SOMATIC CELL COUNTS MASTITIS AND MILK PRODUCTION IN THE PALMERSTON NORTH TOWN MILK SUPPLY HERDS

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ABSTRACT

The study involved work carried out over a period of 12 months from April 1975 to March 1976 with the Town Milk Supply herds of the Manawatu Co-operative Milk Producers, Palmerston North. It could be divided into two parts.

1. Bulk milk samples from all 72 herds of the Town Milk Supply.
2. 19 of the above 72 herds were selected for individual farm visits in May 1975, August 1975, November 1975 and February 1976.

Information on somatic cell counts was obtained from bulk milk samples of the 72 herds; also from the individual cows and farm bulk milk from the 19 herds. Mean cell numbers for the 72 herds was 430,000 cells per ml and the percentages of herds below 250,000; between 250,000 and 500,000 and over 500,000 cells per ml averaged 21, 45.5 and 33.5 and showed wide variation during months. The survey showed that there were marked differences from month to month and the lowest average cell counts were in July 1975 and highest in November and December 1975. On a between herd basis a relationship was demonstrated between milk yield; milk fat yield and total milk protein yield and herd bulk milk cell count ($P < 0.01$). The analysis indicating a loss of .6 litres in milk yield; 0.03 kg in fat yield and 0.02 kg in protein yield per cow per day for each increase of 250,000 cells per ml. Information from the 19 herds showed that the percentage of cows with clinical mastitis were lowest in May (1.9%) and highest in November (2.6%) and this difference was not significant between and within herds.

The relationship between the herd bulk milk cell counts

and the percentage of cows in the herd with less than 250,000 cells per ml, 250,000 to 500,000 cells per ml and above 500,000 cells was described and discussed from the 19 herds and the correlations obtained were -0.91; 0.57 and 0.95 respectively. A relationship between the herd bulk milk cell count and the incidence of clinical mastitis was reported ($P < 0.01$).

Linear regression analysis between the production index and cell counts of individual cows revealed a significant relation ($P < 0.01$) which indicated that a decrease of 0.14 litres of milk per cow per day for each 100,000 cells per ml increase.

Chi square analysis indicated that there was a relationship between age of cows and level of cell counts; an increase in age was associated with an increase in mean somatic cell count of cows.

Daily measurement of bulk milk cell counts for 19 herds for a month in May 1975 and February 1976 showed an average coefficient of variation of 21% and 20% respectively.

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CHAPTER ONE

INTRODUCTION:

The use of somatic cell counting systems for monitoring mastitis status of herds and the udder health of individual cows is being widely accepted in many countries throughout the world. The introduction of new and improved methods now permits greater ease and accuracy in the determination of somatic cell counts. Milk somatic cells are comprised of leucocytes from the blood and epithelial cells from the mammary epithelium.

Quarters that are inflamed can be detected by measurements of the concentration of somatic cells in the milk or by the associated changes in the composition of the milk (e.g. lactose, sodium,et.) but infected quarters can be detected only by bacteriological tests. Although bulk milk somatic cell counts may not necessarily be an accurate indicator of the incidence of infected quarters (Postle et. al., 1971; & Westgarth 1971) the numbers of cells however do indicate the number of damaged udders in the herds (Pearson & Greer, 1974 ; Reichmuth 1975; Reichmuth et. al., 1976). Hence the somatic cell count of a milk sample is a useful index of both udder health and milk quality and most control programmes for abnormal milk are based on the limits of 500,000 cells per ml in milk (International Dairy Federation 1975 Publication on Mastitis Control. No. 85).

The purpose of this study was to monitor the cell counts of the 72 suppliers of the Palmerston North Milk Supply with the view of obtaining information with regards to milk production and the incidence of mastitis under commercial farming conditions.

CHAPTER TWO

REVIEW OF LITERATURE:

2.1 INTRODUCTION:

Inflammation of the milk secreting tissue of the bovine udder, commonly referred to as mastitis, causes a considerable loss to the dairy industry. A large body of survey data and analytical results is available from many countries that substantiate this loss associated with mastitis. Annual losses of 125-150 million krones have been estimated in Denmark (Olsen, 1971), £15-50 million in Britain (Weitz, 1971), $365 - 660 million in the U.S.A. (Dobbins, 1977) and over $16 million in New Zealand (Reichmuth et. al., 1976).

The major losses due to mastitis result from (1) loss in milk production, (2) increased replacement costs, (3) discarded milk, (4) drug costs, (5) veterinary fees, (6) extra labour and (7) loss of genetic potential.

As mastitis is a disease of the udder, the milk from the gland can be used to diagnose the disease. The results of cytological, enzmatic, biochemical and bacteriological analysis of milk will show the responses of the udder to mastitis. The International Dairy Federation (IDF) (Tolle, 1971) have put forward a definition of mastitis on the basis of the presence or absence of one or more abnormalities detected by routine procedures. The categories are:

"1. Normal Udders

are those which show no outward signs of a pathological condition and the milk from which is free from pathogenic organisms and has a normal cell count.

2. Latent Infections are present when the milk shows the presence of pathogenic organisms, but nevertheless has a normal cell count.

3. Subclinical Mastitis shows no macroscopic evidence of inflammation but examination of the milk reveals udder infection, and increased cell count and also alterations in the chemical properties of the milk.

4. Clinical Mastitis:

4.1: Acute Mastitis is present when there are obvious symptoms of inflammation of the udder such as pain and swelling. The milk is macroscopically abnormal and the animals may have a high body temperature.

4.2: Sub-acute Mastitis is present when there are no obvious changes in the udder but when there are persistent clots especially in the foremilk.

5. Non Specific or Aseptic Mastitis is present when there is no recognisable infection and the symptoms may be subclinical or clinical.

Chronic Mastitis occurs when a quarter fails to respond to treatment over a period of time. The quarter may atrophy or show abnormal clinical changes for the rest of the animal's life."

The most important criterion used in the above definitions is the cell count of milk. A threshold value of more than 500,000 cells/ml in milk from single udder quarters is suggested as indicating that the cell count is abnormal and that a diagnosis of mastitis has been established.

Tolle (1971) further elaborates on the definition that "Mastitis is an inflammatory change of the mammary gland which along with physical, chemical and microbiological changes is characterised by an increase of somatic cells, especially leucocytes in the milk and by

pathological changes in the mammary gland."

The IDF definition can be summarised as shown in Table 2.1.

TABLE NO. 2.1: Assessment of Cytological-Bacteriological findings in Mastitis Diagnosis.

CELL COUNT PER ML MILK	PATHOGENIC MICRO-ORGANISMS	
	NOT ISOLATED	ISOLATED
< 500,000	normal secretion	latent infection
> 500,000	non-specific mastitis	mastitis

This definition applies to the examination of quarter milk samples taken at the usual milking time from the foremilk after discarding the first few squirts of milk.

The main drawback of this definition is that udder infection is not defined and this has led to a variety of sampling methods and cultural procedures resulting in considerable confusion in the diagnosis of the disease. In fact, due to the considerable variation in milk sampling techniques one is left wondering if the conclusions on mastitis definition are not conclusions arising from differing sampling methods.

In conclusion, the diagnosis of mastitis in lactating cows is currently based on

1) the clinical examination of the udder and its secretion.
2) the bacteriological examination of milk.
3) the somatic cell count of milk.

2.2. THE CLINICAL DIAGNOSIS OF MASTITIS:

The diagnosis of mastitis by clinical means is the oldest method used to examine udders for mastitis. Schalm et. al., (1971) described a method of physically examining the lactating udder immediately after milking. They reported that thorough evacuation of the udder is necessary since presence of milk reduces the chances of detecting scar tissues and fibrosis. Here comparisons are made mainly between the front quarters with each other and rear quarters with each other. They also suggested that the detection of mastitis by manual palpations is becoming a lost art.

Giesecke & Van Der Heever (1974) cite the work of a number of scientists who regarded clinical examination of the udder to be very useful as it detected not only acute conditions but also permanent udder changes due to chronic mastitis. Narayan & Iya (1954) showed that tissue abnormalities could be detected by deep palpations of the udder. In contrast, Giesecke & Van Der Heever (1974) have reported work of others who recommended clinical diagnosis mainly for acute mastitis and laboratory tests as supplementary methods and the reverse for chronic mastitis.

Doubts have been raised about the efficiency of physical examinations. Giesecke & Van Der Heever (1974) report the work of Heidrich & Renk (1963) which showed that with few exceptions, the physical examination of the udder is inadequate for diagnosis of mastitis because quarters with no induration may be bacteriologically positive. Giesecke & Van Der Heever (1974) noted that discrepancies between chronic tissue alterations and the presence or absence of inflammatory changes in the udder seem to result from the fact that in some indurated udders epithelial lesions persist whereas in others they have healed. An incorrect positive diagnosis of mastitis may be made in udders in which inflammation has subsided and the quarters may be indurated rather

than mastitic.

King (1974) using clinical examination by external palpation showed that quarters with distinct or marked fibrosis gave lower yields. The author stated that the effect on yield was due to udder damage.

The strip cup represents another form of clinical examination in which the foremilk is examined using a wire mesh screen or a black plate placed within the cup and the presence of small flakes and milk clots suggest the presence of mastitis. Neave et. al.,(1954) reported that 12 - 13% of quarters showing clots in milk were not infected with the more common pathogenic organisms and hence its reliability as an indication of infection is questionable. However Giesecke & Van Der Heever (1974) report Brown & Bryan (1950) and Merchant & Packer's (1952) work that negative tests do not indicate absence of infection. This therefore indicates that there may be instances where clots occur in milk without actual inflammation.

From this discussion, clinical examination of the udder can be considered to be a very subjective technique depending on the experience and judgement of the examiner. However when considering individual farm conditions and daily examination of milk from individual cows by the farmer, it is probably the only practicable method for diagnosing mastitis.

In the diagnosing of mastitis it is important to alert the dairy farmer of impending clinical problems before they become fully acute clinical cases and as such clinical cases develop from sub clinical ones, more importance should be paid in the diagnosing of mastitis at sub clinical level. Bacterial assessment and somatic cell counting are the two main daignostic criteria that are used for the assessment of

sub clinical mastitis.

2.3 BACTERIOLOGICAL DIAGNOSIS:

Milk freshly drawn from a quarter which is infected contains micro-organisms which may have entered the milk either during milk or during the intervals between milking (Hammer & Babel, 1957 and Schalm et. al, 1971) As intrammary microbial infection is one of the major causes of mastitis in dairy cattle it can be diagnosed by the recovery of causative pathogens from milk samples, which is done by using culturing techniques to identify the different bacteria. The National Mastitis Council Publication (Brown et. al., 1969); the International Dairy Federation publication (Tolle, 1971) and Schalm et. al. (1971) describe some of the bacteriological methods that are commonly used.

Bacteriological methods are advocated for diagnostic purposes because 90% of mastitis of economic importance is due to the main pathogens, Staphylococcus aureus, Streptococcus agalactiae, Streptococcus dysgalactiae and Streptococcus uberis, (Neave, 1975); and the most common methods detect these bacteria only. These bacteria have been called 'primary mastitis pathogens'. and the intramammary infections which these organisms are responsible for as 'primary infections'. The other bacteria such as coagulase-negative staphylococcus (e.g. Staphylococcus epidermis), micrococci and corynebacterium have been referred to as 'secondary infections'. As the terms 'primary' and'secondary' could imply sequence rather than severity of infection of economic importance, these are gradually being replaced by the terms 'major' and 'minor' respectively (Griffin et. al.,1977).

A Danish investigation of 2 years(Klastrup, 1975) in contrast to Neave's (1975) report that 90% of the mastitis is caused by the primary pathogens, showed that the primary pathogens were present in about 57% of the affected quarters. Pearson & Greer (1974)observed in their

study that 80% of the pathogens were primary and 15% were secondary. In other words, the principal criteria to be satisfied of bacteriological examination seems not to be fulfilled in all environmental circumstances. Also different sampling techniques could be responsible for different results. In New Zealand, staphylococci and the streptococci have been reported to be more commonly associated with abnormal udders, Rapid Mastitis Test positivity and high somatic cell count than other bacteria, (Elliot, et. al., 1976).

The aim in bacteriological assessment is to obtain a milk sample free of contamination by extraneous organisms. Precautions must be taken when milk samples are drawn so that bacteria do not enter the milk from extraneous source and render it unsuitable for diagnostic purposes. A reliable method for determing whether pathogens are derived from an intramammary source is by sampling through the teat wall by puncture (Neave, 1975). However on commercial herds this method is not very practical.

The presence of micro-organisms does not necessarily indicate an infection of the udder tissue. The teat orifice, the skin and the teat canal may all yeild organisms that are not necessarily associated with any overt inflammatory process and hence do not necessarily indicate their presence in the cistern or udder tissue (Munch-Peterson, 1971). Earlier Newbould & Neave (1965) had demonstrated that the presence of organisms in milk might not indicate an infection of intramammary source unless the milk sample also contained a significantly higher number of cells than that found in a normal quarter of a cow. From this it should be recognised that the recovery of mastitis pathogens or any other micro-organisms from milk taken aseptically does not necessarily indicate that they are of intramammary origin. This has led to confusion about bacteriological diagnosis and efforts to develop a working definition of infection.

At the International Dairy Federation Seminar on Mastitis Control 1975, a system based on bacteriological tests was proposed by Neave, (1975). He showed that when the teat duct is colonised many foremilk samples will be heavily contaminated, however when the teat ends are scrubbed for 15 - 20 seconds with ethanol (70 - 80%), 76-98% of the samples showed no evidence of contamination by bacteria from the teat canal in 0.05 mls of plated milk. Neave further points out that if an efficient teat dip is used regularly it will halve the number of samples yielding pathogens from uninfected quarters; where a teat dip is not regularly used it is advisable to introduce one for at least a week before taking any foremilk samples. However these procedures do not completely overcome the problem of teat canal infections, since dipping solutions do not penetrate the whole length of the teat canal (Davis, 1935). Giesecke and Van der Heever,(1974) report the work of Birkner,(1964) and Hendrich,et. al., (1964) in which the bacterial content of samples collected aseptically via the teat canal was compared with that of gland cistern samples taken via canulae; it was found that 100% of the teat samples, but only 6.5% of the cistern samples showed bacterial growth. Giesecke,et. al., (1968) similarly showed significant differences between bacteriological findings in sample drawn aseptically via the teat canal and those drawn directly from the gland cistern. From these and recent findings of Black, et. al., (1972) it can be seen that presence of bacteria from aseptically collected samples via teat canal may more frequently be associated with infection of the teat canal rather than udder infections.

The diagnosis of teat canal infections is not yet satisfactorily achieved although research in South Africa by Giesecke,(1975) suggests a method of solving the problem using a monovalent radial immunodiffusion test based on the immunodiffusion of bovine serum albumin in agarose containing antibovine albumin serum.

Neave, (1975) reported that his system of mastitis diagnosis confirmation of infection required isolation of the primary pathogens from 2 consecutive samples taken a week apart. More recently this criterion has been modified by Griffin, et. al.(1977) who proposed that quarters should be considered infected if the major pathogens were found in 2 out of 3 samples. Quarters diagnosed as infected by this method usually give rise to a marked increase (>10 fold) in somatic cells indicating inflammation (Neave, 1975). However cell counts fluctuate quite widely at each sampling period and Neave does not take them into consideration in the diagnosis of mastitis. Giesecke & Viljoen, (1974) report that infections of the teat duct can result in a leucocytosis in the milk and because of this about half the udder quarters classed as infected by I.D.F. (Tolle, 1971) definitions are in fact teat duct infections. This implies that a quarter with a teat canal infection which does not cause a subsequent increase in cell count, would be classified as an abnormal quarter under the I.D.F. definition. However Giesecke (1975) considers such conditions as 'irrelevant' teat canal infections.

The suitability of the I.D.F.'s own definition (Tolle, 1971) for measuring intrammary infections and assessing levels of infection has been questioned by Neave,(1975), since infected quarters can pass through the definitions 'sub clinical', 'latent', 'clinical', and even 'non-specific' in the course of a few weeks. From this definition, quarters that do not show outward signs of a pathological condition, whose milk is free from pathogens, or having a cell count 500,000 cells/ml are considered normal..

