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TO STUDY THE ASSOCIATION BETWEEN SCC AND
BACTERIA NUMBERS, AND MILK COMPOSITION IN FARM
BULK MILK SUPPLIED TO TUI MILK PRODUCTS COMPANY
FOR 1992/93 SEASON.

**A thesis presented in partial fulfilment of the requirements
for the degree of Master of Agricultural Science in Animal
Science at Massey University, Palmerston North
New Zealand.**

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ABSTRACT

The association between bulk milk SCC and bacteria numbers and milk composition were studied using data containing test records of 1200 farms which supplied bulk milk to TUI Milk Company Limited, for 1992/93 season. Three data sets were created, (1) data set A (N = 4623) with all measurements recorded for each herd for the same milk sample; (2) data B (N = 30 120) with all measurements of BMSCC and milk composition recorded for each herd within a 10 day period but not necessarily on the same sample of bulk milk; and (3) data set C (N = 33 800) with all measurements of bulk milk bacteria numbers and milk composition recorded for each herd within a 10 day period but not necessarily on the same sample of bulk milk. Correlation was used to determine the association between bulk milk SCC and bacteria numbers and milk composition. Multiple regression analysis was also carried to determine the association between bacteria numbers (dependent variable) and SCC and milk composition for early lactation and whole lactation. The results showed the overall average of the mean BMSCC of 280 000 cells/ml of all the farms studied. Approximately 85 % of the farms supplied bulk milk with SCC <250 000 cells/ml, while 1 % of the farms supplied bulk milk with SCC >500 000 cells/ml. Both bulk milk SCC and bacteria numbers were higher in early and late parts of lactation. Highly significant but low positive correlations occurred between the mean bulk milk SCC and bacteria number in early ($r = 0.24$; $r^2 = 0.06$; $P < 0.001$) and whole lactation ($r = 0.15$; $r^2 = 0.02$; $P < 0.001$). Thus 2 to 6 % of the variation observed in bacteria count was accounted for by variation in bulk milk SCC. The mean fat %, protein % and total solids % increased from mid-lactation to the end of lactation. In contrast, the mean lactose % showed a decrease as the lactation progressed. On the average for the whole lactation, low positive correlations occurred between the mean bulk milk SCC and fat % ($r = 0.18$), protein % ($r = 0.26$) and total solids % ($r = 0.15$). However, a moderate but highly significant negative correlation occurred between bulk milk SCC and lactose % ($r = -0.43$; $P < 0.001$).

In conclusion the overall low average BMSCC suggests that good quality bulk milk was supplied to the company, which also meets the EC standards. Significant low correlation between BMSCC and bacteria numbers suggests that mastitis bacteria were only a small but significant contributor to the high bacteria count in the bulk milk particularly in early lactation, with dirty milking machines or poor cooling being the most likely major contributor. Finally, lactose % was more sensitive to mastitis effective than fat %, protein % and total solids % in the bulk milk.

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CHAPTER ONE

INTRODUCTION

Universally there is an increasing demand for high quality milk, free from cellular contamination or with low cell count. This is attributed to (a) public health concern that a sample of milk contaminated with somatic cells carries with it images of diseased udders and (b) in part due to lower product yields and inflated processing cost to the manufacturer. Despite the fact that it is not likely that mastitis pathogens will have survived pasteurisation, there is a public feeling that milk should be clean at the source -farm level. In recent times, the demands of buyers have increased, which include specified somatic cell counts. For example, the European Economic Community (EEC) somatic cell counts standards for raw cows milk for the manufacture of milk based products have been set at < 500,000 cells/ml since the beginning of 1994 (Frank, 1994). Thus all countries wishing to export dairy milk products to the community will have to show that their standards measure up to those required within the EC. This is a challenge to which the New Zealand dairy industry attaches utmost importance since about 85 % of its total milk products is exported (NZDB, 1994). It is therefore imperative that New Zealand dairy farmers supply the dairy factories with raw milk of high quality with low somatic cell counts for the manufacture of finest milk products to meet the high standard requirements of the export market (SAMM Plan, 1995).

Primarily, milk Somatic Cell Counts (SCC) are being used in the diagnosis of subclinical forms of bovine mastitis, which is part of a greater disease complex -mastitis as a whole (IDF, 1975; Fetrow, 1984; Bramley, 1991). An infection of mastitis in the bovine udder is associated with a commensurate increase in somatic cells to fight the causative pathogens (Schalm, 1977; Bramley, 1992). Bovine mastitis and somatic cell counts (SCC) have therefore been associated with decreased milk yield (Janzen, 1970; Hoare, 1982; Deluyker et al, 1993) and changes in milk composition (Wheelock et al., 1966; Janzen, 1970; Schalm

et al., 1971; Kitchen et al., 1981; Munro et al., 1984), which in turn constitute an economic loss to the dairy farmer (Boothe, 1989; Harmon, 1993; Holdaway, 1993). Mastitis costs about \$14,000 annually due to on farm increased costs and decreased milk sold in the average New Zealand herd (Holdaway, 1993). In addition problems with manufacturing properties and product defects have been observed for most dairy products made from mastitic milk (Mitchell et al., 1985). Regular monitoring of the number of somatic cells in farm bulk milk at both farm and factory levels has therefore become a regular practice in the New Zealand dairy industry. Bulk milk somatic cell count (BMSCC) is increasingly becoming a key parameter in determining raw milk quality (Frank, 1994). It is now being used by some manufacturing companies as one of the measurement factors for milk payments to farmers in New Zealand. Furthermore BMSCC has permitted the adoption of regulatory programmes such as the 5-Point Plan worldwide (Bramley, 1992) and the SAMM Plan in New Zealand (The SAMM Plan, 1995) to identify mastitis status in a herd and its control. In addition, it is possible for mastitis causing organisms to increase the bacterial count of the farm bulk milk to such an extent that the count exceeds the dairy company's bacterial standards for finest milk. This may attract a penalty in monetary terms by the dairy companies.

The objectives of this work were to:

- study the association between SCC and bacteria quality and milk composition - fat, protein and lactose concentrations in farm bulk milk supplied to the Tui Milk Products Company for 1992/93 season; and
- assess the importance of mastitis in the herd as a cause of failure by the herd's milk to meet the bacterial quality standards.

CHAPTER TWO

REVIEW OF LITERATURE

INTRODUCTION

Mastitis is recognised as the most costly disease of the dairy cattle. Its far-reaching impact on the dairy industry has generated extensive efforts to understand and control the disease (Fetrow, 1984).

2.0 DEFINITION OF MASTITIS

Mastitis is an inflammation of the mammary gland (Tolle, 1975; Guidry, 1985; Bramley, 1992). The term mastitis is derived from the Greek words, **MASTOS**, meaning *breast*, and **ITIS**, meaning *inflammation* (Guidry, 1985; Philpot, 1977).

2.1 FORMS OF MASTITIS

There are 2 forms of mastitis namely (a) clinical and (b) subclinical mastitis (Tolle, 1975; Guidry, 1985; Bramley, 1992), although these may be caused by the same pathogen.

2.1.1 Clinical mastitis

Clinical mastitis is characterised by detectable abnormalities in the milk such as flakes, blood and clots and the affected udder may be hot, hard, swollen and red (Tolle, 1975; Guidry, 1985; Bramley, 1992). Where the signs are accompanied by the systemic signs such as fever, depression, shivering, loss of appetite and loss in weight, the mastitic condition is said to be *peracute*; with lesser systemic signs (fever and depressions), it is said to be *acute*; and when the obvious signs of inflammation but no systemic signs are visible it is

said to be **subacute** (Guidry, 1985). A condition in which the cow's milk periodically shows abnormalities and then returns to normal is referred to as **chronic** clinical mastitis. In a small proportion of extreme cases recumbency and death may occur (Bramley, 1992).

2.1.2 Subclinical mastitis

It refers to the presence of infectious organisms within the udder, but no visible abnormalities are present either in the cow, udder or milk (Schmidt, 1971; Fetrow, 1984). The subclinical form of mastitis is important because it constitutes a reservoir of organisms that often prompts infection of other animals within the herd and it is 15 to 40 times more prevalent than the clinical form (Philpot, 1977; Fetrow, 1984). It also accounts for more than 90 % of all cases of mastitis (Fetrow, 1984). Subclinical mastitis is more subtle and can be detected only by special tests on milk which measure particular changes in milk such as:

- increases in the concentrations of sodium and chloride ions and serum albumin in milk;
- an increase in the electrical conductivity of milk;
- a decrease in the concentration of casein, lactose and potassium ions in milk;
- an increase in the number of somatic cell counts (SCC) in milk.

However, determination of sample milk SCC is now widely used to monitor the occurrence of subclinical mastitis in herds or individual cows (Dohoo and Meek, 1982; Leslie et al, 1984; Philipson et al, 1993).

2.2 CAUSES OF MASTITIS

Mastitis is not a single disease but a collection of diseases with differing causes, usually microbial infection (Bramley, 1991). More than 99 % of all bovine udder infections are caused by bacteria (Neave, 1975; Tolle, 1975; Fetrow, 1984), with the remainder caused by algae and yeast (Guidry, 1986). However, infections caused by algae and yeast are not common but can cause individual cow and herd problems (Guidry, 1986). The most important bovine mastitis causing bacteria in New Zealand are predominantly *staphylococci*; to a lesser extent the *streptococci*; to a minor extent, the *coliform* and *pseudomonas* groups Cooper (1974) and cited by Milne (1978). A nation-wide survey of mastitis causing bacteria in milk in New Zealand revealed that 9-16 % of cows were infected with *Streptococcus agalactiae*, 2-3 % with *Streptococcus dysgalactiae*, 0.5-3.3 % with *Streptococcus uberis* and 25-40 % with *Staphylococcus aureus* (Brookbanks, 1966; Elliot et al., 1976) and cited by Holmes and Wilson (1987). *Staphylococcus aureus* and *Streptococcus agalactiae* are generally found on the cow's skin and particularly in sores and teat orifices and are readily transmitted between cows and udder quarters during milking either by the teatcups or the milker's hand (Fetrow, 1984; Bramley, 1992), and hence they are termed **contagious pathogens** (Bramley, 1991). These organisms survive well on the teat surface and can colonize and grow in teat lesions. When found on a culture of bulk tank milk, these mastitis causing pathogens are strong indicators of the presence of intramammary infections in the herd (Gonzalez et al., 1986) and cited by Godkin and Leslie (1993). *Staphylococcus aureus* is one of the major bacterial pathogens of concern in New Zealand dairy herds, and it usually produces mastitis infections distributed throughout the lactation (Williamson et al., 1993).

Environmental bacteria such as coliform and *Streptococcus uberis* while they may be transferred at milking time, more typically contaminate teats in the interval between milking, from sources such as cattle litter, faeces and water (Bramley, 1991). *Streptococcus uberis* is ubiquitous for it is found in various

sites of the cow such as on the skin, in the rumen and in the environment such as in the soil, litter and faeces (Fetrow, 1984; Williamson et al., 1993). Streptococcus uberis, is in most cases the cause of mastitis in late lactation (early dry period) and then again in early lactation in New Zealand dairy herds (Day, 1990; Williamson et al., 1993). It is frequently responsible for high bulk tank SCC in early lactation (Williamson et al., 1993). Streptococcus dysgalactiae may also be found in various sites of the cow and other mucosal membranes. The presence of these environmental pathogens in cultures of milk from the bulk tank may relate to the general level of environmental and milking hygiene in the herd (Cousins, 1972). However, their presence has been shown to be independent of the prevalence of mastitis (Gonzalez et al., 1986) and cited by Godkin and Leslie (1993). Udder infections with these environmental pathogens are predominantly of short duration and characterised by clinical disease which make their inadvertent introduction to the bulk tank less likely (Smith et al., 1985).

2.3 METHODS OF DIAGNOSIS OF BOVINE MASTITIS

Many tests are available that attempt to detect bovine mastitis. These are categorised into namely, (a) direct bacterial culture and (b) indirect measurements. Some of the more widely employed tests include culture of bacteria and bacteria counts (Eberhart et al., 1987), changes in p^H (Eberhart et al., 1987), electrical conductivity (Fernando et al., 1982), catalase and NAGase enzymes activities (Kitchen, 1981) or protein levels (Kitchen, 1981; Schultze, 1985). Bacteriological testing is both time consuming and expensive, requiring skilled personnel both to take samples and interpret growth (Guidry, 1986). However, the most widely used indirect test is the measurement of total somatic cells, the somatic cell count (SCC) (Eberhart et al., 1982, Dohoo and Meek, 1982, Fetrow, 1984). This is discussed in detail below.

