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Bovine mastitis in New Zealand

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Abstract

This thesis represents an aggregation of knowledge on bovine mastitis in the New Zealand dairy industry. Firstly, the thesis reviews the factors influencing the economic impact of bovine mastitis. Secondly, it provides information on the incidence of clinical and subclinical mastitis, as a prerequisite for estimating these costs. Thirdly, it investigates the effects of experimentally-induced *Streptococcus uberis* mastitis early in the dry period on milk production in the subsequent lactation.

In the review of factors influencing the cost of bovine mastitis, it was clear that neither farmers nor farm advisors have a good understanding of its full economic impact. In order to better understand these costs, it is necessary to have a clear idea of the incidence and consequences of clinical and subclinical mastitis: areas of knowledge which were identified as being deficient. Hence, two studies were conducted to investigate these areas.

In the first study, the incidence of clinical mastitis in Northland, New Zealand, was estimated. Furthermore, the aetiological agents causing mastitis were elicited and their chronological distributions in lactation were described. The average incidence of clinical mastitis was 0.19 cases per 305 cow-days at-risk, which is higher than previously reported in New Zealand. There were approximately equal numbers of isolations of *Staphylococcus aureus* (23.7%), and *Strep. uberis* (23.3%) from clinical cases: a pattern that is remarkably different to elsewhere in the country. Clinical mastitis due to *S. aureus* or *Strep. uberis* differed between age groups, with the highest incidence of *S. aureus* isolations from older cows (0.043 cases per 305 cow-days-at-risk) and lowest from 2-year old cows (0.014). The incidence of *Strep. uberis* was similar in first calving (0.034 cases per 305 cow-days-at-risk) and older cows (5 year-old: 0.039 cases, 6 year-old: 0.030 cases). Overall, 12% of cows were temporarily removed from supply and 1% were culled for mastitis. The differences between the study in Northland and these reported elsewhere from NZ highlight the need for a national survey on the aetiology and epidemiology of bovine mastitis.

A second study evaluated the effects of *Strep. uberis* clinical mastitis in the early dry period on milk production in the subsequent lactation. In a previous study,

Strep. uberis mastitis was experimentally induced and then promptly treated. This experiment provided a data set from which the impact of *Strep. uberis* clinical mastitis early in the dry period on milk production in the subsequent lactation could be estimated. Results of this study indicated that an early dry period clinical mastitis due to *Strep. uberis*, when promptly treated, did not affect production in the subsequent lactation. For cows that suffered mastitis episode during early dry period compared to those that did not, there was no difference in milk yield (5126 vs. 5010 litres), fat yield (267 vs. 264 kg), and protein yield (182 vs. 179 kg), respectively. It was considered that the short duration of intramammary infection did not cause permanent damage to the mammary secretory tissue.

It was concluded that the current estimates of the economics of mastitis in New Zealand are probably under-estimating the real cost of mastitis to its dairy industry. This was based on the higher incidence of clinical mastitis in Northland than elsewhere in the country and a failure of previous studies to take into consideration the costs associated with animals that were temporarily removed from supply (i.e. rather than culled). Additionally, as the highest frequency of new intramammary infections occurs in the first week or two after drying off, it may prove beneficial for farmers to pay more attention to checking for clinical mastitis during the early dry period.

Preface

It is more than 3 years ago that I started designing a project for my Masters degree. At that time I was working for Dr Ross D Woods in a mixed animal practice in Dargaville, Northland, New Zealand, and I held a special interest in bovine mastitis and dairy cattle reproduction. I was fascinated by how many cases of clinical mastitis or mastitis problem herds were in the area. Anecdotal evidence from veterinarians and laboratory workers from the region suggested that there might be different patterns of mastitis in Northland to those observed elsewhere in New Zealand. The idea to conduct a study on the aetiology of bovine mastitis for the region was thus born. It took more than a year to find someone willing to provide financial support and to start the first study. After that, it was easier to find extra financial support. At this stage I contacted Prof Tim (Timothy) J. Parkinson, who invited Assoc Prof Cord Heuer also to be involved. At this stage both veterinary practices in Dargaville amalgamated causing an additional pressure of changing the working environment and habits. Conducting the study, I found why people are reluctant to work on larger scale studies while employed full time in a practice. There was no time for a private life, especially since I was recently married. Fortunately, my wife, Paulina Rodriguez, was also preoccupied as she was preparing for the New Zealand National Veterinary Examination registration exams. She did not complain (hugely) about the extra hours that I spent with my “girlfriend” (my computer) or with my “love” (dairy farms).

Only due to the valuable time contributed by farmers and farm personnel on the study farms made it possible to conduct and complete this study. I truly enjoyed the discussions with them and the farm visits.

Furthermore, browsing the literature, I found that there is a great deal of literature available on the economics of bovine mastitis, but there is a lack of review/continuing education articles that provide a list of the factors associated with the cost of bovine mastitis to the dairy industry. This provided the inspiration for me to review the literature for these factors and subsequently write an article on continuing education. After a year or so of work, with help of two of my friends from my University days - Prof Metodija Trajcev and Prof Gjoko Buneski of Univeristy “St Cyril and Methodius”,

Skopje, Macedonia, the first version was sent to the editor of the Journal of the South African Veterinary Association.

In late January 2006, my wife and I left Dargaville and moved to Palmerston North, where I joined the team of the Institute of Veterinary, Animal and Biomedical Sciences, Massey University. My close collaboration with Prof Norman B. Williamson at Massey University completed the expansion of my supervisors team. My interest in the economics of bovine mastitis formed the basis of the second study included in this thesis – investigating the effects of clinical mastitis occurring in the early dry period on the milk production in the subsequent season. A study on the efficacy of an experimental and a modified existing external teat sealant, conducted over a previous dry period by Cecilia Fernandez, provided a great opportunity to investigate any production effects after *Streptococcus uberis* challenge and infection.

At this stage I started with my biggest saga during the preparation of this thesis - the analytical part of the studies. I learned that data preparation and proper analyses are very important. Collecting the data is easier than the data entry that can be a very difficult task, particularly when the collected data are on hand-written forms. This was succinctly stated by Prof Ynte Schukken from Cornell University, USA, at a February 2007 Epidemiology workshop when he said “You enter your data preparing it for analysis, not just to be entered”. This is something that I had to learn the hard way, during my Masters preparation.

Analyses in the second study were conducted with guidance and assistance on the use of SAS software from Dr Nicolas Lopez-Villalobos. Mr Alex Grinberg was also involved in the study, providing some information in the absence of Cecilia Fernandez, who had returned to Argentina. I found computer software to be very helpful and fast, however, I am not sure that in my next life I would like to be a statistician.

This Masters project has been one of the most enjoyable educational experiences that I have undertaken. The knowledge gained and the techniques learned (in particular biostatistics), have provided an opportunity to investigate problems that were previously impossible for me to tackle. I am now able to approach problems in a more logical manner. I have gained skills and confidence to learn many more techniques of

the discipline under my own guidance and this will be basis for my further development.

Writing is an essential part of any research and it is often very difficult to bring the observations and analyses to other people, particularly in a foreign language as English is my second (or so) language. Ross Woods, George Tharakan, Kathy Dropulich, Kylie Martinovich, Donald Thomas, Graeme Ewenson, Simeon Pollock and Joyce DeMoulin from Dargaville, Prof Colin Holmes, Assoc Prof Duncan Mackenzie, Mr Allan Thatcher, and my supervisors, at Massey have been of tremendous help reading papers, manuscripts and this thesis and have suggested useful corrections. Prof Williamson once mentioned that my first versions of manuscripts are in Macedonian with English words.

Dedication

I would like to dedicate this thesis to my parents Risto and Verica Petrovski for their guidance and encouragement through my life and my wife Paulina Rodriguez for her love and patience.

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I wish to express my sincere gratitude to my supervisors Prof Tim Parkinson, Prof Norm Williamson and Assoc Prof Cord Heuer, for their patience, sound advice, encouragement, dedication and for leading me through the intricacies of a master's degree thesis.

I am indebted to all the farm owners and farm personnel in the enrolled dairy farms for the friendship, cooperation and interest in my research. Without their full support and cooperation this work could not have been carried out.

I would also like to thank the Dargaville Veterinary Centre crew for their assistance and encouragement during the field work on the second study.

I would also like to thank the New Zealand Veterinary Pathology laboratory crew of Michelle McKeany and Rae Pearson for their assistance in culturing the samples and identification.

Assoc Prof Duncan Mackenzie and Prof Colin Holmes are also thanked for their guidance and encouragement through my post graduate studies. Andrea Coleman has provided computer support and Dr Nicolas Lopez-Villalobos, Dr Richard Laven and Kevin Lawrence have provided assistance with the statistical analysis of the data.

Other Institute of Veterinary, Animal and Biomedical Sciences, Massey University staff, although not directly involved in my studies, have also provided support and friendship through my studies.

The financial support provided by the Northland Community Foundation in cooperation with the Northern Wairoa Vet Club, Dargaville Field days and Mangatapere Vet Club for the Northland study is also gratefully acknowledged.

The study of the effects of early dry period mastitis on milk production in the subsequent season and the printing of this thesis were financially supported by Bomac Laboratories Ltd.

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List of Abbreviations

Abbreviation	Meaning
A.	<i>Arcanobacter</i>
BTSCC	Bulk Tank Somatic Cell Count
CM	Clinical Mastitis
CNS	Coagulase-negative staphylococci
CR-FVR	Case-ratio of clinical mastitis in front <i>versus</i> rear quarters
CR-RVL	Case-ratio of clinical mastitis in right <i>versus</i> left quarters
DAR	Days at risk
DIM	Days in milk
E.	<i>Escherichia</i>
EDPCM	Early Dry Period Clinical Mastitis
HF	Holstein-Friesian
ICSCC	Individual cow somatic cell count
IMI	Intramammary Infection
IVABS	Institute of Veterinary, Animal and Biomedical Sciences
LIC	Livestock Improvement Corporation
MCO	Mastitis-Causing Organism
MS	Milk solids
MUAEC	Massey University Animal Ethics Committee
S.	<i>Staphylococcus</i>
SAS	Statistical Analysis System
SCC	Somatic Cell Count
SCM	Sub-clinical Mastitis
SCS	Somatic cell score
<i>Strep.</i>	<i>Streptococcus</i>
UK	United Kingdom
USA	United States of America

Chapter one

General Introduction

1. 1 General Introduction

Bovine mastitis is defined as an inflammation of the mammary gland mainly in reaction to infection by mastitis-causing organisms (**MCO**). It is one of the most prevalent economically important production diseases affecting the dairy cattle industry worldwide (Blosser 1979; Fetrow 1987; Gill et al 1990; DeGraves and Fetrow 1993; Hortet and Seegers 1998; Seegers et al 2003).

Bovine mastitis is a multi-factorial and complex disease, resulting from interaction between cows, microorganisms and the environment (Figure 1.1; Watts 1988; Schukken and Kremer 1996). Most cases of clinical mastitis (**CM**) in dairy cows are infectious in aetiology (Craven 1987; Sandholm et al 1990). Despite intensive research and the implementation of various mastitis control strategies over the decades, bovine mastitis has not disappeared and the reduction in the prevalence of sub-clinical mastitis (**SCM**) has been minimal (Pyorala 2002) to significant, country dependant. In New Zealand the reduction of SCM is evident from the decrease in national bulk milk somatic cell count to around 200,000 cells/mL lately. On the other hand, there has been a considerable decrease in the incidence of clinical cases of bovine mastitis worldwide.

More than 137 species of organisms have been implicated as causal agents of bovine mastitis (Watts 1988). Many bacteria, yeasts, viruses and fungi have been isolated from bovine mammary glands but only a small group of them cause elevated somatic cell counts (**SCC**) and mastitis (Watts 1988; Malinowski et al 2002; Wellenberg 2002). More than 90% of all new intra mammary infections (**IMI**) are caused by a few MCOs, namely, *Staphylococcus aureus*, coagulase-negative staphylococci (**CNS**), *Streptococcus agalactiae* *Strep. dysgalactiae*, *Strep. uberis* and *Escherichia coli*. However, there are many other miscellaneous species of organisms capable of causing mastitis and finding one of these causing mastitis may be highly significant for a particular dairy herd.

Financial losses due to mastitis are substantial and arise from direct and indirect losses and expenditure. Bovine mastitis is considered one of the most economically important diseases for the dairy industry in developed countries (Blosser 1979; Fetrow 1987; Gill et al 1990; Schepers and Dijkhuizen 1991; McInerney et al 1992; DeGraves

and Fetrow 1993; Allore and Erb 1998; Hortet and Seegers 1998; Fetrow 2000; Pyorala 2002; Seegers et al 2003). Bennett et al (1999) estimate that the total costs of each disease can be much higher than the direct expenditure (Bennett et al 1999).

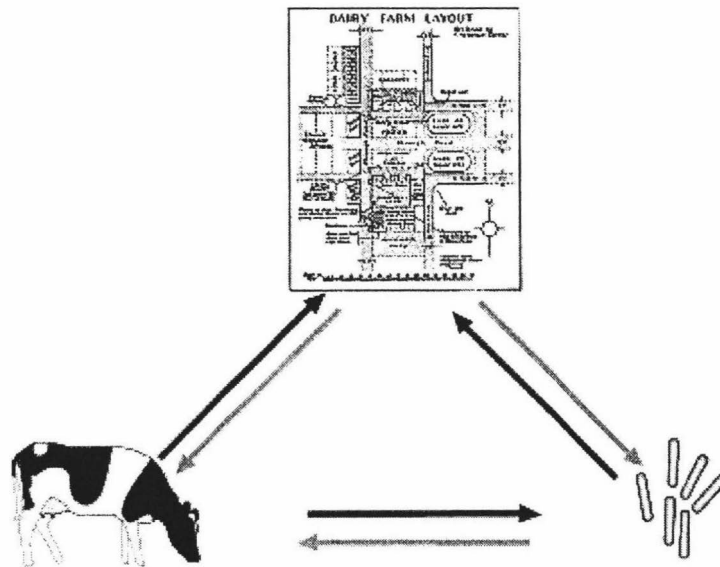


Figure 1.1. Bovine mastitis epidemiological triangle - cow, mastitis-causing organisms and the environment

Mastitis cost the USA dairy industry approximately US\$2 billion/year in 1992 and 1993 (Miles et al 1992; Cullor 1993). According to Ott (1999) the total production loss due to mastitis in the USA is \$108.00 per cow for herds with average Bulk Tank Somatic Cell Count (**BTSCC**) of 200,000-399,999 cells/ml and \$295.24 per cow for herds with average BTSCC 400,000 and above. This amounts to losses of approximately \$1 billion to the USA dairy industry, based only on BTSCC, as a measure of SCM (Ott 1999). Morin et al (1993), monitoring four Illinois herds for twelve months, reported mastitis-associated economic losses ranging from US\$161.79 to \$344.16 per lactating cow/year. Similarly, the total financial cost of mastitis to the average Scottish dairy herd in 1996 was estimated to be £140/cow/year (Yalcin 2000), of which a loss of £100/cow/year was due to SCM in high BTSCC herds (Yalcin et al 1999). Bovine mastitis has been estimated to cost the New Zealand dairy industry around NZ\$180 million/year in 2005/06 (Anonymous 2006).

Many factors have been associated with the cost of mastitis to the dairy industry and they have been recently reviewed (Petrovski et al 2006). The main factor causing

economic losses, due to both clinical and SCM, is a more or less persistent decrease in milk yield. The pathogenesis of mastitis, in many cases, includes damage to secretory tissue and its replacement with fibrous tissue, leading to a permanent decrease in milk yield from the affected quarter (Benites et al 2002). During mastitis episodes and recovery periods milk composition is also affected (Bishop et al 1984; DeGraves and Fetrow 1993; Auldist 1995; Seegers et al 2003) influencing yields of milk solids.

In a case of mastitis during lactation, usually there will be a short-term depression in milk yield of variable severity. In the case of no microbial cure and recovery, there is a longer-lasting effect, sometimes carrying over into the next lactation/s. Regarding short-term effects, it is generally accepted that the earlier mastitis occurs in the lactation the greater the losses of milk yield (Beck et al 1992; Houben et al 1993; Hortet and Seegers 1998; Rajala-Schultz et al 1999; Fetrow 2000).

Long-term effects of CM on milk yields are an area that need more attention from research (Petrovski et al 2006), although available estimates generally indicate that after an episode of CM there are both short- and long-term decreases in milk production, particularly associated with chronic bovine mastitis (Smith et al 1968; Fetrow et al 1991; Lescourret and Coulon 1994; Rajala-Schultz et al 1999; Fetrow 2000).

The effects of CM during the dry period on milk production parameters do not appear to have been systematically investigated. Nonetheless, the dry period is an important part of the lactational cycle during which the mammary gland should recover and prepare for the next lactation. A clinical case of mastitis during this period may therefore be expected to disturb the process of normal involution and preparation for lactogenesis.

Most of the available estimates take into account only a part of the real cost of mastitis, as estimating the true costs associated with mastitis is notoriously difficult. It is even more difficult to quantify the losses associated with SCM, because they are not visible to farm owners. To avoid underestimating the consequences of mastitis in evaluations of economic-loss it is important to account for all of the cost factors involved.

Many techniques and methods have been used to estimate production losses from mastitis in dairy cattle, but none of the techniques used are perfect, due to lack of a direct measure of how much milk a cow would have produced if there had been no occurrence of mastitis during lactation. Furthermore, all have a degree of inherent bias which, in most cases, tends to underestimate the actual milk yield decrease that has occurred (DeGraves and Fetrow 1993). Methods commonly used include producer surveys, regression analyses relating milk somatic cell counts, between-herd comparisons, between-cow yield comparisons, within-udder yield comparisons, within-cow yield comparison and studies between identical twins.

Estimation of the economic costs associated with mastitis depends on having the following data:

1. *An estimate of the incidence and prevalence of mastitis in the population* is a prerequisite for the estimation of its real cost to the dairy industry. There is currently a general demand through regular monitoring, recording and research to establish the incidence and prevalence of mastitis.

McDougall (2002) reported the incidence rate of CM in New Zealand dairy herds to be 14 cows/100 cows per annum, with the majority of cases occurring around calving (McDougall 2002a). Intervention studies in New Zealand found that 10% of cows acquired new infections during early lactation (Pankey 1982; McDougall 1998). Brookbanks (1966) reported that 32% of cows had a positive rapid mastitis test (RMT) during a study conducted in 130 herds throughout New Zealand (Brookbanks 1966). No references to other nation-wide surveys of the incidence of clinical or subclinical mastitis were found in the literature.

2. *The severity of the physical effects of mastitis on milk production*, depends on many factors, such as type and virulence of the MCOs, stage of lactation, age of the cow and udder defence mechanisms.