In the I.D.F. Bulletin document 85, Griffin, (1975) reported the results showing the repeatability of different methods measured over

12 weeks. When using bacteriological methods alone the repeatability for quarters assessed as infected was 89%, for quarters assessed as non-infected 96%, and for all quarters 95%. Using a cell count threshold of 800,000 cells/ml the repeatability of quarters above threshold was 73%, for quarters below threshold 87% and for all quarters 85%. When the cell count threshold was reduced to 500,000 cells/ml then the respective repeatability values were 77%, 83% and 81%. Repeatability values using the I.D.F. definition (Tolle, 1971) were as follows: - Quarters showing normal reaction 74%, 'non-specific mastitis' 41%,'latent infection' 20%, 'mastitis' 80% and for all quarters 78%. The data mentioned show that bacterial methods alone gave results that are least likely to result in false conclusions and cell counts. Threshold method is better than the I.D.F. method which combines both cell count and bacteriological methods.

A number of workers (Philpot, 1969; Kingwell et. al.(1970)Ward and Schultz 1972 and Natzke, et. al.1972) depend mainly on two consecutive samples to be bacteriologically positive as evidence of infection, but despite their elaborate methods of preparing teats prior to sampling they may inevitably be recording streak canal infections as infections of the udder. Pearson, et. al.,(1971) and (1974) suggest that as in the definition of the I.D.F. (Tolle, 1971) that it is perhaps preferable to discard the first 10 to 15 mls of foremilk and use as the testing sample the milk which follows. The reason for this according to them is to flush out the streak canal bacteria and hence reduce the number of teat canal infections that are recorded as udder infections. Pearson,et. al., (1974) based their definition of infection on California Mastitis Test reactions of 2 or greater on single samples combined with bacterial isolations of at least 26 colonies per 0.05 mls of milk. The sub-clinical form of the mastitis according to Pearson, (1975) involves critically the inclusion of strong cellular reactions using the California Mastitis

Test but bacterial isolation on its own is not considered to be adequate.

Whilst the primary pathogens are commonly regarded as pathogenic, there is some suspicion that the secondary pathogens such as coagulase negative micrococci and coynebacterium bovis may be involved in some cases of mastitis (Blackburn, 1969; Holmberg, 1973; Bramley, 1975 and Eberhart 1975). Tj de Vries (1976) also points out some communications, most of them originating from veterinarians that state special cases of mastitis could be caused by corynebacteria. Bramley (1975) reports that these organisms are capable of producing a mild degree of udder irritation, which is evidenced by slight leucocytosis and also probable reduction in milk yield. Overall little is known of the secondary pathogens yet their significance to mastitis should not be disregarded as Neave's (1975) report has done. In a recent publication (Reichmuth et. al. 1976) noted that the presence or absence of organisms in the milk sample conveys little information as the presence of pathogens which do not overcome the protective mechanisms of the udder will not necessarily indicate a disease.

In conclusion, some of the differences that are associated with bacteriological assessmentare due to the variability in the methods of sampling and many workers take more account of bacterial isolations than they do of the cytological changes. However, Neave (1975) criteria does offer an approach but no mention is given to teat canal infection which may play an important part where bacterial isolation may be streak canal infection and not associated with udder damage. Probably one of the biggest limitations of bacteriological diagnosis is that it has the ability only to discover the more specific mastitis pathogens like the primary pathogens, but other organisms have been shown to be responsible for mastitis and some are increasingly becoming important.

2.4 SOMATIC CELL COUNTING:

2.4.1 TYPE AND OCCURRENCE OF SOMATIC CELLS IN MILK:

The number of somatic cells in milk is widely used as a basis for defining milk abnormality both in individual cows samples and in bulk milk. The functional state and health of the mammary gland from which the milk is drained can be measured by its cell content (Tolle et. al.,1971).

The two types of cells present in milk are the epithelial cells from the udder and leucocytes from the blood (Zlotnick, 1947).

Epithelial cells are derived from the mammary duct and ascini as a result of normal breakdown and repair during milk secretion or tissue injury (Schalm et. al., 1971) and also probably due to (premature) regression of the udder (Giesecke & Van der Heever 1974). Epithelial cells generally increase in late lactation due to regression of the gland or as a consequence of injury due to mastitis (Wright, 1977 and Schultz, 1977). Estimates of the percentage of cells which are of epithelial origin in a milk sample are highly variable, being in the range of 35 - 70% in most reports (Schultz, 1977). Normal milk has reported levels of 65 - 70%, chronic mastitis milk approximately 50%, and more severely mastitic milk lower levels (10-45%) due to dilution with leucocytes from the blood (Schalm et. al., 1971 and Schultz, 1977).

Leucocytes are of 5 distinct morphologic types, (i) basophils, (ii) eosinophils, (iii) monocytes (iv) and neutrophil polymorphonuclear (PMN) leucocytes all of which function as phagocytes and protect the host from infectious diseases (Merchnikoff,1905) and (v) lymphocytes that are nonphagocytes and comprise B & T cells that function in humoral and cell mediated immunity (Paape & Wergin, 1977). Cullen (1966) reports that lymphocytes and monocytes increase considerably more with inflammation than do PMN - leucocytes and are characteristic of more chronic

lesions. However in the lactating gland inflammation is unique in that products of inflammation enter the milk and are readily available for study. Of importance here to inflammation of the udder is generally a significant increase in the leucocytes of which neutrophil polymorphonuclear (PMN) leucocyte is predominant (Schalm et. al.,1971). In this regard Schalm et. al.,(1971) consider milk to be normal even with relatively high cell counts provided the PMN - leucocyte remain below 12 per cent. Further Schalm et. al.,(1971) suggests that the term leucocyte count should be confined to the number of PMN-leucocyte only and for convenience all other cell types should be referred to as mononuclear cells.

Waite & Blackburn (1957) have noted a close relationship between total cell count and the number of PMN-leucocytes i.e. polymorph count = 0.75 x total cell count - 74,000 and Reichmuth (1975) considers this correlation essential for the interpretation of the total cell count as a signal of inflammation.

One of the basic host responses to injury or bacterial infection is the release of chemotatic factors that draw the PMN leucocyte from the blood into the area of injury. This is accomplished through the process of diapedesis and chemotaxis (Schalm, 1970 and Schalm et. al.,1971). Complement components such as C3a, C5a, and C567, serum factors and bacteria or their toxins, acting singly or in complexes functions as chemotatic agents (Schalm, 1977). Once the process of PMN leucocyte emigration is set in motion in mastitis, it continues long after the irritant has been neutralised or removed. Jain et. al.,(1972) noted that it is possible that leucocytes could themselves participate in an autocatalytic fashion in perpetuating leucocyte emigration into the milk. It is postulated that inhibators of inflammation eventually come into play, leading to recovery (Schalm, 1977). The extent of tissue injury is important as it determines the

time it would take for the animal to resume normal secretion of milk. This inflammatory response of leucocytes is an attempt on the part of the gland to overcome the attack by mirco-organisms and if successful will cause a return to normal. If it fails there will be a reduction in the performance of the secreting tissue with a resulstant fall in milk yield, accompanied by changes in composition.

2.4.2 INTERPRETATION OF SOMATIC CELL COUNT:

In the interpretation of somatic cell count, it is important to know the origin of milk samples and the factors which may effect numbers of somatic cell levels in a milk sample.

1. ORIGIN OF SAMPLES For carrying out somatic cell counts in milk, samples may originate from the following sources.

(i) Quarter Samples:- Here foremilk samples are usually used though occasionally whole quarter milk samples are taken. Reitchmuth (1975 & 1976) reports a correlation of 0.86 between total and foremilk counts for quarter milk samples. This indicates that the additional effort in collecting samples representing the whole milk from the quarter only increases the reliability of the information by 14%. The IDF definition presented earlier makes use of foremilk samples to indicate inflammation (Table 2.1).

(ii) Composite Udder or Cow Samples:- These are pooled or composite quarter samples that are collected in the course of milking. A convenient way to identify these cows is to determine the somatic cell count by the system of herd testing that is used for individual cows.

From this it is realised that cow samples can be easily obtained and that cell counts can be used to detect the worst cows in the herds and depict the mastitis status of the herd. If the samples are taken on a number of occasions through a lactation, greater precision is obtained.

(iii) Bulk Milk Samples:- Cell Counts in bulk milk are being used by regulatory agencies as an indicator of milk quality and to monitor the mastitis status in herds. The bulk milk of the herd is the economic output that is sent to the dairy plant and it can be used as a diagnostic criteria representing the whole herd. However in small herds, one high cow can have a disproportionate effect on the cell count in bulk milk and in large herds the milk of several bad cows can be diluted out to acceptable levels in the bulk tank. Hoare,(1976) showed in a 30 day trial where 3 cows with subclinical mastitis produced 51% of the total cells in the bulk milk from a 20 cow herd. The use of geometric means in such a condition is generally recommended. However Reichmuth, (1975) in his analysis of 21,000 herds where less than 4% contained more than 15 cows showed that the Arithmetic mean was only 4.3% higher than the Geometric mean. Booth, (1975) too, reports that majority of herds according to his experience had arithmetic mean which was within 5% of geometric mean. So working with arithmetic mean is not always wrong for bulk milk but much depends on the rate at which individual cows with extreme cell counts contribute to the bulk milk cell count.

2. FACTORS INFLUENCING SOMATIC CELL COUNTS:

It is logical to expect variation in cell counts from day to day or milking to milking because the lactating mammary gland is a very active metabolic organ. Possibly the greatest influence on

cow composite and bulk milk cell count is the type, stage and degree of infection. The response to infection is generally a significant increase in the leucocytes and a smaller parallel increase in epithelial cells (Schalm et. al., 1971).

Natzke et. al.,(1972) using the presence of bacteria in two consecutive samples to indicate infection showed that the average cell count of cow composite milk samples to be 214,000/ml for cows uninfected at the time of sampling and for each infected quarter there was an approximate doubling of cell counts of composite milk. (See Table 2.2.)

TABLE 2.2: Average somatic cell counts for composite milk samples in relation to the numbers of quarters infected.

(Natzke et.al, 1972).

No. of quarters infected	Cell Count (10^3 cells/ml)
0	214
1	507
2	701
3	1470

Earlier the International Dairy Federation meeting of 1966 in Munich (Kastli, 1967) resulted in 14 countries proposing that fore-milk samples collected at normal milking and in mid-lactation should be less than 300,000 cells/ml and 5 countries proposing 300,000 - 500,000 cells/ml of milk as being normal. Finally a threshold value of more than 500,0000 cells/ml was accepted by the IDF indicating abnormal milk.

Ward & Schultz (1972) in a study involving 874 quarters with a quarter milker and sampled for type of bacteria. reported the cell counts of milk (See Table 2.3).

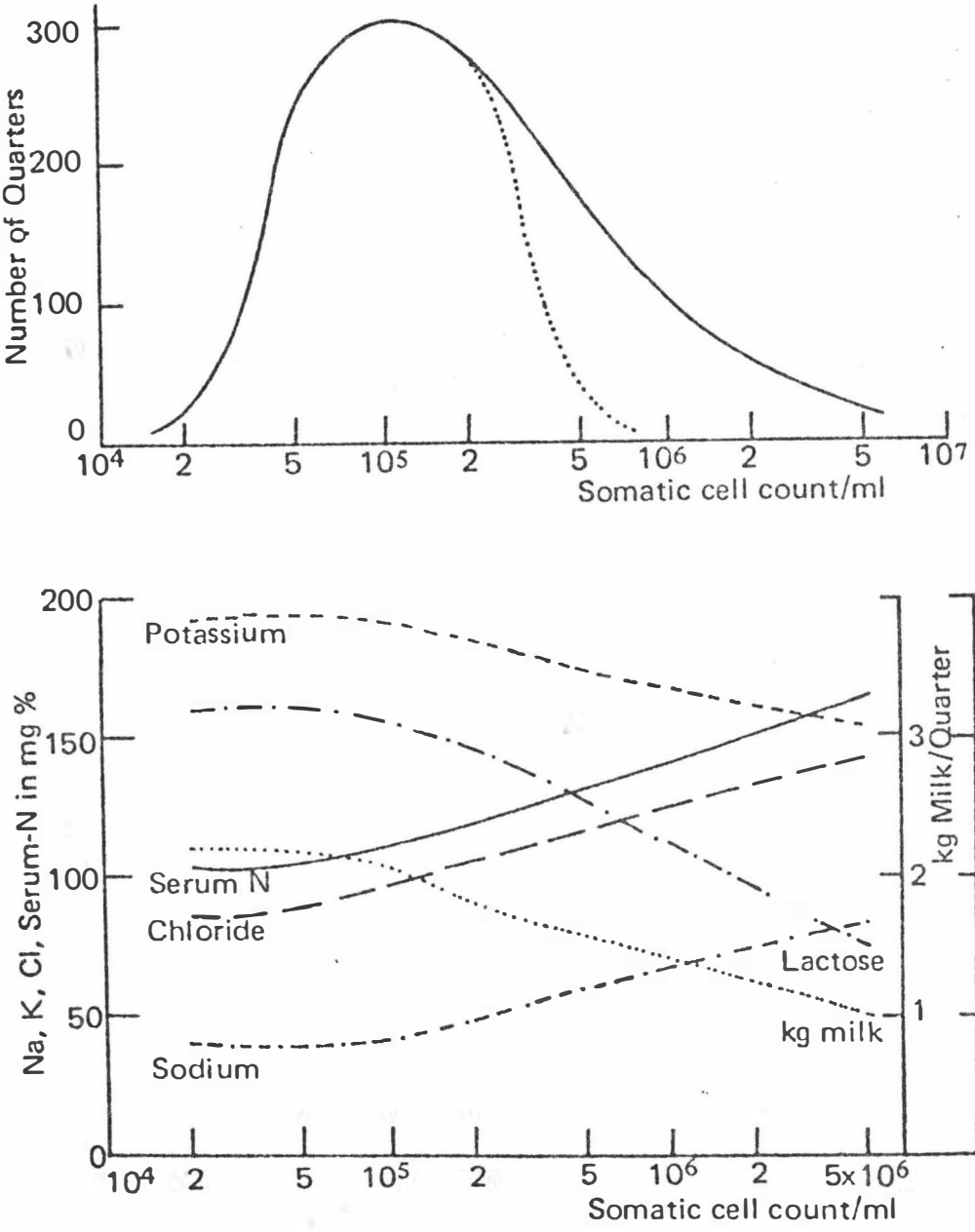
TABLE 2.3: Somatic Cells in milk from quarters with various types of bacteria. (Ward & Schultz 1972).

Bacteria	Cell Count (cells/ml)
None	310,000
Streptococcus agalactiae	900,000
Streptococcus uberis	1630,000
Staphylococcus aureus	1500,000
Coagulase negative staphylococcus	1430,000
Micrococci	360,000
None (previous clinical mastitis)	600,000

They further reported that there was a marked increase in cell response in quarters with a previous history of clinical mastitis. In fact for those quarters with no organisms, mean cell count was 600,000 cells/ml if there had been a prior occurrence of clinical mastitis. From this data it is concluded that pathogens cause the greatest cell response but there is variation in the response to the different pathogens and that previous mastitis infections although cleared, can cause cell counts to be maintained at high levels. Though different bacteria can cause differences in cell counts these authors did report a decrease in milk yield with elevated cell counts thus indicating abnormality in the quarters.

Heeshen, (1975) and Reichmuth (1975) using discriminatory analysis selected six parameters; sodium, potassium, chloride, lactose, whey - N and quarter yield which together with cell count they formed a seven dimensional system. They showed that up to a cell count of 100,000 cells/ml all parameters move in parallel but from 100,000 to 150,000 cells/ml the slopes of these parameters change. The curves

FIG. 2.1: The cell count in relation to different milk components and quantity of milk delivered (n = 2636 quarter milk samples). Heeschen (1975).



for sodium, whey-N and chloride rise while quarter milk yield, potassium and lactose fall.

