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Genetic analysis of incidence of clinical mastitis in New Zealand dairy cattle

A thesis presented in partial fulfilment of the requirements for the degree of

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ABSTRACT

The aim of this thesis was to estimate genetic parameters and examine the effect of different dairy breeds and heterosis on the incidence of clinical mastitis in New Zealand dairy cattle. The data set used in this study was records of clinical mastitis collected during 2005/06 to 2008/09 seasons. The data set consisted of 92,961 lactations from 53,419 Holstein Friesian, Jersey and HF x JE crossbred cows. The cows were the progeny of 641 sires from 167 dairy herds that participated in a progeny-testing programme for sires. Cows with at least one event of clinical mastitis during the season were coded 1 and cows without mastitis were coded 0. The collective incidence of clinical mastitis was 11% for 92,961 lactations. A mixed model was used to estimate heritability, repeatability and breed effects for the incidence of clinical mastitis. The model included the fixed effects of contemporary group (herd and year), calving month, breed, parity, breed composition and heterosis effect of crossbred cows. The random effects included were additive animal and permanent environment of cow.

Heritability for the incidence of clinical mastitis was 0.015 ± 0.003 and repeatability was 0.070 ± 0.005 . By breed comparison, Jersey cows had 2.9% less incidence of clinical mastitis than Holstein-Friesian cows and the heterosis effects in crossbred cows had 13.4% less than the average of the parental breeds. The results from this study suggest that selection for resistance to clinical mastitis will result in a low rate of genetic gain but using Jersey sires of low breeding values can be an alternative to increase genetic resistance to clinical mastitis in New Zealand dairy cattle.

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LIST OF ABBREVIATIONS

Abbreviations

AY	Ayrshire
BS	Brown Swiss
BV	Breeding value
BW	Breeding worth
CG	Contemporary group
CM	Clinical mastitis
CMT	Californian Mastitis Test
DairyNZ	Dairy New Zealand
EBVs	Estimated breeding values
F ₁	First cross
HF	Holstein-Friesian
IMI	Intra-mammary infection
JE	Jersey
kg	Kilogram
LIC	Livestock Improvement Cooperation
MAS	Marker-Assisted Selection
ms	Milksolids (fat + protein)
NR	Norwegian Red
SAS	Statistical Analysis Software
SCC	Somatic cell count
SCM	Subclinical mastitis
SCS	Somatic cell score
SD	Standard deviation
<i>Staph. aureus</i>	<i>Staphylococcus aureus</i>
<i>Strep. agalactiae</i>	<i>Streptococcus agalactiae</i>
<i>Strep. dysgalactiae</i>	<i>Streptococcus dysgalactiae</i>
<i>Strep. uberis</i>	<i>Streptococcus uberis</i>
WMT	Wisconsin Mastitis Test
XB	Crossbred of Holstein-Friesian and Jersey

CHAPTER 1

INTRODUCTION

1. INTRODUCTION

The main focus of most dairy cattle breeding programmes has been to increase milk or milksolids (ms; fat plus protein) production per cow. This breeding strategy has produced negative side effects, with the animals more susceptible to health problems such as poor fertility, metabolic disorder and other health diseases that include mastitis. Mastitis is an inflammation of the mammary gland that occurs as a response to an infection, usually caused by the infection of bacteria or a physical trauma such as excessive vacuum during mechanical milk extraction (IDF, 1999). A wide variety of microorganisms and an array of multiple environmental factors facilitate the development of mastitis (Bramley and Dodd, 1984). The National Mastitis Advisory Committee (2008) introduced a program to control mastitis and reduce somatic cell count (SCC) in New Zealand dairy herds. The adoption of the programme has been successful at reducing the number of mastitis cases caused by contagious pathogens. Despite this, reports indicated that the incidence of mastitis caused by bacteria found in the environment has either remained constant or increased (Holmes et al., 2002).

In conjunction with the programme implemented by the National Mastitis Advisory Committee (2008), the industry initiated a long-term plan to reduce incidence of mastitis through the breeding of dairy cows with reduced levels of SCC (Harris et al., 2005). The main reasons for choosing SCC as the indirect trait for mastitis resistance are that SCC is routinely recorded in the herd-testing programme, it has higher heritability than clinical mastitis (CM), and genetic correlations between these traits are moderate to high. Therefore, it is possible that selection to decrease SCC would reduce the incidence of CM and subclinical mastitis (SCM).

Breed and heterosis effects can be exploited in a crossbreeding programme to reduce the incidence of CM. Washburn et al. (2002) reported results from a farmllet experiment comparing Holstein-Friesian (HF) and Jersey

(JE) cows under two feeding systems, confinement and grazing. Jerseys had half as many clinical cases of mastitis per cow as HF: 31.4 versus 51.0% in confinement and 17.0 versus 34.6% in grazing. McDowell and McDaniel (1968) estimated heterosis effects for health traits on first lactation cows. Average percent deviation of two-breed crosses from parental mean for incidence of CM at any stage of the first lactation was -7% in Ayrshire (AY) x Holstein, -46% in Brown Swiss (BS) x Holstein and 6.9% in AY x BS crossbred cows. Estimates of crossbreeding effects, for the incidence of CM between HF and Norwegian Red (NR) breeds under Irish grazing conditions were reported by Buckley et al. (2008). Incidence of CM was 13% in HF, 10% in NR cows. Heterosis effects expressed as a percent deviation of the two parental breeds was -4.3%. The same authors also reported estimates of heterosis for incidence of CM in a crossbreeding experiment involving the HF and JE breeds. The incidence of CM was 29% in HF x JE cows. The estimate of heterosis expressed as a percent deviation of the two parental breeds was -60.7%.

Mastitis is recognised as a complex disease, one that is caused by both environmental and contagious pathogens that are difficult to completely eradicate from dairy herds. It is one of the most common and a costly disease in dairy cattle, which causes great concern within the dairy industry. The aim of this thesis was to estimate breed and genetic effects on the incidence of CM in New Zealand dairy cattle. The subsequent chapter of this thesis will describe the background of mastitis and the significance of the disease to the dairy industry. In the same chapter alternative approaches to combat mastitis will be explained. However, this thesis will focus on three different approaches – selection for breed with mastitis resistance, crossbreeding or heterosis effect for mastitis resistance and genetic selection for mastitis resistance.

CHAPTER 2

LITERATURE REVIEW

2. LITERATURE REVIEW

2.1 Mastitis in dairy cattle

Mastitis is defined as an inflammation of the mammary gland, which occurs as the response to infection (Harmon, 1994), usually caused by the infection of bacteria or a physical trauma often caused by an excessive vacuum during mechanical milk extraction. However, in terms of quantitative genetic analyses of lactation records, “mastitis” can be defined in several ways. It is often considered as a binary trait, diseased or not diseased, across the whole lactation (Zwald et al., 2004; Heringstad et al., 2006). All methods of commercial milk production, both good and bad practices, provide suitable conditions for mastitis pathogens to spread from cow to cow thus it is important to recognise that mastitis is an infectious and extremely complex disease (Philpot, 1984). Pathogens causing mastitis come from multiple sources, many of the strains involve environmental factors to spread the infection, and it can cause variety kinds of physiological response in the animal. Mastitis is a disease that shows different infection patterns; from subclinical with no clinical signs to acute CM that may cause death of the animal. The duration of a mastitis case also varies from a few days to weeks or months (Heringstad et al., 2000).

Normally, mastitis begins as a result of pathogenic bacteria that have gained entrance through the teat canal and into the gland. The severity of the reaction to the penetration of the bacteria depends on the type of pathogens and immune response of the host gland. The rate of infection within the herd could increase by management factors that favour the spread of bacteria from one cow to another (Jarrett, 1981). There are two broad categories of mastitis, CM and SCM. Clinical mastitis usually shows physical abnormalities in the udder or milk. The udder is hot, swollen and tender to touch; the cow may have a fever and be off its feed. Milk secretion is suppressed and abnormal in appearance e.g. clots or abnormality in colour and less opaque. Subclinical mastitis is not easy to detect, as it does not show physical abnormality, as

does CM. It is characterised by reduced milk production, altered milk composition and the presence of inflammatory components and bacteria in milk (Heringstad et al., 2000). It can be identified only by special tests on the milk such as the SCC, Californian Mastitis Test (CMT) and electrical conductivity (Holmes et al., 2002). For each case of CM in a population of herds, there will be 15 to 40 SCM cases depends on the herd size, and most of CM cases starting from infections at the subclinical level. Subclinical mastitis tends to cause problem at a herd level because its long duration reduces milk production and lowers milk quality (Philpot, 1984).

2.1.1 Pathway of infection

Infection in the mammary gland may occur through the bloodstream, particularly in the case of mastitis caused by *Mycobacterium bovis* and *Brucella abortus*, but the most important route by far is via the teat canal. Most pathogens responsible for mastitis are all non-motile. The teat canal, which provides physical and chemical protection, provides a strong barrier to the penetration of the bacteria into the teat cistern (Holmes et al., 2002). However, there are several ways by which the bacteria can enter the teat canal and cause infection in the mammary gland. These are, a) changes in pressure that occur at the teat-end during milking resulting in contaminated milk moving up through the teat canal, b) teat cup slip, poor technique in cup removal or inadequate vacuum pump capacity, c) distended quarters to the extent that milk dribbles from teats are at risk of infection because the milk can be exposed to pathogens outside the teat then contaminated milk can be drawn back into the teat cistern as the pressure in the udder eases. Once in the teat cistern, and mixed with milk in the cistern, the bacteria spread throughout the teat and gland cistern and eventually up into the duct system. The severity and progression of the disease varies widely with different strains (Holmes et al., 2002).