Research on the distribution of MCO in New Zealand is also limited. Early surveys of MCO in milk reported that 14 to 18% of the cows were infected with *Strep. agalactiae*, 27 to 41% with *S. aureus* 2 to 4% with *Strep. dysgalactiae*, fewer than 3.0% with *Strep. uberis* and 25% with CNS (Brookbanks 1966; Elliot 1976). More recently, the

prevalence of *Strep. agalactiae* and *S. aureus* has fallen, whilst *Strep. uberis* is now reported as the most common cause of bovine mastitis in New Zealand. Reports from intervention surveys in the Waikato report the following proportions of mastitis-causing organisms from cows with CM: 27 to 75% of *Strep. uberis*, 9 to 10% CNS, 2 to 5% coliforms, 1.5 to 4% *Strep. dysgalactiae* and 3 to 5% *S. aureus* (McDougall 1998, 2003).

Mastitis-causing organisms are categorised as contagious or environmental, based on the source of infection, mode of transmission and the tendency to cause persistent or transient IMI (Bradley and Green 2001; Makovec and Ruegg 2003). The primary reservoir of contagious MCOs is the bovine mammary gland and they are commonly transmitted among cows during milking. *S. aureus* and *Strep. agalactiae* have historically been most important. Lately, particularly in the USA, *Mycoplasma* spp. are increasingly reported to cause major mastitis problems in affected herds (Makovec and Ruegg 2003). Environmental MCOs have a primary reservoir in the cow's environment. The difference between contagious and environmental MCO is less clear than originally thought (Figure 1. 2), since environmental organisms have been shown in some cases to persist in the mammary gland throughout the dry period and beyond, and transmission from cow to cow has been proven in the case of *Strep. uberis* and *Strep. dysgalactiae* (Grommers et al 1985; Zadoks et al 2003). The most important environmental MCOs are streptococci (other than *Strep. agalactiae*), CNS and Gram-negative bacteria.

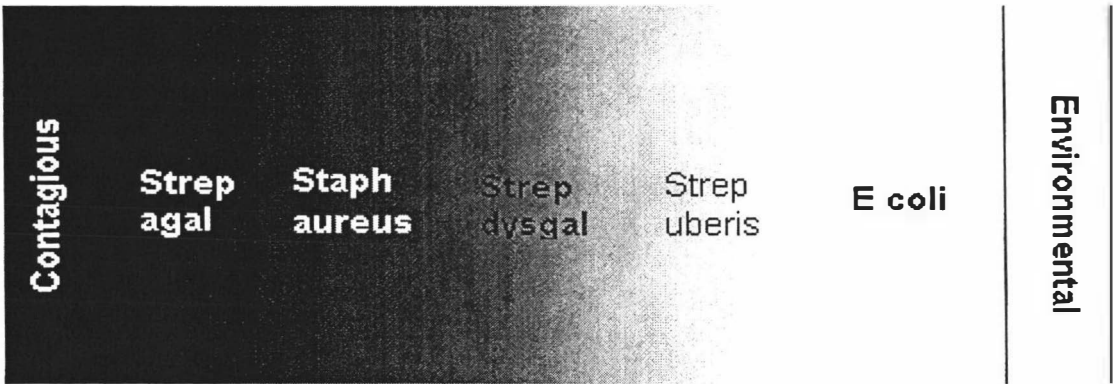


Figure 1.2. Sliding scale from contagious to environmental epidemiology of mastitis-causing organisms.

Streptococcus agalactiae almost entirely transmitted cow-to-cow; *Staphylococcus aureus* largely contagious, partly environmental; *Strep. dysgalactiae* classified as environmental by some, as contagious by others; *Strep. uberis* largely environmental, partly contagious; *Escherichia coli* (almost) entirely environmental.

Re-drawn from Zadoks and Schukken 2003.

Streptococcus uberis is a widely occurring MCO which is commonly isolated from IMIs in many countries, including New Zealand, Australia, the United Kingdom, the Netherlands, the United States, and Canada (Douglas et al 2000; Phuektes et al 2001; Zadoks et al 2003; McDougall et al 2004). It accounts for a significant proportion of subclinical and clinical intramammary infections in lactating and nonlactating cows (Douglas et al 2000; Phuektes et al 2001). *Strep. uberis* is probably the most significant cause of bovine mastitis in New Zealand and Australia, where the dairy industry is predominantly pasture-based (Pankey et al 1996; Douglas et al 2000; Phuektes et al 2001; McDougall 2002b).

3. *Identification of the prevention and treatment measures undertaken and estimates of their efficacy.* Generally, there is no difficulty in finding the expenditure on mastitis control but the influence on the incidence of mastitis, its duration and the resulting impact on production is more difficult.

4. *Valuation of the production losses, treatments and expenditures on prevention and control incurred.* Estimates of milk yield loss are still the subject of debate; however, they are likely to be influenced by the age, breed and type of cow, morphological characteristics of the udder, stage of lactation, pregnancy status, milk yield before mastitis occurred, milk price, premiums and penalties, MCO, inflammation grade, duration and distribution, timeliness of diagnosis (in relation to the onset of the occurrence), treatment and prevention costs, feeding practices, season, recurrence of mastitis during the same or a previous lactation, comparison model (what is the control group) and the analytical model. Milk production losses are typically estimated to account for 70 to 80% of all mastitis losses in a herd (Kirk 1984; Gill et al 1990; Schepers and Dijkhuizen 1991; DeGraves and Fetrow 1993; Morin 1993; Lehenbauer and Oltjen 1998; Fetrow 2000).

5. *Other cost factors- e.g. farm management, culling, replacement and fatalities.*

Extrapolating observations from other countries can be difficult due to the differences in the dairying system in New Zealand where dairy farms are pasture-based with seasonal production, lower milk yields and shorter lactation periods.

This thesis reviews the factors influencing the cost of bovine mastitis, provides information on some of the prerequisites for estimation of the cost of bovine mastitis to the New Zealand dairy industry and reports the effects of experimentally induced and promptly treated *Strep. uberis* CM early in the dry period (EDPCM) on milk production in the subsequent lactation.

In the review of the factors influencing the cost of bovine mastitis, areas of deficient knowledge were identified and two studies were conducted that contribute information to the deficient areas of knowledge.

The first study estimated the incidence of bovine clinical mastitis in the Northland region of New Zealand and describes the aetiology and distribution in lactation of MCOs. The rationale for this study is that knowing the incidence of CM is one of the main pre-requisites for estimation of the economic losses associated with bovine mastitis. It was undertaken from the hypothesis that the particular circumstances of the dairy industry in Northland would give rise to different patterns of mastitis to those observed elsewhere in New Zealand (a view that was supported by anecdotal reports from veterinarians and laboratory workers of the Northland region).

The second study evaluated the effects of experimentally-induced and promptly-treated *Strep. uberis* CM in the early dry period on milk production in the subsequent lactation, by re-evaluation of data from Fernandez (2007). The rationale for this is the general dearth of knowledge about the effects of CM during the dry period. It is well known that the dry period is an important part of the lactational cycle during which the mammary gland prepares for next lactation. Therefore, an episode of CM during this period may impede the process of mammary tissue remodelling, thereby adversely affecting milk yield in the subsequent lactation. *Streptococcus uberis* was elected for this study as it is regarded as the most common cause of mastitis in New Zealand (Douglas et al 2000; McDougall 2002b; McDougall 2002a).

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Chapter two

A review of the factors affecting the costs of bovine mastitis

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References in this chapter are unusually placed at the start of the headings/subheadings accordingly to the rules of publishing instructed by the *Journal of the South African Veterinary Association*. Example of the published section is inserted:

Cost of the drugs

(Refs 1, 2, 10, 11, 14–16, 23, 28, 31, 38, 42, 43, 49, 52, 53)

This part of the mastitis cost is easily calculated from the invoices of purchases of drugs on the farm. A similar calculation may be done for the expenditure on the treatment of individual cases.

Discarded milk

(Refs 1, 6, 11, 14–16, 23–25, 31, 36, 38, 39, 42, 43, 52, 53, 66, 67, 74, 78)

The assessment of the cost of discarded milk should include the milk withdrawn during and after the treatment, which depends on the withholding periods of the drugs used and current regulations. The cost of discarded milk is usually estimated easily. In cases where mastitic milk is used for calf-rearing, estimation of the cost of mastitis should be carefully assessed. The system for accounting for

2. A review of the factors affecting the costs of bovine mastitis

2.1 Abstract

Mastitis is one of the most prevalent production diseases affecting the dairy cattle industry world-wide. Its occurrence is associated with direct and indirect losses and expenditures. When estimating the cost of mastitis to the dairy industry the cost of the control programmes must be added. The direct losses of mastitis are the only costs obvious to the farmer. The difference between the costs of mastitis on one side and the benefits of mastitis control on the other side will give us a picture of the economic efficacy of the mastitis control programme. Continuing education of the farmer is needed for better mastitis control programmes. This article is an attempt to review briefly all relevant factors included in the economics of bovine mastitis and to show the authors' view of some of the costs.

Estimating the costs associated with mastitis is notoriously difficult. The economics of mastitis needs to be addressed at the farm or herd level and depends on local, regional, epidemiological, managerial and economic conditions. Some of the costs and expenditures are easy to calculate and they should be included in research projects dealing with the modelling of the economics of the disease. However, some of the costs are not countable, such as cases of human diseases, farmers' stress etc. To be able to consider the real cost of mastitis to the dairy industry, the prevalence and incidence of mastitis on a national level should first be established. Then estimations of all relevant countable costs and expenditures should be made, and the last step will be to include all of them in one large model for mastitis cost estimation.

2.2 Introduction

Mastitis is defined as an inflammation of the mammary gland. It is a complex disease involving many factors, which is mainly caused by bacteria and there is no simple model that encompasses all possible facets (Scheepers and Dijkhuizen 1991; Fetrow 2000). Despite intensive research and the implementation of various mastitis control strategies over the decades, bovine mastitis has not disappeared and the reduction in the prevalence of sub-clinical mastitis has been minimal (Pyorala 2002). On the other

hand, there has been a considerable decrease in the incidence of clinical cases of mastitis worldwide as a result of these control measures.

Bovine mastitis is considered one of the most economically important diseases for the dairy industry in developed countries (Blosser 1979; Fetrow 1987; Gill et al 1990; Schepers and Dijkhuizen 1991; McInerney et al 1992; DeGraves and Fetrow 1993; Allore and Erb 1998; Hortet and Seegers 1998a; Fetrow 2000; Pyorala 2002; Seegers et al 2003). Morin et al (1993), monitoring 4 Illinois herds for 12 months, reported mastitis-associated economic losses ranging from US\$161.79 to \$344.16 per lactating cow/year. The magnitude of the economic losses to the dairy industry in the USA due to mastitis was around \$1.3 billion in 1979 (Blosser 1979) and around \$2 billion in 1988 (Miles et al 1992) and 1993 (Cullor 1993) (not factored for inflation). The total financial cost of mastitis to the average Scottish dairy herd in 1996 was estimated to be £140/cow/year (Yalcin 2000) of which a loss of £100/cow/year was due to sub-clinical mastitis alone in high Bulk Tank Somatic Cell Count (**BTSCC**) herds (Yalcin et al 1999). According to Ott (1999) the total production loss due to mastitis in the USA is \$108.00 per cow for herds with average BTSCC of 200,000-399,999 cells/ml and \$295.24 per cow for herds with average BTSCC 400,000 and above or losses of approximately \$1 billion to the USA dairy industry, based only on BTSCC, as measure of sub-clinical mastitis (Ott 1999).

There is common confusion between the terms 'loss' and 'cost', so it is important to clarify the terminology. In this article the following terms will be used as defined:

a) **Loss** implies a benefit that is taken away (e.g. the production loss experienced because contaminated milk must be discarded); alternatively, it represents a potential benefit that is not realised (such as an evident decrease in the milk yield) (McInerney et al 1992; Seegers et al 2003).

b) **Expenditure** represent some economic effects of disease that are manifested as extra inputs into livestock production (such as treatment and prevention of mastitis) (McInerney et al 1992; Bennett et al 1999; Seegers et al 2003).

c) **Economic cost** is the monetary value of all the economic effects, both losses and expenditures, consequent upon the occurrence of disease (McInerney et al 1992; Bennett et al 1999; Seegers et al 2003).

This article briefly reviews all the relevant factors influencing the economic cost of bovine mastitis.

2.3 The cost of mastitis to the dairy industry

There have been many articles published worldwide on the economics of mastitis. When considering the cost of any disease, it must be kept in mind that every disease has direct and indirect costs. Bennett et al (1999) estimate that the total costs of each disease can be much higher than the direct expenditure. Most of the available estimates take into account only a part of the real cost of mastitis, as estimating the true costs associated with mastitis is notoriously difficult. It is even more difficult to quantify the losses associated with sub-clinical mastitis, because they are not visible to farm owners. To avoid underestimating the consequences of mastitis in evaluations of economic-loss it is important to account for all of the cost factors involved.

The estimation of the economic costs associated with mastitis depends on having the following data:

1. An estimate of the incidence and prevalence of mastitis in the population is a prerequisite for the estimation of its real cost to the dairy industry. There is currently a general demand of regular monitoring, recording and research to establish the incidence and prevalence of mastitis.
2. The severity of the physical effects of mastitis on milk production, which will depend on many factors, such as virulence of the mastitis-causing organisms, stage of lactation, age of the cow and udder defence mechanisms.
3. Identification of the prevention and treatment measures undertaken. It is generally easy to calculate the expenditure on mastitis control.
4. Valuation of the production losses, treatments and expenditures on prevention and control incurred. Production losses caused by mastitis are likely to be influenced by the age, breed and type of the cow, stage of lactation, milk yield before mastitis occurred,

milk price, premiums and penalties, mastitis-causing organism, inflammation grade and distribution, diagnosis, treatment cost, prevention cost and analytical model.

5. Other costs' factors- e.g. farm management, culling, replacement and fatalities.

Many techniques and methods have been used to estimate production losses from mastitis in dairy cattle. Methods commonly used are: producer surveys, regression analyses relating milk somatic cell counts, between-herd comparisons, between-cow yield comparisons, within-udder yield comparisons, within-cow yield comparison and studies between identical twins. De Graves and Fetrow (1993) state that none of the techniques used are perfect, due to lack of direct measure of how much milk a cow would have produced if there was no occurrence of mastitis during lactation, and they all have a degree of inherent bias, which, in most cases, tends to underestimate the actual milk yield decrease that has occurred.

The economics of mastitis needs to be addressed at the farm or herd level, and depends on local, regional, epidemiological, managerial and economic conditions. At the herd level, as stated by Seegers et al (2003), some compensation or buffer mechanisms can act and this should be taken into consideration in the estimates. An example is a farmer who decides to cull cows with high somatic cell count (SCC), based on the BTSCC and the milk pricing system, rather than on the absolute values of the individual SCC results of the cows. Another example is a farmer who decides to cull an extra cow to decrease the BTSCC and give up selling a heifer (Seegers et al 2003).

2.4 Losses caused by mastitis

2.4.1 Direct losses of mastitis

Direct costs of mastitis to the dairy industry include the costs of treatment (veterinarian's time and drugs), discarded milk (during both the course of treatment and with-holding periods) herdsman's time, fatalities and the costs associated with repeated cases of mastitis. In many cases direct losses are the only cost of mastitis realised by the farmers.

2.4.1.1 Treatment cost

(Blosser 1979; Bishop et al 1984; Kirk 1984; Fetrow 1987; Lightner 1988; Holdaway 1990; Fetrow et al 1991; Beck et al 1992; Miles et al 1992; DeGraves and Fetrow 1993; Miller et al 1993; Lescourret and Coulon 1994; Kossaibati and Esslemont 1997; Lehenbauer and Oltjen 1998b; Fetrow 2000; Pyorala 2002; Seegers et al 2003; Berry et al 2004; Shim et al 2004; Swinkels et al 2005)

The cost of treatment of clinical cases is an important element in the expenditure on mastitis. Very commonly, the size of the veterinarians' bills tends to be seen as 'the cost of disease' in the farmers' eyes. In fact, in general, the treatment of a disease, such as mastitis, is only a small proportion of the disease cost (Kossaibati and Esslemont 1997).

When estimating the treatment cost, the efficacy and cost effectiveness should be taken into account. For example Shim et al (2004) comparing two treatment protocols found that the addition of antimicrobials to supportive treatment is more efficacious and cost effective than supportive treatment alone, projected that cows without mastitis will produce 8.265 kg of milk (305-days in lactation). Cows treated with supportive treatment only produced at 7838 (3.064-11.111) kg of milk, while cows with added antimicrobial to the treatment produced 7975 (5.002-11.163) kg, when discarded milk was included (Shim et al 2004). Assuming that none of the unmarketable milk was fed to calves the cost of mastitis was 3 times higher in the group treated only with supportive treatment (Shim et al 2004).

There are two elements of the treatment cost: veterinarians' fees and the cost of drugs. In addition to the financial considerations, the treatment of cows with clinical mastitis is disruptive to the normal milking routine (Holdaway 1990; Fetrow 2000).

2.4.1.1.1 Veterinary time and consultation fees

(Blosser 1979; Bishop et al 1984; Kirk 1984; Craven 1987; Fetrow 1987; Morse et al 1987; Lightner 1988; Holdaway 1990; Beck et al 1992; Hillerton et al 1992; Cullor 1993; DeGraves and Fetrow 1993; Miller et al 1993; Morin 1993; Allore and Erb 1998; Fetrow 2000; Shim et al 2004)

The veterinary time and consultation fees can vary considerably in a mastitis control programme. These services are charged on an hourly basis, per-cow-per-year basis, or other methods. They can be applied to the individual cow, group of cows or the whole herd.

Veterinary time for the treatment of individual cows with clinical mastitis usually involves a minimal amount of herd-level consultancy and the cost per cow can be calculated from the invoices. The cost at the farm level may depend on the number of visits by the veterinarian. For example in the Nordic countries all mastitis cases are attended by a veterinarian. In most other countries, such as South Africa, USA, Australia and New Zealand, the veterinarians attend only some cases of mastitis. In such situations, the calculation of costs on a per cow basis, from data collected at the farm level, needs some modelling.

Group level service includes treatment and prevention of mastitis in a specific group, such as age-categories, heifers or newly purchased cows. In this case, part of the veterinary time is clinical work, and usually there will be some consultancy time as well. Calculation of the estimated cost per cow in such a case is difficult, as it is unknown how much consultancy time has been spent per individual animal. The usual approach is to divide the amount on the invoice by the number of animals attended.

Most of the time when dealing with herd problems is spent on consultancy work, for example dealing with high BTSCC herds or mycoplasma mastitis affected herds. In this case only a small amount of time is utilised as real veterinarian clinical work. The calculation of the estimated cost of this element is from the invoices. The fees are usually charged at the farm level, and if individual cow-cost is required, then some modelling can be utilised, or the amount on the invoices is simply divided by the numbers of cows in the herd.

2.4.1.1.2 Cost of the drugs

(Anderson and Blackshaw 1977; Blosser 1979; Kirk 1984; Craven 1987; Fetrow 1987; Morse et al 1987; Lightner 1988; Holdaway 1990; Hillerton et al 1992; Cullor 1993; DeGraves and Fetrow 1993; Miller et al 1993; Morin 1993; Lescourret and Coulon 1994; Allore and Erb 1998; Berry et al 2004)

This part of the mastitis cost is easily calculated from the invoices of purchases of drugs on the farm. A similar calculation may be done for the expenditure on the treatment of individual cases.