2.4 ORIGIN AND FUNCTIONS OF SOMATIC CELLS IN MILK

Somatic or body cells in milk include leukocytes mainly neutrophils from the blood and sloughed epithelial cells from the udder (Schultz, 1977; Guidry, 1986). Epithelial cells are present in normal milk as a result of normal breakdown and repair, but increase in late lactation as the gland prepares to be non-functional or as a consequence of stress associated with milking (Schultz, 1977), and as milk volume decreases (Schalm, 1977). Somatic cells of normal milk and late lactation milk are therefore mainly of epithelial in origin (Schalm, 1977). Normal milk contains levels of 65-70 % epithelial cells, chronic mastitis milk about 50 %, and more severely mastitic milk level lower (10-45 %) due to the presence of a high proportion of leukocytes from blood (Schalm and Lasmanis, 1968) and cited by Schalm (1977). Thus in a healthy udder, the dominant somatic cells are epithelial cells whereas leukocytes predominate during mastitis infection. In the event of injury or mastitis infection, the invading bacteria release chemical substances which attract leukocytes from blood to enter milk (Schalm, 1977; Schultz, 1977; Guidry, 1985). The leukocytes phagocytose and destroy the bacteria, resulting in breakdown products of both bacteria and leukocytes. The breakdown products also serve as chemotactic agents to increase the influx of leukocytes, causing an inflammatory response (Guidry, 1986). The continual recruitment of leukocytes into milk provides a protective role in defending the bovine mammary gland (Schalm et al., 1964). A target of 500,000 cells/ml of milk has been suggested as a SCC that can afford protection against intramammary bacterial challenge (Schalm et al., 1964). Some of the bacterial toxins also damage the tight junctions that cause an increase in permeability of vascular and secretory systems (Linzell, 1975; Schultz, 1977). This allows blood components including, serum albumin, immunoglobulins and sodium and chloride ions to enter milk (Linzell, 1975; Schalm, 1977; Schultz, 1977), and hence cause changes in the biochemical composition of mastitic milk. This is discussed further in section 2.8.2.

2.4.1 Use of SCC as a diagnostic test for subclinical mastitis

The somatic cell counts (SCC) measure the number of “somatic” or body cells per millilitre of milk. The cell count of milk is of particular interest in the diagnosis of subclinical mastitis, as this form cannot be recognised by the cardinal signs characteristic of clinical mastitis (Tolle, 1975; Guidry, 1985, Bramley, 1922). Based on the premise that elevated levels of pathogenic bacteria in milk is closely followed by marked increases in the number of somatic cells, SCC are widely used as indirect diagnosis of subclinical mastitis in quarters, cows or herds (Neave, 1975; Schultz, 1977; Dohoo and Meek, 1982; Fetrow, 1984; Bramley, 1992). Various methods have been used over the years to measure somatic cells of milk. These include:

A. ***Direct microscopic somatic cell counts*** of stained milk samples (National Mastitis Council, 1968).

B. ***California Mastitis Test (CMT)*** and ***Whiteside Mastitis Test*** which measure the degree of precipitation or gel formation which occurs when milk samples are treated with detergent reagents (Schmidt, 1971). The degree of precipitation is related to the somatic cell count of milk. A modification of these test is the Wisconsin Mastitis Test (WMT), which quantitatively assesses the degree of gel formation (Thompson and Postle, 1964) and cited by (Schmidt, 1971). This is based on the measurement of viscosity of milk-detergent mixture by allowing it to flow for 15 seconds through a narrow hole in the cap of a plastic tube, and measuring the amount of the mixture left in the tube.

C. ***Automated/Electronic somatic cell count*** devices are now used for rapid estimation of SCC of milk samples. The most commonly used are (1) Coulter counter - a device that counts particles as they flow through an electric field, and (2) Fossomatic - a device that stains cells with a fluorescent dye and then counts the fluorescing particles (Schmidt Madsen, 1975; Leslie et al, 1984;

Bramley, 1992). Both instruments are capable of rapid, inexpensive determination of SCC in large numbers of samples (Leslie et al, 1984).

2.5. RELATION BETWEEN SCC AND SUBCLINICAL MASTITIS

Somatic cell count has become the usual mastitis monitoring parameter applied to individual cows or whole herds milk (IDF, 1975; Bramley, 1991). Several studies have shown that there is a close association between subclinical mastitis and SCC in milk from quarters, individual cows or whole herds (Bulk herd milk) (Klastrup, 1975; Reichmuth, 1975; Gill and Holmes, 1978; Fetrow, 1984; Leslie et al., 1984; Holdaway, 1991). High cell counts indicate an immunological response to bacteria infection.

2.5.1 Relation between SCC and subclinical mastitis of individual cows

Milk from uninfected mammary glands may contain 20 000 to 200 000 cells/ml (National Mastitis Council, 1968; Guidry, 1986; Holmes and Wilson, 1987). Secretory disturbances, including declining milk yield, have been reported to start once cell counts exceed 100-150 000 cells/ml, and the probability of isolating a major pathogen is increased with counts above 200 000 cells/ml (Reichmuth, 1975). The SCC of milk have been reported to average from 260 000 cells/ml in quarters with no previous history of mastitis to 600 000 cells/ml in quarters with a previous history of infection (Ward and Schultz, 1972). The recommended threshold for classifying a quarter as being infected has been set at 300 000 cells/ml (Klastrup, 1975; Dahoo and Meek, 1982). Quarters producing milk containing in excess of 300 000 cells /ml have a high probability of being infected, while SCC value of > 500 000 cells/ml in quarter milk may occur in very severe mastitis infection (Holmes and Wilson, 1987). In general low cell counts suggest a good level of udder health of cows in herds (Leslie et al., 1984).

Reports also indicate that SCC values of uninfected individual cows are generally below 250 000 cells/ml (Natzke et al., 1972; Schultz, 1977; Eberhart et al., 1979; MacMillan and Duirs, 1980; Dohoo and Meek, 1982; Holdaway, 1991). For example a New Zealand report has indicated that 66 % of all cows had SCC values below 200 000 cells/ml (MacMillan and Duirs, 1980). Schultz (1977) also found that the mean SCC for cows with no udder infection, infected with non-pathogens only or infected with pathogens were 169 000 cells/ml, 225 800 cells/ml and 997 800 cells/ml respectively. Data by Natzke et al (1972) (See table 2.1) also showed that composite milk from an uninfected udder contained about 200 000 cells/ml. There was an approximate doubling of the SCC with each additional quarter that was infected (See table 2.1). It is clear that the SCC in the composite milk from all four quarters will depend on the dilution of milk from infected quarters by milk from normal quarters.

Table 2.1 The average values for SCC in composite milk samples from uninfected and infected cows

Number of quarters infected	Mean SCC (cells/ml)
0	214 000
1	507 000
2	701 000
3	1 470 000

(Adapted from Natzke et al., 1972)

This should therefore be taken into consideration in the interpretation of individual cow somatic cell count data (Leslie et al., 1984). A threshold value of about 250 000 cells/ml has been suggested for differentiating an uninfected and infected cows (Dohoo and Meek, 1982; Leslie et al., 1984). More recently Holdaway (1990) has suggested a lower value of 185 000 cells/ml as a threshold for distinguishing between infected and uninfected quarters.

2.5.2 Relation between bulk milk SCC (BMSCC) and subclinical mastitis

On a herd basis, some indication of the extent of subclinical mastitis can be obtained from the bulk milk somatic cell counts (BMSCC). A majority of cows with clinical mastitis tend to produce milk with high SCC and therefore herds which have a high percentage of cows with clinical mastitis are more likely to have high SCC values.

High BMSCC values are associated with a high incidence of subclinical mastitis in herds (Pearson et al., 1971; Gill and Holmes, 1978; Eberhart et al., 1982; Dohoo and Meek, 1982; Holdaway, 1991). Gill and Holmes (1978) observed that herds with BMSCC of 200 000 cells/ml was associated with 1.2 cows with clinical mastitis per 100 cows milked, whereas there were 2.6 clinical cases per 100 cows in herds with a BMSCC of 600 000 cells/ml. More recently, Holdaway (1991) reported that as the BMSCC increased from 200 000 to 1.5 million cells/ml, the percentage of cows infected also increased from 31 to 63 respectively (See table 2.2).

Table 2.2 The relation between herd milk and percentage of cows herds infected with mastitis.

	Bulk milk cell count (cells/ml) x 1000					
	200	250	400	750	1 000	1 500
Percentages of cows infected (%)	31	35	42	52	57	63

(Adapted from Holdaway, 1991).

In general, a herd with BMSCC under 250 000 cells/ml has a low level of subclinical mastitis, whereas counts over 500 000 cells/ml indicate a herd problem with subclinical mastitis (Dohoo and Meek, 1982; Leslie et al., 1984). A BMSCC over 750 000 indicates a very high subclinical mastitis problem. Although BMSCCs do not provide any information about which individual cows

are affected, they can be used as a general indication of the proportion of cows in the herd with low or high values for SCC (Gill and Holmes, 1978; Dohoo and Meek, 1982).

The degree of association between BMSCC and prevalence of mastitis has also been estimated by correlation coefficients. Correlation coefficients have demonstrated significant associations and the estimates vary considerably with values ranging from 0.46 to 0.96 (Pearson et al., 1971; Pearson and Greer, 1974; Reichmuth, 1975; Westgarth, 1975; Schmidt Madsen et al., 1976; Emmanuelson and Funke, 1991). Emmanuelson and Funke (1991) reported a range of 0.53 to 0.77 correlation between prevalence of mastitis and average bulk milk SCC. Westgarth (1975) and Hogan et al (1988) reported low correlation ($r = 0.5$ and 0.58 respectively), while others such as Reichmuth (1975) and Pearson and Greer (1974) reported high correlation ($r = 0.89$ and 0.83 respectively) between bulk milk SCC and the number of quarters with mastitis. The high correlation between bulk milk SCC and the number of quarters affected was influenced by the fact that the infected quarters which were not accompanied by significant increase in SCC were deemed to be uninfected (Pearson and Greer, 1974).

BMSCC is used in several countries (Bramley, 1991) and particularly the SAMM Plan (1995) in New Zealand in mastitis control and screening of farms for mastitis. Furthermore, BMSCC is now being used by some milk factories such as TUI Milk Products Company in Palmerston North, New Zealand as one of the measurement factors for bulk milk payments to farmers (TUI Milk Products Company, 1995). In TUI's grading system, a supplier's milk with BMSCC over 400 000 cells/ml attracts monetary penalty (See table 2.3).

When interpreting BMSCC it is important to remember that elevation of the count may result from a few cows having exceptionally high cell counts or from general elevation of counts in many of the cows in the herd (Dohoo and Meek, 1982). The effect of a few cows with very high SCCs is particularly noticeable

in small herds (Schultz, 1977). It may be more of a problem with some organisms than with others. For example, a lower and non-significant ($r = 0.24$, $P < 0.05$) correlation occurred between quarters infected by Staphylococcus aureus and BMSCC than that between quarters infected by Streptococcus spp ($r = 0.88$, $P < 0.001$) (Holdaway, 1991).

Table 2.3 The TUI Milk Products Company grading system according to BMSCC in farm bulk milk.