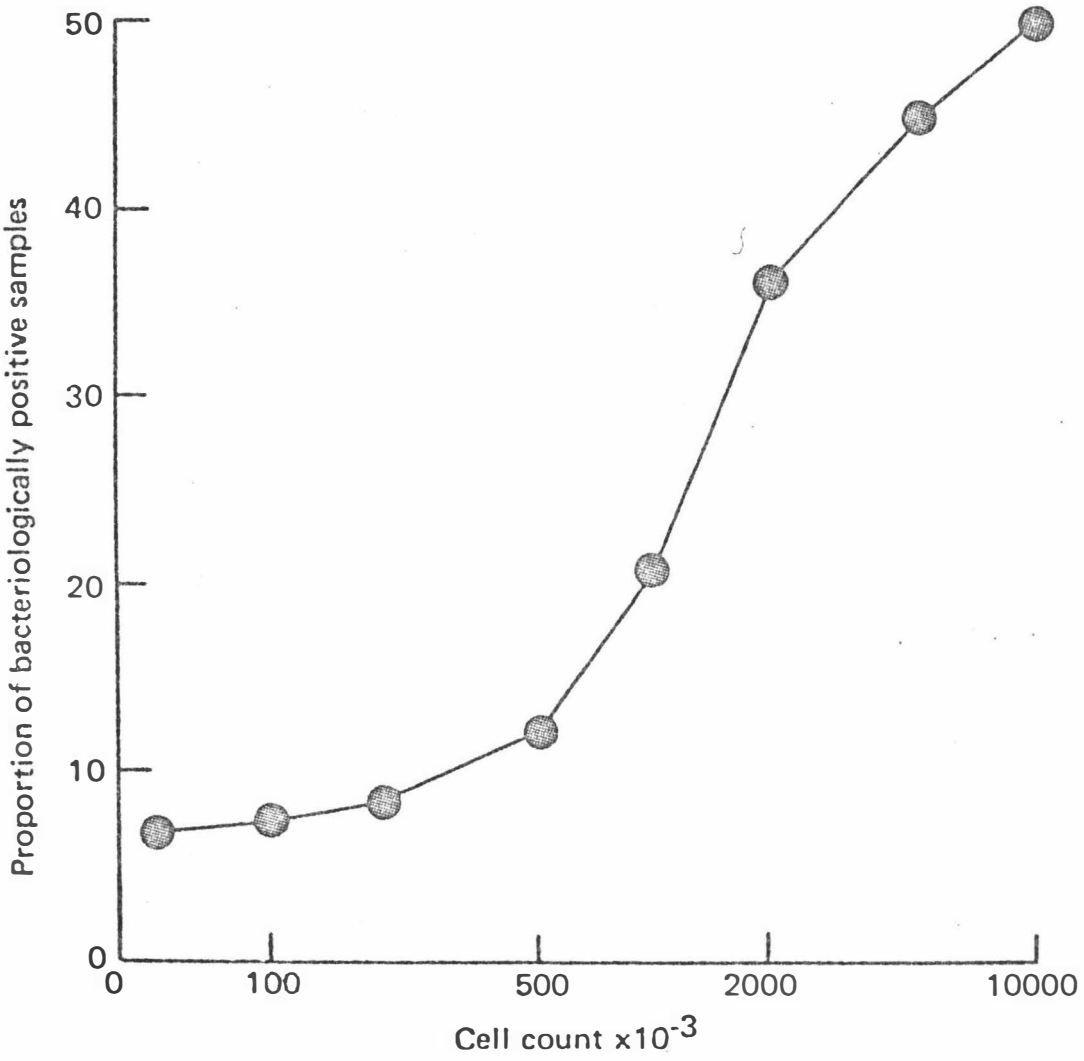
If the rising line of frequency distribution of cell counts (Fig 2.1) is assumed to be an approximation of a gausian bell-shaped curve with its mode at about 150,000 cells/ml there is a two fold standard deviation ranging to about 500,000 cells/ml of foremilk. Based on this data, the definition of mastitis according to Reichmuth (1976) should be the condition of the mammary gland such that "there has been an evocation of the cytological defence mechanism with a consequent rise in somatic cell level to above 150,000 cells/ml."

Linzel,(1976) reported that quarters that had high cell counts and repeatedly shed bacteria had lower lactose and potassium but higher sodium and chloride concentrations than the unaffected quarters of the same cows.

Reichmuth (1975) has also reported the probability of bacteriological detection by quantitative cytological examination (see Fig. 2.2). Above a cell count of 200,000 cells/ml the probability increases.

The correlation between somatic cell count and the incidence of mastitis in herds have varied from figures of 0.52 (Westgarth 1971), 0.50 (Postle et. al., 1971) to 0.88 and 0.96 (Pearson et. al., 1971). This wide difference primarily due to different definitions of mastitis. Thus Westgarh (1971) and Postle et. al., (1971) based their definition of infection on bacterial isolation and with no reference to cellular components whereas Pearson et. al., (1971) based their results only on cells and cellular reactions with no reference to bacteriological detection. Pearson & Greer (1974) using different combinations of cellular reactions and degrees of bacterial isolation from milking quarters reported that

FIG. 2.2: The proportion of bacteriological positive foremilk samples (*streptococcus agalactiae*, *streptococcus dysgalactiae*, *streptococcus uberis*, coagulase + staphylococci) with regard to the cell count (10010 quarter milk samples). Reichmuth (1975).



correlation of 0.71 to 0.88 between herd somatic cell counts and the incidence of mastitis.

The relationship between milk yield and cell count have been well documented by a number of workers (Forster,1964; Daniel & Fielden 1972; Pearson 1971; Ward and Schultz,1972 and Sendelbach et. al., 1976), showing that increased somatic cell count is associated with decreased milk yield.

It is apparent from this discussion that there is a general lack of understanding and agreement about conclusions to be drawn from cell counts. Nevertheless there is substantial data to indicate that regardless of whether or not high cell counts are caused by infective organisms or not they are associated with reduced milk yield. This together with changes in milk composition indicates that there is some disturbance in the milk secretory tissue; cell numbers in milk should therefore be considered as a measure of the integrity of secretory tissue. For secretory activity mastitis is probably the most important factor causing disturbances in the secretory tissue..

LACTATIONAL FACTORS:

(i) Stage of Lactation

It is generally agreed that the somatic cell count is high in colostrum but soon decreases and remains low for the first few weeks after which it gradually increases until the end of lactation (Cullen, 1966; Schalm et, al., 1971), MacLeod and Anderson (1950) showed that in healthy cows the cell counts dropped from 1,000,000 cells/ml to 70,000 cells/ml in the first three weeks of lactation. The average cell count according to Waite & Blackburn (1975) was lowest and least variable from day 70 to day 130 of lactation. The reported increase in cell counts in the beginning of lactation is due to the presence of large amounts of epithelial cells and PMN (Cullen 1966); Table 2.4.

TABLE 2.4: Cell Counts in Normal Milk (millions/ml) (Cullen 1966)

Stage of Lactation	Epithelial Cells	Polymorphs	Lymphocytes	No. of Samples
Colostrum (0.5 days)	1.19	2.28	0.72	44
Early (5 days-8 wks)	0.14	0.14	0.09	40
Mid	0.12	0.10	0.05	84
Late last 4 wks before drying	.85	.28	.59	36

Blackburn (1966) reported that the increase in cell count at the end of lactation is probably due to the shedding of epithelial cells from the alveoli into the milk during the course of involution of the udder. This together with the decreasing volume of milk towards the end of lactation results in the exaggeration in cell numbers in the smaller volume of milk (Schalm et. al., 1971).

Natzke et. al.,(1972) using the presence of bacteria in two consecutive samples as indicator of infection showed that in late lactation uninfected quarters did not necessarily show elevated cell counts in composite udder milk. This confirmed the suggestion of Schalm & Lasmanis (1968) that the increase cell counts at drying off was the result of new infections.

In a herd situation, as colostrum is rarely included in the supply it should not affect the cell counts of either the bulk milk samples or individual cow samples. The reported trend in increase cell counts with advancing lactation is probably due to an increase in new infection rate through lactation. Hence a sudden rise in cell numbers may be attributed to a new infection.

(ii) Age or Lactation Number

It is well documented in literature that the average cell count increases as the lactational age of the animal increases (Van Resburg, 1947; Blackburn, 1966, Cullen 1966; and Blackburn, 1968).

This is probably a reflection of the past infection history of the udder since Natzke et. al.(1972) found that uninfected animals showed no upward trend over five lactations. Earlier Blackburn (1966) over a seven year period found that the increase in cells from one lactation to the next was due mainly to an increase in PMN leucocytes. This increase was associated with bacterial infection resulting in inflammation of the ducts and also an increase in the severity of lobular lesions.

Tolle in the IDF Bulletin (1971) reports that there is a decrease in cell counts averages in cows older than six years. This may be interpreted to selection in terms of udder health with advancing age. From this discussion it appears that the increase in cell count with age is mainly due to PMN leucocytes and past infections play an important role.

(iii) Diurnal Variation and Milking Interval

Considerable variations in cell counts have been observed between morning and evening milk samples particularly when irregular milking intervals are in effect (Cullen, 1967). These diurnal variations were found at both high and low somatic cell counts, the greatest variation occurring when the counts were greater than one million. White and Rattray (1965) also observed that high cell count cows had the greatest variation.

Schalm et. al.,(1971) are of the opinion that the diurnal variation in cell count is the result of pressure changes in the ascini. Towards the end of milking the pressure is reduced which results in large numbers of cells passing into the ascini, however this migration gradually slows down as pressure is built up again towards the next milking. Giesecke and Van der Heever (1974) suggest the cyclic variation may be related to damage

of the apical epithelium ca ed by fat secretion which is itself related to pressure changes.

In Milne's (1973) review it is reported that foremilk samples from shorter milking intervals generally show higher somatic cell counts. This variation in cell numbers can probably be explained in terms of the total milk volume secreted leading to greater concentration of somatic cells during shorter milking intervals. Longer milking intervals will show a greater dilution of somatic cell counts and hence a lower somatic cell count.

Bay (1973) showed no difference in cell counts of morning and evening milking. He obtained a correlation of .968 between the two milkings. Although the correlation is high, there may have been an absolute difference here.

Contrary to the above mentioned reports (Hoare, 1977) reported that there may not be a true diurnal variation. He showed that the number of cells shed per milking tends to be constant and the higher milk production generally obtained at the morning milking results in a lower cell concentration. (See Table 2.5).

TABLE 2.5: Counts in Glenfield Dairy Average (Hoare (1977)

	a. m.	p. m.
Production (litres	7.8	4.1
Log Cell Count (per ml x 10^3)	123	246
Total No. of cells/milking	9.84×10^8	10.8×10^8

From the foregoing it would appear that the cell count at evening milkings will be higher than at morning milking

particularly when there is a long interval preceeding the morning milking. Bulk samples for testing should be composites of both morning and evening milkings.

(iv) Milk Fraction:

The distribution of cells in milk fractions show individual variation Schalm et. al.,(1971) reported that cell counts of foremilk are generally higher than midstream samples and lower than strippings. PMN leucocytes and mononuclear cells are greater in strippings than in foremilk but the differences with regard to mononuclear cells are not as great as with PMN leucocytes. Paape and Tucker (1966) showed that the cell counts in residual milk are higher than in foremilk but lower than in strippings. On the contrary Natzke and Schultz (1967) reported higher counts in residual milk than in strippings. Schultz et. al., (1973) showed that samples taken before letdown had higher cell counts than samples after letdown.

It is important to standardise the time of sampling in the milking routine when taking samples from individual cows.

STRESS RELATED FACTORS:

There has been considerable controversy on the effect of "stress" on the cell counts in cows' milk. MacAdam (1954) reported a 5 fold increase in cell counts when milk was drawn from cows after their transportation by truck to an abattoir. Whittlestone et. al.,(1970) showed that stressful situation such as isolations from the herd, chasing by a dog and sudden thunderstorms can result in an increase in cell counts. Wiersma and Stott (1969) showed that thermal stress resulted in increases in somatic cell levels in milk. All these studies however have

not ensured that cows were free of infection. Stress results in the release of ACTH from the anterior pituitary gland so activating the adrenal cortex to release corticosteriods (Paape et. al.,1973). Hence the administration of ACTH mimics some of the physiological responses to stress.

Schalm et. al., (1965) found injecting of corticoids caused a rise in leucocytes in circulating blood but not in the milk. Paape et. al., (1973)observed no increase in cell counts in non infected quarters when cows were injected with ACTH. They also failed to show a leucocyte response in uninfected animals which had been subjected to thermal stress. Similarly Bandraryake and Holmes (1976) showed that no change in somatic cells takes place under thermal stress.

Earlier Whittlestone et. al.,(1970) showed that quarters which had a previous history of mastitis, injection of ACTH resulted in increases in milk somatic cell counts.

In concluding the earlier work on stress resulting in an increase in cell count seems to be lacking in sufficient detail to enable valid conclusions to be drawn. It now seems that stress may have an effect on cell count if a previous or existing infection has occurred in an udder. This means that under stress infections otherwise not detected will show up as increase in somatic cell level. So stress induced cell count increases have diagnostic value because they occur with animals harbouring infection.

MISCELLANEOUS FACTORS:

Breed

Reichmuth (1975) reported that slight differences have been shown to occur between breeds in Germany. The Highland breeds

are 50,000 to 100,000 cells/ml lower in count than lowland breeds. This seems to be a genetic difference which is also demonstrated within breeds (Lush,1950 and Young, et. al., 1960) However Reichmuth et. al.,(1976) reports that there are no basic differences between breeds in the nature of the response of the mammary gland to infection.

Season:

No definite seasonal effect has been noted and is often complicated by calving patterns.

In concluding, factors other than infections may be related to elevated cells under some circumstances especially if previous infection is present. However there is lack of convincing evidence that the relationship is significant enough to alter the mastitis interpretation under field conditions in other circumstances. The sources of error are further reduced if cell counts are carried out at regular intervals over a period and running means calculated to minimise the anomolies.

2.4.3 DETERMINATION OF SOMATIC CELL COUNT:

A number of methods have been described for determining the somatic cell content of milk. Depending upon whether they are used for precised counting or as a screening test, the methods are referred to as direct methods cr indirect methods.

The direct methods involve either the microscopic or electronic counting of somatic cells. The indirect methods depend on tests for the detection of D.N.A. from cellular nuclear material or the reaction between cell D.N.A. and certain chemical

reagents (Alkyl aryl sulphonate or secondary akyl sulphate) which brings about a marked change in viscosity. There are also other indirect methods that test for altered composition of milk. Included in this group are conductivity measurements, pH measurements and enzymatic procedures.

1. DIRECT METHODS FOR CELL DETERMINATION:

(i) Microscopic Counting:-

The direct microscopic counting of somatic cells was originally described by Prescott and Breed (1910). It involves the spreading of 0.01 ml of milk over 1 cm^2 on a glass slide and staining the dried smear with methylene blue. The cells are then counted in a number of fields with an oil immersion lens and expressed as the numbers of cells per ml. Schalm et. al., (1971) has reviewed this technique fully. Prescott and Breed (1910) expressed each cell counted to represent 5000 cells/ml. The results for 31 duplicate samples of 100 microscopic fields each gave a total variation of 14.5% and a within sample variation of 15% though 2 of the samples had errors of 42.9% and 63.3%.

An improved version of Prescott and Breed's technique was developed by the Sub-Committee on Screening Test, National Mastitis Council (1968) and referred to as the Direct Microscopic Somatic Cell Count (D.M.S.C.C.) This method involves counting the stained cells present in strips across the milk film rather than selected microscopic fields and uses a system of counting dependent on an estimation of the number of cells present in the milk sample (Brazis et.al. 1967;Smith,1969;Ward & Postle,

1970 and Schultz et. al., 1971).

The primary object of microscopic counting is to screen other counting methods and at present the direct visual identification and counting of somatic cells appears to be the best reference method. There is however, an inherently large variance within one sample association with microscopic counting which will ultimately determine the accuracy of the procedure in practical application. It is further hampered because somatic cell determination by D.M.S.C.C. involves human variation by the technician. Moreover the optical and manual operations are tedious and time consuming and hence virtually preclude their use in large scale screening programmes.

(ii) Electronic Counting of Somatic Cells

Electronic counters have been used successfully by several workers to estimate milk somatic cells. Included is the particle counter using current interruption (Coulter System) or light beam interruption (Autoanalyser) and also the microscopic detection of stained cells (Fossmatic).

The principle of the Coulter system is based on the counting of number of particles (somatic cells) singly over a certain size in a suspension. In case of milk, it must be fixed to stabilise the cells and to prevent bacterial growth.