2.1.2 Major Pathogens causing mastitis

- *Staphylococcus aureus* (*Staph. aureus*) is one of the most common pathogens causing mastitis in New Zealand dairy herds (McDougall, 2002; Petrovski et al., 2009). It is quite contagious and very difficult to control once it has become widespread within a herd. It is not dependent on the mammary gland for its survival. Improper milking machine function or over-milking causing injury to the teat allowing *Staph. aureus* to colonize the teat and udder skin. The pathogen does not, however, survive well on healthy, intact skin. The primary source of *Staph. aureus* is the infected glands and the infection is often spread by the common transmitting fomites included the milking machine inflations, common udder cloths, contaminated udder wash water, strip cups, and the milker hands (McDonald, 1984). *Staphylococcus aureus* has the ability to invade the mammary tissue causing deep-seated abscesses of infection. These abscesses are usually very resistant to intramammary treatment with antibiotics and penicillin (Jarrett, 1981). Vaccination with a bacterin-toxoid may decrease the severity of the acute mastitis but may have little effect on prevention of new infection (McDonald, 1984). However, judicious identification, isolation of infected animals, dry-cow therapy, culling of incurable cows, and sanitary precautions hold great promises to eradicate this pathogen from a herd (Jarrett, 1981).
 - *Streptococcus agalactiae* (*Strep. agalactiae*) is highly contagious. It does not invade the glandular tissue like *Staph. aureus* but remains on the epithelial surfaces where it causes tissue damage by lactic acids production. *Streptococcus agalactiae* is totally dependent on the mammary gland for its survival. It spreads from gland to gland and from cow to cow by common transmitting fomites that are contaminated with milk from infected glands. It is readily destroyed by penicillin and is not an active invader of tissue. Therefore, this pathogen is most susceptible to an eradication programme for the entire herd, based on careful detection and the separation of infected cows, appropriate milking
-

hygiene, and antibiotic treatment of the cow. Once eliminated, it cannot reappear unless it is reintroduced to the herd from outside sources (Jarrett, 1981).

- *Streptococcus uberis* (*Strep. uberis*) can be found free-living in the soil and can be recovered from the cow's lips, sex organs, rumen, faeces, and from the udder and teat skin. The most important reservoir is contaminated dirt and bedding. Some infections are chronic and nonclinical but *Strep. uberis* is now a common cause of CM and SCM in New Zealand, especially around the time of parturition (McDougall, 2002). The rate of new infection is high during the second half of the dry period, particularly in herds that do not treat the cows with dry-cow therapy. Many new gland infections following the injury of the teat end are due to *Strep. uberis*. Because it is not dependent on the mammary gland for its survival, it is not as susceptible to the eradication programme as *Strep. agalactiae* (McDonald, 1984).
 - *Streptococcus dysgalactiae* (*Strep. dysgalactiae*) is not as contagious as other pathogens, so the herd infection rate is much lower. The infection rate tends to become clinical at an early time following infection and consequently most infections are treated and eradicated. Faulty milking machine function can increase the rate of infection. If teat lesions are prevented, *Strep. dysgalactiae* can be eradicated from a dairy herd (McDonald, 1984).
 - Other organisms such as coliforms, species of *Corynebacterium*, yeasts, and molds are less commonly involved in infected quarters, and their control programme often needs different procedures of management. However, the control programme of streptococcal and staphylococcal infections can help to control these infections as well (Jarrett, 1981).
 - *Brucella abortus* (causing contagious abortion) and *Mycobacterium bovis* (causing tuberculosis) also cause mastitis but *Brucella abortus* has been
-

eradicated, and the incidence of bovine tuberculosis is very low in New Zealand (Holmes et al., 2003)

2.1.3 The association between mastitis and SCC

Somatic cell count is one of the indicators of the milk quality. Thus many countries use SCC to monitor udder health, and in many countries, SCC is included with milk recording (Poso and Mantysaari, 1996). Some SCC are an indication of uninfected udder or quarter and others are reflective of SCM or a recovery from an infection (ten Napel et al., 2009). Somatic cell consists of many types of cells but in the healthy mammary gland, most viable somatic cells are macrophages and lymphocytes with a few neutrophils and epithelial cells. It is quantified as cells per ml. As a response to pathogenic bacteria invasion to the mammary gland, the number and the predominant types of somatic cells change rapidly with the SCC exceeding 10^6 cells/ml and over 95% of somatic cells consisting of neutrophils. The transition from a healthy, low SCC milk to a secretion containing a higher SCC and possibly clots or flakes, only take a few hours (Kehrli and Shuster, 1994). In the New Zealand dairy system, milk from two-year old and older cows that have SCC exceeding 120,000 cells/ml, and 150,000 cells/ml respectively, indicates that the cow is probably affected by SCM in one or more quarters (Holmes et al., 2003).

2.1.4 Factors affecting mastitis

The incidence of mastitis in a dairy herd is associated with both cows' continually exposure to bacteria in the environment and the cows' resistance to pathogens. Risk factors causing mastitis can be differentiated into two types; 1) Management factors e.g. poor milking procedures, poor function and maintenance of milking machines, poor hygiene and bedding, poor feeding practice and inconsistency of teat spray. There are also; poor treatment of infected cows, poor management of clinical cases and high SCC, feeding of mastitic milk to replacement stock, purchase of replacement stock, culling policies, selective rather than whole herd treatment of dry cow therapy (Carlén, 2008; McDougall, 2007; Petrovski et al., 2009). 2) Non-management factors

e.g. cows dripping milk before calving or prior entry to the milking parlour, breed, lactation stage, age, poor udder conformation, teat damage, increase in milk production and milk flow rate, low vitamin E concentration, low body condition score, ketosis, heat stress, hormonal cycle of the cow and season (Carlén, 2008; de Haas et al., 2008; McDougall, 2007).

Most causative pathogens require mediums to enter the teat canal in order to cause infection. The common medium is milking machine, flawed milking machine can both aid the pathogen to enter the teat canal and spread the causative pathogens from cow to cow. The adoption of the five-point SAMM plan is known to reduce the incidence of mastitis in dairy herds (Holmes et al., 2003). A study conducted on HF and JE cows to compare the effect of feeding systems (confinement and pasture-based systems) on mastitis, reproduction and body condition score. The results showed that cows in confinement had 1.8 times more CM cases and eight times the culling rate for mastitis than did cows on pasture (Washburn et al., 2002). Breed differences have been reported in several studies; JE had half as many clinical cases of mastitis per cow compared to HF (Bannerman et al., 2008a; Washburn et al., 2002; Youngerman et al., 2004). High producing cows reported to have increased risk of CM incidences compared to lower producing cows (Poso and Mantysaari, 1996; Rupp et al., 2000), high producing cow is also associated with faster milk flow. Older cows have a higher incidence of CM than younger cows (Petrovski et al., 2009; Schukken et al., 2009). In late lactation, multiparous cows with SCM have greater production losses than primiparous cows (Hagnestam-Nielsen et al., 2009). There are reports on increased mastitis incidence in early lactation (Holmes et al., 2003; McDougall, 1998; Petrovski et al., 2009). During this period local mammary immune defence is dramatically compromised by parturition, leading to the hypothesis that immune deficiency causes increased mastitis susceptibility in periparturient cows (Burton et al., 2003). The increase in SCC is seen in late lactation in seasonally-calving herds as milk production declines, feed quality and quantity decline, milking frequency is reduced to once daily and teat

spraying is often discontinued (McDougall, 1999). There are also strong genetic correlations between mastitis incidence and udder conformation, e.g. fore udder attachment, udder depth and dairy form. Cows with fewer mastitis infections tend to have stronger fore udder attachment and higher udder depth. Dairy form has an unfavourable genetic correlation with mastitis ranging from 0.29 – 0.54, which means that selection for improved dairy form will result in more mastitis (Rupp and Boichard, 2003; Sorensen et al., 2000). These examples are an indication that both management factors and non-management factors influence mastitis infection rate in a dairy herd.

2.1.5 Costs of mastitis

Mastitis is one of the most common and costly diseases in dairy cattle (Bradley, 2002; Colleau and Le Bihan-Duval, 1995). In herds without an effective mastitis control programme, about 40% of the cows are infected with an average of two quarters and about 70% of reduced milk production is associated with mastitis (Holmes et al., 2002). A high producing cow with high milk yield prior to mastitis can be expected to lose more milk (both in kilogram and percentage) than a low producing cow (Hortet and Seegers, 1998). An economic evaluation reported by the National Mastitis Advisory Committee (2006) estimated that the cost of CM for a representative New Zealand dairy herd was \$36.50 per cow, \$11,500 for a herd with 315 cows and approximately \$180 million for the New Zealand dairy industry. Economic losses result from reduced milk production (from both CM and SCM), discarded milk during the withholding periods, treatment associated costs, increase in labour and vet costs, reduced milk price due to high SCC and the culling of persistently mastitis infected cows (Carlén et al., 2004). Processing costs increase because milk with high SCC is more perishable, cheese yield and quality are reduced e.g. by reduction in curd strength, fat, moisture, protein and cheese yield, and increased coagulation time. The increase of production cost motivated manufacturers to impose penalties on milk with high SCC (Philpot, 1984). Each doubling of bulk tank milk SCC results in 1.8% production loss or 5.8 kilogram (kg) of ms for a herd with 350 cows

(Anonymous, 2006). According to Beaudeau et al. (1993) mastitis is responsible for 5-24% of the reasons for culling, while 7% of forced drying-off was due to CM cases (Lescourret and Coulon, 1994). Evidence from various studies showed that the occurrence of CM during lactation was associated with a reduction in fat and protein yield (Harmon, 1994; Hortet and Seegers, 1998; Pyorala, 2003). This further increases economic loss particularly in the current milk pricing system which is based on fat and protein yield. Therefore, significant productivity gains could be achieved through a reduction of both CM occurrences and bulk tank milk SCC.

2.1.6 Effects on milk yield and milk composition

Mastitis changes the milk composition, and the extent to which various compositional changes occur depends on the inflammatory response. The degree of the changes depend on the pathogen causing mastitis infection and the amount of the affected tissue in the gland; especially the affected epithelial area (Pyorala, 2003). The main changes in the udder include (i) increased permeability resulting in leaking of ions (chloride and sodium), proteins (serum albumin and immunoglobulin) and enzymes from the blood into the milk thereby increasing the pH level in the milk from normal of 6.6 to 6.9 (Harmon, 1994; Hortet and Seegers, 1998), (ii) the invasion of phagocytic cells into the milk section, resulting in elevated milk SCC and clots in the milk, and (iii) a decrease of the synthetic capacity of the gland resulting in decreased concentrations of certain milk constituents (Pyorala, 2003) e.g fat, lactose, casein, calcium, phosphorus, and potassium (Hortet and Seegers, 1998; Jarrett, 1981). Some of the changes in milk composition are greater than others, which are shown in Table 2.1. They are potentially useful as indicators of mastitis but the standard indication of mastitis is the rise of SCC that usually accompanies compositional changes and inflammation in an infected mammary gland (Harmon, 1994).