BMSCC Category (cells/ml)	Grade	Penalty (Cents/litre)/Remarks
<400 000	Finest	No penalty
401 000-600 000	1	1.7 Penalty
601 000-800 000	2	3.7 Penalty
>800 000	2	10.0 Penalty

(Adapted from Raw milk standards, Tui Milk Products Company, 1995)

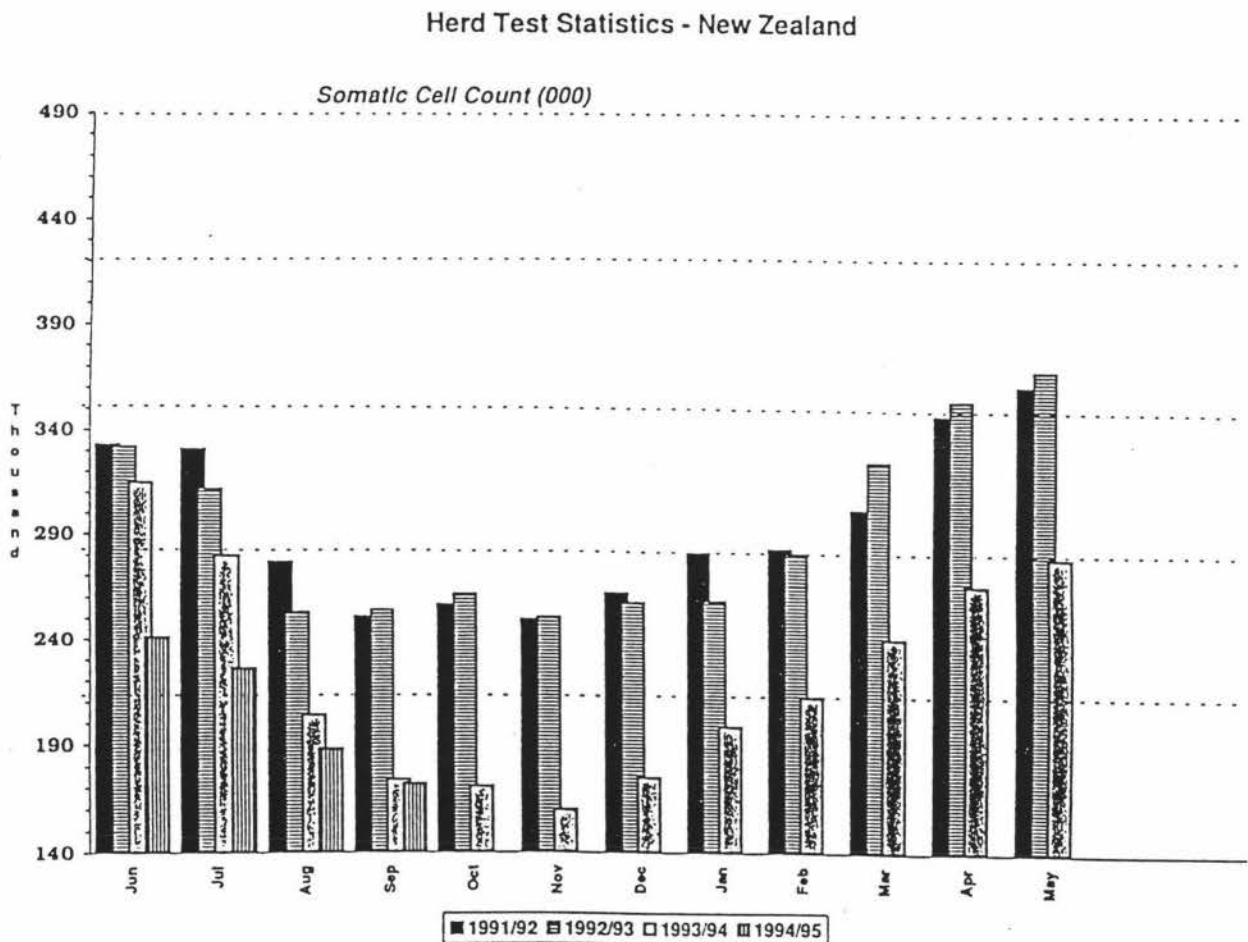
2.6 SEASONAL TREND OF SCC IN HERDS

Somatic cells counts of individual cows are elevated in both early and late lactation regardless of whether the cows are infected or not (Dohoo and Meek, 1982; Schultz, 1977). Elevation of SCC of cows in early lactation has been attributed to a high concentration of somatic cells in colostrum milk (Reichmuth, 1975; Auld et al, 1993) and mastitis infection not treated during the dry period (Pankey, 1994). Higher cell count of cows in late lactation has also been attributed to a decrease in milk volume giving apparent increase in cell numbers from mere concentration of cells in the smaller volume of milk (Schmidt, 1971; Schultz, 1977).

Spring calving dairy herds in New Zealand generally show higher BMSCCs in early lactation, a decrease in mid-season and then an increase in late lactation (See fig. 2.1) (Pankey, 1994; Frank, 1994; LIC, 1994). The average bulk milk somatic cell count for all herds in New Zealand is estimated to be 300 000

cells/ml (SAMM Plan, 1995; LIC, 1994; Franks, 1994). This is comparatively lower than <500 000 cells/ml EC standard for BMSCC (Frank, 1994). The high BMSCC in early season due to undiagnosed mastitis infection during the dry period, new cases of mastitis that developed during the dry period and mastitis in heifers at calving (Pankey, 1994).

Fig. 2.1 showing the seasonal trend of BMSCC in New Zealand dairy herds from 1991/92 to 1994/95 seasons (Adapted from LIC, 1994)



This can be prevented by treating all cows during the dry period and maintaining heifers on clean paddocks at calving (Pankey, 1994). The elevation of somatic cell counts in late lactation is not only a function of decreased dilution effect as milk volumes decline (Schmidt, 1971; Pankey, 1994; Franks; 1994), but also due to mastitis that develops during the season (Schultz, 1977; Franks, 1994; Pankey, 1994). The decreased dilution effect as

milk volumes decline can be prevented by drying-off cows before their yields have decreased to very low levels at the end of the season (Franks, 1994).

Also mastitis that develops during the course of the season can be reduced by a combination of post-calving teat spraying and treatment of mastitis infections as they occur (Frank, 1994).

2.7 BACTERIA COUNT IN BULK MILK

Bacteria have been recognised as the major bovine mastitis-causing agents (Neave; 1975; Tolle, 1975; Fetrow, 1984). (This has been discussed in section 2.2). When found in a sample from bulk tank milk, these mastitis-causing pathogens are strong indicators of subclinical mastitis infection in the herd (Godkin and Leslie, 1993). Bacteria in raw milk may originate from:

- contamination from udder infections.
- contaminations from exterior surfaces of the udder and teats, and
- contamination from milking and storage equipment (Hubble and Manners, 1985; Bramley, 1992).

Contamination from udder infections does occur when udder quarters with both subclinical and clinical infections shed bacteria into milk. A subclinical quarter may contain <10 000 to 100 000 bacteria/ml (Tolle, 1980), whereas clinically infected quarter may contain >10 million bacteria/ml (Mabbit et al.; 1982). If this is inadvertently included in the bulk milk, the total plate count may be increased particularly if there is a high percentage of infected quarters in the herd (Mabbit et al., 1982).

Contamination from exterior surfaces of the udder and teats. This may occur when contaminated water runs down over the udder and teats and drawn into the teatcups during washing prior to milking, or from the milker's hands and dirty water (Hubble and Manners, 1985 and Bramley, 1992). Several

experiments in New Zealand and Australia have shown no significant differences for milk quality (Evans-Scott, 1979; Ridler, 1982 and Hubble and Mein, 1985) and milkfat production (Ridler, 1982) when no washing was compared with washing of udder and teats prior to milking. The recommendation therefore is that where washing is used, the udder surfaces above the teats should be dry, but not necessarily clean, with only the dirty teats cleaned with water (Hubble and Manners, 1985).

Contamination from milking and storage equipment. The milking machine is a potential source of contamination of raw milk with bacteria (Cousins, 1977; Cousins and Bramley, 1981; Bramley, 1992). Such contamination may come from the surfaces of the milk-line, the receiving vessel, the cooler, the milk pump and the vat due to improper cleaning of the milking machine (Holmes and Wilson, 1987). Furthermore, incomplete cooling of bulk milk and storage in the vat above 7°C will enhance bacteria growth.

The presence of particular types of bacteria such as staphylococci and streptococci pathogens in milk can therefore be an indication of mastitis infection. For this reason bacteriological testing is being used as one of the diagnostic tool for bovine mastitis and also as a measure of milk quality (See section 2.7). The most widely used type of bacteriological test is the Standard Plate Count (SPC). In this test a sample of milk is cultured in a special agar gel for the bacteria present to grow and multiply for 72 hours at 30°C to form a colony (Holmes and Wilson, 1987 IDF, 1990). These colonies are counted and the number of bacteria in each millilitre of the original milk is calculated. Recently a device called Foss BACTOSCAN is being used by some laboratories and dairy companies as a rapid alternative to SPC for counting bacteria in milk (Smith and Hill, 1988). The Bactoscan is an automatic instrument which counts the bacteria in a milk sample by staining them with a fluorescent stain, then scanning them with an incident light fluorescent microscope. This instrument can produce a bacterial count in only 7 minutes compared to 3 days taken with the SPC (Smith and Hill, 1988).

2.7.1 Relation between bacteria count in bulk milk and mastitis

Some studies of bulk tank milk results have been used to predict the prevalence of cows infected with specific pathogens within the herd (Gonzalez et al., 1986; Pearson et al, 1979; Greer and Pearson, 1973). The frequency of positive monthly bulk milk cultures for Streptococcus agalactiae was found to have a correlation of 0.53 with the prevalence of infected quarters within herds, and 0.48 with the prevalence of infected cows (Pearson et al., 1979). Statistically significant correlations were also found between the streptococcal count in the bulk milk tank and the rate of streptococcal clinical mastitis ($r = 0.35$) and between the gram-negative bacterial count of the bulk tank and the incidence of clinical coliform mastitis ($r = 0.46$) (Hogan et al, 1988). Milk somatic cell count was correlated with total incidence of clinical mastitis ($r = 0.58$). These correlations indicated that monitoring the bulk tank milk may be an effective means of detecting management changes in herds with low or high bacterial and somatic cell counts.

2.7.2 Relation between bacteria and BMSCC

As discussed in section 2.2, the major factor responsible for elevation of SCC in milk is the presence of bacterial pathogens in the udder. Some studies have shown the association between bacteria and SCC (Ward and Schultz, 1972; Greer and Pearson, 1973; Emmanuel and Funke, 1991; Hogan et al., 1988; Godkin and Leslie, 1993). A highly significant correlation ($r = 0.75$, $P < 0.01$) was observed between Streptococcus agalactiae and BMSCC of some Irish herds supplying bulk tank milk to a dairy company (Greer and Pearson, 1973). However, in a follow-up study, in which tank samples of 51 herds were cultured monthly for one year, a lower correlation of 0.53 was found between the frequency of herd isolation and percent of quarters infected with Streptococcus agalactiae (Pearson et al., 1979). The bracketing of infected herds into cell count ranges showed that only 21 % had annual means of <300 000 cells /ml, in contrast to 61 % with means of greater than 1 millions cells/ml in the same

study (Greer and Pearson, 1973). Also low correlation between Streptococcus agalactiae and SCC in 2 herds were 0.51 (Bennett, 1987) and cited by Godkin and Leslie (1993) 0.37 (Hogan et al., 1986) respectively were recorded. For Staphylococcus aureus, low correlation coefficients of 0.36 (Bennett, 1987) and 0.37 (Hogan et al., 1986) were also reported. These generally low correlations between herd SCC and specific pathogen colony numbers may occur because herd SCC can be influenced by the presence of cows infected with pathogens not belonging to the group of bacteria under investigation (Godkin and Leslie, 1993), or because herd SCC is affected by milk yield (Emmanuel and Funke, 1991), or the numbers of bacteria shed may vary periodically (Sears et al., 1990)

2.8 EFFECTS OF SUBCLINICAL MASTITIS ON THE FARMER

Subclinical mastitis may affect the dairyfarmer in various ways which include:

- A reduction in milk yield by infected cows in the herd
- A change in milk composition
- Increasing the incidence of clinical condition causes an increase in veterinary costs
- Infected cows may act as a reservoir of pathogenic bacteria for uninfected cows of the herd
- Increase in bulk tank milk bacteria as a result of bacteria shed by infected cows, which may attract penalty in bulk milk payment.
- Increase the culling of low-yielding cows

2.8.1 Reduction in milk yield

The major effect of mastitis on the dairyfarmer is the milk production losses due to reduction in milk yield. The production losses have been measured in relation to many variables, including pathogenic bacteria involved, CMT scores

and SCC which are indicators of mastitis. The effects of mastitis on milk yield of have been measured in many studies for:-

1. individual infected quarters of the udder relative to equivalent uninfected quarters
2. individual infected cows relative to uninfected cows in a herd and
3. herds with varying degrees of infection.