Since fat globules are of the same size range as somatic cells, it is necessary that they be removed before the latter can be counted. This is accomplished by either centrifuging (Cullen, 1965; Phipps & Newbould, 1965 and Read et. al., 1967) or dispersing the milk fat using a non-ionic wetting agent

(Cullen, 1967). Chemical dispersion of fat is preferred to centrifuging because it is more rapid, economical, reliable and adaptable to automation (Dijkman et. al., 1969 and Pearson et. al., 1970). A good account on how to measure somatic cell counts in milk using a Coulter Counter has been reported by Tolle, (1971).

A number of workers have reported the correlation coefficient between electronic cell counts and direct microscopic counts; Phipps (1969) reported a correlation of 0.97; Pearson et. al., (1970) 0.978; Philpot and Pankey (1973) 0.854 to 0.977 and Newbould (1974) 0.853 to 0.924.

Heeschen (1975) and Hoare (1976) have reported methods whereby procedures using Electronic counting can be standardised. These workers have also described the continuous flow analysis (Autoanalyse System) which is essentially an Automated Coulter Counter and the Optical Fossmatic instrument. The Fossmatic is an automated microscopic cell count, using a DNA-Specific fluorescent dye.

Schmidt Madsen (1975) have compared the Fossmatic with the Coulter Counter and direct microscopic count. The Fossmatic appeared slightly superior to the Coulter in precision and in ease of operation.

While the electronic counting of somatic cells by the different instruments are accurate, and large numbers of samples can be processed in a work day, standardization continues to be a problem in addition to the high initial cost of equipment.

2. INDIRECT METHODS OF CELL DETEMRINATION:

(i) Whiteside Test:-

The reaction between milk from mastitic cows and sodium hydroxide resulting in the formation of a viscid mass was described by Whiteside (1939). At room temperature the reaction takes place rapidly but when normal, mastitic boiled milk or when milk contains preservatives such as formalin, mercuric chloride and potassium dichromate, the reaction does not occur (Schalm et. al, 1971). Murphy and Hanson (1941) modified the test to the form in which it is used today which involves the addition of 1 drop of normal sodium hydroxide to 5 drops of cold milk on a glass plate followed by vigorous stirring for approximately 20 seconds. The degree of reaction was graded from 0 - 4.

Correlation of 0.48 - 0.8 have been reported between Whiteside Test and Direct Microscopic of somatic cells in milk samples (Milne, 1973).

Nageswaro and Derbyshire (1969) reported that the reaction was dependant on precipitation and the milk components in the reaction ranked in the order of importance are: leucocyte nuclei, calcium ions, leucocyte protein, fat globules and fibrinogen.

Milne (1973) mentions the Negretti Field test which is similar in action to the Whiteside Test and is regarded as a more reliable one; the reagent is a mixture of sodium carbonate, calcium chloride and sodium hydroxide.

(ii) California Mastitis Test (C.M.T.)

The test was first described by Schalm & Noorlander

(1957). They showed that when an anionic surface active agent was added to milk and stirred it resulted in a viscous mixture. The degree of viscosity depending on the number of somatic cells present in the milk i.e. as the somatic cell content increased, viscosity increased. The reagent proposed was alkyl-aryl sulphonate, with the addition of bromcresol purple to indicate the pH of the mixture. This test was designed for use as a cow's side test where milk from the 4 quarters was squirted into 4 different depressions on a plastic paddle. To the milk is added an equal amount of C.M.T. reagent and the paddle is swirled approximately 10 times and the degree of viscosity of the contents assessed.

The C.M.T. is an approximate method for the estimation of the cell content of the milk samples and the reactions are scored as:- (From Schalm et. al. (1971)

C.M.T.	Cell range (cells/ml)	Appearance
- ve	0 - 200,000	No evidence of ppt
Trace	150,000-500,000	slight ppt. may disappear
1	400,000-1,500-000	distinct ppt. no gel formation
2	800,000-5000,000	mixture thickens immediately with some gel formation.
3	5000,000	a thick gel forms.

Most of the attention with regard to the mechanism of the C.M.T. reaction has been focused on the somatic cells (mainly Leucocytes) themselves and their contribution to the reaction. Jaartsveld (1962) reported that Deoxyribonuclic acid (D.N.A.) in the cell nucleus was responsible for the viscous reaction. Carrol & Schalm (1962) found that when nucleated cells were added

to normal milk it produced the C.M.T. reaction whereas non-nucleated cells did not; and the addition of deoxyribonuclease prevented this viscous reaction. Milne (1977a) showed that the viscosity developed in the C.M.T. reaction is correlated ($r = 0.94$) to the D.N.A. content of milk samples which is apparently involved in the formation of a gel matrix; similar results have been reported by Nageswaro & Derbyshire (1969). Hence in this test reagents such as alkyl aryl sulphonates have the ability to break down the cell nucleus creating a fibrillar network containing D.N.A.

(iii) Brabant Mastitis Test:-

This test utilises the C.M.T. reactions but viscosity is measured by recording the flow rate of the milk and detergent mixture through capillary tube of diameter 1.3 mm, and length 2 cm (Jaartsveld, 1962) An automated system allows 100 tests to be conducted. 0.06 ml of milk is mixed with 0.4 ml reagent (20 gm sodium lauryl sulphate and 15 ml of 10% sodium hydroxide) in the funnels of each capillary tube and rotated longtitudally at 15 rotations/min. After 6 rotations the mixing ceases for 30 sec., and is then repeated for another 6 rotations. The tubes are then opened and the results are recorded photographically at 5, 10, 20 and 60 seconds.

(iv) The Michigan Mastitis Test:-

This test described by Paape et. al. (1962) combines the reactions of both the C.M.T. and the Whiteside Test. They reported the use of 190 gms sodium alkyl aryl sulphonate, 13.5 gms of sodium hydroxide, 1.5 gm methylene blue chloride and 3.78 litres of soft water. The correlations obtained with somatic cell count

by these workers was 0.95.

(v) The Wisconsin Mastitis Test:-

Thompson & Postle (1964) reported this test which involves the gel formation of the C.M.T. reaction and determines the viscosity by measuring indirectly the rate of flow of the mixture through a small orifice. The test is fully described in Appendix 1.

The Wisconsin Mastitis Test (W.M.T.)is a modification of the Brabant Mastitis Test and employs the C.M.T. reagent diluted 1:1 with distilled water. In the U.S. this test is extensively used and is officially recognised as a screening test for abnormal milk (American Public Health Association, 1972).

Daniel et. al. (1971) assessed the reliability of the W.M.T. using the rapid mastitis test reagent and a correlation coefficient of 0.91 was shown with direct microscopic counts. The repeatability values for scores between 1 and 10 mm was approximately 0.9, for scores between 11 and 20 mm. 0.76 and above 21 mm was 0.9. This investigation showed that weakly positive milk samples have a lower repeatability.

The problem with the W.M.T. test is that small clots can block the orifice and give falsely high readings.

(vi) Rolling Ball Viscometer:-

Ruakura scientists developed the rolling ball viscometer. The instrument consists of a calibrated glass tube mounted in such a way that it can be tipped by a clock motor for a fixed period of time to 25^{o} and then returned to horizontal position (approx 3.5 sec.). The ball position is then read on the scale below the

tube, the instrument having a range from 200,000 to 2 million cells/ml. The reagent used is 2% teepol (10 mls) which is added to 5 ml. milk sample.

This instrument is a sensitive viscometer designed to measure the viscosity developed in the Rapid Mastitis Test (RMT) reaction.

Milne & Smyth (1976) have reported a correlation of 0.92 between the reciprocal of the viscometer reading and direct microscopic cell counts.

(vii) Enzymatic Procedure:-

The enzyme catalyase liberates oxygen, gas and water from hydrogen peroxide and the percentage gas has been correlated with somatic cell number (Paape et. al. 1965 and Schalm et. al. 1971). Schultz et. al. (1973) showed that that catalase test was less satisfactory than the Wisconsin Mastitis Test, Whiteside Test and the California Mastitis Test.

More recently Kitchen (1976) used the enzyme N - catyl - β - D - glucosaminidase to measure cell counts in milk. However more information is needed before it can be considered to be suitable as a diagnostic test.

2.4.4. SOMATIC CELLS IN RELATION TO MILK COMPOSITION:

The four independent quarters of the udder secrete milk of similar composition when no disturbances are present. The secretory epithelial cells of the mammary gland have very specific activities. They synthesise lactose, casein, milk fat, α-lactalbumin and β - lactoglobulin on one hand and are involved in the selective uptake of vitamins, trace elements minerals, serum albumin and immunoglobulins from the blood stream on the

other (Schalm et. al.1971)

Milk composition changes associated with mastitis have been reviewed by Wheelock & Dodd, (1969) Newstead(1973) Newbould (1974 and more recently by Schultz (1977).

Increases in somatic cells are generally accompanied by either no significant change (Haenlein et. al. 1973) or small increases (Ashworth et. al. 1967 and Weaver & Kruger 1977) in total protein content. The concentrations of some protein components such as - casein, β - Casein, β - lactoglobulin and α- lactalabumin which are synthesised in the gland decrease while that of immunoglobulins and serum albumin which are derived from blood increase with higher cell counts (Haenlein et. al. 1973 and Randolph et. al. 1974).

An inverse relationship between number of somatic cells and the fat content of milk from individual quarters is well documented (Ashworth et. al. 1967; Philpott 1967 and Randolph & Irwin 1974). Mein et.al.(1977) in a survey in Victoria (Australia) found that milk fat yield from bulk milk decreased with an increased cell count.

The lactose concentration decreases with increasing cell count (Ashworth et. al. 1967; Tolle 1969 and Muldoon & Lizka, 1971) and this is usually accompanied by a change in mineral compositions. Thus Tolle (1969) and Tallamy & Randolph (1970) report decreases in potassium and increases in sodium with increasing cell count while Ashworth et. al. (1967) reported an increase in chloride content. Calcium and phosphate may decrease but not in all cases. (Tolle, 1969; and Tallamy & Randolph 1970).

The pH of normal milk is usually 6.6 compared with values of up to 6.9 for milk with higher cell counts (Ashworth et. al., 1967).

The changes in composition of milk with increased cell counts are the result of increased vascular permeability for blood components and decreased synthesis of casein, lactose and fat (Schalm et. al, 1971), (Schalm 1977 and Schultz 1977). Table 2.6 summarises some of the general changes in milk composition.

TABLE 2.6: General changes in milk composition associated with elevated somatic cells.

Decrease	Reason for Change
Lactose	Dilution due to increased (Na) + and (Cl) - leading to Reduced synthesis.
Fat	
Casein (total)	
Minor Change	
Total protein	Opposite changes in components
Increase	
Whey proteins (total)	Leakage from blood
Chloride	Leakage from blood
Sodium	Leakage from blood
pH	Alkaline materials from blood.

In concluding, most of the information here is based on comparisons of results from mastitis screening tests on milks from individual quarters and hence may not apply to bulk milk cell count.

2.4.5: THE NATIONAL MILK QUALITY ADVISORY SERVICE:

The New Zealand Milk Quality Advisory Service (N.M.Q.A.S.) was initiated by the Ministry of Agriculture and Fisheries. The specific objective of this scheme was to ensure continued marketability and market access for New Zealand dairy produce and has three specific goals.

1) To reduce the levels of somatic cells in bulk milk,
2) To reduce the national level of antibiotics in milk,
3) To control mastitis in the national dairy herd.

Pilot schemes were started involving 8 Dairy Companies and these respective dairy companies carry out regular bulk milk cell counts on all suppliers to monitor herd levels of sub clinical mastitis and the suppliers, are notified every month of their cell counts.

The schemes are co-operative ventures involving the Waikato Dairy Laboratory (Dairy Division), the Dairy Advisory Officer (Farms), a Ministry employed specialist in milking management, the Farm Dairy Instructor who carries out checks on the efficiency of its milking machines and the local veterinary surgeon who considers the animal health aspects.

Three monthly rolling means are calculated to minimise fluctuations in bulk milk cell counts and on the basis of these results the following actions is taken:-

Group 1: Cell counts below 250,000 cells per ml - no action taken

Group 2: Cell counts between 250,000 - 500,000 cells per ml - these suppliers are notified that they have a mastitis problem and awareness

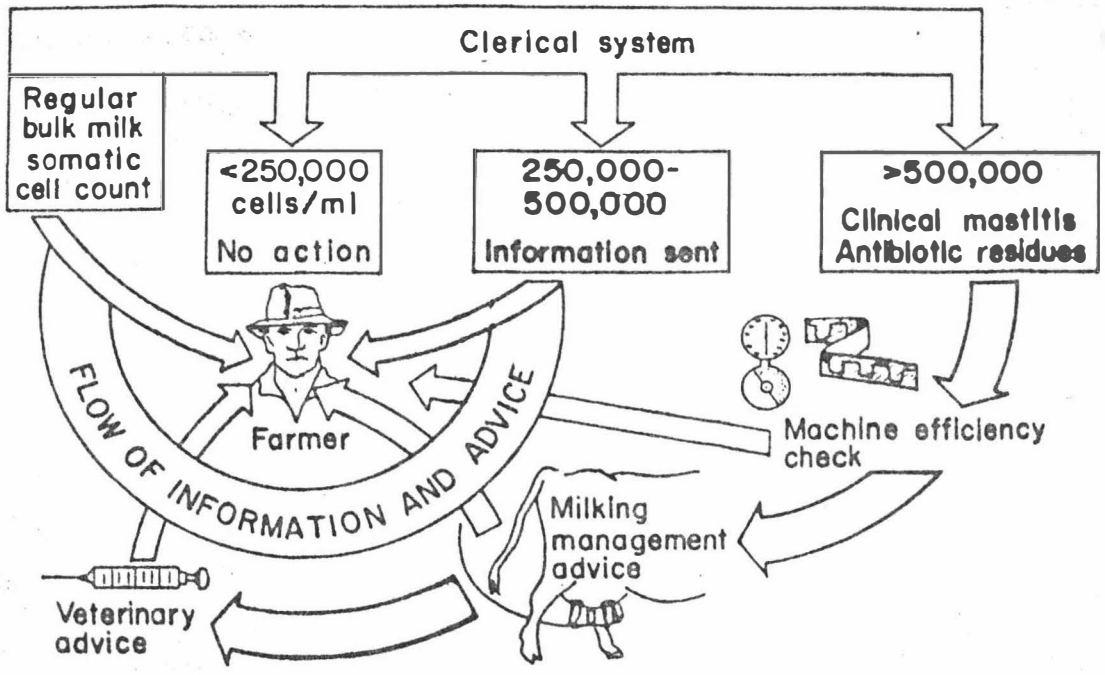


FIG. 2.3. A schematic representation of the operation of the National Milk Quality Advisory Service.

information sent out.

Group 3: Cell counts over 500,0000 cells per ml -these suppliers are placed into a problem herd category and to assist the farmers in this category a co-ordinated plan is initiated. The Farm Dairy Instructor (Dairy Division) is notified and asked to visit the farmer to carry out a milking machine test. He is followed by the Dairy Advisory Officer (Farms) who discusses all aspects of milking management and hygiene pertaining to mastitis with the farmer. The farmer's veterinarian is then advised and advises on the veterinary aspects of dealing with the problem.

Farms experiencing a high incidence of clinical mastitis with a low cell count and those with consistent antibiotic residue problems are also classified as problem herds. The criteria for a problem in clinical mastitis being a 5% infection per 100 cows per week (Milne, 1977B).

A schematic representation is shown in Fig. 2.3.

MATERIAL AND METHODS:

The study involved work carried out over a period of 12 months from April 1975 to March 1976 with the Town Milk Supply herds of the Manawatu Co-operative Milk Producers, Palmerston North. It could be divided into two parts.

1. Bulk milk samples from all 72 herds of the Town Milk Supply.
2. 19 of above 72 herds selected for individual farm visits.

1. BULK MILK SAMPLES FROM ALL 72 HERDS OF THE TOWN MILK SUPPLY

This involved 72 herds ranging in size from 24 to 240 cows with a mean of 100 cows. Bulk milk samples were collected by tanker drivers from farm bulk milk tanks monthly from April 1975 to March 1976 and delivered to the milk processing plant. The milk samples were collected from here and transported to the laboratory at Massey University. They were taken from tanks containing the milk from 2 - 4 milkings at the time of pickup and were kept at approximately $4^{o}C$ during the interval between collection and testing. They were analysed immediately after arrival at the laboratory for:

(i) Somatic Cell Count: determined by the Wisconsin Mastitis Test (WMT) as described by Thompson & Postle (1964) with the exception that the original WMT reagent was replaced by undiluted RMT reagent (Rapid Mastitis Test Reagent, I.C.I. (NZ) Ltd., Wellington). The test is described in Appendix 1. The somatic cell counts results were sent each month to the individual milk producer.