Table 2.1: Main changes in the production and composition of milk caused by mastitis.

Decrease	Degree of change	Increase	Degree of change
Quarter milk yield	-(--)	Somatic cell count	+++
Dry matter	-	Whey proteins	+++
Lactose	-	Bovine serum albumin	+
Fat	-	Immunoglobulins	+++
Long-chained fatty acids	-	κ casein	+(+)
Total casein	--	Proteose peptones	++
α_{s1} casein	--	Free fatty acids	++
β casein	---	Short-chained fatty acids	+
α lactalbumin	-	Sodium	++
β lactoglobulin	---	Chloride	++
Calcium	---	Lactate	+++
Magnesium	---	Enzyme activity	
Phosphorus	---	Lipase	++
Zinc	-	Lysozyme	+++
Potassium	-	NAGase	+++
		β glucuronidase	+++
		Plasmin	+++

Source: Pyorala (2003)

Clinical and SCM also cause reduction in milk yield. A previous study showed that at the lactation level, the milk yield loss due to a CM case varies from 0 - 750 kg or 0% - 9.5% (Hortet and Seegers, 1998). Short-term loss (on a daily basis) varied from 0 – 3 kg. The results from their study were lower than expected, a consequence of the difficulty in obtaining accurate data; e.g. an infection could start and the milk yield could be reduced before mastitis was detected (Hortet and Seegers, 1998). The same study also showed that during lactation, CM was associated with a reduction in fat yield ranging from 3 – 22 kg (1.5 – 7.5%) because of reduced synthetic activity of mammary tissue. The magnitude of the effect depended on breed, parity and number of cases. The effect was larger in JE and in cows experiencing several clinical cases within the same lactation. Also at the lactation level, CM caused the reduction of protein yield of 0 – 15 kg (0 – 8.5%). The severity of this effect depended on parity and number of cases. Although some studies have shown little change

in total protein content, the types of proteins present in the milk change dramatically. The content of casein decreased while whey proteins increased (Hortet and Seegers, 1998).

Subclinical mastitis is often associated with an increased SCC, which is commonly used as an indicator of mastitis. The National Mastitis Advisory Committee (2006) used herd test records in the database to obtain predicted values for the loss of milk production. However, because the distribution of SCC is highly skewed, with a majority of low values and a small proportion of very high values, SCC is transformed to the logarithmic scale for estimating the loss of milk production values (Rupp and Boichard, 2003). The loss of milk yield associated with a \log_2 increase in SCC is shown in Table 2.2.

Table 2.2: Loss of production due to increased SCC for an average mixed age cow in New Zealand.

	Milk yield (litre)	Fat yield (kg)	Protein yield (kg)	Total Solids (kg)
Lactation Production	3574	176	132	308
Loss/Lactation	88.5	3.2	2.39	5.59
Relative Loss	2.5%	1.8%	1.8%	1.8%

Source: National Mastitis Advisory Committee (2006)

Note: Mixed age cows are cows included in the study regardless of their ages.

Table 2.2 shows increased SCC causes a loss in milk yield, fat yield and protein yield and it is clear that the loss in milk volume is greater than the loss in ms. The impact of total solids reduction is diluted and can be considered insignificant on the whole lactation for a short-term infection, but it is important and can cause considerable loss for persistent or frequently recurring severe infections. Following the successful treatment of mastitis with antibiotics during lactation, milk composition returns to pre-infection values. However, the rate of recovery depends on the severity of the infection and the components. Therefore, the milk composition and conductivity may recover within a few days while SCC and composition of proteins in the milk may remain elevated

for a week or more (Holmes et al., 2002).

2.1.7 Mastitis detection

The diagnosis of mastitis is based on the examination of the udder and its secretions. Preliminary detection can be done by palpation of the udder, physical signs of CM are: tenderness of the udder, swelling and gross abnormalities in acute cases make it easy to observe. However, SCM can be difficult to observe and easily go undetected though some routine examination of the milk helps. The most commonly used method is to observe the inline filters to detect clots in milk or strip milking prior putting the milking cup on the cow (Holmes et al., 2002). It may be easy to observe CM cases but with the herd size growing, cows with acute mastitis may be missed, as less time is available to pay attention to individual cows. The rate of inaccurate classification depends to a great extent on cow managers and milking personnel and is therefore strongly herd dependent. Subclinical mastitis can only be detected by special tests, which fall into two broad categories, direct tests (e.g. bacterial culture) that detect the presence of bacteria in the milk and indirect tests (e.g. CMT, electrical conductivity, changes in milk composition, and changes in SCC) that measure a change in milk composition that are correlated with infection (Holmes et al., 2002).

Subclinical mastitis detection methods

- Direct tests

→ Bacterial culture is the most useful tool for evaluating a mastitis problem and the only test for identifying causative pathogens in a dairy herd. The results from a herd culture can be the basis determining the source of infection and establishing an effective control procedure. Milk cultures are not necessary to initiate a mastitis prevention programme but are essential to develop control methods in a problem herd (Sears and Heider, 1981).

- Indirect tests

→ Californian Mastitis Test (CMT) is a quick test for the veterinarian and the milker; it is used at “cow side” to examine the milk. The reagents used to test the milk contain an anionic detergent and a pH indicator. When combined with milk and slightly agitated, they cause gelation and a possible change in colour, depending on the concentration of leukocytes and pH level in the milk sample. The results range from no reaction in samples containing less than 150 – 200,000 cells/ml, which is considered to be the upper limit for normal milk, to very strong reaction in samples containing more than 3 to 5 million cells/ml. There is a high correlation between the increase in number of leukocytes and the presence of pathogens but this is not always the case. Thus CMT has limitations and should not be used as the only criterion in a programme for treatment (Jarrett, 1981).

→ Wisconsin Mastitis Test (WMT) is the adaptation of the gel reaction not unlike CMT but its most common application is to evaluate bulk tank milk samples. The estimation of leukocytes in bulk tank milk give an indication of the level of udder inflammation in the entire herd (Jarrett, 1981).

→ Electrical conductivity in the milk from infected glands rises because mastitis increases the concentration of sodium and chloride ions and decreases the concentration of potassium ions in the milk. Conductivity of milk also rises for reasons other than mastitis but the absolute changes are smaller and the difference of milk samples from each quarter is similar. Thus when a conductivity meter is used to detect mastitis, milk samples from each quarter and the absolute value should be consider. If the difference between the lowest reading for a quarter and that for any other quarter is greater than a certain value, then mastitis is indicated in the quarter with the higher reading (Holmes et al., 2003). Although specific milking devices have been developed, large-scale recording of conductivity has not yet been implemented and further work on the interpretation and data modelling is still required (Rupp and Boichard, 2003).

→ Changes in milk composition e.g. lactose chloride ions, sodium ions, serum albumin and various enzymes, in milk have all been proposed and used as test for mastitis. However, SCC is firmly established as the preferred method used to date (Holmes et al., 2003).

2.1.8 Mastitis control

Prevention and control of new infections can be directed toward three main areas of interest:

- Environment – the management and sanitation of the environment play a highly significant role in the reduction of the new infection rate in a given herd. Regardless of the type of environment (confined or pasture-based farm) the environment sanitation remains a key area in the prevention of mastitis. Pathogenic organisms use moisture as a means of transportation to move toward the teat end, giving them the opportunity to penetrate the mammary gland. This indicates that it is almost impossible to maintain low rate of new infection unless the cattle are maintained in a very clean and dry condition at all times (Jarrett, 1981).
 - Milking procedures – it is believed that most bacterial penetrate through the teat canal occur just prior to, during, or just after milking. Thus milking procedures and hygiene are very important in controlling mastitis. Proper milking procedures to minimize mastitis infections are: (i) keep the animals in an environment that allow the least amount of contamination possible. (ii) If the animals are to be washed prior to milking, use paper towels to dry the udder or allow time for them to drip dry before milking them. (iii) Pre-strip any suspicious quarters before putting the cups on and, (iv) the udder should be sanitized thoroughly immediately after detaching the milking units; this is often supported by post - milking teat dip (v) in order to reduce the new infections during the first part of dry period and at calving or shortly after calving, it is recommended that all quarters of all milking cows that had CM or SCC was high at their last
-

milk test be infused with dry cow therapy following the last milking of the season (Jarrett, 1981).

- Milking machines – can be associated with increased rates of new infection by: (i) transporting pathogenic organisms from one cow to another, (ii) damaging the teat ends making it easy for the bacteria to penetrate into the quarter, and (iii) pumping or propelling the bacteria through the teat canal and into the udder. An adequate milking machine consists of the following qualities: (i) maintains a stable vacuum, (ii) does not stress the teat by stretching or ballooning, (iii) massage the teat without harsh action, and (iv) the entire system can be sanitized efficiently and satisfactorily (Jarrett, 1981).

The mastitis control programme can be summarised in to five main points

- 1 The correct use of properly functioning milking machines
- 2 Dipping teats after milking
- 3 Correct treatment of clinical cases
- 4 Treatment at drying off
- 5 Culling of cows with chronic infections

It is shown that focussing on the rate of new infections and the duration of each infection reduces the average level of infection by 75 percent (Holmes et al., 2002). Alternative approaches have been proposed to control mastitis. This includes: i) inserting a plastic coil into the gland cistern to increase cell counts based on the hypothesis that cows with high SCC should have high resistant to infection, ii) immunising cows against mastitis and, iii) breeding for mastitis resistance. These methods have potential to reduce mastitis incidence but to date they have yet to prove effective (Holmes et al., 2002).