Many studies have shown that milk production from infected quarters is depressed compared with that from uninfected quarters (Wheelock et al., 1966; Philpot, 1967; Smith et al., 1968; Janzen, 1970; Morris, 1973). The reported losses in milk yield at quarter level range from 9 % to 45 % (Janzen, 1970; Philpot, 1967; Dohoo and Meek, 1982). Philpot (1967) reported that milk yield was reduced by 3, 11, 26, and 46% in quarters with CMT reactions of Trace, 1, 2, and 3. Similarly individual quarters which have a high SCC produce less milk than other quarters on the same udder but with low SCC (Ward and Schultz, 1972; Schultz, 1977; Jones et al, 1984). On a quarter basis, loss started at 500 000 cells/ml, progressed to 7.5% at 1 million, and 30% at 5 million (Schultz, 1977). Other workers have also reported that at quarter level milk yields start to decline at 100 000 cells/ml (Reichmuth, 1975), 200 000 cells/ml (King, 1978), and 500 000 cells/ml (Schultz, 1977).

Janzen (1970) reported that losses in total milk yield from infected individual cows ranged from 5% to 25%. Substantial losses in yield are also associated with more positive reactions to CMT. For example losses at the cow level were 6%, 10%, 16% and 25% for CMT reactions respectively of Trace, 1, 2, and 3 (Gray and Schalm, 1962). Several studies have also shown that cows with high SCC produce less milk than cows with lower SCC (Daniel and Fielden, 1971 MacMillan and Duirs, 1978; Gill and Holmes, 1978; King, 1978; Youl and Nicholls, 1987). Gill and Holmes (1978) reported that a loss of 1.4 litres/cow/day was associated with an increase of 1 000 000 cells/ml in cows in a herd in New Zealand. A study in Australia also showed that a doubling of the

geometric mean cell count of individual cows was associated with a loss of 4 kg of milkfat per cow or 2% in production index per lactation (Youl and Nicholls, 1987). In general, elevated somatic cell counts are indicative of production losses due subclinical mastitis. It appears that although that there is a considerable variation in literature as to the magnitude of the loss, it may frequently exceed 20 % of a cow's potential production (Schultz, 1977; Gill and Holmes, 1978; Eberhart et al., 1982; Dohoo and Meek, 1982)

The level of mastitis in a herd, as measured by bulk milk cell count, has also been shown to be associated with milk production of the herd (Gill and Holmes, 1978; Hoare, 1982; Jones et al., 1984; Emmanuel and Funke, 1991).

Table 2.4 The relation between BMSCC for a herd and the average milk production per cow in the herd.

BMSCC (cells/ml)	Decrease in average milk production (%)
<200 000	0
300 000	-4
500 000	-7
700 000	-11

(Adapted from Gill and Holmes, 1978)

It is obvious from table 2.4 that when the herd's BMSCC was partitioned according to average milk production (<200 000, 300 000, 500 000, and 700 000 cells/ml), decrease of milk yield was associated with increasing BMSCC (Gill and Holmes, 1978). Dohoo et al. (1983) found that a unit increase in the log SCC resulted in a loss of 1.44 kg of milk in 32 Ontario herds. Similarly when herds were partitioned according to herd milk production (>7700, 6500, 7700, and <6500 kg/yr), decrease of milk production was linear with increasing somatic cell counts for herds averaging below 7700 kg milk (Jones et al., 1984). Also studies have demonstrated that herds on mastitis control

programme produced more milk than infected herds (Natkze et al., 1972b, Benson and Dettman, 1977).

2.8.2 Effect of subclinical mastitis on milk composition

In a typical lactation curve, while fat and protein concentrations are at their lowest at the peak of milk production in spring and early summer and then gradually rise over the remainder of the season, lactose concentration decreases throughout the season in New Zealand dairy cows (Holmes and Wilson, 1987). Also Jersey cows produce milk higher in fat (5.6 vs 4.5 %) and protein (4.0 vs 3.5 %) concentrations than Friesian cows due to genetic variations (Holmes and Wilson, 1987).

An intramammary infection destroys some or all of the secretory cells and also impairs the integrity of the functioning epithelial tissue (Guidry, 1986). As a result there is an influx of ions and blood constituents such as serum proteins and somatic cells into milk and equivalent movement of lactose and potassium ions from milk into blood (Schalm, 1977; Linzell, 1975), causing changes in the milk composition (Kitchen, 1981; Hoare, 1982; Holmes and Wilson, 1987). Several workers have reported that in mastitic milk the concentrations of major milk constituents namely fat, lactose and casein are decreased but the concentration of total whey protein is variable (Schultz, 1977; Kitchen, 1981; Hoare, 1982; Holmes and Wilson, 1987). Thus the constituents which are synthesised in the mammary gland decrease and those which come from the blood may increase. Some of these changes in individual components are as follows:

2.8.2.1 Total solids and solid-not-fat (SNF) percentages

Total solids (TS) % change with mastitis infection due to changes which occur in lactose, protein and fat. Ashworth et al (1967) observed a decrease in TS % in all quarters with high mastitis infection as measured by CMT. Correlation

coefficient of 0.29 ($P < 0.05$) was found between logSCC and total solids % in farm bulk milk (Mitchell et al., 1986). The decrease in TS was mainly due a decrease in lactose which had a negative correlation of -0.31 ($P < 0.05$). Dawson et al (1974) found a -0.14% change in TS % with each increase in log SCC. This decrease in TS % was mainly due to a decline in lactose % and protein %.

Solid-not-fat (SNF) % also tend to decrease with high cell count in milk due to a decreased lactose % in milk, while there may be little change in protein %. King (1978) found a significant ($P < 0.05$) decrease in the SNF % when comparing samples with an SCC $> 500\ 000$ cell/ml to samples with an SCC $< 500\ 000$ cells/ml. LogSCC was negatively correlated with the SNF % ($r = -0.58$, $P < 0.01$) and lactose % ($r = -0.59$, $P < 0.01$) (Rogers et al, 1989). The decrease in SNF % was mainly due to a decrease in lactose %.

2.8.2.2 Fat concentration

Most reports generally indicate that milkfat % decreases with mastitis infection. This occurs because the fat synthesis is usually reduced more than milk yield in mastitic milk due to damaged secretory cells (Schultz, 1977). Ashworth et al. (1967) reported a decrease in milkfat % with mastitis (4.2 and 3.7% for milk with CMT-negative and CMT 3 respectively in opposite quarters), and Philpot (1967) gave a decrease of 13.7% in milkfat % for CMT 3. Randolph and Erwin (1974) found cows with WMT greater than 20 had a lower milkfat % than cows with WMT less than 10. Other reports indicate that fat % may increase in mastitic milk. For example low positive correlation coefficients have been shown between herd bulk milk cell counts and herd fat content by Gill and Holmes (1978) with $r = 0.06$ ($P < 0.01$), and Mitchell et al. (1986) with $r = 0.41$ ($P < 0.01$). Ng-Kwai-Hang et al. (1982) also reported a low positive correlation in infected quarter milk, while Natzke et al. (1965) reported a non-significant negative correlation of -0.174 between cell count and fat %. This increase in fat % with increase in cell counts may occur in some situations where milk yield

is reduced to a greater degree than the yield of milkfat synthesized (Schultz, 1977). However, when considered over a long period of time, a clinical mastitis infection can result in a large loss in total milkfat yield produced, because of reduced milk yield (Schultz, 1977).

Apart from a decrease in milkfat %, its composition also changes with increase in cell counts (Schultz, 1977). Reports indicate that increase in cell counts causes a reduction in milk fat globule membrane (MFGM) and consequently its phospholipids contents (Erwin and Randolph, 1975; Randolph and Erwin, 1974). Less MFGM leads to physical changes in cream, longer churning time and weaker bodied butter (Munro et al., 1984). The reduction in the phospholipids content of the MFGM makes the fat globules more susceptible to the lipolysis due to lipase and thereby increasing fatty acids and acid degree values (Randolph and Erwin, 1974; Erwin and Randolph, 1975). Free fatty acid content was correlated with SCC ($r = 0.64$, $P < 0.01$) in milk samples obtained from cows with mastitis infection (Gudding, 1982). Some of these free fatty acids can cause off flavours or hydrolytic rancidity in cheese made from such milk (Erwin and Randolph, 1974).

2.8.2.3 Milk protein concentration

Generally there appears to be no significant change in total milk protein concentration with increasing somatic cell counts (Haenlein et al., 1973). But significant changes in the milk protein components have been reported. In general, the caseins (α -casein, β -casein, β -lactoglobulin and α -lactalbumin) have been reported to decrease, while concentration of whey proteins (bovine serum albumin and immunoglobulins) increase with increase in somatic cell counts (Haenlein et al., 1973; Randolph et al., 1974). The various increases and decreases apparently balance one another and therefore the total protein concentration is relatively constant (Schultz, 1977). The changes in composition are directly proportional to the severity of infection, as measured by cell count. Natzke et al (1965) found a significant positive correlation ($r =$

0.122) between cell count and protein concentration. Ng-Kwai-Hang et al (1982) also reported that for each 1 million increase in cell count, total protein concentration increased by 0.18%.

Total casein concentration generally decreases with increasing somatic cell count (Schultz, 1977; Munro et al., 1984). Also during mastitis infection proteolytic activity increases within the udder leading to degeneration of the caseins (Bramley, 1992). Haenlein et al. (1973) found lower concentrations of casein in Holsteins and Guernseys as cell count exceeded 500 000 cells/ml. Rogers et al (1989) found a negative correlation ($r = -0.40$; $P < 0.01$) between total casein concentration and logSCC in bulk milk. On the hand whey proteins concentration has been found to increase as cell counts increased (Haenlein et al., 1973; Schultz, 1977; Rogers et al., 1989). Rogers et al (1989) found a positive correlation ($r = 0.54$, $P < 0.01$) between whey protein concentration and logSCC of bulk milk samples. Heanlein et al (1973) reported that as SCC increased from <250 000 to >1 million cells/ml whey protein concentration also increased from 0.82 to 1.31%. In summary the increase in total whey proteins concentration appears to compensate for the fall in the casein fraction, resulting in little change in total protein concentration until mastitis becomes relatively severe, when the leakage of serum proteins over-compensates for the losses of proteins produced by the gland, with the result that the total protein content of milk increases.

2.8.2.4 Lactose concentration

Generally there is a decrease in lactose content of milk when somatic cells are elevated (Ashworth et al., 1967; Schultz, 1977; Kitchen, 1981; Munro et al., 1984; Mitchell et al., 1986). Lactose is synthesized in the Golgi apparatus of the mammary gland secretory cell (Linzell and Peaker, 1971; Schultz, 1977, Peaker, 1978). Mammary gland infection results in tissue damage and decreased synthetic ability of the secretory cells (Wheelock et al., 1966). The decrease in lactose concentration may also be partly due to a fall in a milk

protein, α -lactalbumin - a component of an enzyme involved in the final step of lactose synthesis, with mastitis infection (Schultz, 1977), and in part to increased permeability of lactose down the concentration gradient between milk and blood. Renner (1975) suggested a lactose concentration for herd milk at 4.49%, and anything below this is associated with mastitis infection. Ashworth et al. (1967) reported lactose concentration from opposite quarters were 4.88, 4.83, 4.73, 4.50, and 3.95% respectively for milks rated negative, trace, 1, 2, and 3 on the California Mastitis Test (CMT). It is obvious that the fall becomes greater as the reaction increases. Bergmann (1979) and cited by Kitchen (1981) reported that milks with <3.8% lactose had SCC usually >1000 000 cell/ml while milks with >5% lactose had low cell counts (<100 000 cells/ml). Negative correlations between lactose concentration and cell counts of -0.53 ($P<0.01$) (Rogers et al., 1989), -0.31 ($P<0.05$) (Mitchell et al., 1986), and -0.6 to -0.81 (Natzke et al., 1965) have been found in farm bulk milk. However, higher negative correlation ($r = -0.89$, $P<0.01$) between lactose concentration and somatic cell count was found in quarter milk samples (Kozanecki et al., 1982).