(ii) Milk Fat Percentage: determined by a photometric method after the milk sample had been diluted by a chelating agent and homogenised. The instrument used was Milk-O-Tester Mk III 12800 (A/S

N. Foss Electric, Hillerod, Denmark). Its accuracy was stated to be 0.06% fat between 0-6% fat and its reproducibility was stated to be 0.02% fat.

(iii) Total Milk Protein Percentage: determined by amido black dye binding utilising the PRO-MILK MK 11 12500 (A/S N. Foss Electric, Hillerod, Denmark). Its accuracy was stated to be 0.045% protein and its reproducibility 0.015% protein.

Information regarding the daily milk yield of cows was obtained from these herds. Printed cards were sent monthly to the farmers who completed the information required and returned it via the milk tanker driver. A sample card is shown in Table 3.1 and from this information the average daily milk yield per cow was calculated for each month. 56 of the 72 farmers regularly returned these printed cards.

TABLE 3.1: PRINTED CARDS THAT WERE SENT TO FARMERS

MASSEY UNIVERSITY

RESEARCH PROJECT

MASTITIS CELL COUNT

INFORMATION CARD

Supplier's No. _ _ _ _ _ _ _ _ _ _

Date _ _/_ _ /_ _

No. of Cows milking today _ _ _ _ _ _

No. of milkings in vat at collection time _ _ _ _ _ _ _ _

Milk in vat today _ _ _ _ _ _ _ litres

2. NINETEEN OF THE 72 HERDS INVOLVED IN VISITS FOR DETAILED EXAMINATION

Of the 72 herds in the Town Supply, 19 were selected and each was visited on four occasions during the experimental period (April 1975 to March 1976). These visits were made in May 1975, August 1975, November 1975 and February 1976). The herds were selected on the previous year's (January 1974 to January 1975) bulk milk cell count (Holmes, 1975 unpublished data) and represented herds with a variety of cell counts (low, medium and high). Of the 4 visits to each individual farm, 2 were in the evening and 2 in the morning. The herds ranged in size from 71 to 203 cows with a mean of 110 cows; the majority of the cows were Friesian or Friesian cross. On each visit to the 19 farms individual composite milk samples were collected from each lactating cow that had calved more than 5 - 7 days. The individual composite milk samples were collected from milk meters (Tru-Test Distributors Ltd. Auckland N.Z.), placed between the teatcup assembly and the milkline and were, representative samples from the whole milk yield at that milking; the milk yield of each cow at that milking was recorded. A sample of milk was also collected from the farm bulk tank on each visit; these samples will be referred to as FARM BULK MILK SAMPLES.

The number of cows showing clinical mastitis were recorded during each of these visits. A case of clinical mastitis was judged either by the presence of clots in the milk along with swelling of the quarter on the day of visit (such quarters were treated with intramammary antibiotic treatment) or quarters that were already on intramammary antibiotic treatment on the day of visit; these having already been diagnosed by the farmer as clinical cases.

The milking machines were tested before milking; the vacuum pump capacity and reserve air flow capacity were measured using Ruakura Air Flow Meter. The volume of air admitted via the pulsators and air

admission holes in the claws were also measured. The pulsators were tested using a fast speed clockwork recorder. A vacuum driven slow speed recorder was placed in the end of the milkline to record vacuum fluctuations during milking.

The milking routine including methods of hygiene at milking time was also recorded. No attempt was made to change the milking procedures used by the farm operators.

All milk samples were transported immediately following collection to the laboratory and stored at approximately 4°C until tested within 16-18 hours of collection from the farm. Somatic Cell counts were determined by the Wisconsin Mastitis Test by one operator at a rate of 40-60 samples per hour.

15 of the 19 herds were associated with the Dairy Herd Improvement Association and the age, calving date and production index (P.I.) of each individual cow in these herds was obtained from the Wellington-Hawke's Bay Livestock Improvement Association. The P.I. was calculated as shown in the LIVESTOCK IMPROVEMENT ASSOCIATION, HERD TESTING MANUAL, July 1977 compiled by the Dairy Production Division of the New Zealand Dairy Board.

Daily Variation in Bulk Milk Somatic Cell Counts:

During the months May 1975, August 1975, November 1975 and February 1976, bulk milk samples were also obtained from the milk processing station for these 19 herds. These milk samples were tested daily for the Somatic Cell Count by the Wisconsin Mastitis Test.

TABLE 4.1: THE ARITHMETIC MEAN AND STANDARD ERRORS OF BULK MILK SOMATIC CELL COUNT FOR 72 HERDS MEASURED MONTHLY FROM APRIL 1975 TO MARCH 1976

HERD NO.	MEAN BULK CELL COUNT	STD. ERROR OF MEAN	HERD NO.	MEAN BULK CELL COUNT	STD. ERROR OF MEAN	HERD NO.	MEAN BULK CELL COUNT	STD.ERROR OF MEAN
	- 10^3 cells/ml -			- 10^3 cells/ml -			- 10^3cells/ml -	
1 *	196	24	25	298	37	49	308	56
2 *	163	13	26	394	39	50	287	41
3 *	95	4	27	553	79	51	253	27
4 *	137	20	28	250	40	52	193	40
5 *	759	65	29	223	30	53	466	88
6 *	326	45	30	345	30	54	343	58
7 *	162	20	31	533	67	55	765	110
8 *	177	24	32	328	28	56	300	36
9 *	302	25	33	366	54	57	423	24
10 *	841	79	34	342	52	58	204	41
11 *	82	4	35	583	71	59	238	20
12 *	276	30	36	785	116	60	138	30
13 *	1105	75	37	537	41	61	453	96
14 *	349	34	38	278	66	62	316	78
15 *	182	29	39	418	38	63	951	135
16 *	896	121	40	328	58	64 *	579	81
17 *	673	68	41	293	30	65	495	80
18	687	88	42	548	63	66	284	49
19 *	825	66	43	391	77	67	224	44
20	643	57	44	613	106	68	613	82
21	547	68	45	755	99	69	396	54
22	278	23	46	437	32	70	642	109
23	358	36	47	558	58	71	662	103
24	355	70	48	411	42	72	133	34

* 19 Herds selected for individual visits.

CHAPTER FOUR

RESULTS

4.1. BULK MILK SOMATIC CELL COUNTS FOR 72 HERDS:

4.1.1 Mean Values

The average bulk milk cell counts for each of the 72 herds involved in the study from April 1975 to March 1976 are shown in Table 4.1. The Arithmetic mean of the somatic cell counts for all herds was 430,000 cells per ml., and the counts for individual herds ranged from 82,000 cells per ml to 1,105,000 cells per ml. Table 4.2 shows the distribution of the 72 herds according to their average bulk milk cell counts.

TABLE 4.2: Distribution of 72 herds according to average bulk milk somatic cell count for 12 months (April 1975-March 1976).

	Bulk Milk Cell Counts (cells per ml.)	Number of Herds in each group	Percentage of herds in each group
Group 1	Under - 250,000	15	21
Group 2	250,000 - 500,000	33	45.5
Group 3	500,000 - 750,000	15	21
Group 4	Above 750,000	9	12.5

The monthly mean values (± Standard Error), for the 72 herds is shown in Figure 4.1. The cell counts were highest in November and December with a mean of 582,000 ± 49,000 cells per ml and 586,000 ± 49,000 cells per ml respectively. The lowest cell counts were recorded in July and the mean was 297,000 ± 24,000 cells per ml.

FIG. 4.1: Monthly Mean Herd Bulk Milk Cell Counts ± Standard Error for 72 herds from April 1975 to March 1976.

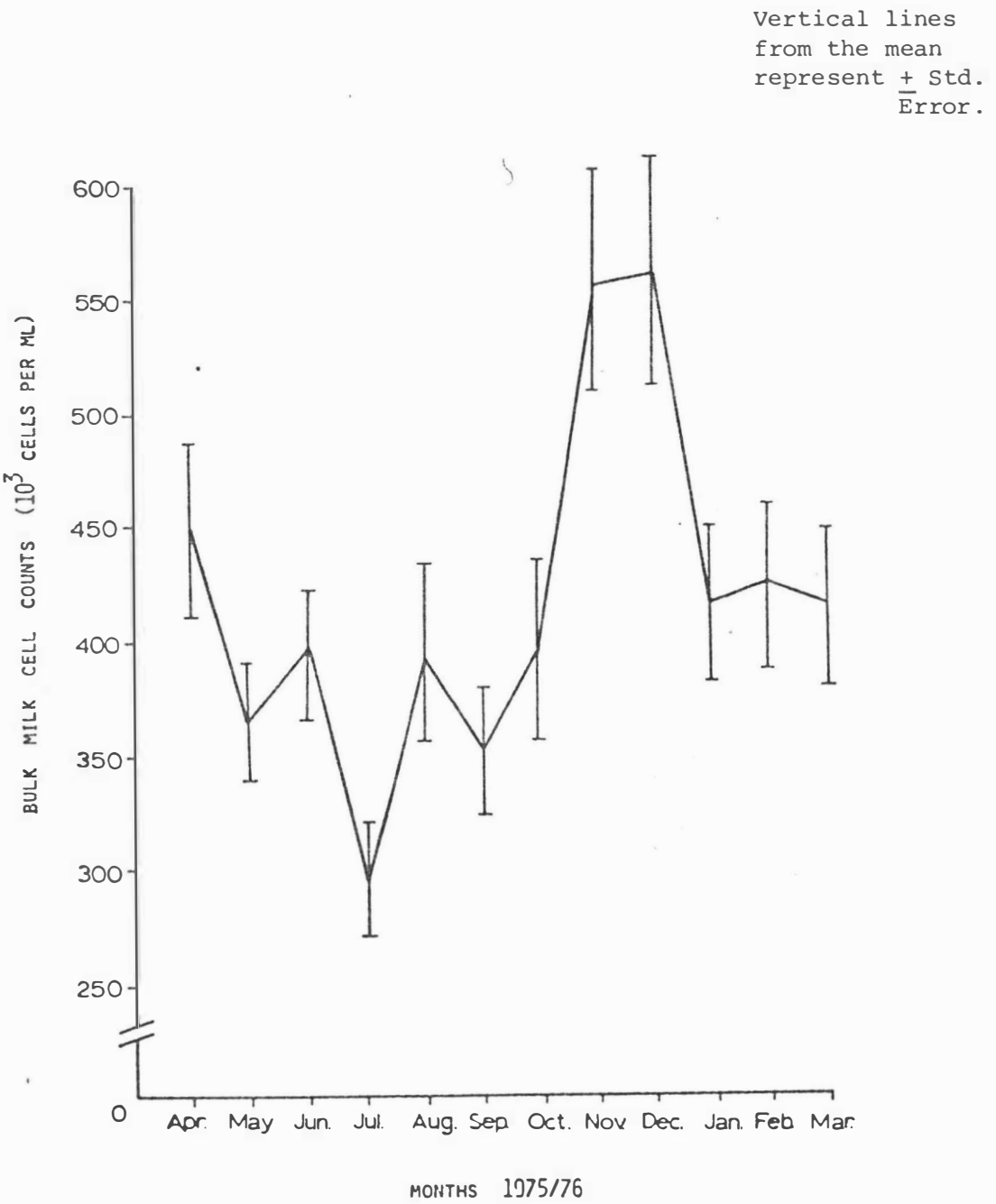


Figure 4.2 shows the distribution of the herds according to their bulk milk cell counts within each month. These are expressed as the percentage of herds in each of the four groups. The percentage of herds above 750,000 cells per ml increased from four percent in July 1975 to twenty five - thirty four percent in November and December 1975 whereas for counts under 250,000 cells per ml there was a decrease from fifty one percent to twenty seven percent of herds during this period.

From the data in Table 4.1 the five herds having the highest mean somatic cell counts (Herd No. 10, 13, 16, 19 and 63) and the five herds with the lowest mean cell counts (Herds No. 2, 3, 4, 7 and 11) were selected and their monthly distribution somatic cell counts for the twelve months is presented in Fig. 4.3. The higher cell count herds had 564,000 cells per ml in July 1975 and this increased to 1,252,000 and 1,234,000 cells per ml in November 1975 and December 1975 respectively. The lower cell count herds had values of 84,000 cells per ml in July and in November 126,000 and December 108,000 cells per ml.

FIG. 4.2: Distribution of 72 herds according to their Herd Bulk Milk Cell Counts within each month; expressed as the percentage of herds in each of the four groups.

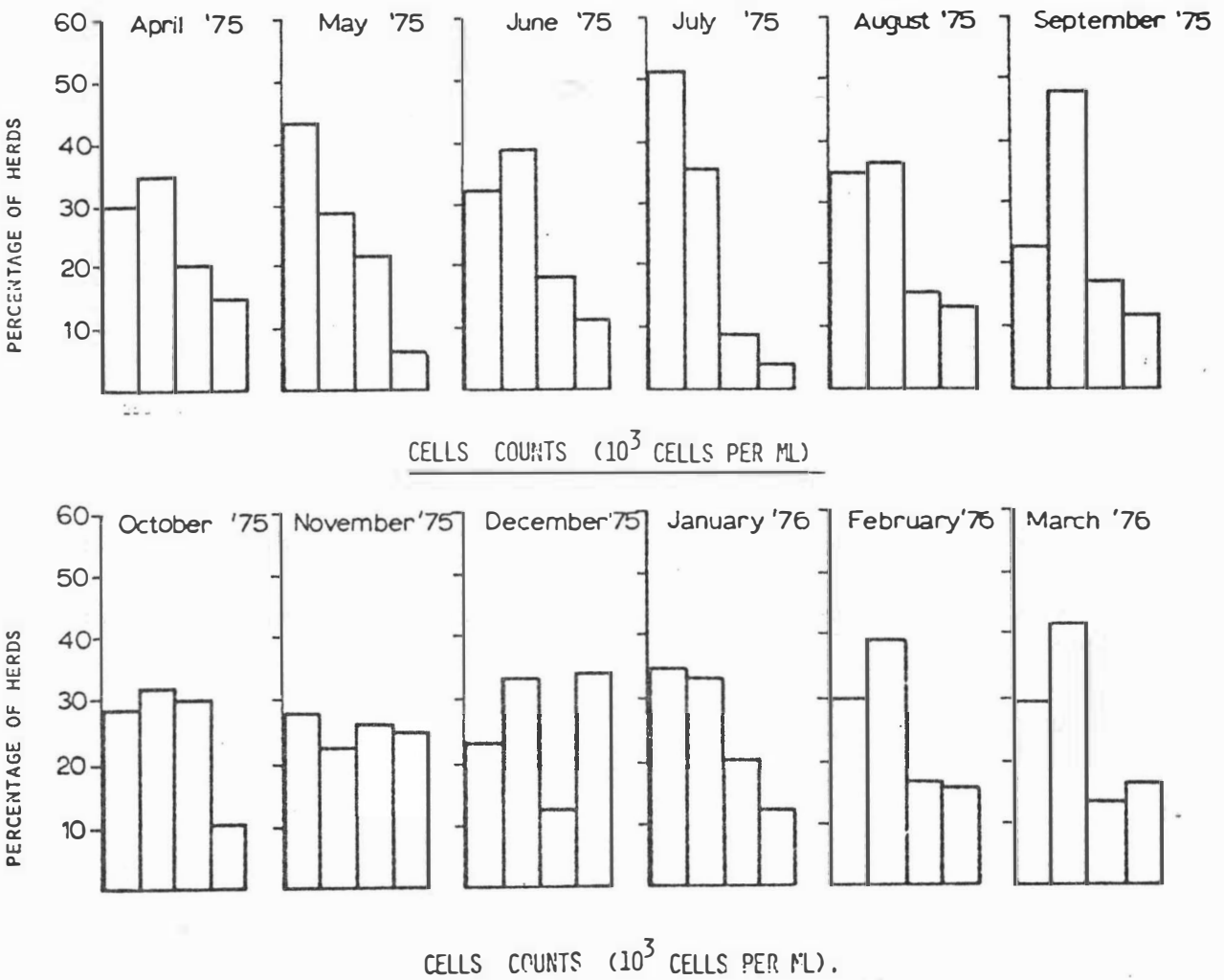


FIG. 4.3: Monthly Mean Herd Bulk Milk Cell Counts and ± Standard Error for the 5 herds with the highest and the 5 herds with the lowest annual Mean Bulk Milk Cell Counts.

