairy cows. *Veterinary Record,* 113(3): 60 (Abstract).

Zwald, N. R.; Weigel, K. A.; Chang, Y. M.; Welper, R. D. & Clay, J. S. 2004. Genetic selection for health traits using producer-recorded data. I: Incidence rates, heritability estimates, and sire breeding values. *Journal of Dairy Science,* 87(12): 4287-4294.