2.8.2.5 Concentration of minerals or ions

In normal milk secreted by the mammary gland, the concentrations of Ca^{2+} and K^+ are high with low Na^+ and Cl^- concentrations, while the converse is the case in blood (Linzell and Peaker, 1971; Peaker, 1978). However, mastitis infection of the udder causes damage to the secretory epithelium and opens up the 'tight junctions' between the secretory cells. This results in an influx of Na^+ , Cl^- and serum proteins such as immunoglobulins and albumin into milk, and simultaneous movement of Ca^+ and K^+ and phosphorus into blood in order to maintain osmolarity (Wheelock et al., 1966 and Peaker, 1978). The lactose concentration also decreases, but as lactose is un-ionised, the increase in sodium and chloride ions more than compensate for the decrease in potassium ions, with the result that the electrical conductivity of the milk increases (Linzell, 1975). The electrical conductivity of mastitic milk is used in the diagnosis of subclinical mastitis in quarters (Linzell and Peaker, 1975;

Fernando et al., 1982; Guidry, 1985). The published correlations between electrical conductivity (ER) and SCC are mainly between 0.4 and 0.6 (Graupner et al., 1992). But the correlation between ER and mammary infection is not always consistent and therefore not a reliable mastitis diagnostic tool on commercial basis (Shoshani and Berman, 1992). It is better if used for individual quarters and when quarters are compared within the cow.

Ashworth et al. (1967) found a 61% increase in Cl^- in CMT-3, compared to negative milk. Significant correlations of 0.415 (Natzke et al, 1965) and 0.52 (Renner, 1975) have been found between Cl^- and cell count. Similarly positive correlation of 0.56 (Renner, 1975) and 0.57 (Rogers et al., 1989) have been found between SCC and Na^+ . Tellamy and Randolph (1970) reported a 38% increase in Na^+ and a 9% decrease in K^+ comparing milk with low and high WMT reactions.

Calcium and phosphorus concentrations of milk are considerably higher than that of the blood and both decrease slightly with increase in cell count (Schultz, 1977). Calcium is of importance in rennin coagulation, therefore a change in content will have effect in cheese making.

The trace minerals such as iron, copper and zinc are very low in milk and increase slightly with elevated cell count (Tallamy and Randolph, 1970 and Shultz, 1977). This is probably due to a decrease in milk yield as a result of mastitis infection (Schultz, 1977).

2.8.3 Economic loss due to mastitis

Bovine mastitis is recognised as one of the most costly diseases of the dairy industry. The financial losses to the dairyfarmer are due to (a) Milk production loss (b) Discarded milk (c) Veterinary fees (d) Increased replacement costs (e) Drug costs and (f) Extra labour cost (Janzen, 1970; Fetrow; 1984; Holdaway, 1993). In New Zealand it is estimated that mastitis costs about NZ\$14,600

annually in average dairy herd (170 cows per herd), with about 62% losses due to reduced milk yield (Holdaway, 1993) (See table 2.5).

Table 2.5 The estimated cost of mastitis to the average New Zealand dairyfarmer annually.

	Cost in NZ Dollars (NZ\$)
Milk production loss	9095
<i>Clinical mastitis:</i>	
Labour	164
Antibiotics	216
Discarded milk	63
<i>Dry cow therapy:</i>	
Labour	28
Antibiotics	459
Culling	4 614
Total cost per herd	14 639
Total cost per cow	87

(Adapted from Holdaway, 1993)

A similar report indicates economic loss in the US is about US\$220/cow/year (Gill et al., 1990), with approximately 75% of all losses to mastitis in US are a result of lost of milk production (Fetrow, 1984; Gill et al., 1990).

In addition the New Zealand seasonal supply dairy farmers are paid on the basis of the amount of milkfat or milkfat and protein produced (Deane, 1992), therefore any change in milk composition due to mastitis will affect farmers income.

2.9 EFFECT OF MASTITIS ON THE MANUFACTURER OF DAIRY PRODUCTS

The survey of literature above indicates that mastitis causes changes in both the concentration and chemical constituents of fat, protein, lactose and minerals of secreted milk. This therefore affects its suitability for manufacturing purposes. Products made from bulk milk with an elevated SCC content have been shown to be inferior to products made from bulk milk with a low SCC. The following are affected:

2.9.1 Whole milk quality

Raw milk

Mastitis infection has been shown to increase the free fatty acids content of milk (Randolph and Erwin, 1974). They found 47% more free fatty acids in milks with WMT greater than 20 compared with WMT less than 10. The increase in free fatty acids content of milk contributes to rancid flavour of raw milk which may render it unsuitable for manufacturing purposes.

Pasteurised milk

Pasteurised milk made from milk with high SCC has been shown to have adverse effect on flavour quality. Janzen (1972) observed significant ($P < 0.01$) negative correlations between SCC and flavour scores of pasteurised milk samples at 1, 7 and 14 days storage at 3-6°C. He also found that these were mainly caused by flavour differences in milk containing $>1\ 000\ 000$ cells/ml. After the pasteurised milk was held at 3-6°C flavour defects ranged from cooked, lacking freshness to rancid and unclean. In a similar study Roger and Mitchell (1989) found that pasteurised milk processed from milk with cell count $<250\ 000$ cells/ml had a significantly ($P < 0.05$) higher organoleptic grade than those processed from milk containing $>500\ 000$ cells/ml and $>1\ 000\ 000$ cells/ml

after 14 days storage at 4°C. The flavour defects were saltiness associated with samples with SCC levels >800 000 cells/ml and uncleanness with samples with SCC levels >500 000 (Rogers and Mitchell, 1989).

2.9.2 Reconstituted milk

The milk compositional changes associated with mastitis infection may affect recombined products quality when such milk is processed. High levels of SCC in milk cause poor heat stability of dried milk produced from such milk (Feagan et al., 1966a). This property is important in determining the quality of recombined evaporated and condensed milk manufactured from milk powder. Milk with poor heat stability coagulates and milk product is unacceptable to the consumer. Kizza and Krus (1970) observed that mastitic milk had heat coagulation time at 140°C of 4.5 minutes compared with 7.8 minutes for normal milk. Spray-dried whole milk produced from milk with high cell count had reduced keeping quality (Abbot et al., 1974), and its solubility index is extremely high (Brus and Jaartsveld, 1971a). They also found that, when compared to skim milk powder manufactured from milk with an SCC of 200 000 cells/ml, skim milk powder manufactured from an SCC of 1 200 000 cells/ml develops a slightly burnt flavour when freshly mixed, changing to a tallowy flavour on storage.

2.9.3 Butter

Butter made from milk with high cell count has impaired taste, less aroma and long churning time (Munro et al., 1984). Kiermeier and Keis (1964a) and cited by Munro et al. (1986) reported that butter made from milk with high SCC content had a lower flavour score than butter made from low SCC content milk. Brus et al (1966) also found that after 2 months storage butter made from high cell count (1-2 millions cells/ml) milk showed signs of oxidation and of inferior quality than normal butter.

2.9.4 Cultured dairy products

High cell count may influence the suitability of milk for use in the manufacture of certain cultured dairy products, such as cheese and yoghurt (Munro et al., 1986, Rogers and Mitchell, 1994).

CHEESE

Altered milk composition due to mastitis, especially decreased casein and fat contents, can affect the cheesemaking properties (Munro et al., 1984; Rogers and Mitchell, 1989). Milk from mastitic cows used in cheesemaking have been shown to have increased rennet clotting time (Ali et al., 1980; Munro et al., 1984; Mitchell et al., 1989); decrease curd firmness and rate of acid formation (Hampton and Randolph, 1969); and a decrease in whey components (Rogers and Mitchell, 1994). Ali et al (1980) found that rennet clotting time(RCT) increased by 50% as the SCC of quarter milk increased from 45 000 to 1 020 000 cells/ml, while Rogers et al. (1986) found that RCT increased by 19% as SCC increased from <500 000 to 500 000 cells/ml in farm bulk milk. Rogers and Mitchell (1994) also reported that RCT increased by approximately 25% when milk containing >500 000 cells/ml was used. The use of high SCC in the manufacture of cheese is also associated with increases in losses of fat and protein in the whey, and increases in moisture content in the final product (Ali et al., 1980; Mitchell et al., 1986; Politis and NG-Kwai-Hang, 1988). Both cheese texture and the flavour are also lowered when milk containing high SCC is used for the manufacture of Cheddar cheese(Ali et al., 1980; Rogers and Mitchell, 1994). The cheeses made from high SCC milk may be softer and more brittle than those cheeses from low SCC milk (Mitchell et al., 1986).

The yield of cheese has also been found to decrease when manufactured from milk with high SCC content (Ali et al, 1980; Mitchell et al, 1986; Rogers and Mitchell, 1994). The use of milk containing SCC >500 000 cells/ml resulted in decrease in cheese yield of about 8.9% when compared to cheese yield

containing SCC <250 000 cells/ml (Rogers and Mitchell, 1994). Also with high SCC milk, cheese yield was at least 1% lower, cheese moisture was about 1% higher, renneting time was up to 50% longer and wheying-off time was higher than with low SCC milk (Mitchell et al., 1985).

It has been suggested that improvement in cheese quality, composition and yield are possible by using milk with a cell count of under 500 000 cells (Politis and NG-Kwai-Hang, 1988; Rogers and Mitchell, 1994) and possibly under 300 000 cells/ml (Politis and NG-Kwai-Hang, 1988).

YOGHURT

There is not much information available on the effects of mastitic milk on the manufacture of yoghurt. Schott (1967) found that yoghurt manufactured from high SCC milk was slightly yellow but otherwise did not differ organoleptically from yoghurt produced from low SCC milk. Rogers and Mitchell (1994) in a recent study also found a conflicting result. In one trial they found that skim milk yoghurt manufactured from milk containing <250 000 cells/ml was organoleptically superior to that manufactured from milk of >500 000 cells/ml. However, in the other trial they found no significant relationship ($P < 0.05$) between milk logSCC and organoleptic grade score of yoghurt.

CONCLUSIONS

High SCC in bulk milk is associated with high incidence of mastitis in individual cows or herds. SCC is therefore widely used as a diagnostic test for mastitis in herds or individual cows and as a parameter to measure milk quality at both farm and factory levels in several countries.

CHAPTER THREE

3.0 MATERIALS AND METHODS

Data used in this study were obtained from the database of the Tui Milk Products Company Limited, situated at Longburn, near Palmerston North. The data contained test records of 1200 farms which supplied bulk milk to the company for the 1992/93 season. The data include records of bulk milk samples of the following:

- somatic cell counts (SCC) (cells/ml)
- bactoscan counts of bacteria numbers (bacteria/ml)
- milk composition concentration - viz fat %, protein %, lactose % and total solids %

The test records were measured every 10 days (3 times per month) throughout the lactation period from August 1992 to May 1993, although not every parameter was always measured on the same sample and same day.

3.1 STATISTICAL ANALYSIS

Mean values were determined for each of the parameters measured, namely SCC, bacteria numbers and milk composition (fat %, protein %, lactose % and total solids %), using the MEANS procedure (Statistical Analysis System, 1985). All statistical analyses were carried out untransformed, because there is considerably lesser effect of total variation in herds bulk milk SCC than in individual cow SCC and therefore logarithmic transformation of SCC data is not necessary (Eberhart et al., 1982; Emmanuel and Persson, 1984). All statistical analyses were carried out using SAS (Statistical Analysis System, 1985) computing package.

A. Correlation analyses were carried out to determine the associations between SCC and bacteria and milk composition as follows:

- correlation between mean bulk milk SCC and bacteria numbers using 4 623 data, with all measurements recorded for each herd, for the same milk sample (data set A).
- correlation between bulk milk SCC and milk composition using 30 120 data, for which all measurements were recorded for each herd within a 10 day period but not necessarily on the same sample of bulk milk (data set B).
- correlation between bacteria numbers and milk composition using 33 800 data, for which all the measurements were recorded for each herd, within a 10 day period but not necessarily on the same sample of bulk milk (data set C).
- correlation between SCC and bacteria numbers, and milk composition for means calculated for each month of the lactation using all data in A, B and C.
- correlation between bacteria numbers and milk composition for means calculated for each of month of the lactation using all data in B.