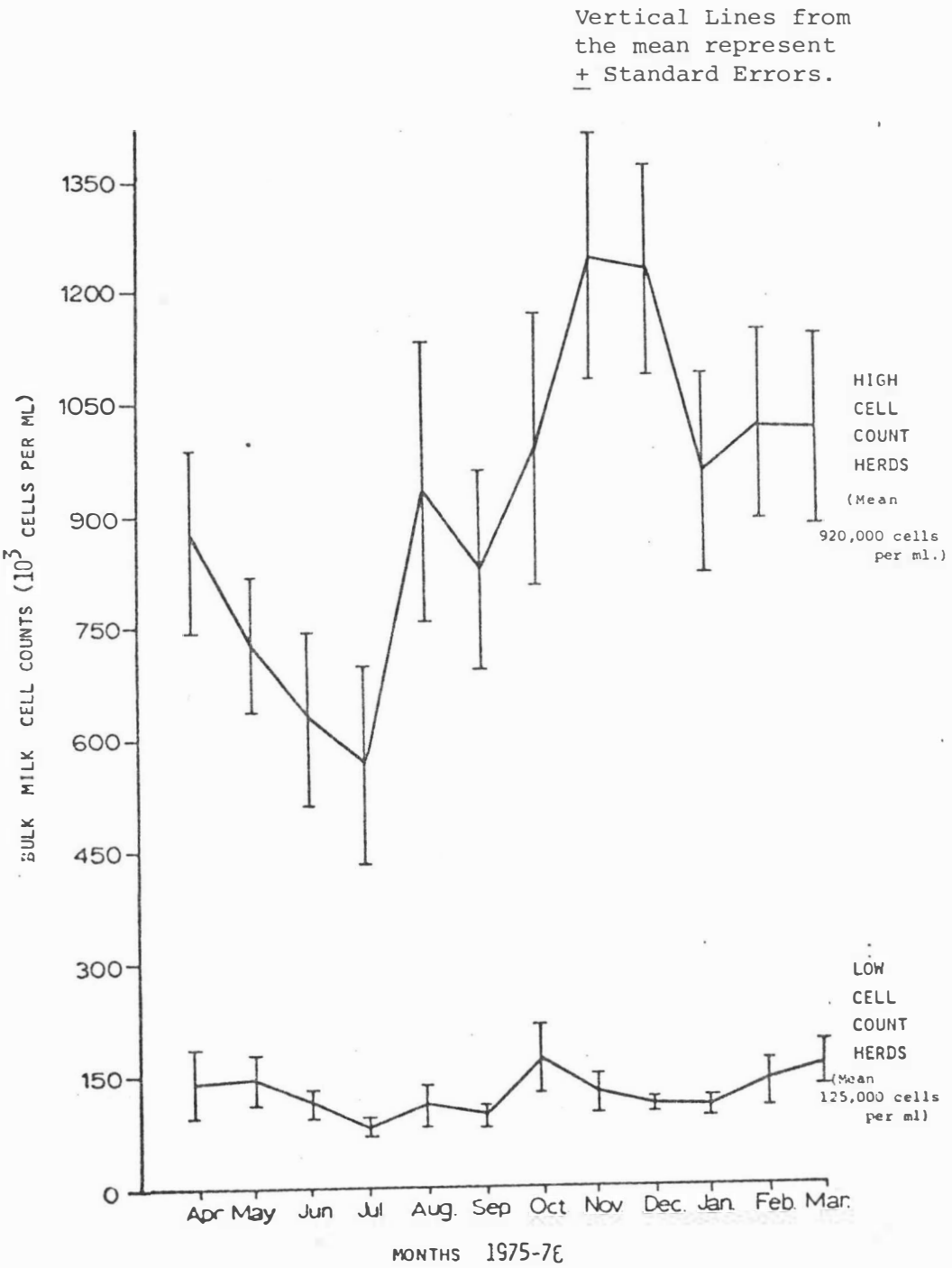


TABLE: 4.3

Mean values for each of 56 herds calculated from twelve monthly measurements on bulk milk for each herd analysed for somatic cell count, milk yield fat percentage, fat yield, protein percentage and protein yield.

Herd Number	Numbers of cows	Bulk mean cell count (10^3cells/ml)	PER COW PER DAY				
			Mean milk yield (litres)	Average fat percentage	Mean fat yield (kg)	Average protein percentage	Mean protein yield (kg)
* 1	145	196	13.4	4.42	0.592	3.47	0.465
* 2	89	163	11.1	4.17	0.463	3.48	0.386
* 3	114	95	12.2	4.37	0.533	3.38	0.412
* 4	203	137	14.0	4.20	0.588	3.41	0.477
* 5	77	759	10.1	4.43	0.447	3.51	0.355
* 6	98	326	11.0	4.23	0.465	3.42	0.376
* 7	139	162	14.2	4.23	0.601	3.26	0.463
* 8	129	177	18.6	3.84	0.714	3.48	0.616
* 9	117	302	14.6	4.06	0.593	3.35	0.489
* 10	102	841	12.7	4.13	0.525	3.38	0.429
* 11	82	82	11.1	4.41	0.490	3.49	0.387
* 12	104	276	12.7	4.31	0.547	3.42	0.434
* 13	91	1,105	11.3	4.08	0.461	3.37	0.381
* 14	160	349	13.4	4.17	0.559	3.26	0.424
* 15	71	182	12.9	4.56	0.588	3.56	0.459
* 16	70	896	10.4	3.84	0.399	3.27	0.340
* 17	91	673	10.5	4.17	0.438	3.50	0.368
18	82	687	12.8	4.13	0.529	3.29	0.421
* 19	130	825	11.1	4.35	0.483	3.47	0.385
20	82	643	12.7	4.11	0.522	3.45	0.438
21	24	547	11.1	4.16	0.462	3.41	0.379
22	144	278	11.3	4.21	0.476	3.48	0.393
23	120	358	11.9	4.50	0.536	3.54	0.421
24	87	355	10.9	4.19	0.457	3.32	0.362
25	130	298	11.8	4.41	0.520	3.42	0.404
26	101	394	10.5	4.18	0.439	3.39	0.356
27	140	553	11.2	3.95	0.442	3.44	0.385
28	107	250	12.0	4.46	0.535	3.43	0.412
29	240	223	13.7	4.13	0.566	3.29	0.451
30	61	345	12.0	4.12	0.494	3.38	0.406
31	93	533	12.1	4.42	0.535	3.54	0.428
32	121	328	11.1	4.06	0.451	3.42	0.380
33	95	366	12.1	4.16	0.503	3.46	0.419
34	96	342	12.4	3.94	0.489	3.35	0.415
35	125	583	10.4	4.12	0.428	3.35	0.348
36	27	785	10.8	4.03	0.435	3.29	0.355
37	100	537	10.9	4.30	0.469	3.38	0.368
38	88	278	10.6	4.43	0.470	3.53	0.374
39	100	418	10.4	4.39	0.457	3.42	0.356
40	80	328	13.9	3.93	0.546	3.32	0.461
41	55	293	12.0	4.17	0.500	3.54	0.425
42	55	548	12.7	4.28	0.544	3.51	0.446
43	52	391	12.2	4.31	0.526	3.48	0.425
44	87	613	11.2	4.06	0.455	3.49	0.391
45	146	755	12.1	4.28	0.518	3.49	0.422
46	96	437	12.6	4.22	0.532	3.42	0.431
47	127	558	10.9	4.18	0.456	3.47	0.378
48	44	411	10.5	4.30	0.452	3.38	0.355
49	50	308	10.9	4.57	0.498	3.48	0.379
50	80	287	11.8	4.09	0.483	3.29	0.388
51	98	253	12.6	3.92	0.494	3.36	0.423
52	52	193	13.0	4.15	0.540	3.26	0.424
53	95	466	13.8	4.39	0.606	3.37	0.465
54	115	343	13.6	4.26	0.579	3.38	0.460
55	77	765	10.9	3.96	0.432	3.34	0.364
56	78	300	10.3	4.35	0.448	3.42	0.352

* Farms involved in individual visits.

4.1.2 Data from herds which provided information about milk production:

The annual mean results for some of the parameters that were measured monthly for each of the 56 herds are presented in Table 4.3 and Table 4.4 shows the general overall information for these herds.

TABLE 4.4 : Mean values and range for bulk milk of 56 herds (April 1975 to March 1976).

Item	Mean	Range
Herd size (no. cows)	100	24 to 240
Mean Bulk milk cell counts (10^3 cells per ml)	430	82 to 1,105
% counts under 250,000 cells per ml.	32	21 to 51
% counts 250,000 - 500,000 cells per ml	36	22 to 48
% counts 500,000 - 750,000 cells per ml	18	9 to 30
% counts above 750,000 cells per ml	14	4 to 34
Daily milk yield (litres per cow)	12	10.1 to 18.6
Milk fat percentage	4.20	3.84 to 4.57
Milk protein percentage	3.41	3.26 to 3.56
Daily milk fat yield (kg. per cow)	0.506	0.399 to 0.714
Daily milk protein yield (kg. per cow)	0.409	0.340 to 0.616

The data in Table 4.3 were subject to linear regression analyses. The results of this regression analysis between annual mean bulk milk cell count (BMCC) for each herd (Independent Variable) and the several dependent variables are shown in Table 4.5.

TABLE 4.5: Results of regression analyses between annual mean bulk milk cell counts (BMCC) for each herd (Independent Variable) and several dependent variables.

	Linear Regression Equation	Correlation coefficient	Significance of correlation
1. Milk Yield (litres/cow/day).	$= 13.05 - 24.1 \times 10^{-7} \times$ BMCC	-0.37	$P < 0.01$
2. Milk Fat yield (kg./cow/day)	$= 0.56 - 1.2 \times 10^{-7} \times$ BMCC	-0.46	$P < 0.01$
3. Total Protein yield (kg/cow/day)	$= 0.44 - 0.8 \times 10^{-7} \times$ BMCC	-0.39	$P < 0.01$
4. % Milk Fat (per cow)	$= 4.3 - 1.9 \times 10^{-7} \times$ BMCC	-0.25	$P < 0.1$
5. % Protein (per cow)	$= 3.4 - 0.09 \times 10^{-7} \times$ BMCC	-0.03	N.S.

Figures 4.4, 4.5, 4.6 and 4.7 show the relation between bulk milk cell counts and the daily milk yield, daily fat yield, daily total milk protein yield and percentage of milk fat for the 56 herds. The mean value for each herd was calculated from 12 monthly measurements from April 1975 and March 1976.

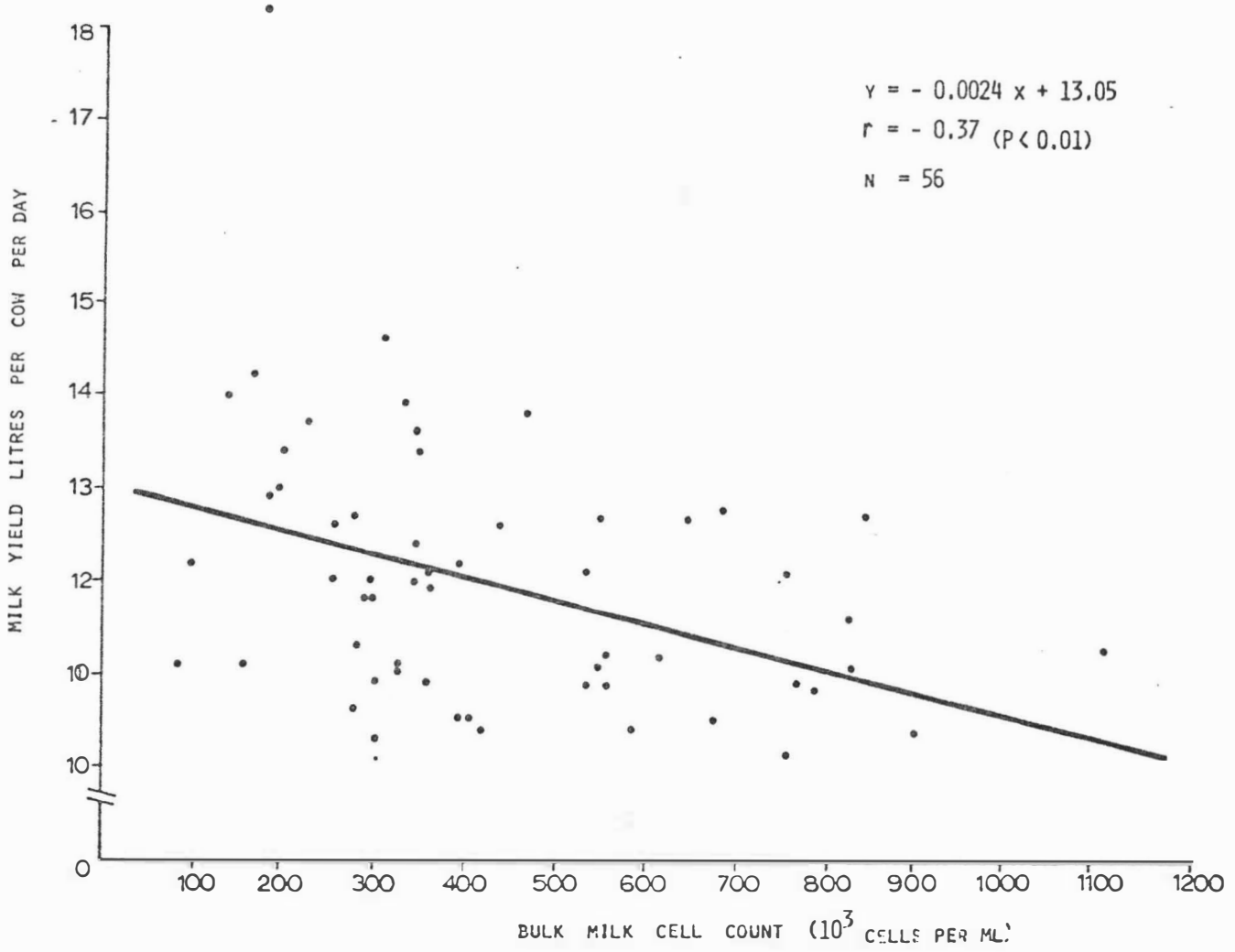


FIG. 4.4: Relation between Mean Milk Yield (Y) and Mean Herd Bulk Milk Cell Count (X) calculated for 56 herds from 12 monthly values from April 1975 to March 1976.