2.2 Proposed approaches to improve mastitis resistance

2.2.1 Breed differences for CM

Genetic differences between breeds of animals are known to influence disease resistance (Bannerman et al., 2008a). Dairy breeds originating from eastern France (Montbéliarde, Abondance) or central Europe (Simmental and BS) have lower SCC and CM frequency than Holstein breeds (Rupp and Boichard, 2003). Udder health, as characterized by SCC, and incidence of CM was significantly lower in NR than HF (Begley et al., 2009). Regarding the two most popular dairy cow breeds in New Zealand – HF and JE - several studies have reported a lower susceptibility for mastitis in JE cows than HF cows (Bannerman et al., 2008b; Washburn et al., 2002; Youngerman et al., 2004).

Holstein Friesian cows seem to have the highest frequency of CM and SCM compared to other breeds. This trend is also increased over time as a result from the successful selection based on high productivity despite its positive genetic correlation with mastitis resistance (Rupp and Boichard, 2003). Because mastitis is one of the main reasons for culling dairy cows, lower susceptibility of mastitis in JE cows may contribute to their higher functional longevity than HF cows (Bannerman et al., 2008a).

Milk somatic cells inside the healthy mammary gland may offer some degree of protection against mastitis through their ability to recognize pathogens and induce a quick inflammatory response (Kehrli. and Shuster, 1994). This may account, in part, for the association between lower milk SCC and increased risk of CM and the severity of the disease (Bannerman et al., 2008a; Bradley and Green, 2001). Large-scale surveys have shown that JE cows in the United State and Canada have higher average milk SCC than HF (Caraviello et al., 2005; Sewalem et al., 2006). This information suggests that a higher SCC may protect JE from CM, which would explain the lower incidence of CM rate in JE compared to HF cows. However, according to Bannerman et al. (2008a) the increased milk SCC before mastitis infection

does not give a protective advantage to the JE cows. Regarding pre-infection milk somatic cells, a rapid increase of milk SCC in HF cows by $78,300 \pm 27,690$ cells/ml within 6 hours after the onset of CM did not appear to decrease the severity of CM in HF cows over that of JE cows, which had no increase in milk SCC during this period. During acute mastitis, neutrophils constitute more than 90% of milk somatic cells but the increased number of recruited neutrophils did not correspond to an overall SCC increase that was higher in HF than JE cows (Bannerman et al., 2008b). This finding may therefore be attributed to a change in the composition of cell types rather than an absolute increase of SCC in HF.

The milk production decline may be proportional to the severity of mastitis and the cow's ability to fight off the infection while still producing milk. A study reported that within-breed differences in milk production did not affect the severity of *E. coli* mastitis, as both low and high producing cow coped equally with the demand for milk production (Kornalijnslijper et al., 2003). This finding may also apply to cows of different breeds as several studies showed that both HF and JE cows developed IMI after being experimentally infused with *E. coli* and *Staph. aureus* regardless of the level of milk production (Bannerman et al., 2008a; Bannerman et al., 2008b). Hence, milk production apparently does not affect the severity of mastitis. However, selection for high milk production has also selected for a faster milk flow, which, together with increased pressure due to large volume of milk, may cause increased milk leakage in high producing cows, and that is associated with an increased risk of CM (Bannerman et al., 2008b; Kornalijnslijper et al., 2003). Whether the differences in pre-infected SCC in different breeds is the underlying influence of the ability to response to mastitis infection is still disputed.

2.2.2 Heterosis effects for mastitis resistance

Genetic improvement of dairy cattle results from the selection within breeds, across breeds, and crossbreeding (Harris, 2005). *Hybrid vigour* or *heterosis* is the genetic effect resulting from crossbreeding and is measured as performance of crossbred progeny that could be above or below that of the average performance of their parents depending on the trait (Bryant et al., 2007). The effect is greater in first-crosses (F_1 , coefficients of heterosis > 50%) than in later-crosses animals (coefficients of heterosis > 25%), and greater when the differences between the parental breeds are largest (Holmes et al., 2002).

Heterosis is the result of non-additive heterozygous gene interaction. It usually has low heritability and also exists when the average of the F_1 individual differs from the average of the two parental breeds. This indicated that additive gene action is not responsible for hybrid vigour because the heritability of a trait is low and that the average performance of F_1 would closely approximate the average of the parents (Lasley, 1972). Legates and Warwick (1990) suggested that most traits of economic importance were influenced by additive gene action except for reproductive performance and vigour that were influenced by heterosis. Heterosis can be of additional economic benefit, but the extent of heterosis is not well established for many breed combinations, and effects of heterosis are not heritable. However, some favourable heterosis effects for important economic traits are well understood, and crossbreeding becomes an increasingly common practice (Freyer et al., 2008).

New Zealand farmers began to crossbreed in the 1960s, initially hoping to change from JE to HF. Since the 1980s, the overseas HF genetics became more popular. However, the daughters of these animals were heavier and seemed to be less fertile and had decreased survival rates than JE. Thus, the New Zealand dairy farmers have not moved to overseas HF (Vanderick et al., 2009). In 1985, the increasing trend of using HF bull semen for artificial

breeding levelled out (Montgomerie, 2005). Since then, crossbreeding has been a strategy adopted in itself rather than as a method for changing from one breed to an alternative. Crossbreds (defined as cows with less than 14/16 of single breed ancestry) are increasing in proportion as replacements, reared for the New Zealand national herd (Montgomerie, 2005). In Denmark, 24% of dairy farmers would consider starting crossbreeding programmes within their herds as a result from a Danish crossbreeding experiment. This experiment included 1,680 cows from three breeds and their crosses. The result showed that at least 10% heterosis could be expected for total merit, mainly due to increased longevity and improvement of functional traits. A minor part of heterosis for total merit is due to heterosis for production traits (Sorensen et al., 2008).

New Zealand has had across-breed genetic evaluation since 1996, a common across-breed breeding objective called Breeding Worth (BW), and has extensive linkages between cows of different breeds and crosses in large CG (Harris, 2005). The across-breed genetic evaluation system provides estimates of the average hybrid vigour in all animals. In 2005, Montgomerie (2005) published the estimates of hybrid vigour from the across-breed genetic evaluation system for all the traits in the BW index and for the crosses between HF and JE, HF and AY, and AY and JE.

Heterosis can be measured by comparing average performance of the F₁ offspring with that of the two parental breeds by the following formula.

$$\% \text{heterosis} = \frac{(\text{mean of } F_1) - (\text{mean of parental breeds})}{\text{mean of parental breeds}} \times 100$$

Estimates of heterosis for production, fertility, SCC and longevity in New Zealand dairy are shown in Table 2.3.

Table 2.3: Hybrid vigour estimates for three first crosses of cows, expressed in genetic standard deviation (SD) units.

Traits	SD	HF x JE	HF x AY	JE x AY
Milk fat (kg)	13.3	0.70	0.33	0.73
Milk Protein (kg)	9.1	0.67	0.37	0.69
Milk Volume (kg)	329.3	0.46	0.29	0.49
Live weight (kg)	19.6	0.33	0.003	0.48
Cow fertility %	6.4	0.60	0.59	0.38
SCS (log ₂ SCC)	0.4	-0.14	-0.37	-0.11
Longevity (days)	217.0	0.96	0.58	0.55

Source: Montgomerie (2005).

HF = Holstein Friesian

JE = Jersey

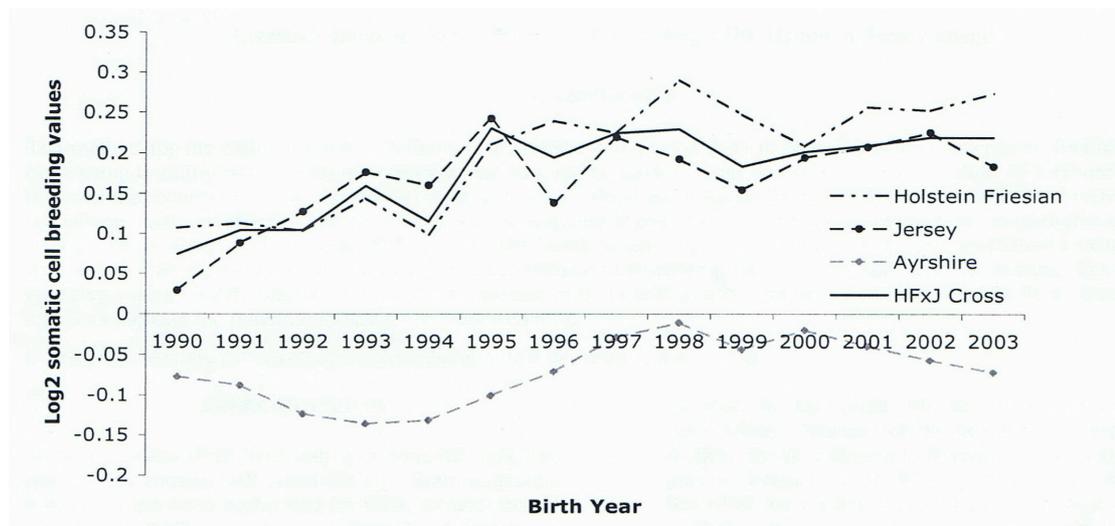
AY = Ayrshire

Table 2.3 shows that there are differences in term of production, fertility, SCC and longevity for different combination of crossbred cows. It also emphasises crossbreeding as an important method to reduce the frequency of IMI. Table 2.3 also shows that HF x AY has the lowest incidence of SCS at -37, which was the best result compared to -14 for HF x JE and -11 for JE x AY. The most important message drawn from this table is that IMI can be reduced through crossbreeding systems and that heterosis effects can be more significant in non-production traits than in production traits.

Another study investigating the effect of crossbreeding on mastitis resistance showed that the first cross between HF and JE has a small reduction of SCS of 0.8% and 0.6% of the SCS mean compared with the average of the parental breeds for first lactation and second/third lactation records, respectively (Harris et al., 2005). This result is similar to that of Montgomerie (2005). Although the difference between the two studies may be that the study of Montgomerie (2005) came from the average of multi-parous cows rather than on first and second/third lactation.