B. Multiple regression analysis was carried out to determine the relation between bacteria numbers (dependent variable) and SCC and milk composition for early lactation and the whole lactation using General Linear Model (SAS, 1985). Included in this model are:

$$\text{BACT} = \alpha + b_1\text{SCC} + b_2\text{FAT} + b_3\text{PROT} + b_4\text{LACT}$$

where:

BACT = bacteria numbers in bulk milk

α = intercept

$b_1\text{SCC}$ = change in bacteria per unit change of SCC

$b_2\text{FAT}$ = change in bacteria per unit change of fat %

$b_3\text{PROT}$ = change in bacteria per unit change of protein %

$b_4\text{LACT}$ = change in bacteria per unit change of lactose %

CHAPTER FOUR

4.0 RESULTS

The results of the mean values of BMSCC, bacteria numbers and milk composition were obtained. Also the relations between mean bulk milk somatic cell count (BMSCC) and bacteria numbers, and milk composition (Fat %, Protein %, Lactose % and Total solids %) using three sets of data were studied (See methods).

4.1 MEAN VALUES FOR BMSCC AND BACTERIA NUMBERS IN THE FARM BULK MILK IN EACH MONTH

Table 4.1 and fig. 4.1 show the seasonal distribution of the mean BMSCC and bacteria in farm bulk milk supplied to the Company.

The mean BMSCC in farm bulk milk in August was 305 500 cells/ml, then decreased to 221 300 cells/ml in December and increased again to 400 200 cells/ml in May (See table 4.1). In general, the mean BMSCC of farm bulk milk supplied to the Company was high in the early lactation, decreased in mid-lactation and then increased again in late lactation (See fig. 4.1). The overall average BMSCC for the whole lactation period was 280 500 cells/ml.

The seasonal distribution of mean bulk milk bacteria numbers showed a gradual decrease from 144 100 cells/ml in August to 51 700 cells/ml in March, and from there they increased sharply to 162 400 in May (See table 4.1).

Thus, the mean bacteria numbers of the bulk milk were generally elevated in both the early and late parts of the lactation and decreased in mid-lactation. However, there was a very sharp increase in bulk milk bacteria numbers in the late lactation (See fig. 4.1)

Fig. 4.1 Seasonal distribution of the mean bulk milk SCC and bacteria numbers

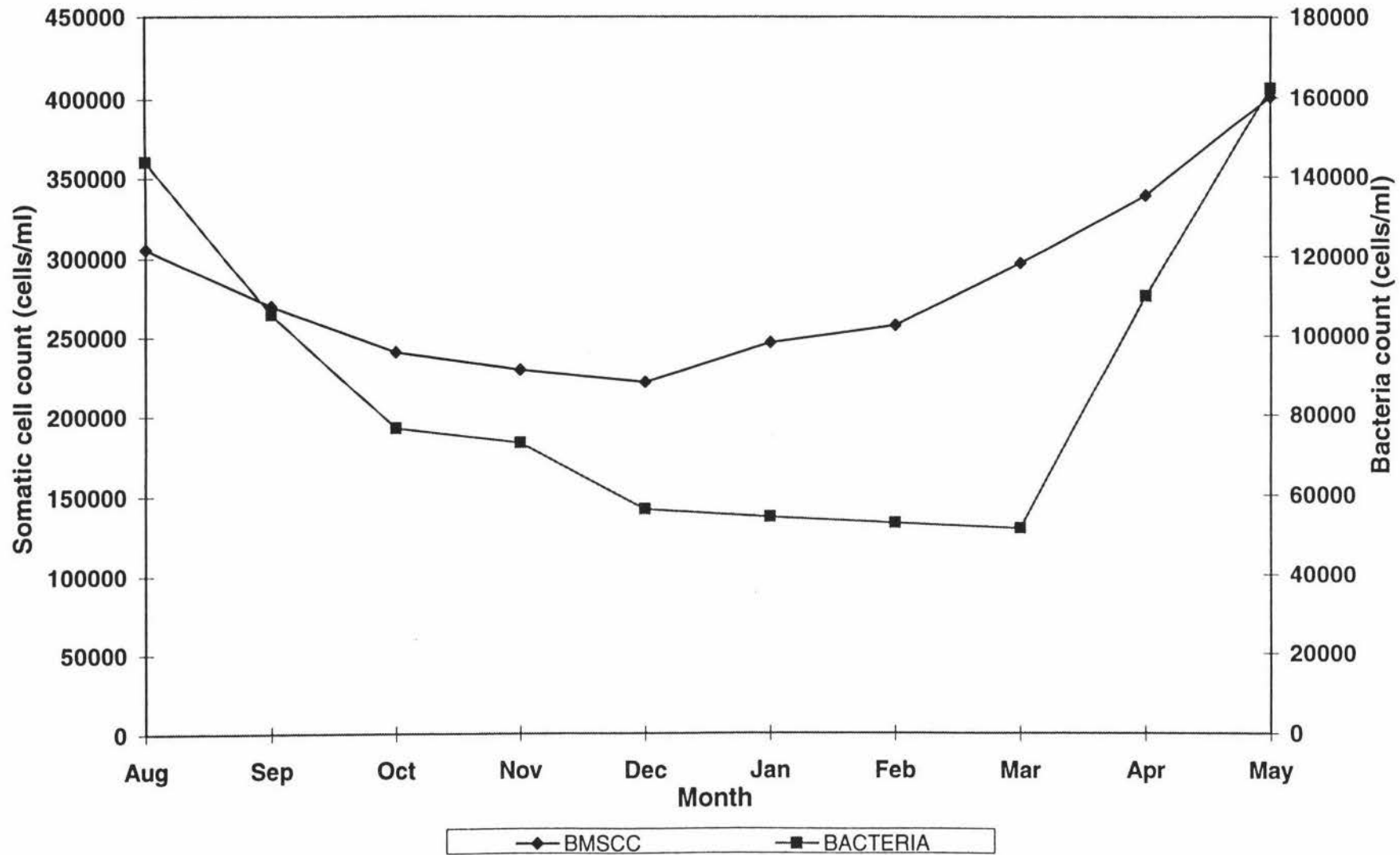


Fig. 4.2 SEASONAL DISTRIBUTION OF FAT, PROTEIN AND LACTOSE PERCENTAGES FOR 1992/93 SEASON

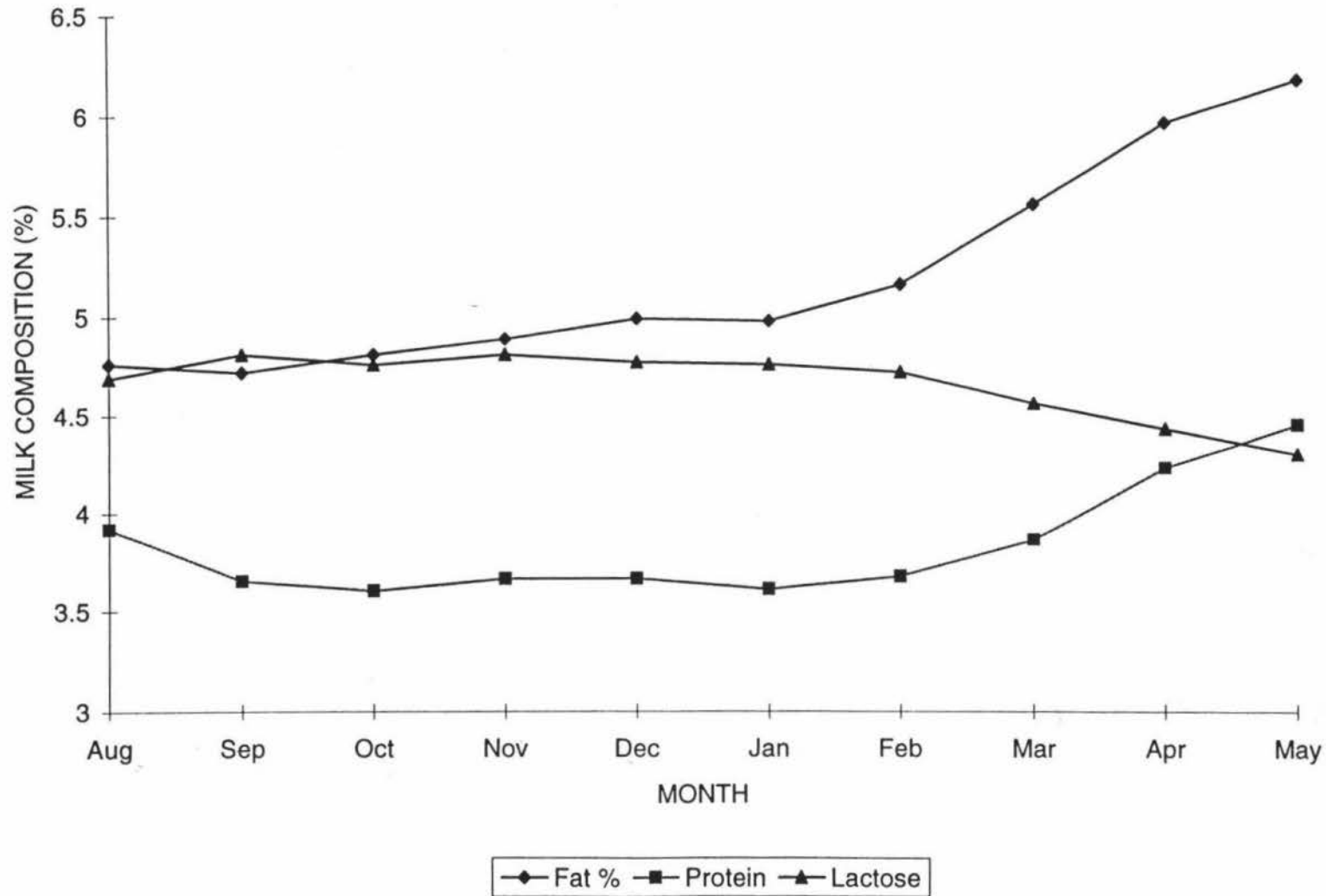


Table 4.1 Seasonal distribution of the mean bulk milk SCC and Bacteria numbers.

Month	Bulk Milk Somatic Cell Counts (cells/ml)	Bacteria count (cells/ml)
August	305 500	144 100
September	269 500	105 800
October	240 800	77 000
November	229 300	73 200
December	221 300	56 600
January	246 200	54 700
February	257 000	53 100
March	296 600	51 700
April	338 300	105 800
May	400 200	162 400

4.2 MEAN VALUES FOR MILK FAT, PROTEIN, LACTOSE AND TOTAL SOLIDS CONCENTRATIONS IN EACH MONTH

Table 4.2 and fig. 4.2 show the seasonal distribution of mean Bulk milk fat, protein, lactose and total solids percentages.

There was a gradual increase in mean bulk milk fat % (4.76 % in August) from the early part of the season to mid-season (4.98 % fat in January), and it then increased sharply to the end of the season (6.18 % fat in May) (See table 4.2 and fig. 4.2). Thus generally there was an increase in bulk milk fat % throughout the season (See fig. 4.2).

The bulk milk mean protein showed a gradual decrease in the early season, and remained fairly constant in the mid-season and then increased gradually in the late season (See table 4.2 and fig. 4.2).

Table 4.2 Distribution of the mean percentages of Bulk milk Fat, Protein, Lactose and Total Solids.

Month	Fat (%)	Protein (%)	Lactose (%)	Total Solids%
August	4.76	3.92	4.69	14.08
September	4.72	3.65	4.81	13.93
October	4.81	3.60	4.76	13.94
November	4.89	3.66	4.81	14.11
December	4.99	3.66	4.77	14.17
January	4.98	3.61	4.76	14.10
February	5.16	3.67	4.72	14.26
March	5.56	3.86	4.56	14.63
April	5.97	4.23	4.43	15.37
May	6.18	4.45	4.30	15.64

There was a slight increase in the mean bulk milk lactose % from the early season to the mid-season, but it showed a gradual decrease towards the end of the season (See table 4.2 and fig. 4.2).

There was no change in the mean bulk milk total solids % in the early season, but it increased gradually from the mid-season to the late season (See table 4.2).

4.3 DISTRIBUTION OF FARMS ACCORDING TO THEIR LACTATION AVERAGE MEAN BULK MILK SCC (BMSCC) VALUES.

Tables 4.3 shows the distribution of farms according to their average bulk milk SCC values.

The data in table 4.3 show that approximately 97 % of all the farms supplied the Company bulk milk with cell count <400 000 cells/ml, while the remainder

(3%) supplied bulk milk with cell count 400-800 000 cells/ml. Also about 85 % of the farms supplied bulk milk with cell count <250 000 cells/ml.

Table 4.3 Distribution of farms according to their average mean bulk milk SCC (cells/ml).