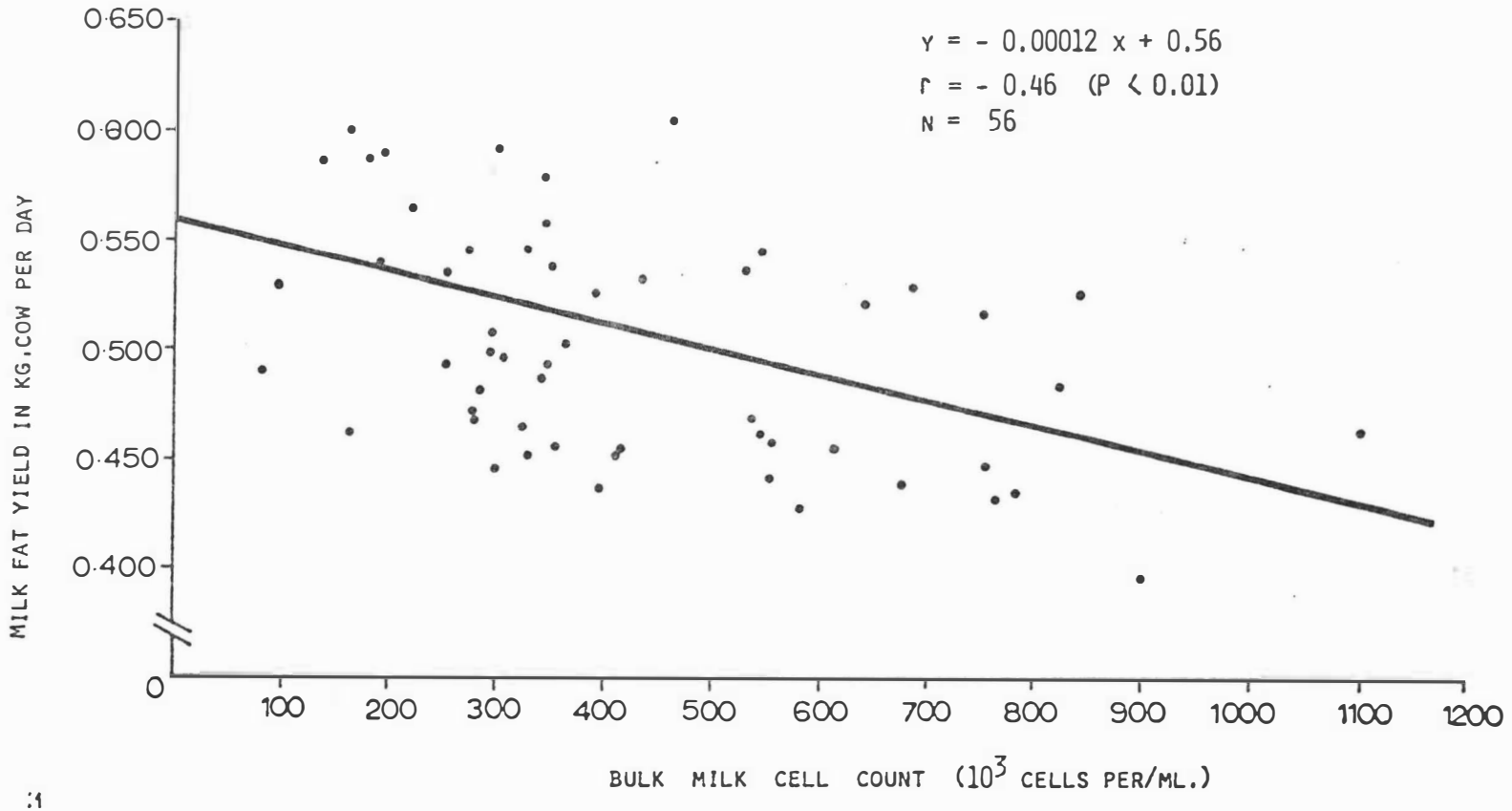


FIG. 4.5: Relation between Mean Milk Fat Yield (Y) and Mean Herd Bulk Milk Cell Count (X) calculated for 56 herds from 12 monthly values from April 1975 to March 1976.

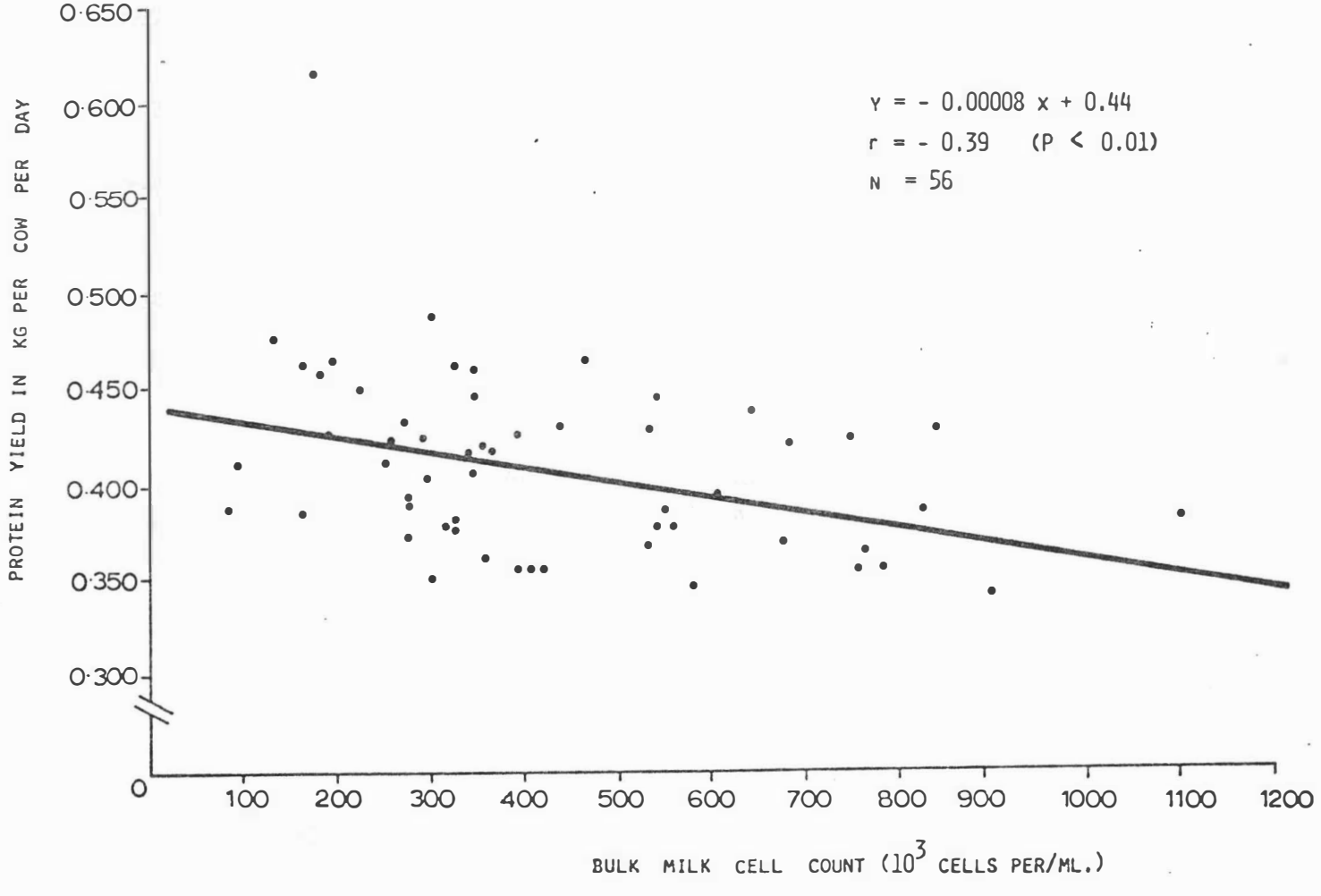


FIG. 4.6: Relation between Mean Total Milk Protein Yield (Y) and Mean Herd Bulk Milk Cell Count (X) calculated for 56 herds from 12 monthly values from April 1975 to March 1976.

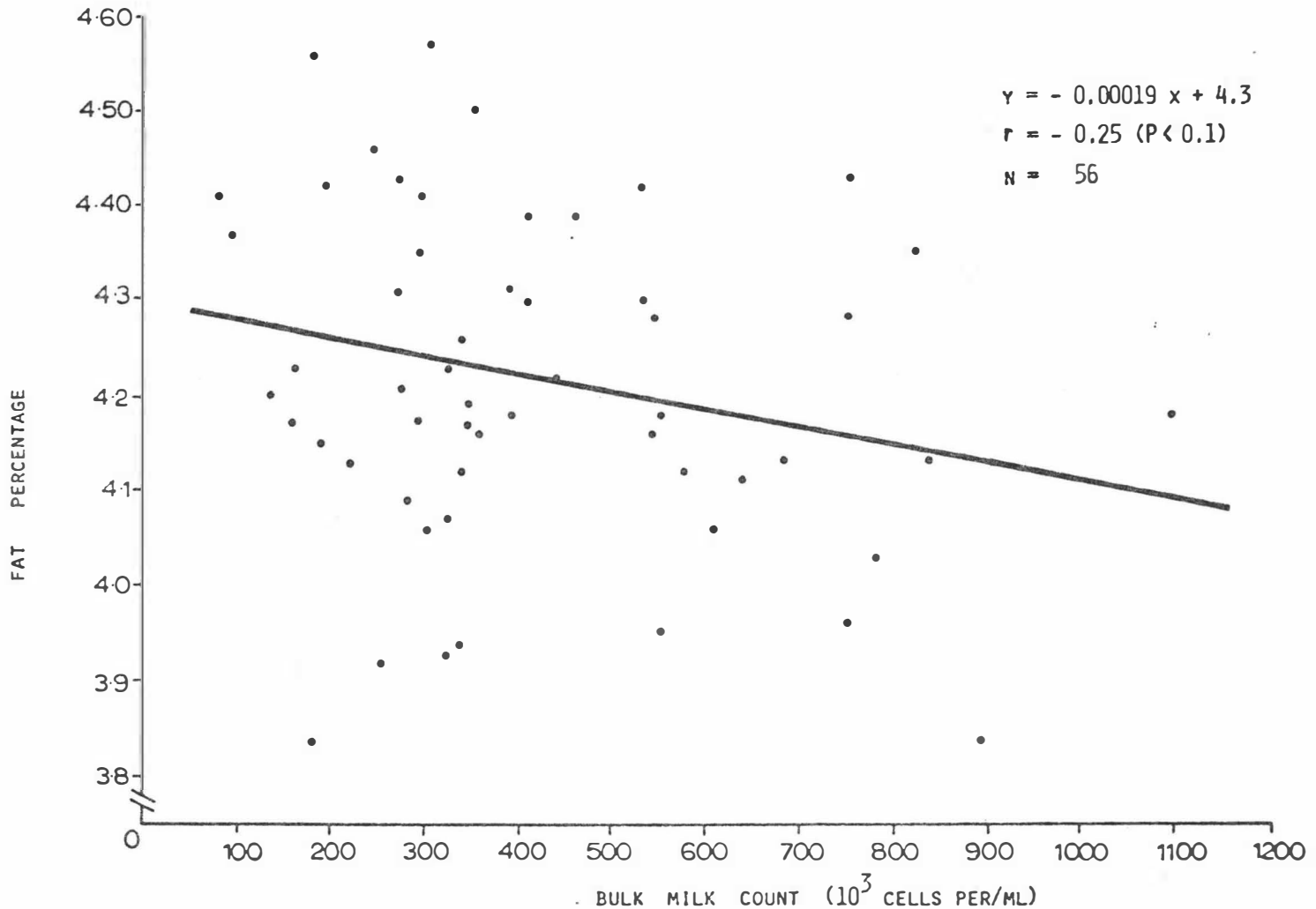


FIG. 4.7: Relation between Mean Fat Percentage (Y) and Mean Herd Bulk Milk Cell Count (X) calculated for 56 herds from 12 monthly values from April 1975 to March 1976.

4.2. DATA FROM NINETEEN OF THE 72 HERDS SELECTED FOR FARM VISITS:

4.2.1 Mean Values

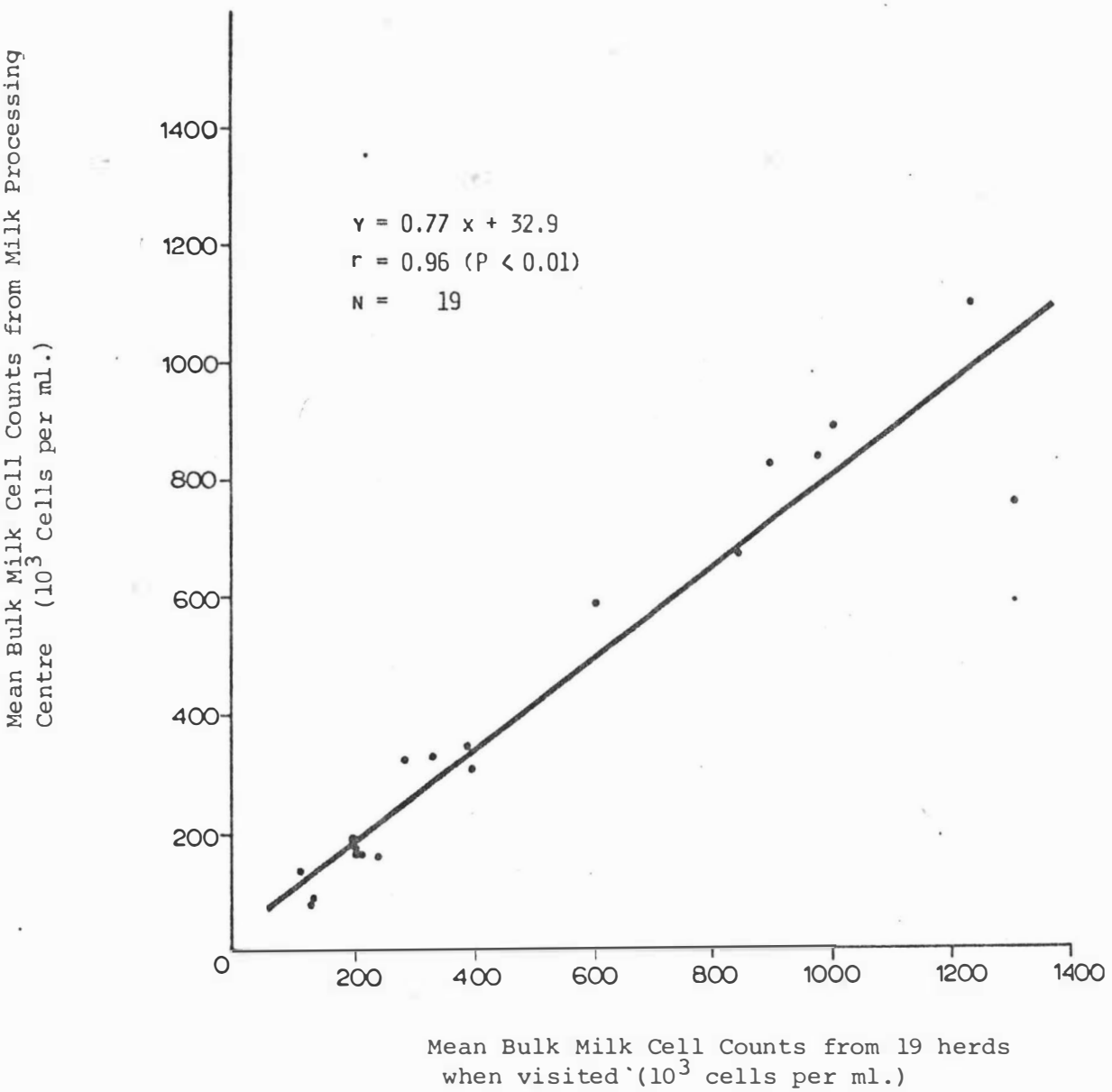
Table 4.6 lists a summary of the general information about the 19 herds that were visited four times during the experimental period. The results are generally similar to those of 56 herds that were involved in the bulk milk analysis (Table 4.4).

TABLE 4.6: Mean values and range for the 19 herds; calculated from twelve monthly values (April 1975 to March 1976).

Item	Mean	Range
Herd Size (no. cows)	110	71 to 203
Mean bulk milk cell counts (10^3 cells per ml)	470	105 to 1240
* % counts under 250,00 cells per ml.	39.5	37 to 47
* % counts 250,000 to 500,000 cells per ml	9.1	0 to 32
* % counts 500,000 to 750,000 cells per ml	25.0	5 to 37
* % counts above 750,000 cells per ml	28.0	16 to 37
Daily milk yield (litres per cow)	12.5	10.1 to 18.6
Milk fat percentage	4.22	3.84 to 4.56
Milk protein percentage	3.42	3.26 to 3.56
Daily milk fat yield (kg per cow)	0.527	0.399 to 0.714
Daily milk protein yield (kg per cow)	0.414	0.340 to 0.616

* Bulk Milk Cell Count of herds.