2.4.1.2 Discarded milk

(Blosser 1979; Bishop et al 1984; Kirk 1984; Craven 1987; Fetrow 1987; Morse et al 1987; Lightner 1988; Holdaway 1990; Fetrow et al 1991; Beck et al 1992; Cullor 1993; DeGraves and Fetrow 1993; Morin 1993; Lescourret and Coulon 1994; Kossaibati and Esslemont 1997; Allore and Erb 1998; Fetrow 2000; Seegers et al 2003; Winkelman 2003; Shim et al 2004; Swinkels et al 2005)

The assessment of the cost of discarded milk should include the milk withdrawn during and after the treatment, which depends on the withholding periods of the drugs used and current regulations. The cost of discarded milk is usually estimated easily. In cases where mastitic milk is used for calf-rearing, estimation of the cost of mastitis should be carefully assessed. The system for accounting for the economic costs associated with 'discarded milk' should be transparent. No matter where it ends up the milk is not sold, so it is a loss of income. A possible solution is to budget for the economic costs of mastitis to be debited with the full costs of the milk not sold and the calf rearing budget to be credited with the value of the milk as a replacement for alternative sources of feed. If the estimates of milk losses are calculated on basis of BTSCC, then discarded milk in many cases is not taken into account, leading to underestimates of real mastitis costs (Lightner 1988). In many dairy countries it is common practice for the farm owners of herds with average high BTSCC to withhold or discard the milk from the cows with highest SCC aiming to control their bulk milk in acceptable levels.

2.4.1.3 Labour cost

There appear to be two main approaches in the literature for dealing with the expenditure on labour for mastitis treatment. The first one is to consider the labour time as a direct cost of the disease and include it in the calculations as such (Blosser 1979; Bishop et al 1984; Kirk 1984; Morse et al 1987; Lightner 1988; McInerney et al 1992; Cullor 1993; DeGraves and Fetrow 1993; Miller et al 1993; Morin 1993; Lescourret and Coulon 1994; Allore and Erb 1998; Seegers et al 2003; Winkelman

2003). The second approach is to calculate the labour cost if a farm specifically employs additional labour to manage treatment, segregation, or other aspects of mastitis control (Fetrow 1987; Fetrow et al 1991; Fetrow 2000). The more usual case is that mastitis control and treatment are handled by existing farm labour (Morin 1993) (i.e. no labour reduction would occur if mastitis cases were reduced). In authors opinion, the workers' time should be included in the calculations of mastitis cost. The estimation of the time spend per case is variable and will depend on many factors, such as type of mastitis, milk yield, farm size, hired labour, farm owner etc. For example, a peracute case of mastitis, associated with general illness, requires more time for treating, nursing and frequent stripping than mild subacute mastitis with only changes in the milk.

2.4.1.4 Fatality

(Kirk 1984; Fetrow 1987; Beck et al 1992; Cullor 1993; Miller et al 1993; Morin 1993; Kossaibati and Esslemont 1997; Hortet and Seegers 1998a; Pyorala 2002; Seegers et al 2003)

Severe cases of mastitis can lead to the death or euthanasia of the affected cow. The cost of a fatality is greater than simply the value of the cow in the market, as it includes the lost margin from the incomplete portion of its lactation. According to Kossaibati and Esslemont (1997) it also includes the cost of a replacement heifer. The mortality rate for clinical mastitis is usually low. Wilesmith et al (1986) reported between 0.3 and 0.6% of mastitis cases to be fatal. Worldwide, higher mortality rates caused by mastitis are seen in specific situations with a high prevalence of Gram-negative infections, particularly coliform mastitis. Menzies et al (2003) recorded a fatality rate of 14% and a further 21% early culling because of the condition in a study involving 264 cases of acute and peracute toxic mastitis in Northern Ireland. In contrast, Bradley and Green (2001) reported a mortality rate of 0.6%, in general, and 2.2% due to Gram-negative organisms in 6 Somerset dairy herds.

2.4.1.5 Repeated cases of mastitis

Some dairy cows suffer repeatedly from mastitis during a single lactation. Kossaibati and Esslemont (1997) found that a typically affected cow suffers on average 1.6 cases

per lactation. The extra costs of that 0.6 repeat case should be taken into account when assessing losses caused by mastitis. For these repeated cases only the relevant direct costs should be included (i.e. cost of drugs, herdsman's and/or veterinarian's time and discarded milk). For the indirect costs, it is important to calculate only additional losses associated with further decrease in milk yield and increased risk of culling. The rest of the indirect costs usually have been already taken into account.

2.4.2 Indirect losses of mastitis

Indirect losses due to mastitis, particularly the subclinical form, are not well-recognised by many farmers. It is generally accepted that subclinical mastitis accounts for the majority of economic costs of mastitis. Education on this matter is necessary because un-recognised indirect losses can be a reason for difficult implementation of mastitis control measures, as farmers usually hold an opinion that their own losses, due to mastitis, are much lower than the estimates provided for the industry by the experts (Brown et al 1988). Indirect losses include the decreased milk production due to clinical or sub-clinical mastitis, decreased milk quality, increased culling, loss of premiums, penalties, pre-term drying-off, animal welfare aspects and other associated health problems.

2.4.2.1 Decrease in milk yield

(Smith et al 1968; Blosser 1979; Bishop et al 1984; Kirk 1984; Craven 1987; Fetrow 1987; Lightner 1988; Bartlett et al 1990; Holdaway 1990; Fetrow et al 1991; Beck et al 1992; Hillerton et al 1992; McInerney et al 1992; Miles et al 1992; Cullor 1993; DeGraves and Fetrow 1993; Miller et al 1993; Morin 1993; Kossaibati and Esslemont 1997; Allore and Erb 1998; Hortet and Seegers 1998a; Lehenbauer and Oltjen 1998b; Fetrow 2000; Pyorala 2002; Seegers et al 2003; Mungube et al 2005; Swinkels et al 2005)

The main factor in causing economic losses due to both clinical and sub-clinical mastitis is a more or less persistent decrease in milk yield. Usually there will be a short-term depression in yield of variable severity and, in case of no microbial cure and recovery, a longer lasting effect, sometimes carrying over into the next lactation/s. Milk production losses are typically estimated to account for 70 to 80% of all mastitis losses

in a typical herd (Kirk 1984; Gill et al 1990; Schepers and Dijkhuizen 1991; DeGraves and Fetrow 1993; Morin 1993; Lehenbauer and Oltjen 1998b; Fetrow 2000).

Losses in milk yield (not including discarded milk) need to be assessed within several time-frames. There are the short-term effects on the current lactation and long-term effects, including carry-over effects into the next lactation or beyond, that are usually estimated using several types of comparison or modelling approaches (Hortet and Seegers 1998a; Seegers et al 2003).

Estimates of milk yield loss are still under debate and likely to be influenced by the age, breed and type of cow, morphological characteristics of the udder, stage of lactation, pregnancy status, milk yield before mastitis occurred, mastitis-causing organism, inflammation grade, duration and distribution, diagnosis (early or late after the occurrence), treatment, feeding practices, season, recurrence of mastitis during the same or previous lactation, comparison model (what is the control group) and the analytical model.

It is generally accepted that mastitis occurring earlier in the lactation will lead to greater milk yield losses. Lescourret and Coulon (1994) reported that milk production curves of about one third of the cows infected early in lactation were little affected and yield recovered in less than 5 weeks. The production curves of the rest of the infected cows were markedly affected or the cows were culled. In contrast, more than half of the cows infected from mid- to late-lactation were not affected by marked modifications in their milk production curves and recovered in less than 5 weeks (Lescourret and Coulon 1994). It has been reported that the milk yields of older cows were obviously affected if mastitis occurred early in lactation, while younger cows' yields are sensitive with carry-over effects seen if mastitis occurred after the peak of lactation (Rajala-Schultz et al 1999; Fetrow 2000). Rajala-Schultz et al (1999) analysed records of over 24,000 Finnish Ayrshire cows, and reported that milk production declined 4 weeks before the onset of clinical mastitis and dropped further below the curve of "healthy" cows during the first week afterwards. Milk yield never reached the pre-mastitis levels if mastitis occurred in early lactation (before peak) (Rajala-Schultz et al 1999). A decreased milk production before the occurrence of clinical mastitis was presumably due to the effects of sub-clinical infection.

A higher level of milk yield prior to mastitis could be expected to be associated with higher losses in milk (both in absolute value and in percentage) (Lescourret and Coulon 1994; Hortet and Seegers 1998a).

The pathogenesis of mastitis, in many cases, includes damage to secretory tissue and its replacement with fibrous tissue leading to a permanent decrease in milk yield from the affected quarter (Benites et al 2002). In addition, it is probable that part of the decrease of the milk production is due to an increased demand for energy by the immune system, a decreased appetite associated with the inflammatory process and lowered feed intake due to pain and decreased mobility.

Some mastitis-causing organisms were shown to have a more profound impact on milk yields than others (DeGraves and Fetrow 1993; Wilson et al 1997; Fetrow 2000). Mastitis caused by *Staphylococcus aureus* generally evolve into persistent but moderate infections, unlike mastitis caused by coliforms. Thus, the mastitis-causing organism may contribute to the residual variation of responses, as well as to the level of intensity. Generally it is estimated that the greater the inflammation the less milk is produced.

There are three broad groups of comparison models: (1) between herd comparison, (2) between-cow or within- herd comparison, and (3) between-quarter or within-udder comparison. A comparison of the relative yields of herds with varying levels of mastitis may be used to estimate the decrease in milk production. However, in this type of study, factors other than mastitis may significantly contribute to any difference in milk yield that may be observed. The herds included in the study must be closely matched for factors such as location, breed, age, and plane of nutrition. The between-cow comparison model is also affected by some non-mastitis compounding factors such as age and breed and the cows must be closely matched for such factors. Within-udder yield comparisons compare a mastitis-infected quarter with an opposing mastitis-free quarter. Generally it is accepted that the contra-lateral quarters of the udder, when both are un-infected, give approximately the same volume of milk. However, while within-udder comparison avoids sources of variation which may confound other estimates of decreased milk production, it is possible that within the infected cow, un-infected quarters partially compensate by producing more milk or both produce less as

the cow is sick. There is evidence that mastitis-free quarters may compensate for quarters with mastitis by increasing milk production (Holdaway 1990; Holdaway 1993; McDougall 2002). If compensation does in fact occur, then this would cause over-estimation of the actual milk loss as a result of mastitis (DeGraves and Fetrow 1993; Holdaway 1993). Hortet and Seegers (1998), using a regression-modelling approach to analyse data from 20 papers published worldwide, predicted the average milk-yield loss over the lactation was 300-400 kg (i.e. 4-6%) per treated case of clinical mastitis in a Holstein Friesian cow producing approximately 7,000 kg/lactation. In primiparous cows, the average loss was lower (200-300 kg) and mild patterns of mastitis were more frequent than in multiparous ones. Cases occurring before the peak of lactation were associated with higher average losses (450-550 kg) than cases occurring later. Similarly Seegers et al (2003) estimated loss of about 375 kg (5%) per average clinical case, occurring in the second month of lactation in a Holstein cow.

The estimates need to be used with caution, especially for breeds other than Holstein Friesian or if unusual mastitis-causing organisms are involved in clinical-mastitis cases (Hortet and Seegers 1998a).

2.4.2.1.1 Short term effects

(Beck et al 1992; Houben et al 1993; Hortet and Seegers 1998a; Rajala-Schultz et al 1999; Fetrow 2000)

For the estimation of short term effects, it is necessary to bear in mind that an infection can start and the milk yield can be reduced before the mastitis is detected. This may lead to underestimation of the real loss from mastitis (Rajala-Schultz et al 1999).

Hortet and Seegers (1998a) using regression models estimated that short-term losses from clinical cases of mastitis varied from 0 to about 3 kg/cow/day, but suggested that the estimates are lower than expected. They suggested that regression models underestimate short-term losses, because of the difficulty in accounting for variable losses occurring before a clinical diagnosis.

Short-term reduction in milk yield is higher for clinical mastitis in early lactation compared to mastitis in mid to late lactation. Losses from 0 to 200 kg/cow/month

were estimated by Hortet and Seegers (1998a) in cases of clinical mastitis occurring before the expected peak of the lactation or 0 to 100 kg/cow/month with occurrences in mid-to late-lactation. Houben et al (1993), using records for over 5,300 lactations of nearly 2,500 black and white cows in Denmark, with approximate calculated production of 7,500 kg, reported the estimated effect of clinical mastitis on production of 527 kg of milk for ≥ 3 cases of clinical quarters in the second lactation (Houben et al 1993). Rajala-Schultz and Grohn recorded, in cows of second parity, mean milk losses of 294, 348 and 110 kg milk if mastitis occurred before peak, between peak and 120 days and later in lactation, respectively (Rajala-Schultz and Grohn 1999). The losses in older cows were significantly higher. For example, in cows over three lactations, mean recorded loss was 555, 329 and 357 kg, respectively (Rajala-Schultz and Grohn 1999).

2.4.2.1.2 Long term effects

This is an area that needs more attention from research, although available estimates generally indicate there are both long-term decreases in milk production after episodes of clinical mastitis and long-term economic losses associated with chronic mastitis (Smith et al 1968; Fetrow et al 1991; Lescourret and Coulon 1994; Rajala-Schultz et al 1999; Fetrow 2000).

Deluycker (1990) considered that the cows affected by clinical mastitis in the first lactation but not in the next do not have a higher, or compensatory increase in milk yield when compared to cows free of mastitis in two successive lactations, even if the infection was eliminated. Fetrow et al (1991) found that the carry-over effect of mastitis and high SCC from one lactation to the next was generally statistically significant but small, amounting to less than one half of the effects of high SCC in the current lactation. When production measures were adjusted for herd effect (rolling herd average), the carry-over effect was less than 20% of the direct effect of increased SCC (Fetrow et al 1991). However, chronic mastitis in 3 or more quarters is associated with long-term economic losses in the following lactation of more than 350 kg in the 2nd and 3rd lactation (Hortet and Seegers 1998a) and up to 381kg of milk, up to and including eight months into the second lactation (Houben et al 1993).

2.4.2.1.3 Elevated Somatic cell counts - SCC

The measurement of the SCC in bulk milk is the most universal method of evaluating the occurrence of mastitis in the dairy herd. There are significant correlations between the BTSCC of a farm and the economic losses associated with decreased milk production and quality. It is evident that an elevated SCC in milk, regardless of cause, is associated with decreased milk yield and economic losses (Fetrow et al 1988; Lightner 1988; Bartlett et al 1990; Fetrow et al 1991; DeGraves and Fetrow 1993; Trajkovski 1997; Fetrow 2000; Bennedsgaard et al 2003; Miller 2004; Mungube et al 2005). There is considerable variation in the estimates of the cost of milk loss in studies that have related milk yield to SCC. Thus, lactation losses of 80 kg and 120 kg by primiparous and multiparous cows respectively for each 2-fold increase in the geometric mean of SCC above 50,000 was estimated by a regression analysis of data from 19 papers (Hortet and Seegers 1998b). Similarly, Bennedsgaard et al (2003), analysing data from 17,500 lactations in 48 Danish organic herds, reported average losses of 0.2, 0.3 and 0.4 kg of energy-corrected milk/day in the first, second and third or later lactations respectively with each 2-fold increase in SCC between 100,000 and 1,500,000 cells/ml. Losinger (2005) estimated loss of US\$810 +/- 480 million to the USA economy as a whole caused by reduced milk production associated with an increase in BTCSS during 1996.

2.4.2.3 Milk quality changes

The economic losses that should be included in the calculations due to milk quality changes are poorer milk composition, zoonotic risk and hygienic milk quality changes leading to public health considerations, lower end-product yields and quality, shorter shelf-life of the final products and a decrease in profitability to both producers and processors.

2.4.2.3.1 Compositional changes

(Smith et al 1968; Bishop et al 1984; Craven 1987; Holdaway 1990; Beck et al 1992; McInerney et al 1992; DeGraves and Fetrow 1993; Morin 1993; Trajkovski 1997; Allore and Erb 1998; Hortet and Seegers 1998a; Pyorala 2002; Seegers et al 2003; Winkelman 2003; Swinkels et al 2005)

Mastitis is responsible for a number of changes in milk composition. While the effects of mastitis on the concentrations of protein and fat in the milk are variable, changes in

the actual composition of these components, especially protein, are more consistent and often quite marked (Bishop et al 1984; DeGraves and Fetrow 1993; Seegers et al 2003). There is a reduction in the synthesis of the main components of milk, namely fat, lactose and protein, which may lead to a change in the relative proportions of these components in the milk. There are also increased concentrations of blood serum components due to the inflammatory reaction, e.g. proteins, (serum albumin and immunoglobulins), chloride and sodium (Beck et al 1992; DeGraves and Fetrow 1993; Hortet and Seegers 1998a). These changes have direct and indirect effects on the manufacturing properties of milk, often decreasing yield (Allore and Erb 1998), quality (Trajkovski 1997) and shelf-life of end-product (Holdaway 1990; DeGraves and Fetrow 1993; Trajkovski 1997; Allore and Erb 1998). Furthermore, the presence of small quantities of antimicrobials in the milk due to mastitis treatment is associated with major losses incurred by the manufacturers when starter micro-organisms are destroyed or their activity is slowed.

The final products, manufactured from milk with changed composition, will potentially command lower prices on the market and therefore will reduce the income for the dairy industry and farmers. The current milk-pricing system mainly relies on total-fat and total-protein yields. Since there is little financial incentive for dairy farmers to do so, mastitis control programmes are not stimulated. Also, due to the withdrawal period after treatment of clinical cases composition changes in bulk milk can, as stated by Seegers et al (2003), be neglected in economic calculations. However, SCC and microbial count play an increasingly important role in many payment systems and therefore a decrease in milk quality, due to mastitis, plays a significant role. The introduction of premiums for milk quality stimulates interest on this matter (examples seen in UK and Australia). The very severe penalties for the presence of antimicrobials in milk are a major incentive for ensuring that effective measures are in place on the farm to prevent contamination.

All costs associated with the compositional changes, at farm level, can be calculated from the statements of milk collecting and processing companies. When the cost of mastitis is estimated on a per cow basis, data collected at the farm level needs some modelling taking into account cow numbers and mastitis occurrence.

2.4.2.3.2 Decreased hygienic quality of milk and Public health considerations

When discussing the financial implications of mastitis, its importance in public health and the effects of mastitis on the consumers should not be overlooked (Holdaway 1990; McInerney et al 1992; DeGraves and Fetrow 1993; Allore and Erb 1998). The risk of zoonotic diseases is also an important issue. This risk, however, is not necessarily associated with mastitis. The potential spread of zoonotic organisms via milk, though rare in the era of pasteurisation, remains a risk especially in the niche markets of un-pasteurised dairy products, and during pasteurisation failures (Pyorala 2002). A number of mastitis-causing bacteria and fungi are potentially pathogenic to humans, causing in many cases severe or even fatal infections or intoxications (e.g., Staphylococcal food poisoning with the thermo-stable toxins produced by the *staphylococci*; *Strep. agalactiae* – human septicaemia and neonatal meningitis etc.) (Holdaway 1990). The extensive use of antimicrobials in the treatment and control of mastitis has possible implications for human health through an increased risk of antimicrobial resistant strains of microbes emerging that may then enter the food chain (Beck et al 1992; Allore and Erb 1998; Pyorala 2002) or through the increased risk of allergic reactions.