Heterosis estimates for diseases in dairy cows from a Danish experiment (Sorensen et al., 2008) found that in later crossbred generations, in which more breed combinations appeared, significant negative heterosis estimates were found for metabolic disease, reproduction diseases and leg and claw disease, whereas large positive heterosis estimates were found for mastitis. The result for mastitis of this study was in contrast to several other studies. This was perhaps due to the use of a repeatability model that did not account for unequal number of lactations among purebred and crossbred cows, with crossbred cows having more frequency of later lactation. Also this experiment had no correction for yield, which will be a disadvantage for high producing crossbred cows because of the negative correlation between mastitis and yield.

According to Harris et al. (2005), the heritability of SCS ranging between 0.06 – 0.21 indicate that selection for reduced SCS would reduce mastitis susceptibility. Also from the same study, the national genetic trend in the cow population for SCS by breed is shown in Figure 2.1. The rate of a genetic trend for SCS are 0.03, 0.02, 0.01 and 0.02 genetic SD units per year for HF, JE, AY and HF x JE crossbred cows, respectively.



Source: Harris et al. (2005)

Figure 2.1: Genetic trend for SCC in the national cow population.

The values of SCS are low compared to the genetic gain for milk production of about 0.2 genetic SD units per year. However, the positive genetic trend in SCS (Figure 2.1) is undesirable for HF, JE, and HF x JE but not for AY (Harris et al., 2005).

In most western countries, the breeding goal has changed in recent years from being primarily focused on milk production and udder conformation to include functional traits such as fertility, health and longevity. The reason for this change comes from observed deterioration of functional traits, a problem that crossbreeding may help to overcome (Sorensen et al., 2008). Although one study showed a negative impact of heterosis effect on mastitis resistance, evidence from other studies encouraged the use of crossbreeding for improved resistance, which could be expected to enhance cow health and welfare.

2.2.3 Genetic selection for mastitis resistance

Effective selection tools have become available in many countries and new developments in the areas of genomics and proteomics promise to improve genetic selection for mastitis resistance effectiveness. This, together with the increasing concerns over the use of antibiotics, the need to reduce costs of production at the farm level, and the restriction of labour on ever increasing farm size, present the challenge of finding the alternative approach that will control mastitis effectively (Hogeveen, 2005). According to Vallimont et al. (2009) increased mastitis incidence was genetically correlated with higher SCS (range 0.66 – 0.88) and was generally correlated with higher yield (range -0.03 to 0.40). This was in agreement with Heringstad et al. (2000) but his result of the estimated genetic correlation between mastitis susceptibility and milk yield was 0.43, which was higher than that of Vallimont et al. (2009) and the mean of 0.30 from the studies reviewed by Emanuelsson et al. (1988). This figure indicates that if mastitis was ignored in a breeding programme, the large weight traditionally placed on increasing milk production would have a negative effect on mastitis resistance. Under a traditional breeding programme

without selection for mastitis, the genetic increase of 0.02 cases of mastitis per cow per year is expected, assuming a genetic correlation between mastitis and milk yield of 0.30. The rate of change in mastitis may seem to be low, but the increase over a long-term perspective is disconcerting (Heringstad et al., 2000).

Previous studies have shown that selection for mastitis resistance is possible because the relevant traits of the immune system are heritable (Colleau and Le Bihan-Duval, 1995; Rupp and Boichard, 2000). However, it is difficult to select CM directly due to poor records (except in the Scandinavian countries) and low heritability (Carlén et al., 2004; Emanuelson and Philipsson, 1988; Rupp and Boichard, 2003; Simianer et al., 1991). Therefore, selection for indirect traits such as SCC has been considered in the selection. Animal breeders use SCC to quantify the cow's inherited sensitivity to CM and SCM (Shook, 1989), while farmers and veterinarians use SCC to detect subclinical intramammary infections (Schukken et al., 2003). Three reasons for choosing SCC as the indirect trait for mastitis resistance are: SCC is routinely recorded in most milk recording systems, SCC has higher heritability than CM, and genetic correlations between both traits are moderate to high. Therefore, it is possible that selection to decrease SCC would reduce the incidence of CM and SCM (de Haas et al., 2008). However, indirect response of CM will depend on the strength of genetic correlations between SCC and CM (Heringstad et al., 2000; Rupp and Boichard, 2003). Estimates of the genetic correlation between SCC and mastitis ranges between 0.3 - 0.8, with an average of 0.7. Heritability of SCC is slightly higher than mastitis at about 0.15 (Emanuelson and Philipsson, 1988; Mrode and Swanson, 1996).

Scandinavian countries (Norway, Finland, Denmark and Sweden) have investigated a genetic selection for mastitis resistance in dairy cattle since 1978, and applied the results into breeding practice in the late 1990s (Sorensen et al., 2000). Accumulated information on selecting against mastitis showed that most studies were carried out in first lactation cows in order to

avoid the bias of culling cows with high SCC, which are relatively rare in first lactation. Since it is desirable to reduce the lactation incidence of mastitis for cows at any parity, it is of interest to estimate genetic correlations for mastitis resistance in different lactation periods (Sorensen et al., 2000). Another study suggested that the frequency of mastitis and the level of SCC would increase with increasing lactations (Carlén et al., 2004). Thus, it is important that selection programmes that have mastitis resistance trait in the farmer-breeding programmes should also consider lactation number (Carlén et al., 2004).

A study showed that cows that had moderate to high SCC before being challenged to mastitis pathogens responded with a lower incidence of infection than cows that had low SCC prior to the challenge (Schukken et al., 1994). These findings were also supported by studies on SCC and CM at herd level: herds with a high proportion of cows with low SCC or low bulk milk SCC had a higher incidence of CM than herds with high SCC (Beaudeau et al., 2002; Elbers et al., 1998; Waage et al., 1998). However, the opposite result was concluded, by researchers investigating the correlation between SCC at a given time and the subsequent occurrence of mastitis, defined by either high SCC or observed CM in later lactation (Rupp and Boichard, 2003). The results of these studies showed that cows with the lowest SCC (> 35,000 cells/ml) at the first test in the first lactation were at the lowest risk to be affected by CM later in the first lactation or at the beginning of the subsequent lactation (Rupp and Boichard, 2003). Cows with highest mean SCC level in the first lactation were at the highest risk of experiencing CM in the second lactation (Rupp et al., 2000).

Thus, studies have reached different conclusions about the risk of low or high SCC cows for subsequent CM. Therefore, further investigation is needed to explain the dissimilarity between studies based either on natural occurrence of infections or experimental infections. Many results tend to show that, at least in natural infections and on individual basis, selection for reduced SCC should be effective to reduce CM incidence in a breeding programme (Carlén

et al., 2004; Emanuelson and Philipsson, 1988; Kehrlı and Shuster, 1994; Rupp and Boichard, 2003). This result is valid under the current situation and will need regular testing for the long term. In addition, it would be helpful to have a better understanding of defence mechanisms that are involved and altered by a selection on phenotypic traits (Rupp and Boichard, 2003).

Although the correlation between SCC and CM indicates that both are expressions of udder health, they are not the same trait (de Haas et al., 2008). High SCC indicates increase cell count for a long period, when recorded frequently, thus reflects long duration of SCM cases, while the use of CM records ignores subclinical cases. Also long-term SCM, which is frequently caused by *Staph. aureus* activates the specific immune system, while CM is of short-term duration being more frequently caused by *E. coli* that activates the innate immune system (Heringstad, 2000). According to Shook and Schutz (1994) SCC monthly sampling only detects about 10 – 20% of the infections. Selection on SCC as an indirect trait against mastitis will be more effective than direct selection on CM when progeny group size are small (Heringstad, 2000). This result is in agreement with Philipsson et al. (1995) and Weller et al. (1992). Although SCC is used widely as a measure of SCM and CM in many countries, there are some disadvantages associated with its use. Studies have shown that the use of SCC alone to identify udder quarters as infected or not infected can be unreliable (Harmon, 1994). A study by de Haas et al. (2008) concluded that to improve the overall udder health (both CM and SCM), it might be more successful to combine several SCC traits in an udder health index than a single SCC measure. In addition, the use of SCC as a predictor of mastitis will depend on the level of mastitis in the herd. It will be a good predictor in high incidence herds, whereas it will be less useful in herds with a low mastitis incidence (Kehrlı and Shuster, 1994). Although indirect selection on SCC involves some risks and limitations, in countries where direct selection for CM is difficult apply, it is better to use indirect selection on SCC than completely ignoring mastitis in the breeding programme (Heringstad et al., 2000; Philipsson et al., 1995).

2.3 Current practice and its limitation

Regarding the complexity of mastitis resistance, the best approach for genetic selection to reduce mastitis susceptibility is to combine the information on CM and SCC, which have proven to be the most effective measures regardless of progeny group size. Where only one of the traits was considered, SCC was more effective than CM when the progeny groups were small, while CM was more effective than SCC when the progeny groups were large (Philipsson et al., 1995). According to Ødegård et al. (2003) the selection based on CM records results in 43% more efficiency than indirect selection using SCS. Therefore, combining SCC, udder type traits and milking speed would improve the efficiency of genetic selection when the information on CM was unavailable (Boettcher et al., 1998). With respect to genetic selection, selection for lower SCC and individual breed to improve mastitis resistance give slower but accumulating effects. Furthermore, it lower cost and less effort for the long-term perspective to combat mastitis problem compared to the management measures that can be difficult to control (Shook and Schutz, 1994).