Average SCC values (cells/ml)	Percentage of farms in each category
0-100 000	2.5
101-200 000	39.1
201-300 000	42.0
301-400 000	13.1
401-500 000	2.6
501-600 000	0.7
601-700 000	0.1
701-800 000	0.1

4.4 RELATIONS BETWEEN BMSCC, BACTERIA NUMBERS AND MILK COMPOSITION FOR THE WHOLE LACTATION

Results of the correlation between mean bulk milk SCC and bacteria numbers and milk composition using data sets A and B are presented in table 4.4.

A highly significant, but low, positive correlation ($P < 0.001$) existed between the mean BMSCC and bacteria numbers for data set A. However, no data was obtained for data set B, because not all records were measured on all days (See table 4.4). Similarly, highly significant but low positive correlations ($P < 0.001$) were obtained between BMSCC and fat %, protein % and total solids % for data sets A and B. The correlations were relatively higher in data set B than in data set A. However, a moderate negative but highly significant correlation occurred between BMSCC and lactose % ($r = -0.43$; $P < 0.001$) in both data sets.

Table 4.4 Correlation coefficients between mean bulk milk SCC and bacteria numbers, and milk composition (Fat %, Protein %, Lactose % and Total Solids %) for data sets A and B.

	Data Set A	Data Set B
	SCC	SCC
Bacteria	0.1521***	Not measured on all days
Fat	0.1660***	0.1986***
Prot	0.2486***	0.2765***
Lact	-0.4087***	-0.4481***
TS	0.1424***	0.1638***

P<0.05 * P<0.001 ***

P<0.01 **

Overall, with the exception of lactose %, the correlation between BMSCC and fat %, protein % and total solids % was low.

4.5 RELATIONS BETWEEN BMSCC, BACTERIA NUMBERS AND MILK COMPOSITION FOR EACH MONTH OF THE LACTATION

Table 4.5 shows the correlations between mean BMSCC and bacteria numbers, and milk constituents, calculated for data set A measured in each month.

There were statistically significant low correlations between the mean bulk milk SCC and bacteria numbers in both early and mid-lactations. The correlation coefficients in the early lactation were comparatively higher than in the late lactation. However, the correlations in April and May were not significant ($P>0.05$).

The correlations between BMSCC and fat % in the early lactation were not significant ($P>0.05$), while low but significant correlations occurred between SCC and fat % from mid-lactation to late lactation (See table 4.5).

Table 4.5 Correlation coefficients for each month between the mean BMSCC and Bacteria numbers, Fat %, Protein %, Lactose % and Total Solids % for data set A.

MONTH	SCC vs BACT	SCC vs FAT	SCC vs PROT	SCC vs LACT	SCC vs TS
August	0.2357***	0.0287 NS	0.1249***	-0.1541***	0.0571 NS
September	0.2355***	0.0382 NS	0.1319**	-0.1962***	0.0366 NS
October	0.1494**	0.0769 NS	0.0306 NS	-0.2044 ***	0.0372 NS
November	0.2446***	-0.1274**	-0.0329NS	-0.1083*	-0.1069NS
December	0.0939 NS	-0.1205*	-0.1124NS	-0.1693**	-0.1321*
January	0.1095*	-0.0592NS	-0.0662NS	-0.1452*	-0.0730NS
February	0.1493**	-0.1408***	-0.1516***	-0.3670***	-0.1881***
March	0.1841***	0.0959**	0.1124**	-0.5033***	0.0357*
April	0.0440 NS	0.0904*	0.1206**	-0.3391***	0.0512 NS
May	-0.0755NS	0.1201 NS	0.1417*	-0.3619***	0.0774 NS

P<0.05 * P<0.01 **

P<0.001 *** P>0.05 NS

In general, BMSCC exhibited low but significant correlation with protein % in both early and late parts of the lactation period, while that in mid-lactation was not statistically significant (P>0.05).

Generally, a significant low negative correlation occurred between BMSCC and lactose % throughout the lactation. However, the correlation between BMSCC and lactose % showed a gradual increase from the early part of the lactation to the end of the lactation (See table 4.5).

In general, there was no association between the mean BMSCC and total solids % throughout the season, except in December and February where low significant correlations occurred (See table 4.5).

4.6 RELATIONS BETWEEN MEAN BULK MILK BACTERIA NUMBERS AND MILK COMPOSITION FOR THE WHOLE LACTATION

Table 4.6 shows the correlation coefficients between mean bulk milk bacteria numbers and milk composition for data sets A and C.

Table 4.6 Correlation coefficients between the mean bacteria content and mean milk composition (Fat %, Protein %, Lactose % and Total Solids %) of farm bulk milk for data sets A and C.

	Data Set A	Data Set C
	Bacteria	Bacteria
Fat	-0.0388**	-0.0204***
Protein	0.0243 NS	0.0378***
Lactose	-0.0508***	-0.0769***
Total solids	-0.0179 NS	-0.0140**

P<0.05 * P<0.001 ***

P<0.01 ** P>0.05 NS

Very low but significant negative correlations occurred between mean bulk milk bacteria numbers and fat %, and lactose % ($P<0.01$, and $P<0.001$ respectively) with both data sets. The association between bacteria numbers and protein %, were also low but positive and significant only in Data Set C, while the relations between bacteria numbers and total solids % were both very low and negative

and significant only for Data Set C. Overall, the correlation between bulk milk bacteria numbers and milk composition was low but significant.

4.7 RELATIONS BETWEEN THE MEAN BULK MILK BACTERIA NUMBERS AND FAT %, PROTEIN %, LACTOSE % AND TOTAL SOLIDS % FOR EACH MONTH OF THE LACTATION

Table 4.7 show the correlation coefficients between bulk milk bacteria numbers and each of the parameters of milk composition for each month of the lactation for data set C.

There was generally very low not significant correlation between the mean between bulk milk bacteria numbers and fat % throughout the lactation, except in August and January where low negative significant correlations occurred (See table 4.7). Similarly there was no significant correlation between the mean bulk milk bacteria numbers and protein % throughout the lactation, except in October where a low significant correlation ($r = 0.11$, $P < 0.05$) occurred.

The correlation distribution between the mean bulk milk bacteria numbers and lactose % was low in the early season, and showed no significant correlation ($P > 0.05$) from the mid-season to the late lactation. Overall there was no association between the bulk milk bacteria numbers and each of the parameters of milk composition (Fat %, Protein %, Lactose % and Total solids %) throughout the lactation period (See table 4.7).

Table 4.7 Correlation coefficients in each month between the mean bulk milk Bacteria and Fat %, Protein %, Lactose % and TS % for data set C.

MONTH	BACTERIA vs FAT	BACTERIA vs PROT	BACTERIA vs LACT	BACTERIA vs TS
August	-0.1019**	0.0740 NS	-0.1708***	0.0163 NS
September	0.0621 NS	0.0125 NS	-0.0903*	0.0311 NS
October	0.0492 NS	0.1059 NS	-0.0029 NS	0.0509 NS
November	-0.0481 NS	-0.0649 NS	-0.1051*	-0.0644 NS
December	-0.0073 NS	-0.0144 NS	0.0613 NS	-0.0037 NS
January	-0.1274 **	-0.0955 NS	0.0114 NS	-0.1114*
February	0.0059 NS	0.0031 NS	-0.0032 NS	-0.0106 NS
March	0.0213 NS	0.0019 NS	-0.1396***	-0.0025 NS
April	-0.0147 NS	-0.0036 NS	-0.0002 NS	-0.0123 NS
May	0.0206 NS	-0.0306 NS	-0.0061 NS	-0.0025 NS

P<0.05 * P<0.001 ***

P<0.01 ** P>0.05 NS

4.8 PREDICTION OF THE EFFECT OF BULK MILK SCC, FAT %, PROTEIN % AND LACTOSE % ON BACTERIA COUNT

A model was developed ($Bact = \alpha + b_1SCC + b_2FAT + b_3PROT + b_4LACT$) to predict the effect of SCC, fat %, protein % and lactose % on bacteria numbers in the early lactation and for the whole lactation.

Table 4.8 Prediction model of the effect of bulk milk SCC, Fat %, Protein % and Lactose % on Bacteria count.

A. EARLY LACTATION (AUGUST-NOVEMBER)

$$BACT = \alpha + b_1SCC + b_2FAT + b_3PROT + b_4LACT$$

$$847.17 + 0.0004 + (-35.12) + 86.64 + (-208.37)$$

Coefficient of determination = 0.08 (P<0.001)

B. WHOLE SEASON (AUGUST 1992- MAY 1993)

$$BACT = \alpha + b_1SCC + b_2FAT + b_3PROT + b_4LACT$$

$$37.13 + 0.0002 + (-50.00) + 71.95 + (-5.82)$$

Coefficient of determination = 0.03 (P<0.001)

The model (See table 4.8) show that in the early lactation 8 % of the variation in bacteria count was accounted for by joint variation in SCC and milk composition. For the whole lactation the ability of the model to predict bacteria count was even lower (3 %).

CHAPTER FIVE

5.0 DISCUSSION

5.1 MEAN VALUES FOR BMSCC AND BACTERIA NUMBERS IN THE FARM BULK MILK IN EACH MONTH

The mean values of BMSCC and bacteria numbers were used to determine their distribution throughout the lactation. The results in this study clearly showed that BMSCC in the farm bulk milk was elevated in the early and late parts of the lactation and decreased in the mid-lactation (See fig. 4.1 and table 4.1). This is consistent with the findings of others (Pankey, 1994; Franks, 1994; LIC, 1994) (See fig. 2.1), which indicate that spring calving dairy herds in New Zealand generally show higher BMSCC in early lactation, a decrease in mid-lactation and then an increase in late lactation. The elevation of BMSCC in the early lactation was probably due to undiagnosed mastitis infections in the herds during the dry period, new cases of mastitis that developed during the dry period (Pankey, 1994) or high concentration of somatic cells in colostrum milk (Reichmuth, 1975; Schultz, 1977; Auld et al., 1993).

The increase in BMSCC which occurred toward the end of the lactation was probably associated with decreased dilution effect as milk volumes decline (Schmidt, 1971; Brolund, 1985; Pankey, 1994; Franks, 1994) and also mastitis that develops during the course of the lactation (Schultz, 1977; Pankey, 1994). It can also be attributed to an increase in the proportion of the epithelial cells in the somatic cells from the mammary gland as a result of a long period of stress associated with machine milking (Schultz, 1977).

The overall average BMSCC of the farms for the whole lactation period was approximately 280 000 cells/ml (See table 4.1). This is comparable to the New Zealand Herd Test average of approximately 300 000 cells/ml (LIC, 1994; Franks, 1994; SAMM Plan, 1995), and comparatively lower than the EC

BMSCC standards of <500 000 cells/ml in milk for the manufacture of milk based products (Franks, 1994). This shows that the raw cows milk supplied to the company for the manufacture of milk based products meets international standards. It is also an indication of how effectively the farmers are identifying and treating mastitis as they occur in their herds. However, a small number of the herds of the suppliers had BMSCC >1 000 000 cells/ml, while some had BMSCC <100 000 cells/ml.

The mean bulk milk bacteria numbers also showed a similar trend; high in the early and late parts of the lactation, and low in the mid-lactation (See fig. 4.1 and table 4.1). This seasonal relationship is similar to that reported in New Zealand seasonal supply dairy herds (Williamson et al., 1993; Pankey, 1994). The elevation of bulk milk bacteria numbers in the early lactation was probably mainly due to bacteria contamination from milking machine or poor cooling and storage in the vat (Cousins and Bramley, 1981; Holmes and Wilson, 1987; Bramley, 1992) and in part due to mastitis pathogenic bacteria (Day, 1990; Williamson et al., 1993).

5.2 MEAN VALUES FOR THE BULK MILK FAT%, PROTEIN %, LACTOSE% AND TOTAL SOLIDS % IN EACH MONTH

In a typical New Zealand seasonal supply dairy herds lactation curve, fat and protein concentrations are lowest at the peak of milk production in spring and early summer (early to mid-lactation) and then gradually rise over the remainder of the season (Holmes and Wilson, 1987). The results of the present study show a similar trend of low in the early to mid-lactation and increased towards the end of the lactation (See fig. 4.1). The increase in the fat % and protein % from mid-lactation to the end of the lactation is most likely due to simple concentration effects in a smaller volume of milk produced (Brolund, 1985; Holmes and Wilson, 1987; Williamson et al., 1993; Frank, 1994). The average fat and protein percentages of 5.2 and 3.8 respectively are comparable to that of Friesian-Jersey crossbred cows (5.0 % and 3.8 %

respectively) (LIC, 1994). This suggests that a greater proportion of the cows in the herds of the farms was Friesian-Jersey crossbred cows.