The excretion of large numbers of mastitis-causing organisms in milk from infected cows adds to the total number of bacteria in bulk milk, regardless of degree of care taken with plant hygiene (Holdaway 1990; Beck et al 1992; McInerney et al 1992; Seegers et al 2003).

The stress to the farmer is considered as a potential public health concern.

The costs associated with the effects of mastitis on milk quality can be estimated from the penalties imposed by the milk processor for failure to meet the quality standards for SCC, microbial content and antibiotic contamination. Some factors of concern for public health, such as exposure to potential pathogens and pathogens resistant to antimicrobials in milk that is used un-pasteurised, or the stress to the farmer are not easily identified or costed.

2.4.2.4 Culling and replacement cost

The term culling describes the removal of an animal from a herd. A significant part of the economic cost of mastitis is related to culling losses (Blosser 1979; Bishop et al 1984; Kirk 1984; Craven 1987; Fetrow 1987; Lightner 1988; Fetrow et al 1991; Beck et al 1992; Hillerton et al 1992; McInerney et al 1992; Miles et al 1992; DeGraves and Fetrow 1993; Miller et al 1993; Morin 1993; Stott and Kennedy 1993; Kossaibati and Esslemont 1997; Trajkovski 1997; Allore and Erb 1998; Hortet and Seegers 1998a; Lehenbauer and Oltjen 1998a; Fetrow 2000; Seegers et al 2003; Winkelman 2003; Berry et al 2004; Swinkels et al 2005) of cows that have or have had clinical mastitis (Bartlett et al 1990; DeGraves and Fetrow 1993; Beaudeau et al 1994; Allore and Erb 1998; Bascom and Young 1998; Hortet and Seegers 1998a; Lehenbauer and Oltjen 1998a) or elevated SCC (Bartlett et al 1990; Beaudeau et al 1994; Seegers et al 2003), and the increased expenditure associated with their replacement (Blosser 1979; Bishop et al 1984; Holdaway 1990; Beck et al 1992; McInerney et al 1992; Cullor 1993; Allore and Erb 1998; Lehenbauer and Oltjen 1998b). Mastitis is usually second only to reproduction as the largest involuntary culling category (Gill et al 1990; DeGraves and Fetrow 1993; Kossaibati and Esslemont 1997; Bascom and Young 1998; McDougall 2002; Seegers et al 2003; Berry et al 2004). Financial losses at the farm level can be attributed to the loss of future income and genetic potential (Holdaway 1990; Hillerton et al 1992; Cullor 1993; Allore and Erb 1998) resulting from culling. Schepers and Dijkhuizen (1991) state that the loss in this case is the difference between the income that a particular animal could earn during her remaining expected life and the expected average income from replacement animals with normal productive qualities and normal probabilities of disposal over the same period of time. However, the loss occurs only when animals have to be replaced before reaching their optimal economic age for culling.

The decision to cull is a complex one. There are different ways of classifying culling according to the motives that lead to the culling decision. The traditional concept distinguishes between voluntary and involuntary culling (Monti et al 1999). A different approach has gained attention that defines biologic and economic culls which allows consideration of all the factors that influence the decision-making process (Gill et al

1990). Lehenbauer and Oltjen (1998a) state that the culling strategies are further influenced by short-term fluctuations in cow numbers as well as by planned herd expansion. However, most cows are likely to be removed from dairy herds only after they have displayed several reasons that would lead to culling. Farmers may consider many cow factors, such as age, stage of lactation, milk production, health status, disposition, reproductive performance, economic factors, such as milk price, the price of culled cows, and the price, genetic merit and availability of replacement heifers when determining whether or not a cow should be culled. The large effect of clinical mastitis before peak lactation on short-term milk yield may partly explain the higherrate of culling of cows infected early in lactation (Fetrow 2000; Seegers et al 2003). When mastitis occurs later than 240 days after calving, the effect on culling is not evident (Rajala-Schultz and Grohn 1999). This could be explained by the fact that when the time of next calving is approaching, farmers are prone to wait until the next time the cow calves and see whether she has recovered from mastitis at the start of the next lactation (Rajala-Schultz and Grohn 1999).

As many of the factors of culling and replacement cost are not easily calculated, particularly the loss of genetic potential, it will be necessary to employ complicated dynamic programming model to estimate the cost of this group of factors. The culling of an infected cow is likely to reduce the risk of spread of infection through the herd (Stott and Kennedy 1993). The benefits of this effect should be included in the dynamic programming models.

2.4.2.5 Premium loss and penalties

(Allore and Erb 1998; Fetrow 2000; Seegers et al 2003; Winkelman 2003; Swinkels et al 2005)

Penalties and premium losses in many countries, particularly the European Community and Australia are an important part of the economic losses caused by mastitis. The stringent standards for a number of quality parameters including contamination with antimicrobial substances, microbes, flavour defects, and concentration of milk components as well as somatic cells count are monitored and penalised or compensated for in different countries. Morin et al (1993) reported 21-40% of the cost of mastitis in 4 Illinois herds resulted from milk quality premium losses.

All costs associated with loss of premiums and incurred penalties at the farm level are easily calculated from the statements of the milk collecting and processing companies. To estimate the cost at cow level, calculations from the data collected at the farm level will need modelling, taking into account cow numbers and mastitis occurrence. In the final model estimating costs of premium losses and incurred penalties error by staff (Fetrow 2000) should be taken into account, as this will lead to over-estimates of the cost of mastitis; for example milking of a cow before withholding period is finished, etc.

In the literature the following authors list or detail the premium loss and penalties as factor associated with losses caused by mastitis: Allore and Erb (1998), Fetrow (2000), Seegers et al (2003), Winkelman (2003), and Swinkels et al (2005).

2.4.2.6 Pre-term drying off

In many cases, particularly from mid-lactation onward, and when there is a recurring case, pre-term drying off the affected quarter of a cow is advocated. To avoid underestimates of mastitis consequences, all those cases should be specifically recorded and accounted for in economic-loss cost evaluations (Hortet and Seegers 1998a; Winkelman 2003).

2.4.2.7 Animal welfare aspect of mastitis

The welfare implications of peracute toxic mastitis are obvious. Allore and Erb (1998) and Pyorala (2002) state that more recent studies, have demonstrated significant secondary hyperalgesia in cows following mild clinical episodes of mastitis. It has been now accepted that mastitis is associated with hyperalgesia, particularly in acute and peracute cases (Fitzpatrick 2004). Allodynia has been demonstrated for approximately 5 and 40 days in the case of mild and moderate cases of mastitis, respectively (Fitzpatrick 2004). Concentrations of bradykinin, cortisol and other kinins change during clinical mastitis (Shuster et al 1991a, 1991b, 1991c; Eshraghi et al 1999). Therefore, supportive treatment of each case of mastitis can be an issue in the near future, leading to increased costs of mastitis.

According to the Milk Hygiene Directive 92/46 EEC it is not allowed to deliver milk from cows suffering from recognisable inflammation of the mammary gland.

2.4.2.8 Associated health problems

Mastitis is commonly associated with other health problems such as reproductive failure (McInerney et al 1992; Kossaibati and Esslemont 1997; Fetrow 2000; Schrick et al 2001; Swinkels et al 2005) and loss of appetite (McInerney et al 1992; Kossaibati and Esslemont 1997; Seegers et al 2003). There will be some indirect costs due to increased risk of these associated health problems.

Recently there has been a trend when estimating mastitis costs to take into account less food consumed to produce less milk; a factor that was not usually considered (Berry et al 2004; Hillerton and Berry 2005). However, trying to differentiate between loss due to inflammation of the secretory tissue and that due to a decreased intake because the cow is not feeling well is an unrealistic sophistication. A healthy udder is more efficient at converting nutrients into milk (Berry et al 2004), so to estimate the real cost of mastitis regarding feed intake will need a complicated modelling.

Schrack et al (2001) reported that cows with clinical or sub-clinical mastitis before the first service had increased days to first insemination, increased days open and increased service to conception. This indicates that some of the losses from associated health problems can be calculated using relatively simple modelling.

2.5 Cost of mastitis control programmes

The cost of mastitis control include expenditures which can be measured directly from invoices or calculated according to standard treatment and prevention costs, and from labour time for monitoring, treatment, prevention (Bishop et al 1984; DeGraves and Fetrow 1993; Seegers et al 2003), and other expenditures.

Expenditure on mastitis control is determined by the methods employed, namely: educational costs, pre-milking preparation of the udders, teat disinfection, dry-cow therapy and mastitis vaccines, monitoring measures, and maintenance of the milking machine. On the other hand, some authors also include the treatment of clinical cases (Gill et al 1990; Morin 1993), the pre-partum treatment of heifers (Oliver et al 2003), culling (Gill et al 1990; Morin 1993) and the management changes in milking routine (Gill et al 1990; Morin 1993; Seegers et al 2003), such as milking infected cows last. For example Oliver et al (2003) reported that pre-partum antibiotic treatment of heifers

yielded net revenue of around US\$200/heifer/year. Contagious mastitis in the herd is associated with shedding of the mastitis-causing organisms during milking and the risk of cross-infection to other cows in the herd (Craven 1987; Holdaway 1990; Morin 1993; Stott and Kennedy 1993; Swinkels et al 2005). In such cases the mastitis prevalence in the herd will change, and consequently mastitis costs will be increased. A proper transition management with the added cost of feed additives, minerals and vitamins in particular, plays an important role in modern mastitis control programmes. A relatively new area in mastitis control is vaccination against different mastitis-causing organisms, and some research on the benefits of this procedure is already available. DeGraves and Fetrow (1991) estimated a benefit of \$57 per cow if the herd is vaccinated, assuming that 1% of the cows would normally contract coliform mastitis during the season.

2.5.1 Educational costs

Continuous education of farmers is a necessary tool in the battle against bovine mastitis (Fetrow 2000). Farmers need to be aware of economic cost of mastitis in the herd and the cost benefits of a mastitis control programme that will increase the farm's net income.

The importance of education is demonstrated by the survey conducted by Gill et al (1990). They found that a regular visit by a veterinarian or udder health specialist, more years of ownership or managing a farm, more education, and frequent attendance at dairy extension seminars were associated with lower SCC, while the increase in the total number of people working on the dairy farm was associated with an increased SCC (Gill et al 1990). In contrast Kuiper et al (2005) found that the education factors were not as important as premiums and penalties applied for milk quality.

The costs associated with education of the farmers and the labour can be partially estimated from the invoices for attended courses. Time spent on education is difficult to estimate. Also decreased SCC were associated with the higher education qualifications of the farmers or workers and not necessarily related to knowledge about mastitis, so to try and attribute this to the cost of mastitis seems somehow unrealistic.

2.5.2 Pre-milking preparations of the udders

(Gill et al 1990; DeGraves and Fetrow 1993; Morin 1993; Seegers et al 2003)

The costs of this procedure include the time the milker takes for pre-stripping, washing and drying the udders; use of water and teat disinfectant for washing, and paper towels for drying the udders. Pre-milking teat disinfection is a relatively new concept in mastitis control. The majority of authors conclude that this procedure is generally effective and not expensive (Ingawa et al 1992; Pankey and Drechsler 1993; Oliver et al 2001). On the contrary, Ruegg and Dohoo (1997) reported that the reduced incidence of clinical cases of mastitis does not justify the added expense incurred from pre-milking teat disinfection, with a benefit to cost ratio of 0.37. Further research is needed to evaluate the economic impact of the procedure.

2.5.3 Post-milking teat disinfection

When assessing the cost of teat disinfection there are three main elements that should be addressed, namely the cost of the teat disinfectant, the installation and maintenance cost, and labour cost (if included as a cost of mastitis). Gill et al (1990) found that the cost of teat disinfectants is quite variable and is influenced by the amount used per cow per year and the cost per litre. On top of this the cost of the emollient used should be added when it is used.

Farms that have equipment for back-flushing of the milking units should include the cost of installation, maintenance and any disinfectant used.

In the literature the following authors list or detail post-milking teat disinfection as a factor associated with mastitis cost: Anderson and Blackshaw (1977), Fetrow (1987), Gill et al (1990), Beck et al (1992), McInerney et al (1992), DeGraves and Fetrow (1993), Morin (1993), and Seegers et al (2003).

2.5.4 Dry-cow treatment and mastitis vaccines

The cost of commercial dry-cow products (antimicrobials and teat sealants) is somewhat variable, being influenced by the product used (Fetrow 1987; Gill et al 1990; Beck et al 1992; McInerney et al 1992; DeGraves and Fetrow 1993; Morin 1993; Seegers et al 2003; Winkelman 2003). The cost will be influenced by the numbers of

cows treated at drying off if selective dry-cow therapy is used. If labour cost is considered as an element of the mastitis cost, then it should be added to the calculations.

The same procedure can be used for calculation of the estimated cost of vaccines against mastitis (DeGraves and Fetrow 1993; Allore and Erb 1998; Pyorala 2002; Seegers et al 2003) or some of the other immuno-modulatory systems (Pyorala 2002) and their application.

2.5.5 Monitoring measures

(Fetrow 1987)

2.5.5.1 SCC monitoring cost

(Fetrow 1987)

The cost of herd testing is calculated easily from invoices. Herd testing is done by different companies around the world and these usually record the volume, protein and fat content, and SCC in the milk from each individual cow. For example herd testing in New Zealand is done by Livestock Improvement Corporation (LIC) and Ambreed. The information gained from herd testing is vital for effective herd management and decision making. If labour cost is considered as an element of the mastitis cost, then it should be added to the calculations. Zepeda et al (1998) estimated that testing and monitoring pays for itself over a short period of time, except when there is very low incidence of mastitis and very low SCC.

2.5.5.2 Culturing

(Gill et al 1990; DeGraves and Fetrow 1993; Morin 1993)

The cost of detecting and characterising mastitis-causing organisms from infected cows or bulk tank milk is variable and depends on the numbers of samples submitted and the laboratory used for culturing. The cost of materials (sample tubes, alcohol, wipes, cotton wool) should be added to the calculations of the mastitis control programme. If labour cost is considered as an element of the mastitis cost, then it should be also added to the calculations.

Some mastitis control programmes may include more extensive culturing. In this case, individual cows that are likely to be sub-clinically infected are identified either by using the somatic cell count or other methods for sub-clinical mastitis diagnosis, and milk samples are cultured. Cows with culture-positive milk can then be treated, based on the mastitis-causing organism, sensitivity results, age of the cow, stage of lactation and productivity.

2.5.6 Milking system and milking procedure analysis

Analysis of milking equipment and procedures is highly variable, influenced by the herd size, past history and management level (Fetrow 1987; Gill et al 1990; McInerney et al 1992; DeGraves and Fetrow 1993; Edmondson 1993; Morin 1993). However, regular milking machine tests lead to better results and have a high cost benefit ratio (Edmondson 1993).

2.5.7 Transition period management

It has been reported that cows suffering from clinical parturient hypocalcaemia have been associated with a nearly 9-fold increased risk for mastitis (Epperson 2005). The diet of a dairy herd plays an important role in cow productivity, and its general ability to resist disease (Pyorala 2002). Nutritional relationships to host defence mechanisms have led to the idea of increasing the resistance of dairy cattle to mastitis through nutrition. Not only gross malnutrition, but also merely suboptimal levels of any one micronutrient is sufficient to adversely affect mammary gland immunity (Hogan et al 1993; Jukola et al 1996; Sordillo et al 1997; Petrovski 2005).

Mastitis control programmes should ensure that proper intakes of all macro- and micro- nutrients are maintained in all cows at all times. The key to ensuring adequate levels of these important micronutrients is direct testing of animals at the herd level to delineate patterns in overall nutrient deficits (Sordillo et al 1997).

Transition period management includes many other procedures associated with mastitis control, for example teat disinfection and pre- or post-calving treatment of heifers.

The cost of the supplements is easily calculated from invoices. The cost of other procedures is calculated in the same way as for normal treatment or teat disinfection.

2.6 Discussion and conclusions

Bovine mastitis is considered as the most costly production disease to the dairy industry worldwide. Estimating the costs associated with mastitis is notoriously difficult. It is even more difficult to quantify the losses associated with sub-clinical mastitis as they are not visible to the farmer. The economics of mastitis needs to be addressed at the farm or herd level and depends on local, regional, epidemiological, managerial and economic conditions. When considering the cost of any disease, it is necessary to keep in mind that every disease has a direct and an indirect cost. Direct costs and expenses are usually the only ones realised by the farmer. Indirect losses due to mastitis are not realised by the farmer in many cases and are a reason why the implementation of mastitis control measures is difficult, so continuous education on this matter is necessary. Some of the costs and expenditures are easy to calculate and they should be included in research projects dealing with the modelling of the economics of the disease. However, some of the costs are not countable, such as cases of human diseases, farmers' stress etc. To be able to consider the real cost of mastitis to the dairy industry, the prevalence and incidence of mastitis on a national level should first be established. Then estimations of all relevant countable costs and expenditures should be made, and the last step will be to include all of them in one large model for mastitis cost estimation.

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Chapter three

The Incidence and Aetiology of Bovine Mastitis in 14 Herds from Northland, New Zealand

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3 The Incidence and Aetiology of Bovine Mastitis in 14 Herds from Northland, New Zealand

3.1 Abstract

3.1.1 AIMS: To investigate the incidence of clinical mastitis over a single lactation (July 2005 to July 2006), the frequency of isolation of different mastitis-causing organisms, the prevalence of subclinical mastitis, and estimation of the predictive values of the first available herd test for prediction of clinical mastitis in herds from the Northland region of New Zealand.

3.1.2 METHODS: The incidence of clinical mastitis was determined by farm personnel recording the identities of affected cows. Composite milk samples of the affected quarters were collected aseptically for microbiology. Mean numbers of affected cows and quarters were compared at the population and herd level per 305 cow-days-at-risk. Subclinical mastitis was determined as the presence of somatic cell counts >250,000 cells/ml during routine herd testing. Individual cow somatic cell counts from the first available herd test for the season were used to predict whether treatment of subclinical mastitis would prevent development of clinical mastitis.

3.1.3 RESULTS: Of 3765 lactating cows, 559 (14.8%) had one or more episodes of clinical mastitis. The average incidence of clinical mastitis was 0.19 cases per 305 cow-days at-risk. The incidence in rear quarters (56.2%) was 1.3 times that of front quarters (43.8%). The incidence of clinical mastitis and numbers of affected quarters was significantly influenced by the stage of lactation (higher in early lactation), age (higher in older cows) and herd. At the cow level, the most common isolates were *Staphylococcus aureus* (23.7%), and *Streptococcus uberis* (23.3%). No causative organisms were present in 27.3% of the samples. The estimated prevalence of subclinical mastitis increased through the season (i.e. from calving to dry-off). Four hundred and twelve cows would have to be treated to attempt to prevent clinical mastitis in only 49 cows.