CHAPTER 3

MATERIALS AND METHODS

3. MATERIALS AND METHODS

3.1 Data

The data set used in this study was extracted from Livestock Improvement Cooperation (LIC) database. Records on CM collected during the seasons 2005 - 2006 and 2008 - 2009 from 53,419 cows of different breeds including HF, JE and crosses of HF and JE (XB). The average number of herd tests per lactation was 3.26. The percentage of cows had repeated lactations were: lactation 1 - 54.98%, lactation 2 - 26.92%, lactation 3 - 12.07% and lactation 4 - 6.03%. About 16.1% of first lactation cows had a record in the subsequent lactation. The cows were the progeny of 641 sires and were distributed in 167 dairy herds used for the progeny testing of bulls. Animal information such as sire, dam, breed of the cow, farm location, season, parity, and herd-test for daily milk yield, fat yield, protein yield and SCC was available for each cow and in each lactation. Data were edited using SAS (Statistical Analysis System, version 9.1 SAS institute Inc., Cary, NC, USA). The structured query language (proc sql) was used to sort, summarise, merge and create new variables and to combine datasets. Cows without any information on their breed composition, calving before the age of two years, or having more than 10 parities were excluded from the study. Herds with less than 100 cows were excluded from the analysis. Clinical mastitis was coded as a binary trait; cows that presented at least one reported case of CM at any day at risk in the season were coded as “1” and “0” for no recorded CM.

3.2 Statistical Analysis

3.2.1 The proportion of genes from each breed was calculated for each animal using the simple equation:

$$p_i = (s_i + d_i)/2$$

where:

p_i is the proportion of genes from breed i in the progeny

s_i is the proportion of breed i in the sire and

d_i is the proportion of breed i in the dam

Breed composition of each cow was described in terms of proportion of HF (proportion of HF \geq 87.5%), and JE (proportion of JE \geq 87.5%) and XB (cows with either HF or JE less than 87.5%).

3.2.2 Coefficient of HFxJE breed heterozygosity was calculated using the following identity:

$$h_{\text{HFxJE}} = p_s^{\text{HF}} p_d^{\text{JE}} + p_s^{\text{JE}} p_d^{\text{HF}}$$

where:

h_{HFxJE} is the coefficient of expected breed heterozygosity between proportion of HF and JE in the progeny

p_s^{HF} is proportion of HF in the sire

p_d^{JE} is proportion of JE in the dam

p_s^{JE} is proportion of JE in the sire

p_d^{HF} is proportion of HF in the dam

The definition of CG was cows that calved in the same herd and year. There were 356 CG in this study.

3.2.3 Descriptive statistics of CM and milk production traits for HF, JE and XB were analyzed using SAS version 9.1. Phenotypic means for each breed group for lactation average of daily yields of milk, fat and protein and SCS were obtained using the GLM procedure with a linear model that included the fixed effect of breed. Multiple comparisons between means of breed groups were performed. Somatic cell score was calculated as \log_2 (SCC)

3.2.4 A repeatability animal model (Mrode, 2005) was used to estimate breed and heterosis effects and variance component as follow:

$$y_{ijkmn} = hy_i + \text{month}_j + \text{parity}_k + \beta_1 p_{JE} + \beta_2 h_{HF \times JE} + \text{animal}_m + \text{cow}_n + e_{ijkmn}$$

where:

- y_{ijkn} is the observation for CM recorded in the cow n , which was in i th CG, calving in the month j , of parity k ;
- hy_i is the fixed effect of CG i ;
- month_j is the fixed effect of calving month j ;
- parity_k is the fixed effect of parity group k ;
- β_1 is the fixed linear regression coefficient of CM on proportion of JE (HF was fixed to zero for comparison);
- β_2 is the fixed linear regression coefficient of CM on coefficients of expected HF x JE breed heterozygosity;
- animal_m is the random animal additive effect of animal m ;
- cow_n is the random permanent effect of cow n ; and
- e_{ijkmn} is the random residual effect unique to observation y_{ijkmn}

The model was used to obtain variance components for incidence of CM using ASReml (Gilmour et al., 2002). The regression on HF breed proportion was excluded from the model in order to avoid linear dependencies with JE. The random effects of animal, cow, and residual were assumed to be normally and independently distributed with mean equal to zero. Heterosis effects for CM were expressed as a percentage of the mean of purebred HF and JE.

3.2.5 Heritability and repeatability of breeding value for CM were calculated using the following formulae:

Heritability was calculated as:

$$h^2 = \sigma_a^2 / (\sigma_a^2 + \sigma_c^2 + \sigma_e^2)$$

Repeatability was calculated as:

$$r = (\sigma_a^2 + \sigma_c^2) / (\sigma_a^2 + \sigma_c^2 + \sigma_e^2)$$

where:

σ_a^2 is additive animal genetic variance

σ_c^2 is cow permanent variance

σ_e^2 is residual variances

3.2.6 Sire breeding values were estimated using the pedigree file that included parents and grandparents of the cow. The sire breeding value was calculated as follow:

$$BV = (\text{breed_effect} \times \text{prop_Jersey}) + u$$

where:

breed_effect is breed effect

prop_Jersey is the proportion of JE in the sire

u is the solution for animal effect after solving the mixed model equations.

CHAPTER 4

RESULTS

4. RESULTS

The comparison of daily milk yield between breeds of New Zealand dairy cattle is shown in Table 4.1. Holstein-Friesian had the highest milk yield, followed by XB and JE. Holstein-Friesian had the same fat yield as XB, which was higher than JE cows by 0.08 kg. Holstein-Friesian had the highest protein yield compared to JE and XB. Somatic cell score was highest in HF followed by JE and XB respectively. The average milk yield for New Zealand dairy cows across breed was 16.67 kg, with fat yield of 0.80 kg, protein yield of 0.62 kg. The average SCS was 6.43.

Table 4.1: Daily milk, fat and protein yields and SCS of breeds of New Zealand cows.

Breed	Milk yield (kg)	Fat yield (kg)	Protein yield (kg)	SCS ¹ (units)
HF	19.16 ^a	0.83 ^a	0.68 ^a	6.49 ^a
JE	13.51 ^c	0.75 ^b	0.54 ^c	6.43 ^b
XB	17.34 ^b	0.83 ^a	0.65 ^b	6.38 ^c

¹SCS = somatic cell score calculated as \log_2 (somatic cell count).

^{a,b,c} means with the different superscripts within the same column differ significantly ($P < 0.001$)

The incidence of CM for HF, JE and XB cows is summarised in Table 4.2. Jersey cows had the lowest incidence of CM followed by crossbreds and HF cows. The cumulative lactation incidence of CM was 11.17% in 92,961 lactations.

Table 4.2: Incidence of CM for different breeds in New Zealand

Breed	0	1	Total
HF (n)	32,148	4,617	36,765
%	87.4	12.6	
JE (n)	9,365	962	10,327
%	90.7	9.3	
XB(n)	41,066	4,803	45,869
%	89.5	10.5	
Total (n)	82,579	10,382	92,961
%	88.8	11.2	

Note: 0 = no CM recorded during lactation
1 = at least one case of CM was reported

Table 4.3 shows correlations between CM and milk production traits. The correlation coefficients between CM and daily yields of milk, fat and protein were close to zero.

Table 4.3: Phenotypic correlations (1st row) and p-value (2nd row) between milk traits, SCS, and incidence of CM in New Zealand dairy cows

	Fat yield ¹	Protein Yield ¹	SCS ²	CM ³
Milk yield ¹	0.85 <.0001	0.96 <.0001	-0.05 <.0001	-0.004 0.293
Fat yield		0.91 <.0001	-0.03 <.0001	-0.025 <.0001
Protein yield			-0.028 <.0001	-0.002 0.493
SCS				0.184 <.0001

¹ Milk traits are daily yields during the lactation of milk yield, fat yield, and protein yield.

² SCS is the average somatic cell score during the lactation, $SCS = \log_2 (SCC)$.

³ CM = clinical mastitis defined as 1 for cows that presented at least one case of clinical mastitis during the lactation and 0 for healthy cows

The correlation between milk yield and protein yield was higher than that of milk yield and fat yield. CM had a small but positive phenotypic correlation with SCS of 0.184 ($P < 0.001$) indicating that as SCS increases incidence of CM increases. The total number of cases of CM in the data set was 12,144 but some cows had repeated cases of CM during the lactation resulting in 10,382 lactations that presented at least one case of CM during the lactation.

The distribution of 12,144 cases of CM during the lactation considering all the 92,961 lactations is shown in Figure 4.1. The majority of cases of CM occurred within the first 30 days of lactation. When comparing mastitis incidence by lactation number (Table 4.4), mastitis incidence was higher for the first lactation cows than for cows in later lactations. However, from the seventh lactation mastitis incidence was higher than that of the first lactation, indicating that as the cows get older the incidence of CM also increases.

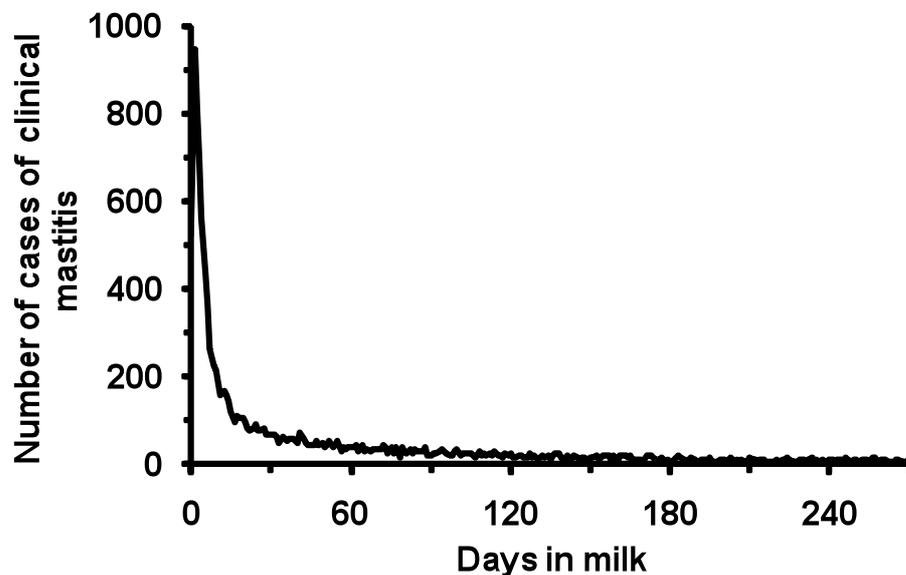


Figure 4.1: Number of cases of CM during the lactation.