Total solids % also showed a similar trend of a gradual increase from the early lactation to the end of lactation in the present study (See fig. 4.1). This is probably due to the simultaneous increases in both fat % and protein %, major constituents of total solids %, which occurred throughout the lactation.

However, the decrease in lactose concentration which occurred throughout the lactation in the present study, is most likely associated with the decrease in milk volumes produced towards the end of the lactation (Schultz, 1977; Holmes and Wilson, 1987; Williamson et al., 1993; Franks, 1994).

5.3 DISTRIBUTION OF FARMS IN RELATION TO THEIR AVERAGE MEAN BMSCC VALUES

In general, herds with BMSCC <250 000 cells/ml have few problems with mastitis, whereas cell count >500 000 cells/ml are likely to have problems with mastitis (Dohoo and Meek, 1982; Leslie et al., 1984). The results in this study (See table 4.3) show that approximately 85 % of the farms supplied bulk milk with cell count <250 000 cells/ml, and this would suggest that these farms had few problems with mastitis (Dohoo and Meek, 1982; Leslie et al., 1984). However, approximately 1 % of the farms supplied bulk milk with cell count >500 000 cells/ml and these were most likely to have problems with mastitis (Dohoo and Meek, 1982; Leslie et al., 1984). Furthermore, the results also show that approximately 97 % of the farms supplied bulk milk with cell count <400 000 cells/ml and the remainder (3 %) (See table 4.3), supplied bulk milk with cell count 500-800 000 cells/ml. Using Tui Milk Products Company grading system (Tui Milk Products Company, 1995), it would imply that about 97 % of the farms with cell count < 400 000 cells/ml had a finest grade with no monetary penalty, while 3 % of the farms with cell count 500-800 000 cells/ml had grades 1-2 and therefore attracted a monetary penalty.

5.4 RELATIONS BETWEEN BMSCC, BACTERIA NUMBERS AND MILK COMPOSITION FOR THE WHOLE LACTATION

Several studies have shown that elevation of SCC in milk is associated with increase in bacteria numbers (Greer and Pearson, 1973; Pearson et al., 1979; Bennet, 1987; Hogan et al., 1988; Emmanuelsen and Funke, 1991; Godwin and Leslie, 1993). In this study, a highly significant but low positive correlation ($r = 0.15$; $P < 0.001$) was obtained between the mean BMSCC and bacteria numbers for the whole lactation. This correlation coefficient was lower than 0.53 and 0.36 obtained by Pearson et al. (1979) and Bennet (1987) respectively. The high significance observed for the correlation between BMSCC and bacteria numbers may be a reflection of the large number of samples used ($n = 4,600$). This highly significant but low correlation obtained, suggests that the incidence of mastitis and mastitis bacteria were only a small contributor to the high total bacteria numbers.

Although the results obtained in the present study for data sets A and B show similar trends in compositional changes and SCC levels, the strength of these relationships vary (See table 4.4). In this investigation the correlation coefficient between BMSCC and fat % was 0.18 ($P < 0.001$) on the average in both data sets A and B. This was comparatively lower than 0.41 ($P < 0.01$) reported by Mitchell et al. (1986) but relatively higher than 0.06 ($P < 0.01$) reported by Gill and Holmes (1978). Similarly on the average a positive correlation coefficient of 0.26 ($P < 0.01$) was obtained between BMSCC and protein %. This was similar to 0.27 ($P < 0.01$) recorded by Mitchell et al. (1986) but higher than 0.12 ($P < 0.05$) reported by Natkze et al. (1965).

On the average lactose % had a moderate negative correlation coefficient of 0.43 ($P < 0.001$) with BMSCC in both data sets. This falls in the range of -0.31 ($P < 0.05$) to -0.53 ($P < 0.01$) reported by Rogers et al. (1989) and Mitchell et al. (1986). This is also consistent with results cited in the literature (Chapter 2), which indicate a decrease in lactose % with BMSCC due mastitis (Schultz,

1977; Kitchen et al.; 1981; Munro et al.; 1984; Rogers et al., 1989; Holdaway, 1990). The highly significant moderate negative correlation therefore strongly suggests a consistent effect of mastitis on lactose %.

A highly significant but low correlation existed between BMSCC and total solids % ($r = 0.15$, $P < 0.001$) in data sets A and B. This relation was most likely associated with fat % and protein %, which also showed positive correlation with BMSCC (See table 4.4).

Overall, the high significant but low correlation between BMSCC and fat %, protein % and total solids % in data sets A and B, suggest that mastitis was not a major cause of variation in bulk milk composition, except for lactose %, which was more sensitive to the effects of mastitis. Furthermore, the highly significant correlations observed in both data sets is most likely due to the large numbers of farm records involved in this study.

5.5 RELATIONS BETWEEN MEAN BULK MILK BACTERIA NUMBERS AND MILK COMPOSITION FOR THE WHOLE LACTATION

Mastitis infection due to bacteria pathogens causes changes in the concentration of major constituents of milk namely fat %, protein % and lactose % (Schultz, 1977; Kitchen et al., 1981; Hoare, 1982; Munro et al., 1986). On the average the results in the present study showed very weak association between bulk milk bacteria numbers and fat % ($r = -0.03$), protein % ($r = 0.03$), lactose % ($r = -0.06$) and total solids % ($r = 0.02$) in both data sets (See table 4.6). This very weak association between bacteria numbers and milk composition, suggests that total bacteria (mastitis or spoilage) were not major causes of variation in bulk milk composition. Also the high significance observed particularly in data set B, was partly due to the large numbers of records included in the analysis.

5.6 RELATION BETWEEN BMSCC AND BACTERIA NUMBERS AND MILK COMPOSITION FOR EACH MONTH OF THE LACTATION

An analysis was made to determine the association between BMSCC and bacteria numbers and milk composition for each month of the lactation period for data set A (See table 4.5).

Several studies have shown that high BMSCC is a reliable indicator of high mastitis incidence in dairy herds (Ward and Schultz, 1972; Greer and Pearson, 1973; Pearson et al., 1979; Hogan et al., 1988; Emmanuelson and Funke, 1991). The results in the present study showed that the correlation between bacteria numbers and BMSCC was generally low but highly significant in the early lactation ($r = 0.22$, $P < 0.001$) and relatively very low but not significant in the late lactation ($P > 0.05$) (See fig. 4.5). This suggests that higher incidence of mastitis in the herds of the farms was not a major cause of high total bacterial numbers in the bulk milk. The results for early lactation also showed about 5 % variation in bacteria numbers due to variation in BMSCC associated with mastitis. This figure is comparable to 8 % variation in bacteria numbers predicted by the full model in early lactation (See table 4.8). This implies that mastitis bacteria had a small but significant effect on the high total bacteria numbers in early lactation. The elevation of BMSCC in early lactation is most likely due to an increased incidence of mastitis in early lactation (Schultz, 1977; Pankey, 1994). However, in late lactation, the higher BMSCC may be due to the effects of sloughing epithelial cells (Schultz, 1977).

In general, very low correlations also existed between BMSCC and fat %, protein % and total solids % throughout the lactation (See table 4.5). This weak association between BMSCC and fat %, protein % and total solids % throughout the lactation once again confirms the above suggestion that mastitis infection was not an important cause of compositional changes in the bulk milk. Furthermore, the results also suggest that the general increases in fat %, protein % and total solids % observed throughout the lactation in section 5.2

(See fig. 4.1) was typical of New Zealand seasonal supply dairy herds lactation curve (Holmes and Wilson, 1987), and therefore not associated specifically with mastitis infection.

The results of this study showed highly significant but low correlations between lactose % and BMSCC throughout the lactation ($P < 0.001$). The correlation coefficients, however, increased in late lactation. This suggests that mastitis caused a small but significant decrease in lactose % particularly in late lactation, which is a very consistent effect of mastitis reported by several workers (Schultz, 1977; Kitchen et al., 1981; Munro et al., 1984; Mitchell et al., 1986; Rogers et al., 1989; Holdaway, 1990). This also confirms a similar result obtained for the whole lactation for data sets A and B discussed earlier in section 5.4

5.7 RELATIONS BETWEEN THE MEAN BULK MILK BACTERIA NUMBERS AND FAT %, PROTEIN %, LACTOSE % AND TOTAL SOLIDS FOR EACH OF THE MONTH OF THE LACTATION

Correlation analysis was used to determine the effect of bulk milk bacteria numbers on fat %, protein %, lactose % and total solids % for each of month of the lactation for data set C (See table 4.7). The overall results showed very low non-significant correlations ($P > 0.05$) between bulk milk bacteria numbers and fat %, protein % and total solids % throughout the lactation. However, there was a very low significant negative correlation between lactose % and bacteria numbers in early lactation. This suggests that mastitis bacteria did not have significant effect on milk composition, except lactose % which showed a small but significant decrease in early lactation.

CHAPTER SIX

6.0 SUMMARY AND CONCLUSION

The results in this study showed that the overall average of the mean of the farms bulk milk SCC of 280 000 cells/ml is comparable to the New Zealand Herd Test average of approximately 300 000 cells/ml. This is also considerably lower than the upper limit set by the EC (< 500 000 cells/ml.).

Approximately 85 % of the farms supplied bulk milk with cell count <250 000 cells/ml, which suggests that they had low incidence of mastitis infection. However, approximately 1 % of the farms supplied bulk milk with cell count >500 000 cells/ml, suggesting a higher incidence of mastitis infection.

Based on the Tui Milk Product Company's grading system for milk quality standards, about 97 % of the farms supplied bulk milk with cell count <400 000 cells/ml and therefore had 'finest grade' with no monetary penalty, while the remainder (3 %) had cell count >400 000 cells/ml would have grades 1-2 and have attracted monetary penalty.

Both BMSCC and bacteria numbers showed an increase in the early and late parts of lactation, a trend similar that reported in New Zealand seasonal supply dairy herds.

A highly significant but low positive correlation ($r = 0.15$; $r^2 = 0.02$; $P < 0.001$) between the mean BMSCC and bacteria numbers for the whole lactation, would suggest that mastitis had a small but significant effect on total bacterial numbers in bulk milk. This correlation was higher in early lactation ($r = 0.24$; $r^2 = 0.06$; $P < 0.001$), probably due to a higher incidence of mastitis at this time.

The mean percentages of fat, protein and total solids increased from the mid-lactation to the end of the lactation. In contrast, the mean lactose showed a

decrease as the lactation progressed. The average fat and protein percentages of 5.2 and 3.8 respectively are comparable (5.0 % fat and 3.8 % protein) that of Friesian-Jersey crossbreeds. This suggests that a higher proportion of cows in the herds of farms was of Friesian-Jersey crossbreeds.

On the average for the whole lactation, low positive correlations occurred between the mean BMSCC and fat %, ($r = 0.18$), protein % ($r = 0.26$), total solids % ($r = 0.15$). However, a moderate but significant negative correlation occurred between BMSCC and lactose % ($r = -0.43$; $P < 0.001$). The moderate decrease in lactose % in late lactation is a combination of the effect of mastitis and the effect of decreases in milk volumes at the end of lactation.

The conclusions emerging from the present study are as follows:

1. The overall average BMSCC of 280 000 cells/ml obtained in this study suggests that good quality bulk milk was supplied to the company for the manufacture of milk based products, which also meets the EC standards.
2. The significant but low correlation between BMSCC and bacteria numbers, suggests that mastitis bacteria were only a small but significant contributor to the high total bacteria numbers in bulk milk particularly in early lactation, with dirty milking machines or poor cooling being the most probable major contributor.
3. Between 2 and 6 % of the variation observed in bacteria count was accounted for by variation in BMSCC.
4. The very weak association between BMSCC and fat %, protein % and total solids %, and then between bacteria numbers and milk composition, suggests that mastitis was not a major cause of variation in these milk constituents. However, the consistent negative correlation between BMSCC and lactose %, suggests that mastitis did cause decreases in lactose %.

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