3.1.4 CONCLUSIONS: This study demonstrated a higher incidence of staphylococcal infections in clinical mastitis in Northland, compared to findings from other regions of New Zealand.

3.1.5 KEY WORDS: Mastitis, dairy cows, *Staphylococcus aureus*, *Streptococcus uberis*, Northland

3.1.7 ABBREVIATIONS: BTSCC – bulk tank somatic cell count, CNS - coagulase-negative staphylococci, DAR – days at risk, DIM – days in milk, ICSCC - individual cow somatic cell count, IMI – intramammary infection, SCC – somatic cell count.

3.2 Introduction

Bovine mastitis is one of the most common and economically important diseases of dairy cows. Mastitis has been estimated to have cost the New Zealand dairy industry around NZ\$180 million/year in 2005/06 (Anonymous, 2006a) and the United States dairy industry approximately US\$2 billion/year in 1992/3 (Miles et al 1992; Cullor 1993). However, it is likely that these figures are underestimates, as, when considered in detail (Petrovski et al 2006), there are many factors other than those commonly considered by which mastitis causes economic losses.

Mastitis is an inflammation of the mammary gland, which usually occurs in reaction to invasion by mastitis-causing organisms. It is a multi-factorial and complex disease resulting from interaction between cows, microorganisms and their environment (Watts 1988; Schukken and Kremer, 2001). Most cases of clinical mastitis in dairy cows are infectious in aetiology (Craven 1987; Sandholm et al 1990). Although many bacteria, yeasts, viruses and fungi have been isolated from bovine mammary glands, only a small group of these organisms cause intramammary infections (Watts 1988; Malinowski et al 2002; Wellenberg 2002). More than 90% of all new intramammary infections (IMIs) are caused by a few mastitis-causing organisms; namely, *Streptococcus agalactiae*, *Staphylococcus aureus*, *Strep. dysgalactiae*, *Strep. uberis*, coagulase-negative staphylococci (CNS), *Corynebacterium bovis* and *Escherichia coli*.

Mastitis-causing organisms are categorised as contagious or environmental, based on the source of infection, mode of transmission and the tendency to cause persistent or transient IMI (Bramley and Dodd 1984). The primary reservoir of contagious mastitis-

causing organisms is the bovine mammary gland, whilst the predominant means of spread is transmission between cows during milking. *Staphylococcus aureus* and *Strep. agalactiae* have historically been considered as the most important contagious mastitis-causing organisms. Lately, however, particularly in the USA, *Mycoplasma* spp. have been reported with increasing frequency as the cause of major mastitis problems in affected herds (see Makovec and Ruegg 2003). The primary reservoir of environmental mastitis-causing organisms is the cows' environment. The most important environmental mastitis-causing organisms are streptococci (other than *Strep. agalactiae*), CNS and enterobacteria. In New Zealand, the most important environmental organism is *Strep. uberis* (e.g. Douglas 2000). However, the distinction between contagious and environmental mastitis-causing organisms is less clear than originally thought, since some environmental organisms persist in the mammary gland throughout the dry period and beyond. Moreover, for both of *Strep. uberis* and *Strep. dysgalactiae*, transmission from cow to cow also occurs (Grommers et al 1985; Zadoks et al 2003). In consequence, some authors have classified *Strep. dysgalactiae* as a primarily contagious organism (Dodd and Naeve 1970).

Changes control methods for mastitis and of production practices in dairying, together with the availability of potent antimicrobial agents, have resulted in significant changes in the causative factors and the nature of clinical mastitis. A considerable reduction in the overall incidence of clinical mastitis has taken place over the past 30-40 years. For example, in the UK, the national average incidence in 2000 was 43 cases per 100 cows per annum, compared with approximately 140 prior to 1970s (Dodd and Naeve 1970; Wilson and Kingwill 1975; Blowey and Edmondson 2000). Furthermore, *Strep. agalactiae* was the most prevalent streptococcal cause of mastitis before the onset of widespread use of dry cow therapy and post-milking teat disinfection, but is now relatively uncommon in most of the developed dairy countries (Makovec and Ruegg 2003; Pitkala et al 2004). By contrast, there has been a slight increase in the incidence of clinical mastitis due to environmental organisms over the same period that is independent of reporter or location (Bramley and Neave 1975; Blowey and Edmondson 2000; Bradley and Green 2001; McDougall 2002b), so that the relative contribution of *Strep. uberis* to clinical case of mastitis has risen from 7 to 33% over

the period (Wilesmith et al 1986) . It has been postulated that this is occurring because the niche vacated by the contagious mastitis-causing organisms is being occupied by the environmental organisms (Erskine et al 1988; Myllys et al 1998; Phuektes et al 2001). However, it is also evident that many well-managed farms that have successfully controlled contagious mastitis (including *S. aureus*) and that consistently produce milk with low somatic cell count (SCC, below 300,000 cells/ml (USA) or 150,000 cells/ml (Europe, Australia and New Zealand)) have problems with the incidence of clinical mastitis caused by environmental organisms. It therefore appears likely that the control of contagious organisms, with a consequential concurrent decrease in leukocyte counts in milk, has also facilitated a rise in the incidence of mastitis due to environmental organisms (McDougall 1998a; Taponen et al, 2006).

Relatively few studies have examined the overall incidence of mastitis in New Zealand dairy herds. McDougall (2002a) reported the incidence of clinical mastitis to be 14 cows/100 cows per annum, with the majority of cases occurring around calving. Intervention studies in New Zealand have found that 10% of cows acquired new infections during early lactation (Pankey et al 1982; McDougall 1998a). Brookbanks (1966) reported that 32% of cows had a positive rapid mastitis test during a study conducted in 130 herds throughout New Zealand. The authors are not aware of other national prevalence estimates. Early surveys of the prevalence and frequency of mastitis due to different organisms showed that 14 to 18% of cows were infected with *Strep. agalactiae*, 27 to 41% with *S. aureus* 2 to 4% with *Strep. dysgalactiae*, less than 3.0% with *Strep. uberis* and 25% CNS (Brookbanks 1966; Elliott et al 1976). More recently, the prevalence of *Strep. agalactiae* and *S. aureus* appears to have fallen, with *Strep. uberis* now being reported as the most common cause of bovine mastitis. Reports from intervention surveys in the Waikato reported the proportions of mastitis-causing organisms from cows with clinical mastitis to be: *Strep. uberis*: 27 to 75%, CNS: 9 to 10%, coliforms: 2 to 5%, *Strep. dysgalactiae*: 1.5 to 4% and 3 to 5% *S. aureus* (McDougall 1998a, 2003). In these reports only 50% to 70% of samples yielded positive bacteriological cultures. Whether this pattern of mastitis-causing organisms represents that of other parts of the country, in which different climatic and husbandry practices

pertain, has not been established. In particular, anecdotal evidence from veterinarians and laboratory workers suggested that there might be different patterns of mastitis in Northland to those observed elsewhere in New Zealand.

The aim of this project was therefore to estimate the incidence, aetiology and distribution in lactation of mastitis-causing organisms in cases of clinical mastitis in the Northland region of New Zealand.

3.3 Materials and methods

3.3.1 Study Design

A longitudinal prospective study investigating the incidence of clinical mastitis over a single lactation, the incidence and distribution of different mastitis-causing organisms, the prevalence of subclinical mastitis, and estimation of the predictive values of the first available herd test for prediction in prevention of clinical mastitis incidence.

3.3.2 Study Population and Description of Farms

Data were collected from 14 seasonal spring-calving, pasture-based, dairy farms from the Dargaville, Aranga, Waihue, Tangiwhae, Tangiteroria and Ruawai areas of Northland, New Zealand. Farms were selected on the basis of regular herd testing, a history of keeping mastitis treatment records, a willingness to collect samples and farmer consent. The study period was July 2005 to July 2006 and included observations on calving, events during lactation, drying-off and during the dry period of the 2005/06 season. The total study population included 3765 lactating cows with an average 294.1 (95% Confidence Interval 276.2-312) days in milk (DIM). All cows were identified using ear tags (plus freeze branding on one farm).

Management of lactating cows was similar among farms and showed due regard for their welfare. Usual farming practices and feeding were followed during the study period. All study farms predominantly grazed pasture throughout the year, with regular fertilizing of the paddocks. Supplementary feeds were provided for at least part of the season on 13 out of 14 farms (grass silage: 12/14, palm kernel: 9/14, turnip crop: 8/14, hay: 5/14 farms). Cows were milked twice daily on 12 and once daily on two of the 14 farms.

All study farms kept records on forms supplied for the purpose and in note books. Additionally, 12 farms used computer records or ‘Best on Farm Practice’ booklets. Most of the farms also used another recording technique (white board, temporary notes, and hand-held computerised systems, such as M-note or PAM).

Mastitis was managed by farm personnel. Cows that had quarters diagnosed with mastitis by farm personnel were assigned to the clinical mastitis study group. Cow identification, affected quarter/s and treatment/s were recorded.

3.3.3 Procedures

3.3.3.1 Mastitis Diagnosis:

Diagnosis of clinical mastitis in affected quarter/s was undertaken by farm personnel using procedures that were currently practiced on the particular farm. The procedures used to diagnose mastitis and opinion of farms personnel on their importance are presented in Table 3. 1. A questionnaire that had previously been given to the farm staff and owner/share-milker was used to generate the criteria used in this table.

Table 3.1. Diagnostic procedures and their importance in the diagnosis of clinical mastitis on different farms (0- not practiced; 1- rarely; 5- very often) as graded by the farm personnel

Farm Id	Abnormal udder	Herd test	Abnormal milk	CMT ²	Filter socks	Hot quarter	Cow behaviour	Other	Sick cow	EC ¹ meter
1	4	5	4	5	5	4	2	5	2	5
2	4	3	5	4	2	1	2	0	1	0
3	5	5	5	0	4	0	0	0	0	0
4	5	5	5	5	5	5	0	2	1	3
5	4	3	4	0	3	0	3	0	0	0
6	5	5	4	0	0	0	0	0	0	0
7	4	2	4	3	3	0	1	0	0	0
8	4	5	5	5	3	4	0	5	0	0
9	5	3	0	5	0	5	2	5	0	0
10	5	5	5	5	4	5	5	0	5	5
11	5	5	5	5	5	5	5	0	5	0
12	5	5	5	5	5	4	5	0	5	0
13	5	5	1	3	5	5	0	0	0	0
14	5	2	3	4	5	3	4	5	1	0
Total	65	58	55	49	49	41	29	22	20	13
Median	5	5	4.5	4.5	4	4	2	0	0.5	0

¹Electrical conductivity ² California mastitis test

3.3.3.2 Milk Sampling:

Milk samples were aseptically collected, before the commencement of antimicrobial treatment, from quarters that were affected by clinical mastitis. The teats were

washed, disinfected, dried and the fore-milk discarded, prior to collecting a composite milk sample from all affected quarters. Unaffected quarters were not sampled. The milk samples were stored at approximately -20°C in domestic freezers immediately after milking. Samples were later dispatched to the New Zealand Veterinary Pathology Laboratory in Palmerston North.

3.3.3.3 Microbial Culture:

Each milk sample was subjected to routine microbiological culture and isolated mastitis-causing organisms were identified to genus and/or species level. Microbiological examination was performed according to IDF recommendations (Anonymous, 1987).

3.3.4 Statistical Analysis

Raw data were available from 3892 animals. Of these, 127 animals were excluded on the basis of lactations of >365 days. These animals were exposed to more than one lactational period and their risk was assumed to be different. After exclusion of these animals, the data set 'incidence data' included 3765 animals. Only the incidence of clinical mastitis during lactation was determined.

Data sets were incomplete for some cows, lacking information on age (n=95) and individual cow somatic cell count (ICSCC) (n=104). These cows were excluded from the relevant parts of the analysis.

Ages of the cows was recorded in years (range 2 – 16). All animals whose ages were 6 years and over were amalgamated into a single age category (Age ≥6) for statistical analysis.

Herd test data were available for 3689 cows. These data were used in the analyses of the predictive value of the first available herd test for prevention of clinical mastitis by treatment and in the estimation of the prevalence of subclinical mastitis.

Statistical analyses were performed using SAS (Statistical Analysis System, version 9.1; SAS Institute Inc., Cary, NC, USA) and Excel (Microsoft Office, version 2003; Microsoft Corporation, USA).

3.3.4.1 Days at Risk:

The days at risk (DAR) were calculated and manually corrected using the following formulae:

- a) Cows that did not have a clinical mastitis episode:

$$\text{DAR} = \text{Total DIM for the season}$$

- b) Cows that experienced a clinical mastitis episode and no further episode occurred in the 17 day recovery period at the beginning or end of lactation:

$$\text{DAR} = \text{DIM} - (\text{number of mastitis episodes} \times 17)$$

- c) Cows that had clinical mastitis and another episode occurred in the 17 days recovery period (a typical three days of treatment plus 14 days recovery):

$$\text{DAR} = \text{DIM} - (\text{number of mastitis episodes} \times 17) - \text{days between subsequent clinical mastitis episodes when these were } \leq 17 \text{ apart (if they were } > 17 \text{ days apart, the last clinical mastitis was a new event)}$$

3.3.4.2 Incidence of Lactational Clinical Mastitis:

Clinical mastitis was calculated as incidence per 305 DAR. If clinical mastitis recurred in the same cow more than 17 days after the diagnosis of the first occurrence it was considered as a new case or recurrence. If clinical mastitis recurred in the same cow within 17 days after the first clinical mastitis occurrence, it was assumed that was a relapse of the same case. It was assumed that, during the 17-day recovery period the cow was not at risk of re-infection.

The overall incidence of clinical mastitis and the incidence of mastitis due to individual organisms (i.e. SU (*Strep. uberis*), SA (*S. aureus*) and OTH (other mastitis-causing organisms)) per 305-days lactation were calculated at a cow level as:

$$\text{CM Incidence} = \frac{\text{Total number of CM cases} \times 305}{\text{Total number of cow-days-at-risk}}$$

The frequency of recurrence for repeated cases was calculated as:

$$\text{Recurrent CM} = \frac{\text{Total number of cows treated more than once for CM}}{\text{Total number of cows with CM}}$$

The effects of herd and age on clinical mastitis incidence were estimated by a generalised linear model using a Poisson distribution and adjusting variances for over-dispersion (Pearson residual chi-square/degrees of freedom error; proc GLIMMIX in SAS). Over-dispersion correction was required because the mastitis incidence of cows from the same herd was assumed to be more similar than that of cows from different herds; hence observations were not likely to be independent. Total and organism-specific incidence at a cow level was also calculated as a 4-week moving average to examine periods of highest risk throughout the course of lactation.

In addition the following measures were calculated:

i) Case-ratio of clinical mastitis in front versus rear quarters (CR-FVR):

$$\text{CR-FVR} = \frac{\text{Total number of CM cases in front quarters}}{\text{Total number of CM cases in rear quarters}}$$

ii) Case ratio CM in right versus left quarters (CR-RVL):

$$\text{CR-RVL} = \frac{\text{Total number of CM cases in right quarters}}{\text{Total number of CM cases in left quarters}}$$

3.3.4.3 Removal from Supply:

If a cow was removed from supply for the remainder of the season (such as by transfer to a group of nurse-cows, pre-term dry off, culling or death due to mastitis) she was counted as having been removed from supply due to mastitis. The risk of cows removed from supply due to mastitis was calculated as the number of cows removed divided by the number of cows calving. The risk of mastitis resulting in a removal from the herd was calculated as the number of cows removed for mastitis divided by the number of cows with mastitis and the total number of cows in the herd.

3.3.4.4 Predictive Values of ICSCC from the First Available Herd Test for the risk of clinical mastitis:

The risk of clinical mastitis during 60 days after the first herd test date was calculated by logistic regression analysis of the presence of an ICSCC $\geq 250,000$ cells/ml, (adjusted for over-dispersion and for the confounding effects of herd and age) and occurrence of a clinical mastitis episode prior to the first herd test. Predicted proportions were used to define the positive/negative predictive value (NPV/PPV) as PPV = probability of

clinical mastitis given high ICSCC, and NPV=1 - (probability of clinical mastitis given low ICSCC).

3.3.4.5 Prevalence of Subclinical Mastitis, assumed new infection and assumed cure rates:

Subclinical mastitis (i.e. IMI without observed clinical symptoms) was estimated from ICSCC as for predictive values (i.e. ICSCC $\geq$ 250,000 somatic cells/ml). An assumed new IMI was defined as presence of low ICSCC at one herd test followed by high ICSCC at the next herd test. Assumed cure from subclinical mastitis was defined as the presence of high ICSCC at one herd test followed by low ICSCC at the next herd test. If there was no change in the status of the ICSCC level then the cow remained in the same category (i.e. contributed to the denominator only).

3.4 Results

3.4.1 Cows and milk production

Table 3.2. Number of cows and milk production per lactating cow per annum among farms in 3765 cows from dairy farms in Northland, New Zealand.

Farm	Lactating cows	Heifers	Milk Production ¹	Average days in milk
1	166	26	295.6	271
2	249	59	213.1	290
3	340	55	251.9	318
4	312	95	264.6	307
5	213	64	285.6	318
6	198	35	296.4	375
7	173	40	291.4	297
8	221	58	238.9	270
9	247	18	304.4	230
10	423	94	262.3	323
11	212	35	294	295
12	591	99	281.2	268
13	228	32	313.4	278
14	192	0	225.5	277
Total	3765	710	3818.3	4117
Median	224.5	47.5	283.4	292.5
95% CI ²	61.01	15.71	16.12	17.89

¹Kilograms of milk solids per lactating cow per year; ²95% confidence interval

The number of lactating cows per farm is shown in Table 3. 2. A total of 3765 lactating cows from 14 farms were included in the study. The average number of lactating cows

per farm was 269. Herd size varied between 166 and 591 lactating cows per farm. Average milk production per lactating cow and farm, and duration of lactation are shown in Table 3. 2. Average production per cow was 272.7 (95% confidence interval 256.6-288.8) Kg milk solids/cow over an average lactation period of 294.1 (95% confidence interval 276.2-312.0) DIM.

3.4.2 Incidence of clinical mastitis

In the 3765 lactating cows included in the study, clinical mastitis occurred in 1125 quarters in 559 lactating cows (14.8%) during 638 episodes of clinical mastitis. In 222 of the cases (34.8%) more than one quarter was concurrently affected. Each affected cow had an average of 1.8 quarters diagnosed with clinical mastitis per episode. There were 61 (10.9%) cows with recurring cases of clinical mastitis (using the definition of recurrence given above). A cow with clinical mastitis had a 12.2% chance of having at least one recurrence.

Moving averages of the case incidence of clinical mastitis are presented in Figure 3.1. Moving averages of quarter mastitis incidence were very similar (data not shown). The highest incidence of clinical mastitis was in the first week (Days 2-7) post-calving with 1.89 cases per 305 cow-days-at-risk. The incidence then progressively decreased until Week 15, after which it remained low until near to the time of drying off, when a small increase occurred.