Table 4.4: Estimates of incidence of CM in different lactation number.

Lactation No.	Cows (n)	Estimate \pm SE (%)
1	19794	13.0 \pm 0.33
2	16453	7.0 \pm 0.34
3	14298	8.2 \pm 0.35
4	12197	9.6 \pm 0.37
5	9726	10.8 \pm 0.40
6	7612	12.6 \pm 0.43
7	5543	13.6 \pm 0.48
8	3713	14.0 \pm 0.55
9	2310	14.5 \pm 0.68
10	1312	15.4 \pm 0.88

Estimates of variance components for incidence of CM are shown in Table 4.5. Heritability and repeatability for the incidence of CM were 0.015 ± 0.003 and 0.070 ± 0.005 , respectively. Estimates of genetic, permanent environmental, residual and phenotypic variances were 0.001 ± 0.0002 , 0.005 ± 0.0005 , 0.085 ± 0.0006 and 0.091 ± 0.0004 respectively.

Table 4.5: Estimates of variances, heritability and repeatability for incidence of CM in New Zealand dairy cows.

Parameter	Estimate
Genetic variance (σ^2_a)	0.001 ± 0.0002
Permanent environmental variance (σ^2_{pe})	0.005 ± 0.0005
Residual variance (σ^2_e)	0.085 ± 0.0006
Phenotypic variance (σ^2_p)	0.091 ± 0.0004
Heritability (h^2)	0.015 ± 0.003
Repeatability (r)	0.070 ± 0.005

Table 4.6 shows the number of sire across breeds. Figure 4.2 shows considerable variation between estimates of breeding values (EBVs) of sires for CM with values ranging from -8 to +8%. Breeding values of JE sires tended to be negative and lower than breeding values of HF sires most of which were positive, whereas XB sires had intermediate estimated breeding values.

Table 4.6: Descriptive statistics of sires to EBVs for CM and breed

Breed	Total (n)	Mean	Std. Dev.	Min	Max
HF	2426	-0.1	149.5	-8.1	10.0
XB	406	-0.8	19.8	-4.2	4.7
JE	1798	-1.7	124.0	-8.4	6.0

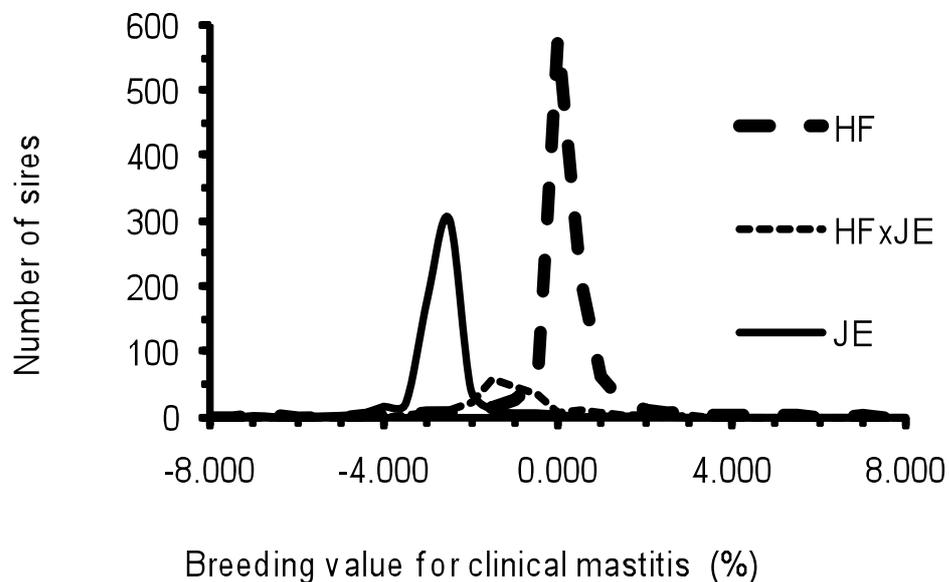


Figure 4.2: Distribution of sires according to EBVs for CM and breed (HF = Holstein-Friesian, JE = Jersey).

Breed and heterosis effects obtained from the repeatability animal model are shown in Table 4.7. Using estimates of the regression coefficients for breed and heterosis it was calculated that incidence of CM in HF cows was 2.9% higher than incidence of CM in JE cows. Crossbreeding between HF and JE further decreased CM incidence by 1.25% compared to the average of CM incidence in HF and JE, which was 13.0% lower than the average of the parental breeds (11.7%).

Table 4.7: Breed and heterosis effects for incidence of CM in New Zealand dairy cows.

Breed effect	%
Holstein-Friesian	13.2 ± 0.31
Jersey	10.3 ± 0.40
Heterosis Holstein-Friesian x Jersey	-1.25 ± 0.40

CHAPTER 5

DISCUSSION

5. DISCUSSION

As a result of efficient selection on production traits as the main breeding objective, it has exacerbated mastitis to become and continued to be an expensive disease for any dairy industry despite the improvement in antibiotic therapies, milking protocols and management practices. Therefore, it is accepted in the dairy industry that production and functional traits, such as mastitis resistance, should be included in the breeding programme in order to prevent further decline in animal welfare as well as to meet the economic and ethical concerns of consumers and farmers. The findings for different dairy breeds and heterosis of the incidence of CM for this study are comparable to the results from other studies.

5.1 Breed differences and mastitis incidence

Breed differences have shown to have significant effects on total milk production ($P < 0.001$); HF cows have higher milk yield, fat yield and protein yield than JE cows (Coulon and Remond, 1991). In this study, HF had higher level of SCS than JE cows, which agrees with results of a genetic evaluation (Harris et al., 2005). However, the result from this study is in disagreement with Berry et al. (2007) who found that JE cows had a higher SCS compared to HF cows. VanRaden and Sanders (2003) also reported a higher SCS in JE than HF in the first lactation but not across all lactations. Similarly, a Canadian study by Sewalem et al. (2006) reported a higher SCC for JE cows than HF cows.

The cumulative lactation incidence of CM in this study was 11.2% in 92,961 lactations; this value is lower than the value of 23.3% reported in Norwegian dairy cattle (Heringstad et al., 2003) and 23.0% of Canadian dairy cattle (Olde Riekerink et al., 2008). The result in this study was similar to 10.1% reported in Swedish dairy cattle (Carlén et al., 2009) but higher than 7.6% reported UK dairy cattle (Kadarmideen et al., 2001). The large variation of cumulative CM incidence can be influenced by many factors including:

selection criteria, country, management practice, confined vs. outdoors grazing, environmental conditions, sampling season, method of data collection, the definition of CM and distribution of pathogens causing mastitis (Olde Riekerink et al., 2008).

Despite the difference of SCS between breeds and cumulative CM incidence compared to other studies, the current study shows that the incidence of CM was higher in HF cows compared to JE cows, which was consistent with previous studies (Berry et al., 2007; Washburn et al., 2002). A lower incidence of CM in the Swedish Red than Swedish HF was reported (Waller et al., 2009). In addition, Myllys and Rautala (1995) found a lower incidence of CM in AY cows compared to HF in a Finnish study. One factor that distinguishes HF from other breeds is high milk production, which has made it the most popular dairy breed in dairy industry worldwide.

It is widely accepted that there is a positive genetic correlation between milk production and SCS and between milk production and incidence of CM (Emanuelson et al., 1988; Lund et al., 1999; Heringstad et al., 2000; Carlén et al., 2004). This means that the continuation of high milk yield selection will result in the increasing rate of CM incidence. Lopez-Villalobos and Spelman (2010), using the same data set as in this study, estimated a genetic correlation between milk yield and incidence of CM of 0.26. The correlation may be small but could accumulate over time with negative impacts on animal welfare and compromise genetic gains in production. Therefore, high incidence of CM that occurs in HF cows compared to other breeds is not unexpected. Other possible factors that may influence the difference for CM between HF and JE cows are: 1) a variation of innate mastitis resistance of each breed, i.e. JE may have more efficient immune response than HF cows: and, 2) differences in management systems between HF and JE herds. For the current study, the lower mastitis incidence favoured JE over HF and crossbreds.

5.2 Lactation periods and the incidence of CM

In the current study, the majority of CM cases occurred within the first 30 days of lactation. A similar pattern was reported in New Zealand (Berry et al., 2007), Danish (Lund et al., 1999), Norwegian (Heringstad et al., 2003) and Swedish (Carlén et al., 2009; Carlén et al., 2004) dairy cattle. The majority of outbreaks that occur in different stages of lactation may occur for different reasons other than exposure to mastitis - causing pathogens. High frequency of CM occurring at the beginning of lactation may be exacerbated by the physiological stress of calving, rapid increase in milk yield and negative energy balance (Lund et al., 1999; Suriyasathaporn et al., 2000). Generally, the average milk production of individual cows in early lactation is higher than that observed in mid-lactation regardless of breed, energy supply, and the cows' physiological disposition because energy requirements are almost never met during this period. Therefore, the milk output depends on the efficiency of utilization of metabolizable energy for milk production in early lactation (Coulon and Remond, 1991).

The composition of colostrum is markedly different from the milk obtained at mid-lactation. The most noticeable difference is a much higher concentration of immunoglobulins, i.e. proteins containing antibody passed from dam to calf. It also contains large numbers of somatic cells. Even in healthy cows not affected by mastitis, the SCC in milk is over 1 million cells/ml up to two days after calving. The proportion of neutrophils, macrophages and lymphocytes change rapidly around the time of parturition: SCC usually decreases to less than 300,000 by day 3, and to less than 100,000 by day 7. After about two days milk composition changes from that of colostrum to milk with a particular increase in lactose concentration (Holmes et al., 2002). This suggests that the high incidence of CM at the beginning of lactation is associated with the cow coping with high physiological demands around calving, which will affect the cow's genetic resistance to CM. Whereas, the incidence of CM later in the lactation much more depends on environmental challenges (Lund et al., 1999).