The age specific incidence of clinical mastitis (Figure 3. 2) was highest in cows ≥ 5 years old (17.3, 95% confidence interval 13.0-23.0), while the lowest clinical mastitis incidence was registered in 3-year old cows (9.8, 95% confidence interval 7.0-13.8). Significant differences in the incidence of clinical mastitis incidence existed between age categories 2 and 6 ($p < 0.05$), and categories 3 and ≥ 5 ($p < 0.05$). Although not statistically significant, it appeared that the quarter incidence in cows 2 or 3 years old was similar but the cow level incidence was lower in 3-year cows suggesting that 3-year old cows had more quarters affected per mastitis event than 2-year old cows.

The case incidence of clinical mastitis (Figure 3.3) varied between farms from 0.04 to 0.45 cases per 305 cow-days-at-risk ($p < 0.0001$) with an average of 0.19 cases per 305 cow-days-at-risk (95% confidence interval 0.13-0.25).

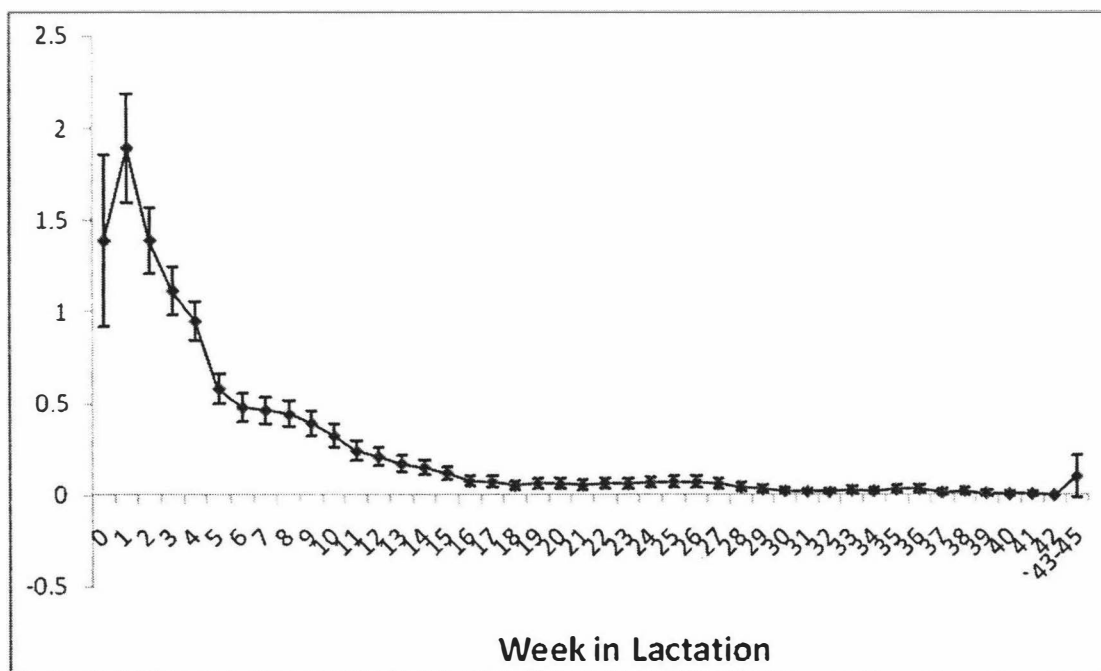


Figure 3.1. Four-weekly moving average and SE of the case incidence of clinical mastitis (cases per 305 cow-days-at-risk) in 3765 cows from 14 dairy farms in Northland, New Zealand. $p < 0.001$.

'Week 0' = Day of calving, 'Week 1' = Days 2-7 smoothed over Day 1-7, 'Week 2' = Days 8-14 smoothed over Day 1-14, 'Week 3' = Days 15-21 smoothed over 1-21 Days, 'Week 4' = Days 22-28 smoothed over 2-28, 'Week 5' = Days 29-35 smoothed over 8-35, etc.

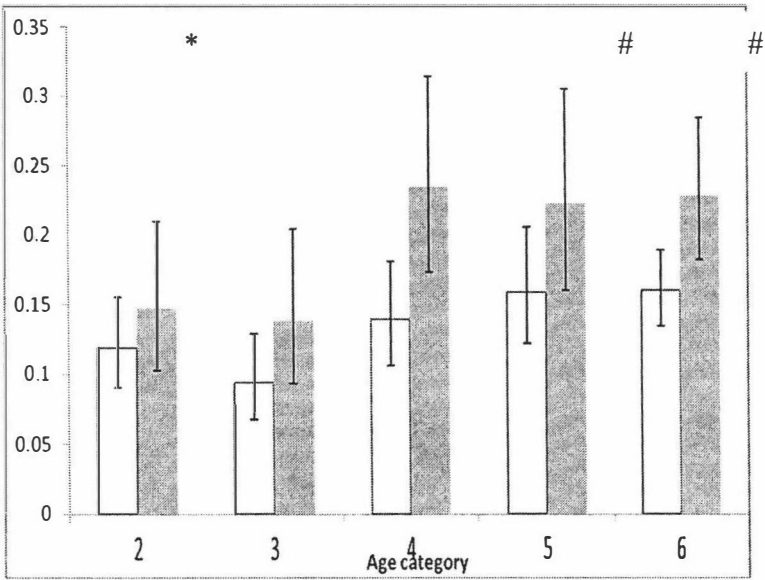


Figure 3.2. Age related incidence of clinical mastitis events in cows (white bars) and quarters (grey bars) and SE in 3765 cows from 14 dairy farms in Northland, New Zealand. * significantly lower than grand mean for all age groups; # significantly higher than grand mean for all age groups ($p < 0.05$)

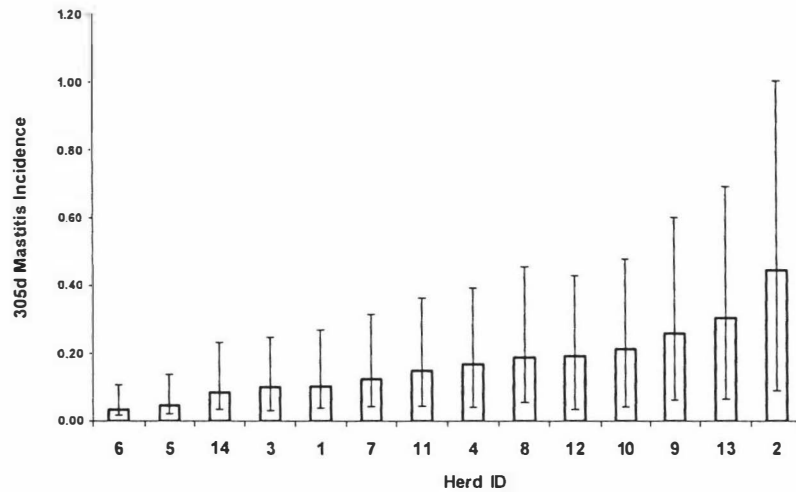


Figure 3.3. 305-days herd incidence of clinical mastitis and SE in 14 dairy farms in Northland, New Zealand

The distribution of quarter location affected by clinical mastitis is presented in Figure 3.4. The number of rear quarters affected by clinical mastitis was 1.3 times higher than that of front quarters ($p = 0.027$). There was no difference in clinical mastitis episodes between left (49.2%) and right quarters (50.8%, $p = 0.13$). The analysis of quarter level clinical mastitis incidence in herds and age groups resulted in similar patterns as did the cow-level analysis (data not shown).

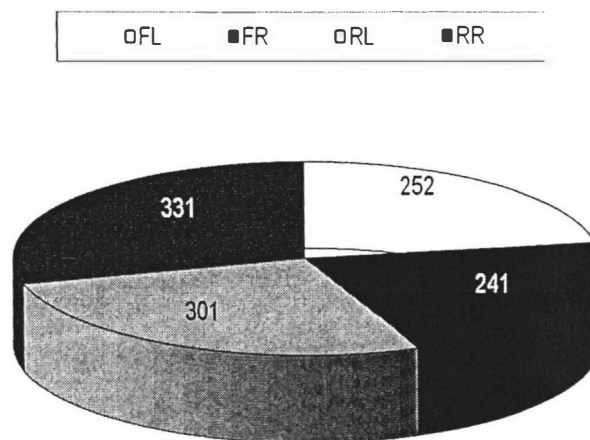


Figure 3.4. Numbers of episodes of clinical mastitis per quarter position.

3.4.3 Distribution of mastitis-causing organisms

Milk samples were collected from 417 out of 638 cases of clinical mastitis (65.4%). The number and percentage of different mastitis-causing organisms isolated from these samples, at a cow level, are presented in Table 3.3. The most frequently isolates were *S. aureus* (23.7%), and *Strep. uberis* (23.3% of isolates). The difference between the total number of *S. aureus* and *Strep. uberis* isolations over the season was not significant ($p=0.77$). No organisms were cultured from 27.3% of the samples and milk samples were not collected from 232 (34.6%) clinical mastitis cases. However, as there was no difference in the proportion of animals from which no causal organism was isolated at different stages of lactation, it was considered that the fraction of clinical mastitis sampled for culture or culture negative was therefore unbiased by stage of lactation.

Table 3.3. Total number and percentage of mastitis-causing organism isolates before and after re-classification (sampled 417 cows with clinical mastitis)

Mastitis-causing organism or not sampled	Re-classified	Total number of isolates	Per cent of total number cases	Per cent of isolates
Not sampled	221		34.64	N/A ¹
No growth	174		27.27	41.73
<i>Staphylococcus aureus</i>	99		15.52	23.74
<i>Streptococcus uberis</i>	97		15.2	23.26
Coagulase negative staphylococci	OTHER	16	7.36	2.51
Mixed growth		8		1.25
<i>Escherichia coli</i>		5		0.78
<i>Enterococcus</i> spp		4		0.63
<i>Streptococcus dysgalactiae</i>		4		0.63
<i>Bacillus</i> spp		2		0.31
<i>Corynebacterium</i> spp		2		0.31
<i>Nocardia</i> spp		2		0.31
<i>Streptococcus agalactiae</i>		2		0.31
<i>Acinetobacter</i> spp		1		0.16
<i>Micrococcus</i> spp		1		0.16
Total		638	100	100

¹Not applicable

The incidence of clinical mastitis, at a cow level, stratified by causative organisms (classified as *S. aureus*, *Strep. uberis* or 'Other') was not significantly ($p>0.05$) associated with age groups (Figure 3. 5) or herd. However, there was a trend for

clinical mastitis due to *S. aureus* to be more common in cows ≥ 6 years (mean: 0.043, 95% confidence interval: 0.028 – 0.066 cases per 305 cow-days-at-risk) and lowest in 2-year old cows (mean: 0.014, 95% confidence interval: 0.005 – 0.042).

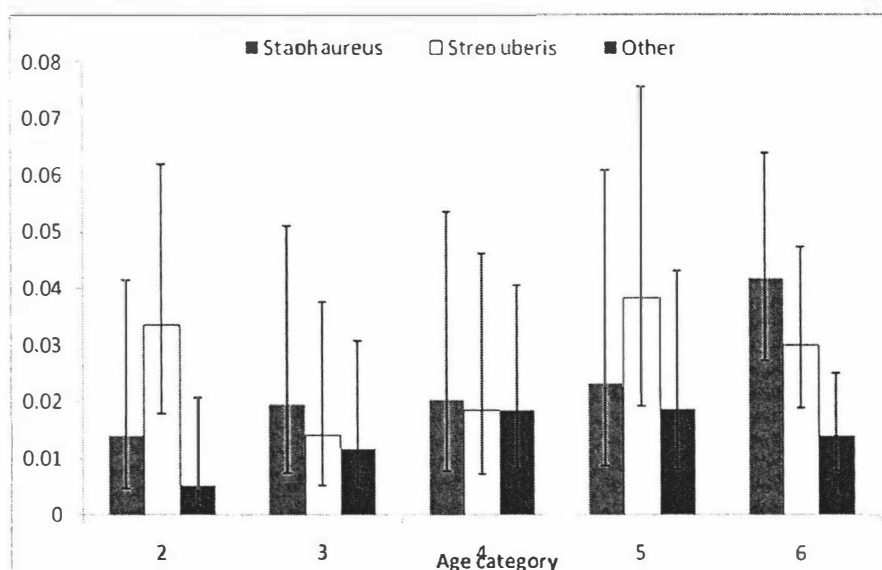


Figure 3.5. Age related isolation of mastitis-causing organisms in 3765 cows and SE from 14 dairy farms in Northland, New Zealand

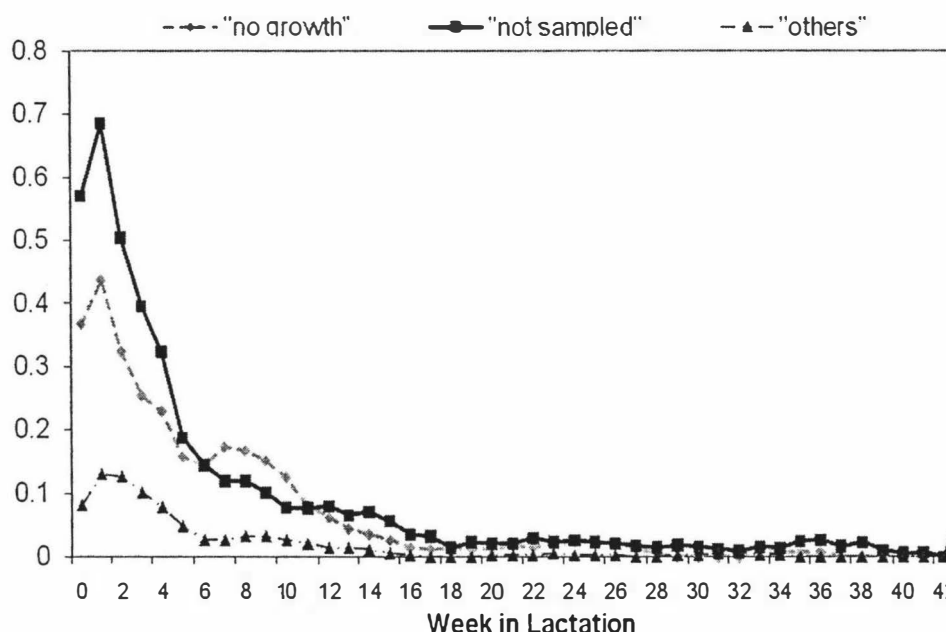


Figure 3.6. Four-weekly average distribution of culture isolates and no sampling from cows with clinical mastitis, excluding cases caused by *Staphylococcus aureus* or *Streptococcus uberis*.

'Week 0' = Day of calving, 'Week 1' = Days 2-7 smoothed over Day 1-7, 'Week 2' = Days 8-14 smoothed over Day 1-14, 'Week 3' = Days 15-21 smoothed over 1-21 Days, 'Week 4' = Days 22-28 smoothed over 2-28, 'Week 5' = Days 29-35 smoothed over 8-35, etc.

By contrast, the clinical mastitis case incidence due to *Strep. uberis* was highest in 2-year old (0.034, 95% confidence interval 0.018-0.064) and in cows ≥ 5 years (mean: 0.039, 95% confidence interval: 0.02-0.079), and lowest in 3- and 4-year olds (mean: 0.014, 95% confidence interval: 0.005-0.038).

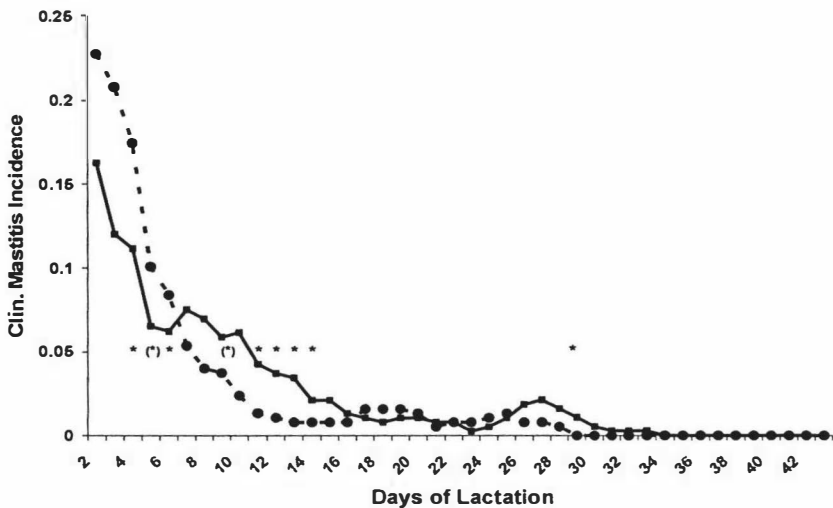


Figure 3.7: Four-weekly moving average of *S. aureus* (solid line) and *Strep. uberis* (dashed line) isolation (numbers/305 cow-days at risk).
 ‘Week 0’ = Day of calving, ‘Week 1’ = Days 2-7 smoothed over Day 1-7, ‘Week 2’ = Days 8-14 smoothed over Day 1-14, ‘Week 3’ = Days 15-21 smoothed over 1-21 Days, ‘Week 4’ = Days 22-28 smoothed over 2-28, ‘Week 5’ = Days 29-35 smoothed over 8-35, etc.; * $p < 0.05$; (*) $p < 0.10$

Four weekly moving averages of the case incidence of clinical mastitis due to individual mastitis-causing organisms are presented in Figures 3.6 and 3.7. The pattern of cases due to *S. aureus* and *Strep. uberis* differed through lactation. The case incidence of *Strep. uberis* was initially high (0.227 cases/305 cow-days-at-risk) and decreased rapidly to a low figure from Week 11 onwards (0.013 cases/305d), whereas the incidence associated with *S. aureus* was initially lower (0.162 cases/305d) than *Strep. uberis*, decreased to Week 5, but thereafter increased again and remained considerably higher than that of *Strep. uberis* during Weeks 7-14 of lactation (Figure 3. 7).

3.4.4 Removal from supply

A total of 66 cows were removed from supply from the 14 farms due to mastitis during or at the end of lactation. This represents 1.8% of all lactating cows and 11.8% of cows with clinical mastitis. The number of cows culled for mastitis during or at the end of lactation was 8, 1.4% of the total number of cows with clinical mastitis.

3.4.5 Predictive value of the first available herd test for prevention of clinical mastitis by treatment

Data were available for 3689 cows. The first available herd test was taken at a mean interval of 49.2 (10th percentile: 15.0; 90th percentile: 85.0) days after calving (DIM). There were 412 cows (11.2%) with ICSCC $\geq$ 250,000 cells/ml at the first herd test.

During the first 60 days after the herd test, 49 (11.9%) of these 412 cows with high ICSCC at first herd test developed clinical mastitis, and 3193 of 3277 cows (97.4%) with a low ICSCC did not develop clinical mastitis. Although high ICSCC was a significant predictor of a subsequent episode of clinical mastitis, the positive predictive value of the first available herd test for predicting clinical mastitis in the subsequent two months was low (mean predictive ability: 11.9%, 95% confidence interval: 9.0-15.5%). The negative predictive value for correctly predicting the non-occurrence of clinical mastitis in this two months period was 97.4% (95% confidence interval: 96.8-97.8%). The practical consequence is that 412 cows would have to be treated to have prevented a maximum of 49 cases, whereas another 84 cases would be expected to occur in the non-treated group (assuming that treatment would be 100% successful).

An episode of clinical mastitis within three weeks before the first available herd test influenced the ICSCC, inasmuch as most such cows had high ICSCC at that test. However, having had clinical mastitis before the first available herd test was of no value for prediction of subsequent clinical mastitis ($p>0.05$). There was no effect of age on the results of the predictive value ($p>0.05$). Both the incidence of clinical mastitis over the 60 day period and the prevalence of high ICSCC at the first herd test differed significantly ($p<0.001$) between herds.

3.4.6 Prevalence of subclinical mastitis

Data were available for 3689 cows. Most of the herds (10 out of 14) were tested 4 times; the others were tested 5 or 6 times during the season. The prevalence of subclinical mastitis, as determined by the presence of ICSCC $\geq$ 250,000 cells/ml, increased significantly from one herd test to another throughout lactation. New IMIs (as defined the appearance of ICSCC $\geq$ 250,000 cells/ml) increased over the course of lactation in parallel with the overall prevalence of subclinical mastitis. These events

were independent of the rising background BTSCC that occurred in the latter stages of lactation. Conversely, the apparent cure rate for IMI, (as defined by a decrease in ICSCC from $\geq 250,000$ cells/ml to below that figure) was consistently low throughout the course of lactation (Figure 3.8). As suggested by a significant ($p < 0.001$) interaction between the rate of increase and age, cows 5 and ≥ 6 years old demonstrated increased new IMI rates to 60 and 75%, respectively, whereas in cows aged 2-4 years the increase was 27-34%.

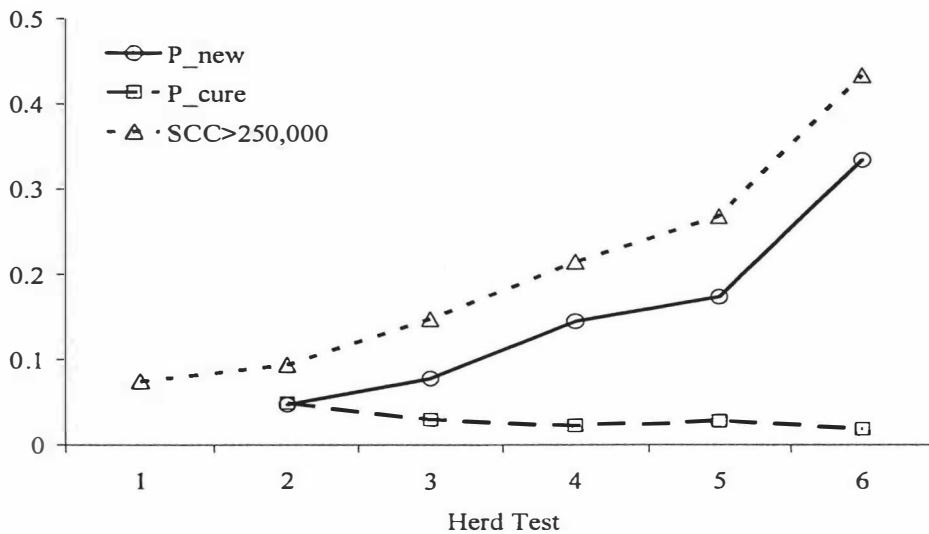


Figure 3.8. Percentage of high individual cow test day somatic cell counts ($\text{SCC} \geq 250,000$ /ml; dotted line Δ) and changes in the percentage of cows with high SCC individual somatic cell counts from low to high (P_{new} ; solid line O) and high to low (P_{cure} ; intermittent line $\square$) approximating rates of new and cure from subclinical mastitis through lactation.

3.5 Discussion

This investigation of the incidence and temporal patterns of clinical mastitis and the distribution of causative organisms is the first such investigation that has been undertaken in the Northland region of New Zealand. It provides additional information on the estimated prevalence of subclinical mastitis and on the value of the first available herd test to predict the development of clinical mastitis over a subsequent period of 60 days. Results from the present study have been compared with those from similar investigations in other regions of the country, in order to evaluate an

opinion held by local veterinarians that the patterns of mastitis in Northland differ from those observed elsewhere in the country.

3.5.1 Incidence of mastitis and mastitis causing organisms

The average number of lactating cows in the study herds (269 cows) and their average yield (272 Kg milk solids/lactating cow/year) were similar to regional (260 cows; 272 Kg milk solids/lactating cow/year) and national averages (Anonymous, 2006b). As in studies of mastitis in other regions of New Zealand (McDougall 2002a; Parker K.I. et al 2005), older cows had a higher incidence of clinical mastitis than did younger animals, with the lowest incidence of clinical mastitis in first calving and 3-year old cows and highest in cows 6 years and older (Figure 3.2).. Likewise, the distribution of affected quarters agreed with previous reports from New Zealand and elsewhere (Adkinson et al 1993; Miltenburg et al 1996; McDougall 1998a). Each cow had an average of 1.8 quarters affected during an episode of clinical mastitis. There was a higher incidence in rear (56.2%) than in front quarters (43.8%). These findings are in agreement with previous non-random distribution of quarter infections from New Zealand and elsewhere (McDougall 2002a; Rainard and Poutrel 1984). The incidence in rear quarters was not as high as previously reported (McDougall 2002a).

The case incidence of clinical mastitis, on a cow level, due to *S. aureus* or *Strep. uberis* differed between age groups, with the highest incidence of *S. aureus* isolations from older (0.043 cases per 305 cow-days-at-risk) and lowest from 2-year old cows (0.014). By contrast, clinical mastitis associated with *Strep. uberis* was similar in first calving (0.034) and older cows (0.039 and 0.030 in 5 and 6 year old cows, respectively). Perhaps this is because older cows have increased susceptibility to mastitis caused by *S. aureus*. The increased susceptibility to *S. aureus* infection in older animals could occur as a result of greater individual susceptibility to develop mastitis due to a longer period of exposure to the organism, or to a decrease in the activity of the defence mechanisms due to mechanical damage and slower repair of the mammary tissues.

The incidence of clinical mastitis, on a cow level, caused by *Strep. uberis* isolations was high during the first 9 weeks and low towards the end of lactation. Likewise, clinical mastitis due to *S. aureus* was more frequent during the first 12 weeks of lactation than

later. However, there was an increase in the proportion of clinical mastitis cases due to *S. aureus* during mid to late lactation while clinical mastitis due to *Strep. uberis* decreased. This cross-over pattern of clinical mastitis due to these two pathogens is consistent with both previous literature (Erskine et al 1988; McDougall 2002a) and the main characteristic of the two organisms as primarily environmental (*Strep. uberis*) and primarily infectious (*S. aureus*) (McDougall 1998a; Khan et al 2003).

However, previous studies of mastitis-causing organisms from clinical mastitis cases in New Zealand have consistently shown that the majority of isolates were *Strep. uberis* and only a small proportion were *S. aureus* (McDougall 2002a; McDougall and Compton 2005; Parker K.I. et al 2005): findings that are at variance with those of the present study. For example, McDougall (2002a) reported that 40% of isolates from herds in the Waikato region of New Zealand were *Strep. uberis* and only 2% were *S. aureus*. By contrast, in the present study there were approximately equal numbers of isolates of the two organisms (*S. aureus*: 23.7%, *Strep. uberis*: 23.3% of cultured samples). The proportion of samples from which no causative organisms was isolated (Table 3.3 and Figure 3. 5) was similar to that reported previously.

Thus, it appears that *Strep. uberis* is less frequent and *S. aureus* more frequent in Northland than elsewhere in New Zealand. This conclusion assumes that the selection of farms and cases selected for isolation were unbiased. In support to this hypothesis comparison of national and regional BTSCC trends demonstrated lower values for Northland early in the season and higher values later in the season that is a typical characteristic of higher prevalence of the contagious organisms (Anonymous, 2006b). It is possible that herd owners who volunteered to participate in this study had a higher proportion of cows with chronic *S. aureus* infection than a typical Northland herd. However, it appears unlikely that the high relative incidence of *S. aureus* was due to biased sample collection and culture. The proportion of cases from which milk samples were collected was unrelated to farm, lactation stage or age. Secondly, milk culturing is more likely to demonstrate *Strep. uberis* than *S. aureus* (Zadoks et al 2001) than *vice versa*. Thirdly, it seems unlikely that the differences between the present and previous studies can be explained with reference to differences in either herd structure or in calving seasons because, even though there is a higher proportion of

non-seasonal calving herds in Northland, none of these herds were included in this study.

Nevertheless, even though the proportion of isolates of *S. aureus* was higher in Northland than elsewhere in New Zealand, *Strep. uberis* remains an important and significant mastitis-causing organism. Its importance has been emphasised in previous studies in New Zealand (Douglas et al 2000), especially with regard to heifer mastitis (McDougall and Compton 2005). There has been much discussion about the epidemiology of mastitis due to this organism in terms of the husbandry practices of pastoral dairying. Traditionally, *Strep. uberis* has been characterised as a purely environmental mastitis-causing organism (McDougall 1998a; Khan et al 2003). However, other reports on the epidemiology of *Strep. uberis* mastitis have demonstrated intramammary persistence (Grommers et al 1985; McDougall et al 2004), intracellular location (Matthews et al, 1994; Tamilselvam et al 2006), induction of subclinical mastitis (Jayarao et al 1999; Khan et al 2003; Zadoks et al 2003) and the persistence of a variety of strains (Phuektes et al 2001; Zadoks et al 2003), all of which are characteristics of a contagious mastitis-causing organism.

The incidence of mastitis is affected by many factors including management practices, culling policies, buying of replacement stock, dry cow therapy, treatment of clinical cases and teat disinfection, as well as the prevalence of mastitis in the herd, management of clinical and high SCC cases, milking procedures, milking machine function and maintenance, age structure and, less probably, the feeding of mastitic milk to replacement stock, (Erskine et al 1988; Myllys et al 1998; Phuektes et al 2001). How these factors explain the differences observed in the incidence of major causative organisms observed in Northland and in previous reports from Waikato (McDougall 1998b, 2002a) is unclear.

3.5.2 Recurrence and removal from supply

The proportion of cows with repeated cases of mastitis was small (10.9% of the cows with mastitis) compared to previous reports from the Northern hemisphere (e.g. Wolfova et al, 2006: 44 % of cows with repeated cases). Interestingly, most of the cows which had recurrent clinical mastitis in this study had more than one repeated episode.

Mastitis is considered a major reason for removal of cows from supply or permanent culling and is second only to reproduction as the largest involuntary culling category (Kossaibati and Esslemont 1997; McDougall 2002b; Seegers et al 2003). In the present study, around 12% of the total number of cows that had an episode of mastitis during the season were removed from supply for the remainder of the season and 1.4% were culled. Approximately 1-3% of cows would be expected to be culled for mastitis (Kossaibati and Esslemont 1997; Xu and Burton 2003).

3.5.3 Prevalence of subclinical mastitis

A threshold of 250,000 ICSCC/ml was used to distinguish likely infected from likely non-infected udders. Based on this approximation of 'infection', a steep increase in both prevalence and incidence of infections between subsequent herd tests through lactation was present. Conversely, assumed cure rates were almost constant and much lower than assumed new infection rates. It was assumed that, in the absence of IMI, the ICSCC should not exceed 250,000 cells/ml, despite the decrease in milk volume towards the end of lactation (Harmon, 1994; Schukken et al 2003). There is no international standard for definition of IMI, based on ICSCC threshold. Recently, most authors use a cut-off of 200,000 cells/ml. It is well known that using this cut-off misclassification is possible, particularly un-infected cows may be classified as infected. Andrews et al (1983), in an Australian based study, reported most satisfactory results with using threshold of 250,000 cells/ml cut-off. Additionally, some authors from elsewhere (for example Rainard et al 1990) have reported that average geometric mean of ICSCC in bacteriologically un-infected quarters was more than 200,000 cells/ml, particularly when major mastitis-causing organisms are most prevalent (for example Schepers et al 1997). Therefore, to be able to distinguish between infected and un-infected cows and reduce the diagnostic error, according to Schukken et al (2003), cut-off of approximately 200,000 to 250,000 cells/ml is optimal. Thus, in the present study a conservative threshold of 250,000 ICSCC/ml was elected. Based on this assumption, it was demonstrated that most assumed new IMI that occurred after the first herd test remained subclinical because the incidence of clinical mastitis decreased whilst that of IMI increased. This finding is consistent with the pattern of *S. aureus* in causing more subclinical than clinical mastitis (Jayarao et al 1999; Khan et al 2003;

Zadoks et al 2003). Even though ICSCC increase with the age and stage of lactation, the high prevalence of ICSS >250,000 cells/mL in the second half of lactation in the present study suggests that many infected cows remained in the milking herd and were a source of infection for other non-infected, susceptible cows (Harmon, 1994).

3.5.4 Predictive value of the first available herd test for the risk of clinical mastitis

Farmers and veterinarians might wish to use ICSCC of the first available herd test for the prevention of clinical mastitis by treating sub-clinical mastitis. Reports from numerous studies have demonstrated that treatment during lactation, based solely on ICSCC is impractical (for example Timms and Schultz 1984, Rose et al 2003 etc). The present results suggest that for every 12 cows treated because of high ICSCC at herd test, only one would have developed clinical mastitis in the following 60 days. It would be hard to demonstrate economic benefit of such a measure. However, expected benefits might include the reduction of transmission of contagious udder pathogens such as *S. aureus*, in addition to possibly preventing up to one third (49 out of 133 cases) of clinical mastitis in the next 60 days in milk (including costs of discarded milk, extra labour, and drugs). A substantial economic impact might be achieved through the reduction of transmission rates. The high observed rates of assumed new infection combined with high prevalence of subclinical mastitis towards the end of lactation suggest that the selection and removal of cows infected with contagious pathogens has great potential in reducing loss from subclinical infection.

3.6 Conclusions

This study described the incidence of clinical and subclinical mastitis, its association with lactation stage, age and herd, and the relatively high importance of *Strep. uberis* and *S. aureus* in 14 seasonally calving dairy farms in Northland, New Zealand. It is strongly hypothesised that these two pathogens cause significant economic losses to the dairy industry in this region. Further study should evaluate risk factors for clinical mastitis, measure the prevalence of subclinical mastitis and associated pathogens more accurately, and use these and future findings to estimate the economic effects of mastitis and its control in this region.

3.7 Acknowledgments

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Chapter four

Effects of Clinical Mastitis caused by *Streptococcus uberis* in the Early Dry Period on Production in the Subsequent Lactation

4.a Introduction to the study used to provide data for analysis of production effects

The data analysed in this study were initially generated by Cecilia Fernandez during the animal phase of her investigations on “The effect of external teat sealants on mastitis incidence during the dry period under experimental challenge with *Streptococcus uberis*.” (Master of Applied Science thesis, submitted, 2007). The data used in the present study were extracted from the regular herd tests in the following lactation from the farm that was used in her study.

The work of Fernandez was undertaken to assess the efficacy of two different substances used at drying off as external teat sealants in prevention of IMI following experimental challenge of the teats with *Streptococcus uberis* in the early dry period. The products used for sealing teats were based on a commercially available teat sealant (tetrahydrofuran: DryFlex) and on a wound sealant for use in humans (2-octylcyanoacrylate: Band-Aid).

Her study was conducted based on the following:

1. There are two main periods of high susceptibility to mastitis during the dry period, namely the periods of active involution and colostrogenesis.
2. From the 1960s the use of dry cow therapy antimicrobials has been practiced in order to eliminate existing and prevent new IMI during the dry period.
3. The extensive use of antimicrobials to treat and control mastitis has consequences for human health, through an increased risk of introducing residues into the food chain and the emergence of antimicrobial-resistant organisms.
4. Non-antibiotic means for the treatment and prevention of mastitis have the potential to create widespread benefits from reducing the need for antibiotics.

One hundred and seventy five cows with four functional quarters, low ICSCC and no evidence of clinical mastitis were enrolled in the study, a few days prior to dry-off. Single-quarter milk samples were taken for microbiological culture, four and one days before dry-off, and again on two occasions within four days after calving. At drying off,

enrolled cows were split into two groups, with 88 cows receiving modified “DryFlex”, external teat sealant available on the USA market, and 87 receiving “Band Aid”, liquid bandage, in two contra-lateral quarters (i.e. LF and RH), while the other two quarters were left as untreated controls. All cows were challenged on two occasions with a broth culture of *Strep uberis*, by dipping all quarters, two and four days post dry-off and treatment. Assessment for clinical mastitis was subsequently performed daily on all quarters.

The results from this study demonstrated that the highest frequency of mastitis occurred between Days 6 and 11 after dry-off and treatment. After adjusting for the effects of treatment and individual cow-level, the daily hazard of clinical mastitis in cows identified as infected at dry-off was 1.64 (95% CI 1.10 to 2.44) times that of uninfected cows. Modified DryFlex provided protection against the bacterial challenge for 50% of the quarters for four days. At the group level, there were 35 CM events out of 176 treated quarters with modified DryFlex, compared to 83 events in the untreated quarters (176). For the Band Aid treated group, 67 CM events occurred in 174 treated quarters, compared to 64 events in the untreated quarters (174).

Based on the results from this study it was concluded that the application of DryFlex at drying-off was beneficial in reducing mastitis caused by *Strep. uberis* challenge. Contrarily, the use of Band aid at dry-off had no benefit in reducing the incidence of mastitis following two experimental *Strep. uberis* exposures.

All of the cows that developed clinical mastitis were treated (with antimicrobials intramammarily or systemically when all four quarters affected) promptly, resulting in a clinical cure within (3-4) days.

This study generated a substantial group of cows that had had a brief period of clinical mastitis in their early dry period. As most of these cows (165) remained in the herd for a further lactation, the opportunity was present to evaluate various parameters of their lactational performance. This was undertaken, as described in the remainder of this chapter, by comparing the performance of cows that developed mastitis with those that did not.

Effects of Experimentally Induced and Treated *Streptococcus uberis* Mastitis Early in the Dry Period on Production in the Subsequent Lactation

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4. Effects of Experimentally Induced and Treated *Streptococcus uberis* Mastitis Early in the Dry Period on Production in the Subsequent Lactation

4.1 Abstract

The effect of experimentally induced clinical mastitis in the early dry period (EDPCM) upon the milk production in the subsequent lactation was examined. Animals with low somatic cell counts at herd test (n=165) were experimentally exposed to *Streptococcus uberis* in their early dry period in an efficacy study of two external teat sealants. Animals that developed EDPCM after challenge (n=127) were treated with an antibiotic after observation of clinical mastitis. Total lactation yields of milk, fat, protein and milk solids were analysed with respect to treatment group (fixed effect) and the covariables: calving week (linear), parity (linear and quadratic) and proportion of Holstein-Friesian genes (linear). For animals that suffered EDPCM and those that did not, there was no difference in production (milk yield; 5126 vs. 5010 litres, fat yield; 267 vs. 264 kg, protein yield; 182 vs. 179 kg). It was concluded that promptly treated EDPCM due to *Strep. uberis* did not affect production in the subsequent lactation.