A concentration effect may explain the increasing SCC that occurs in uninfected quarters towards the end of lactation (Prendiville et al., 2010). Even in uninfected healthy cows, the reduction of daily milk yield to low levels (below 5 L/day) is observed, a consequence of restricted feed availability, a reduction in milking frequency from twice daily to once-a-day, and a consistently high SCC in some cows are contributing factors for an increasing CM incidence in late lactation (Holmes et al., 2002).

First calving uninfected two-year-old cows often have slightly higher post-calving SCC than older cows, and they remain higher until about 10 days in milk. For cows that are sub-clinically infected at calving, SCC usually remains at about 400,000 for at least two weeks or even longer (Holmes et al., 2002). Moreover, the mastitis incidence was also higher in the first than in later lactations. The current study also found that the CM incidence was high at the first, decreased by almost 40% in the second and increased in subsequent lactations until the level of the first lactation was reached at the seventh lactation, and further with increasing parity. A similar pattern was reported by Carlén et al. (2004) though their study only included the first 3 lactations in Swedish HF cows. This pattern coincides with a prime production age of 5 - 6 years (DairyNZ and LIC, 2008).

5.3 Selection for CM

Heritability and repeatability for the incidence of CM were 0.015 ± 0.003 and 0.070 ± 0.005 , respectively. The estimate of heritability is similar to the average value of 0.04 reported by Mrode and Swanson (1996) and in agreement with other studies that had results ranging from 0.001 – 0.06 (Heringstad et al., 2000). The wide range of heritability value for the incidence of CM may be explained by the size of data set. The heritability value for this study was based on a large data set collected under field conditions. Therefore, the heritability value was lower compared to designed field studies that tend to have smaller data set and higher estimates of heritability for CM (Lyons et al., 1991; Pryce et al., 1997). Carlén et al. (2008) indicated that the

low value of heritability has often been misinterpreted as meaning that genetic selection to improve the innate resistance has a limited role to play in mastitis control programmes. Dairy cattle breeding programmes in the Scandinavian countries include low heritability traits such as health and fertility and were associated with a reduction in mastitis incidences. Despite a low CM heritability, direct selection for mastitis resistance may be beneficial and the resulting genetic productivity gain can be substantial, as long as proper recording and the progeny group size is adequately large (Heringstad et al., 2000).

As shown in Figure 4.2, considerable genetic differences exist between bulls. The estimated breeding values (EBV) for CM ranged from -8 to +8% with breeding value (BV) for JE bulls being lower than for HF bulls, and intermediate BV for XB bulls. Similarly, the average BVs for SCC reported by DairyNZ and LIC (2008) were 0.33 for JE and XB bulls, and 0.41 for HF bulls. Nash et al. (2000) reported that sires selected for low SCS BVs had progeny that had low incidence of CM and small number of clinical cases during the first and second lactations. The daughters from sires with high SCS BVs had high CM incidence and total number of clinical cases during the first lactation. This is also in agreement with a study on sire evaluations from the US, Denmark and Sweden, which found that sires with the lowest genetic evaluation for SCS also had the most favourable evaluation for CM (Rogers et al., 1998). Studies by Nash et al. (2000) and Rogers et al. (1998) support selection of bulls with the lowest SCS BVs. This does not support the theory that selection for the lowest SCS will result in dairy cows that are unable to respond to mastitis infection. If this was true then the lowest SCS would be associated with a higher number of clinical cases, and an intermediate SCS would provide optimal resistance to mastitis (Nash et al., 2000). This theory originated from the results of experimental studies controlling for environment, which suggested that elevated SCC before the infusion of mastitis causing pathogens would protect the cow against infection (Nash et al., 2000). In summary, most studies provided evidence that selection for lower SCS may

reduce both the incidence of CM and the number of clinical cases per lactation without reducing the ability to respond to infection.

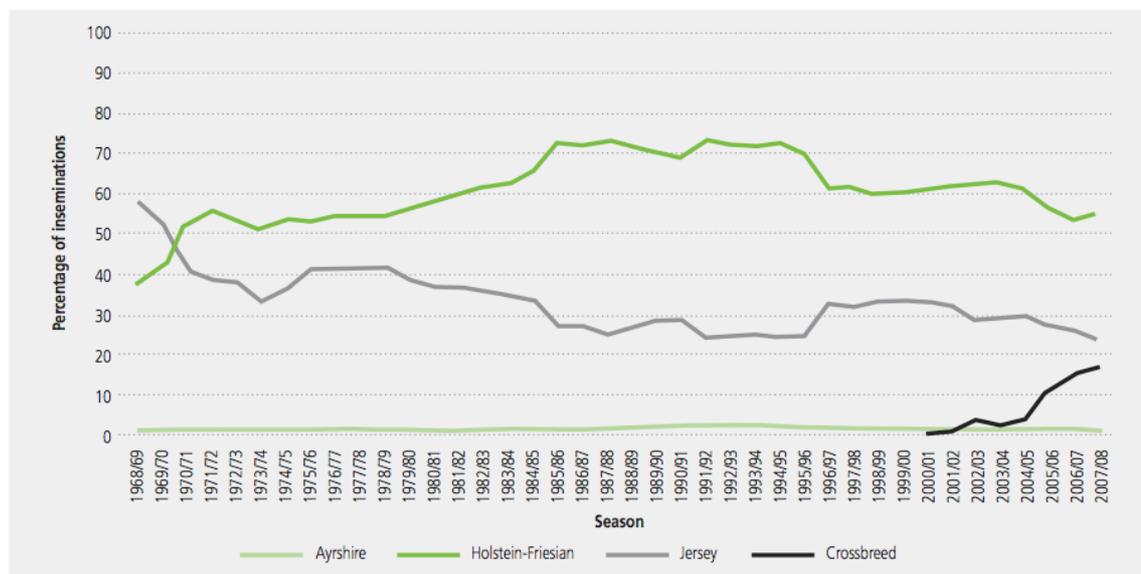
5.4 Heterosis effect and its potential in combatting mastitis in dairy cattle

Heterosis, or hybrid vigour is a measure of the differences between a crossbred and the average of its purebred parents. If a difference is not positive enough to be greater than the best of the purebreds, crossbreeding may not be beneficial. However, the overall economic merit of a combination of traits may justify crossbreeding. Dairy industries were generally trying to use heterosis for improving fertility and longevity rather than milk production. In reality, no breed can compete with HF in terms of milk production. Therefore, HF makes up the vast majority of dairy cows in the population, for example 92% in Canada (Murray, 2002). There have been concerns about high culling rates, low fertility, inbreeding, and poor health and fitness traits in traditional HF dairy breed. All pure breeds are by definition inbred to some extent and increased inbreeding in a population tends to accumulate undesirable recessive genes, which in dairying, depresses performance. As two breeds become more and more inbred, the potential benefit of a heterosis effect from crossbreeding different breeds increases (Murray, 2002).

Many studies found differences between dairy breeds for mastitis resistance by determining genetic and phenotypic correlations between CM and milk production. As the result of crossbreeding researches also found evidence of improved non-production traits (e.g. health) while adequate milk production in crossbred animals. To date however, little crossbreeding research has been done on disease resistance such as mastitis. Cost and time are the main reasons that limit crossbreeding research. Field data from herds that practice crossbreeding may be incomplete or poor quality. Thus any conclusions or recommendations would need further findings from planned studies to support the validity of results.

In the current study, JE cows had an average of 2.9% lower incidence of CM than HF cows (Table 4.6), agreeing with experimental results in Ireland (Buckley et al., 2008) and United States of America (Washburn et al., 2002). The BV for JE bulls was lower than that of HF bulls, and crossbred bulls had intermediate values (Figure 4.2). Crossbreeding between HF and JE was associated with a decreased CM incidence in XB progeny compared with the average of CM incidence in the parental breeds. The heterosis effect of HF x JE was -1.25%, which was equivalent to 11.7% decrease in CM incidence of the parental breeds. This finding agrees with estimates of Buckley et al. (2008) for HF x JE crossbred and HF x Norwegian Red (NR) cows.

Whereas little supporting evidence exists about the effect of crossbreeding on reducing the CM incidence, it may be hypothesised that crossing high producing cows with CM-resistant cows results in crossbred cows with adequate milk production and improved udder health. Other functional traits may be improved by a similar mechanism through crossbreeding.



Source: DairyNZ and LIC (2008)

Figure 5.1: Trend in the percentage of inseminations of each major breed for the last 40 seasons

Figure 5.1 shows that the percentage of inseminations for HF increased slightly in 2007/2008 while artificial breeding in JE continued to decline and the use of Ayrshire breed remained low and constant. The use of XB (HF x JE) semen increased steadily since the year 2000 and went up by 2.2% from 15.2% in 2006/2007 to 15.4% for the 2007/2008. This indicates the popularity of XB bulls in New Zealand dairy industry and the effect of crossbreeding will benefit the farmer in the long run as the heterosis effect will reduce the incidence of CM and increase the cow longevity by improving other functional traits resulting in some economic benefit for the farmer.

In addition, the selection process could be enhanced by the application of new technologies. Advances in DNA chip technology allows the discovery of single nucleotide polymorphisms related to the genes of the immune system and therefore prediction of breeding values for resistance to a specific pathogen may become feasible. This information can be used to increase genetic progress, either by increasing the reliability of EBV, by reduction of the generation interval, or increasing the selection intensity without having to rely heavily on progeny testing (Schrooten et al., 2005).

CHAPTER 6

CONCLUSIONS

6. CONCLUSION

Mastitis is a complex disease caused by a number of pathogens and supporting factors that affect different parts of the udder and produce varying levels of response of the immune system (Bannerman et al., 2004). Breed difference is a contributing factor that influences lower CM cases in JE than HF. A possible reason beside management and environmental factors that cause difference in CM could be innate immune response in JE cows is more efficient than that of HF cows.

One approach to reduce the incidence of mastitis, in addition to adequate udder health management, is the selection for animals with a higher resistance to the disease. Results from this study confirm previous estimates of genetic parameters for CM. Although, limited recording of CM and low heritability may restrict the use of CM as a direct selection for mastitis resistance, use of JE bulls of low breeding values for incidence of CM used for crossbreeding can be an alternative to improve resistance to CM in New Zealand dairy cattle.

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