4.2 Introduction

Bovine mastitis is one of the most economically important diseases affecting the dairy cattle industry internationally (Hortet and Seegers 1998; Seegers et al 2003). Mastitis was estimated to have cost the New Zealand dairy industry around NZ\$180 million/year in 2005/06 (Anonymous 2006). Many factors have been associated with the cost of mastitis, including stage of lactation, pregnancy status, prior yield, mastitis causing organism, severity, diagnosis (early or late after occurrence), treatment and recurrence of mastitis. The main factor (70-80% of all losses) is reduced milk yield. Moreover, as the pathogenesis of mastitis results in irreplaceable loss of secretory tissue, this loss of milk yield can be permanent (Benites et al 2002).

Short-term depression in milk yield occurs when cows develop mastitis during lactation, with more severe losses occurring if it occurs early in lactation and there is a failure of microbial cure or when the effects of the mastitis carry over into subsequent

lactations (Houben et al 1993; Hortet and Seegers 1998; Rajala-Schultz et al 1999). Adverse effects on milk composition (Seegers et al 2003) increase economic losses. Production effects of clinical mastitis result from both short- and long-term decreases in milk production, particularly associated with chronic mastitis (Smith et al 1968; Fetrow et al 1991; Rajala-Schultz et al 1999).

Less is known about the effects of clinical mastitis during the dry period, on milk production in subsequent lactations. The dry period is an important part of the lactational cycle during which the mammary gland prepares for the next lactation. Clinical mastitis during this period may slow the process of mammary tissue remodelling, thereby adversely affecting milk yield in the subsequent lactation.

Streptococcus uberis is the most significant cause of clinical bovine mastitis in New Zealand and Australia (Pankey et al 1996; Douglas et al 2000; Phuektes et al 2001; McDougall 2002) where the dairy industry is predominantly pasture based. Information is lacking about the effects of clinical mastitis in the early dry period (EDPCM) on milk production in the subsequent lactation. A previously conducted study with different objectives provided an opportunity to further understand any impact of treated *Strep. uberis* mastitis on production parameters in the following lactation. In this *post hoc* analysis, the effects of experimentally induced and promptly treated *Strep. uberis* clinical mastitis early in the dry period on milk production in the subsequent lactation have been analysed.

4.3 Materials and methods

4.3.1 Animals and experimental design

A total of 175 cows (Holstein-Friesian (HF) and HF-Jersey crossbreds) were selected from the Massey University #4 herd, located in the Manawatu Region of New Zealand to participate in a prospective, randomised, controlled field trial of the efficacy of two external teat sealants. Ten cows were excluded from analysis in this study due to missing production data, leaving 85 cows (modified DryFlex) and 80 (investigational external teat sealant). The herd was managed at pasture, with supplementary hay/silage as required. Selection criteria for cows in this trial were:

- (i) <200,000 SCC/mL at the April 2005 herd test,

- (ii) four functional quarters and,
- (iii) no clinical signs of mastitis or teat abnormalities at enrolment.

All animal manipulations were approved by Massey University Animal Ethics Committee (MUAEC 04/165).

4.3.2 Challenge protocol

The *Strep. uberis* strain used for the challenge was initially isolated by Douglas et al (2000) in the Horowhenua district, Wellington region, phenotypically identified as 99.9% probable to be *Strep. uberis*, by means of biochemical tests, and kept frozen at -80°C at the Institute of Veterinary Animal and Biomedical Sciences Microbiology Laboratory. All cows were exposed to the challenge broth on two occasions, two and four days after dry off, by dipping of each teat, entirely, for 1-2 seconds in a suspension of 1.15×10^8 cfu/mL of the challenge strain.

4.3.3 Milk sampling

Quarter milk samples were aseptically collected, following National Mastitis Council (NMC) recommendations (and stored on ice) before morning milking on 4 occasions:

- (i) 4 days before drying off,
- (ii) one day before drying off,
- (iii) the day of calving and,
- (iv) 3-4 days postpartum.

Samples were subjected to routine microbiological culture and examination on the day of collection following NMC recommendations.

4.3.4 Clinical assessment and treatment

All quarters were examined daily by an experienced dairy technician for the presence of mastitis from the time of the first exposure until 29 days later. Individual quarters were observed and palpated for the clinical signs associated with mastitis, i.e. heat, swelling, redness, painful quarter/s and if required, by an examination of the secretion. Each quarter was subjectively judged as mastitic or non-mastitic according to the above criteria. Mastitic quarters were sampled for microbiological culture before

treatment was initiated. After sampling each affected quarter was treated as for lactating cow clinical mastitis with penicillin based antibiotics as prescribed on the label.

4.3.5 Statistical analysis

Total yields of milk, fat and protein were estimated from herd-test data during the production season (2005-06), which is the season after the *Strep. uberis* challenge. Somatic cell score (SCS) was calculated as natural log (somatic cell count +1) for each herd-test record.

Statistical analyses were performed using SAS (Statistical Analysis System, version 9.1; SAS Institute Inc., Cary, NC, USA). Frequencies of EDPCM between treatment groups were compared using Fisher's exact test.

Total lactation yields of milk, fat, protein and milk solids and average SCS were analysed with the MIXED procedure using a linear model that considered the fixed effects of treatment group (modified DryFlex and investigational external teat sealant), EDPCM occurrence (cows that suffered EDPCM and those that did not), their interaction and the covariables calving week (linear), parity (linear and quadratic) and proportion of Holstein-Friesian genes (linear). Least squares and their standard errors were used for multiple comparisons.

4.4 Results

4.4.1 Early dry period mastitis occurrence

After *Strep. uberis* exposure, 127 of 165 cows developed early dry period clinical mastitis (76.97%). Sixty four of 85 (75.29%) that developed EDPCM were from modified DryFlex and 63 out of 80 (78.75%) from the investigational external teat sealant group. The difference between groups was not significant ($p=0.13$).

Milk production parameters

Milk production data and SCS are given in Table 4. 1. There was no difference in milk yield, fat or protein production between cows that suffered EDPCM and those that did

not. There was a statistically significant difference ($p<0.05$) in the SCS observed in cows that suffered EDPCM and those that did not.

There was no difference in the milk production parameters between treatment groups.

Table 4.1.: Least squares means and standard errors of milk production and somatic cell score (SCS) of cows affected and not affected with clinical mastitis in the early dry period

Group	Days in milk	Milk yield (L)	Milk solids yield		Protein yield	
			(kg)	Fat yield (kg)	(kg)	SCS
Affected (EDPCM) ¹	277.6 ± 1.4	5126.8 ± 73	449.5 ± 6.5	267.1 ± 4.2	182.4 ± 2.5	4.45 ± 0.07
Unaffected	275.4 ± 2.7	5010.2 ± 135.9	443 ± 12	264.3 ± 7.8	178.6 ± 4.7	4.17 ± 0.12

¹ early dry period clinical mastitis

4.5 Discussion

The bovine mammary gland is particularly susceptible to new infections early and late in the dry period, due to involution and colostrogenesis respectively (Bradley and Green 2004). The pathogenesis of mastitis includes, in some cases, damage to secretory tissue and its replacement with fibrous tissue, leading to a permanent decrease in milk yield from the affected quarter (Benites et al 2002). It is probable that part of the decrease in milk production seen when clinical mastitis occurs during lactation is due to an increased demand for energy by the immune system, a decreased appetite associated with the inflammatory process and lowered feed intake due to pain and decreased mobility (Petrovski et al 2006). These factors may also influence the normal involution of the bovine mammary gland after drying off. If the normal involution is affected there is a possibility of decreased milk production in the subsequent lactation.

Previous studies reported less than 20% milk yield losses in subsequent lactations for cows affected with clinical mastitis (Fetrow et al 1991; Houben et al 1993; Hortet and Seegers 1998). In the present study, no significant effect of induced and treated *Strep. uberis* mastitis soon after drying off was found in cows upon subsequent lactation yields.

Reasons that the results of the present study differ from earlier investigations are not readily apparent. Possibilities include the organisms involved, time of infection, nature and pathogenesis of the intramammary infection (IMI) and the duration of the clinical mastitis episode. All previous reports were based on the natural occurrence of clinical mastitis during lactation, while in the present study, clinical mastitis was induced by a challenge occurring in the early dry period. In naturally occurring infections the numbers of causative organisms are generally lower than in challenge conditions. The development of naturally acquired clinical mastitis is generally slower and clinical form is not always an outcome. It is possible that a longer duration in such circumstances allows damage to extended areas of the bovine mammary gland. In this experiment, the high numbers of causative organisms in challenge conditions may have easily overcome the defence mechanisms of the bovine udder, causing an acute clinical mastitis episode. All EDPCM episodes in the present study were promptly diagnosed and treated. Early detection and treatment of clinical mastitis generally result in higher probability of cure than treatment of chronic infections (Milner et al 1997; du Preez 2000) as was demonstrated for *Staphylococcus aureus* (Sol et al 1997). This experiment suggests that promptly treated *Strep. uberis* clinical mastitis episodes in early lactation do not affect milk production parameters in the following lactation

In field conditions, the infections occurring during the early dry period are more likely to be caused by mixed microbial flora (Bradley and Green 2004). In the present study, clinical mastitis cases were caused by *Strep. uberis* only. Some mastitis-causing organisms have been associated with a more profound impact on milk yields than others, for example: Grohn et al (2004) reported that *S. aureus*, *E. coli*, *Klebsiella* spp., and "no pathogen isolated" among primipara, and *Streptococcus* spp., *S. aureus*, *A. pyogenes*, *E. coli*, and *Klebsiella* spp. in older cows, caused the greatest losses. Hence, while the present study shows that pure *Strep. uberis* infections in the early dry period that were identified and treated do not appear to result in permanent changes to lactation yield, it may not reflect the field situation in which mixed infections may be present and where such rapid identification and treatment is unlikely. As expected, the differences between the SCS between cows that suffered EDPCM and those that did not were significant, due to the increased influx of white blood cells post IMI in each of the affected quarters (Rajala-Schultz et al 1999; Benites et al 2002).

4.6 Conclusions

Results of this study indicate that EDPCM due to *Strep. uberis*, when promptly treated, did not affect production in the subsequent lactation. This is probably because the short duration of the new IMIs did not allow a permanent damage to the mammary secretory tissue to occur. As a majority of new IMIs occur in the first week after calving, it may prove beneficial for farmers to pay more attention to checking for clinical mastitis during the early dry period.

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Chapter five

General discussion

5.1 General discussion

Bovine mastitis is regarded as the most costly production disease to the dairy industry worldwide. The economics of mastitis possibly should be addressed at the farm or herd level, since it depends on local, regional, epidemiological, managerial and prevailing economic conditions. Estimating the total costs associated with mastitis is notoriously difficult. When considering the cost of any disease, it is necessary to keep in mind that every disease has direct and indirect costs. Direct costs and expenses are usually the ones most readily comprehended by the farmer. Indirect losses due to mastitis are not perceived by the farmer in many cases and it is even more difficult to quantify the losses associated with SCM since these are not readily visible. This lack of visibility may explain why the implementation of mastitis control measures is difficult. Continuous education on this matter is necessary. Some of the costs and expenditures due to mastitis are easy to calculate and they should be included in research projects dealing with the modelling of the economics of the disease. However, some of the costs are not readily quantified, such as cases of human disease and farmers' stress.

The primary objectives of this thesis were (a) to describe factors associated with the costs of bovine mastitis, (b) to investigate one of the prerequisites for estimating losses due to mastitis - the incidence of clinical mastitis - and (c) to investigate the effects of mastitis in the early dry period on milk production in the subsequent lactation when it was promptly treated.

While considerable resources have been invested in quantifying the economic effect of bovine mastitis, little attempt has been made to understand the factors that contribute to it, particularly with regard to the incidence of clinical mastitis in New Zealand or to the effects of mastitis in the early dry period upon subsequent milk production. The lack of knowledge in these two instances was confirmed in the literature review of factors associated with the costs of bovine mastitis (Petrovski et al 2006).

Previous studies on the incidence of CM in New Zealand have been undertaken in the Waikato region, but no such data exist for the Northland region. Based on the hypothesis that the particular circumstances of the dairy industry in Northland would

give rise to different patterns of mastitis to those observed elsewhere in New Zealand, a study of the incidence of bovine mastitis and a description of the aetiology and distribution in lactation of organisms causing mastitis in Northland was conducted. This study confirmed the importance of stage of lactation, age and herd on CM incidence and the distribution of the occurrence of MCOs. A striking difference in the proportion and the incidence in stage of lactation of the main MCOs was demonstrated when compared to the previous reports from elsewhere in New Zealand. Additionally, a difference in the incidence of CM during lactation was registered, being higher than reported in the previous Waikato-based studies. It is unlikely that this difference is due to methodology for, in the present study, as to previous work, the incidence of CM was determined by farm personnel recording the identities of affected cows. The average incidence of CM was 0.19 cases per 305 cow-days at-risk. The incidence in rear quarters was 1.3 times higher than in front quarters. The incidence of CM and numbers of affected quarters was significantly influenced by the stage of lactation (higher in early lactation), age (higher in older cows) and herd. This study demonstrated a higher incidence of contagious staphylococcal IMIs in CM episodes from Northland, when compared with findings from other regions of New Zealand.

Subclinical mastitis prevalence was determined as the presence of high ICSCC ($\geq 250,000$ cells/mL) during routine herd testing. It was assumed that, in the absence of IMI, the ICSCC should not exceed 250,000 cells/mL, despite the decrease in milk volume towards the end of lactation. The estimated prevalence of subclinical mastitis steadily increased through the season (i.e. from calving to dry-off). Conversely, self-cure rates were almost constant and much lower than new infection rates. The incidence of CM after the first herd test decreased whilst that of IMI increased, demonstrating that most new IMI remained subclinical. This finding is consistent with the pattern of *S. aureus* in causing more subclinical than clinical mastitis (Jayarao et al, 1999; Khan et al, 2003; Zadoks et al, 2003). The high prevalence of high ICSCC in the second half of lactation in the present study suggests that many infected cows remained in the milking herd and probably were a source of new intramammary infections for other non-infected, susceptible cows.

Many farmers and veterinarians question the usefulness of herd test data to indicate treatments to prevent clinical mastitis in the period between two herd tests. Results from the present study do not support the use of treatment based on the identification of high ICSCC in herd tests as a means of preventing subsequent clinical mastitis. However, the possibility of a decreased transmission rate and thus, an increase in the predictive value of treatment based on herd test results in herds with contagious mastitis problems (i.e. *S. aureus*, and *Strep. agalactiae*), should not be ignored, provided that treatment is efficacious and that the first herd test results are available in the first third of the season (for example, Barlow et al, 2005; Schukken et al, 2007). In the case of environmental causative organisms, the economic benefit of the predictive value of the first available herd test for prevention of CM by treatment is negligible.

The most visible aspect of the losses and costs associated with bovine mastitis are those due to CM. The results of this study indicate that CM is common on the sampled dairy farms; therefore, it seems likely that CM is a reason for significant economic losses to the dairy industry in this region. Economic effects of CM will be influenced by treatment cost, discarded milk, decreased milk production, temporary or permanent removal of cows from supply and other factors listed in the review of the costs of mastitis included in this thesis (Chapter 2).

Mastitis is a major reason for temporary removal of the cows (e.g. by premature drying off or transfer to being a nurse cow) from supply or permanent culling. Financial losses at the farm level due to culling for mastitis can be attributed, at least in part, to the loss of future income and genetic potential (Holdaway 1990; Hillerton et al 1992; Cullor 1993; Allore and Erb 1998). Such economic effects of mastitis are exacerbated by the loss of milk yield from cows that are temporarily removed from supply for the remainder of a lactation. New Zealand farm management practices are often influenced by the milk quality requirements, with the result that farm managers often decide to remove cows with high ICSCC from supply for the remainder of the season as part of the milk quality management. In the present study, both the number of cows temporarily removed from supply (i.e. for the remainder of the season) and the number culled were calculated. This approach has not been taken previously, as earlier

studies have only taken into account permanent removal from supply (culling). In the current study approximately 12% of cows with one or more episodes of mastitis during the season were removed from supply, whilst the percentage of cows permanently removed from the herd (1.2%) was similar to previous reports (1 to 3%: Kossaibati and Esslemont 1997; Xu and Burton 2003). Therefore, based on the higher incidence of CM in Northland and the differences of removal from supply for the remainder of lactation vs. culling, it is not unreasonable to suppose that estimated costs of mastitis in New Zealand of \$180 million per year (Anonymous 2006) are likely to be under-estimated.

The main factor causing economic losses from both clinical and sub-clinical mastitis is a more or less persistent decrease in milk yield. *Streptococcus uberis* is currently considered to be the most prevalent cause of bovine mastitis in the pasture-based dairy industry of New Zealand (Pankey et al 1996; Douglas et al 2000; McDougall 2002a), so an estimation of the impact of mastitis caused by this organism upon milk production is of national interest. Additionally, IMIs caused by *Strep. uberis* are of growing concern to dairy producers throughout the world due to the relative ineffectiveness of current mastitis control procedures to prevent and control mastitis caused by this organism. Long-term effects of CM on milk yields have previously been identified as an area that needs more attention from research (Petrovski et al 2006).

Similarly, few data exist on the effects of CM on milk production, when it occurs during the dry period. However, results from the second study indicate that CM due to *Strep. uberis* in the early dry period did not affect production in the subsequent lactation, if promptly treated, probably because the short duration of the new IMIs did not cause permanent damage to the mammary secretory tissue. As the majority of new IMIs occur in the first week after calving it was concluded that farmers should be encouraged to pay close attention to the occurrence of clinical mastitis in the early dry period.

A number of avenues for future research have been identified as a result of the work contained in this thesis. The ability to determine the real cost of mastitis to the dairy industry depends on a detailed and up-to-date description of the prevalence and incidence of bovine mastitis and the geographic distribution of causative organisms at a national level. As reported in this thesis, there are differences in the CM incidence

and the frequencies causative organisms isolation in Northland, from previous studies undertaken in the Waikato. It thus may be considered inappropriate to take reports from one region as being indicative of nationwide situation. If this is the case, the target population for future studies should be nationwide dairy farms and not be limited to farms from one region. Restricting a study to dairy cattle from one region, in an attempt to reduce extraneous sources of variability has its merits in research settings. However, the development of a national approach would allow a variety of benefits to be achieved in the same observational framework. If such future studies also survey the epidemiological factors that regulate the incidence and affect the costs of mastitis, significant benefit would accrue to the dairy industry of New Zealand.

A detailed, up-to-date, description of the prevalence and incidence of bovine mastitis and spatial distribution of causative organisms and their genetic diversity in New Zealand would also provide useful background information for further development of the SAMM Plan mastitis control programme.

Currently, there is no standardised methodology in the collection of data on the incidence of bovine mastitis, or on its recording and reporting at national level. It is strongly recommended that consideration be given to the development of a centralised system for recording bovine mastitis, since it would be of considerable benefit to the dairy industry in being able to document CM incidence in a standardised format.

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