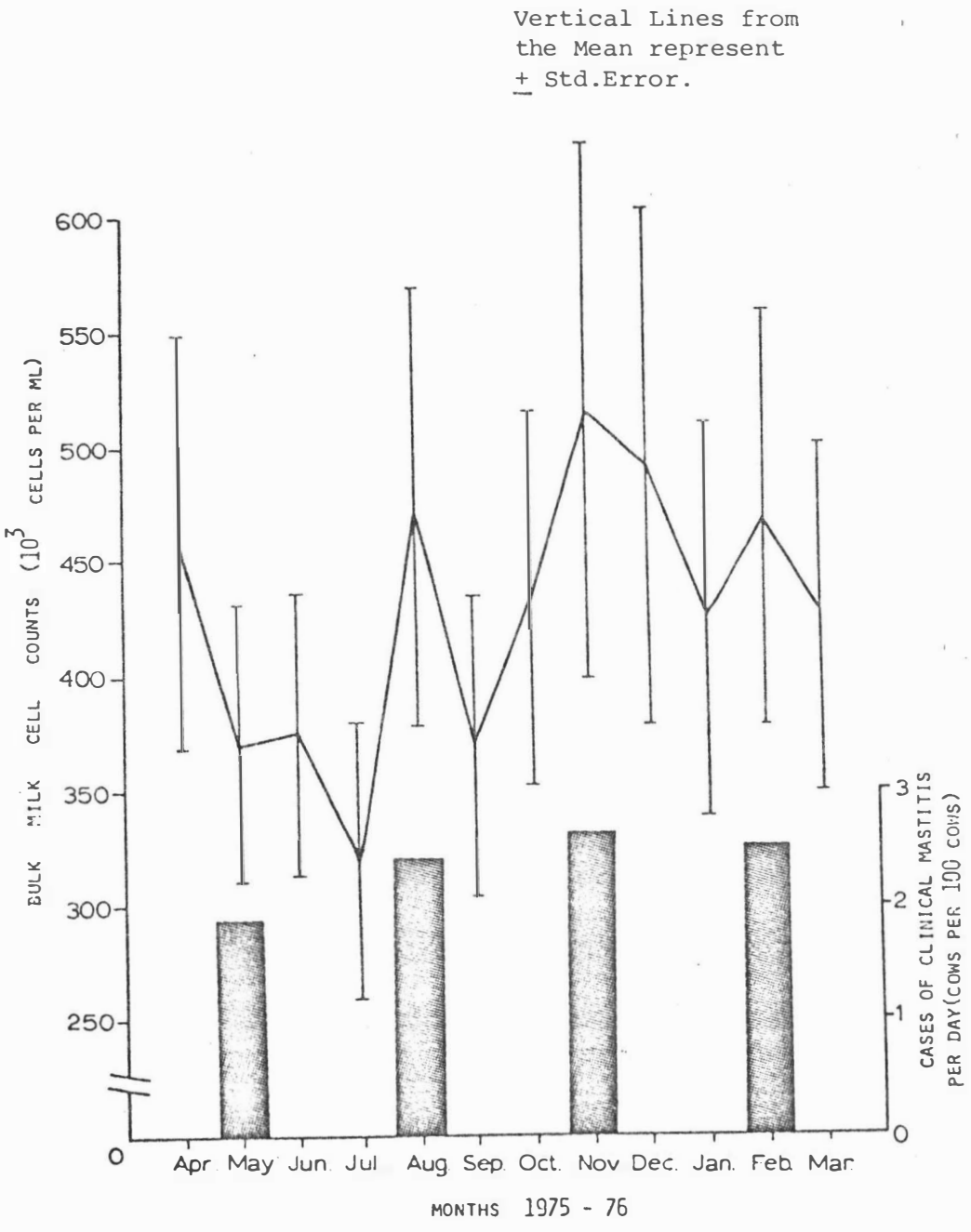
FIG. 4.8: Relation between the cell counts in Bulk Milk tank samples collected from the Milk Processing Centre (Y) and the cell counts of Bulk Milk tank samples collected from the farms on the day it was visited (X); results for 19 herds.



From the nineteen herds analyses of the relation between the cell counts in the bulk milk tank samples collected from the milk processing centre (milk factory) and the cell counts in the bulk milk tank samples collected from the farm itself (on the day it was visited), showed that the relationship was highly significant, (Fig. 4.8). The means of the factory bulk milk and farm bulk milk cell numbers were 430,000 cells per ml and 470,000 cells per ml respectively. The correlation coefficient between these sets of data was 0.96.

The monthly mean values (± Standard Errors) for the nineteen herds is shown in Fig. 4.9 together with the cases of clinical mastitis per 100 cows for one day in each month (May 1975, August 1975, November 1975 and February 1976). The somatic cell counts were lowest in July with a mean of 320,000 cells per ml, and the highest counts were November and December with a mean of 515,000 and 480,000 cells per ml respectively. The percentage of cows with clinical mastitis were lowest in May (1.9%) and highest in November, (2.6%). There was no significant difference between the 4 months (May, August, November 1975, and February 1976) and clinical mastitis.

FIG. 4.9: Monthly Mean Bulk Milk Cell Counts and ± Standard Errors together with the cases of Clinical Mastitis per 100 cows calculated from 19 herds from April 1975 to March 1976.



4.2.2 The relation between Farm Bulk Milk Cell Count and the cell counts in milk of individual cows in the herds; and the incidence of clinical mastitis in the same herd.

In Table 4.7 are shown the average values for the 19 herds which were calculated from the data recorded on the 4 visits during the period April 1975 to 1976 for farm bulk milk cell count, clinical mastitis and the distribution of individual cows according to their mean cell count in each of 3 groups i.e. under 250,000 cells per ml, 250,000 to 500,000 cells per ml and above 500,000 cells per ml.

TABLE 4.7: Mean values for 19 herds (calculated from data recorded on 4 visits during period April 1975 to March 1976) for farm bulk milk cell count, clinical mastitis and distribution of individual cows according to their mean cell count in each of three groups.

Herd No.	No.of Cows	Mean Farm Bulk Milk Cell Count (10^3cells/ml).	Percentage of Cows			Clinical Mastitis (cows per 100 cows per day
			Under 250	250-500	Above 500	
			—— 10^3 cells per ml. ——			
1	145	200	72	12	16	1.8
2	84	200	77	6	17	1.3
3	114	138	86	7	7	0
4	203	105	81	9	10	0.4
5	77	1310	42	13	45	5.4
6	98	288	68	14	18	1.7
7	139	220	60	15	25	1.6
8	129	200	78	9	13	0.7
9	117	405	55	17	24	1.0
10	102	983	44.5	16.5	39	3.4
11	82	125	84	10	6	0.6
12	104	333	63	14	23	3.1
13	91	1240	31	14	55	5.0
14	160	393	65	12	23	2.2
15	71	213	74	11	15	2.2
16	70	1003	33	16	51	4.3
17	91	855	38	14	48	3.4
18	86	602	55	13	32	2.0
19	130	907	43	15	42	3.8

The relation between the percentage of cows within each herd which fell into each of the three groups and the farm bulk milk cell count was subjected to linear regression analyses. The results were;

(1) Under 250,000 cells per ml

Percentage of cows in herd = $81.6 - 0.4 \times 10^{-4} \times$ Bulk Milk Cell Count

($r = 0.91$; $P < 0.01$)

(2) 250,000 to 500,000 cells per ml

Percentage of cows in herd = $10.2 + 0.04 \times 10^{-4} \times$ Bulk Milk Cell Count

($r = 0.57$; $P < 0.01$).

(3) Above 500,000 cells per ml

Percentage of cows in herd = $8.2 + 0.36 \times 10^{-4} \times$ Bulk Milk Cell Count

($r = 0.95$; $P < 0.01$).

These results are illustrated in Fig. 4.10 and show that with an increase in Farm Bulk Milk cell counts the percentage of cows having cell counts above 500,000 cells per ml increase, while the percentage of cell counts of under 250,000 cells per ml decrease. The cows having counts of 250,000 - 500,000 cells per ml however do not show such good relation as the other two groups.

Figure 4.11 shows the relation between incidence of clinical mastitis and farm bulk milk cell count. The regression equation describing this relation was:-

Cases of Clinical Mastitis (per 100 cows) = $0.48 + 0.036 \times 10^{-4} \times$ Bulk Milk Cell Count

($r = 0.92$ $P < 0.01$).

Table 4.12 shows the relation between the cases of subclinical mastitis (cell count greater than 500,000 cells per ml), and the incidence of clinical mastitis per 100 cows.

FIG. 4.10 Regression lines of percentage of cows with low, medium and high somatic cell counts, (Y) on Farm Bulk Milk Cell Counts.

• = Under 250,000 cells per ml (Low)

$Y = 2 - 0.04 X + 81.6.$

$r = 0.91 \ (P < 0.01)$

▫ = 250,000 -500,000 cells per ml (medium)

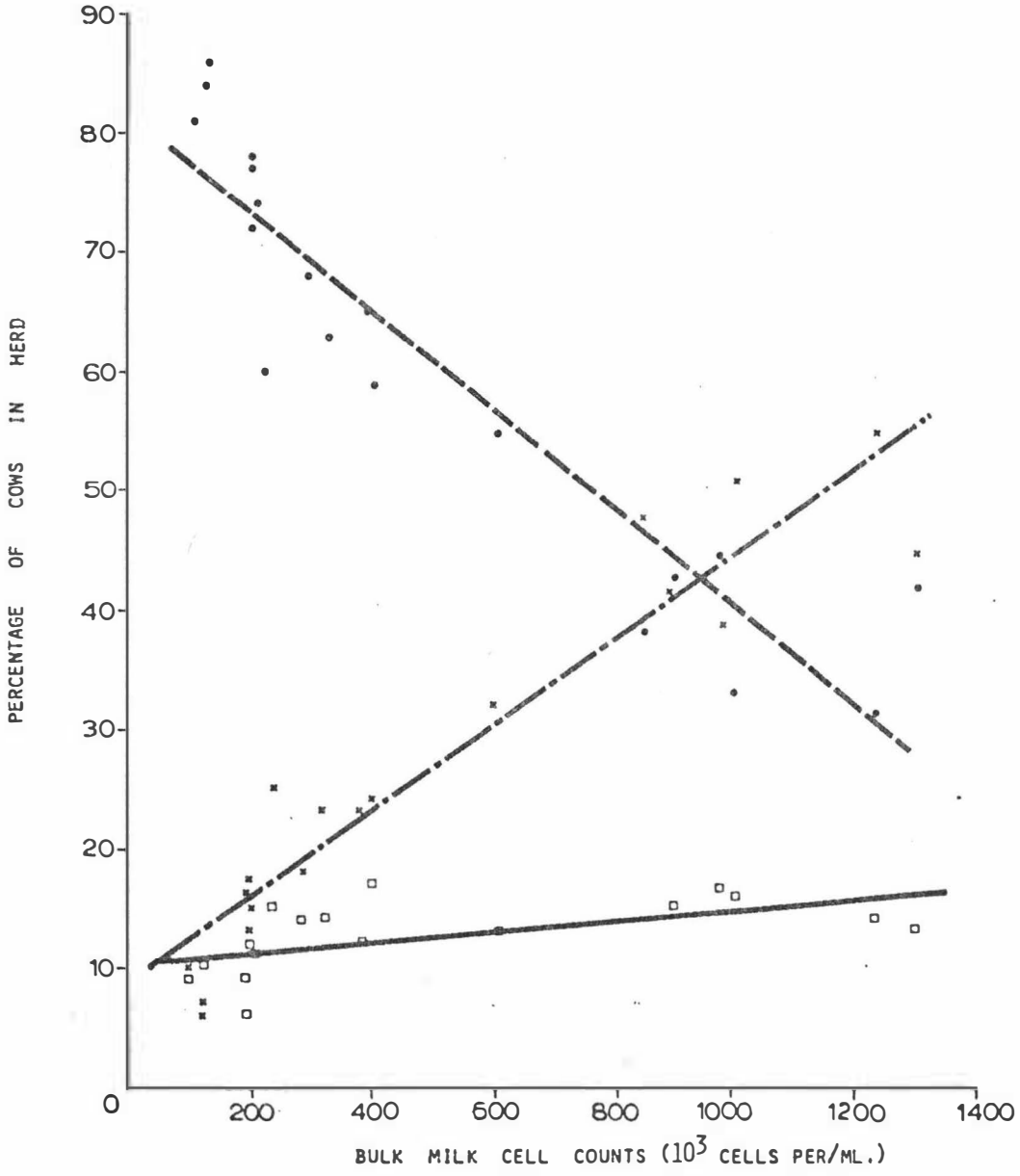
$Y = 0.004 x + 10.2$

$r = 0.57 \ (P < 0.01)$

× = Above 500,000 cells per ml (high)

$Y = 0.036 x + 8.2$

$r = 0.95 \ (P < 0.01)$


PERCENTAGE OF COWS IN HERD
90
80
70
60
50
40
30
20
10
0
200
400
600
800
1000
1200
1400
BULK MILK CELL COUNTS (10^3 CELLS PER/ML.)

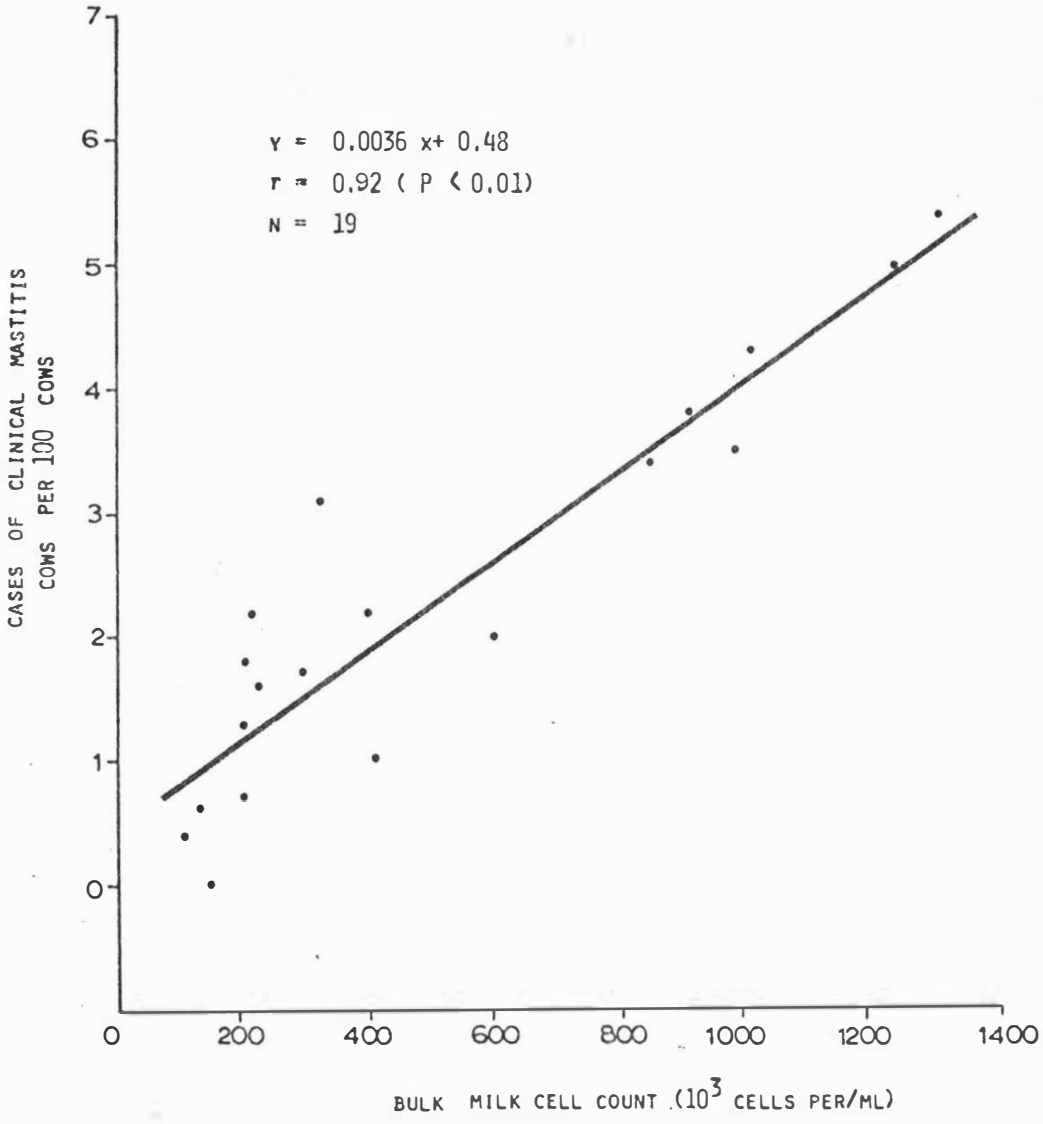


FIG 4.11: Relation between cases of clinical mastitis per 100 cows (Y) and Herd Bulk Milk Cell Count (X) for 19 herds.

FIG. 4.12: Relation of cases of sub-clinical mastitis per 100 cows (above 500,000 cells per ml) and cases of Clinical Mastitis per 100 cows.

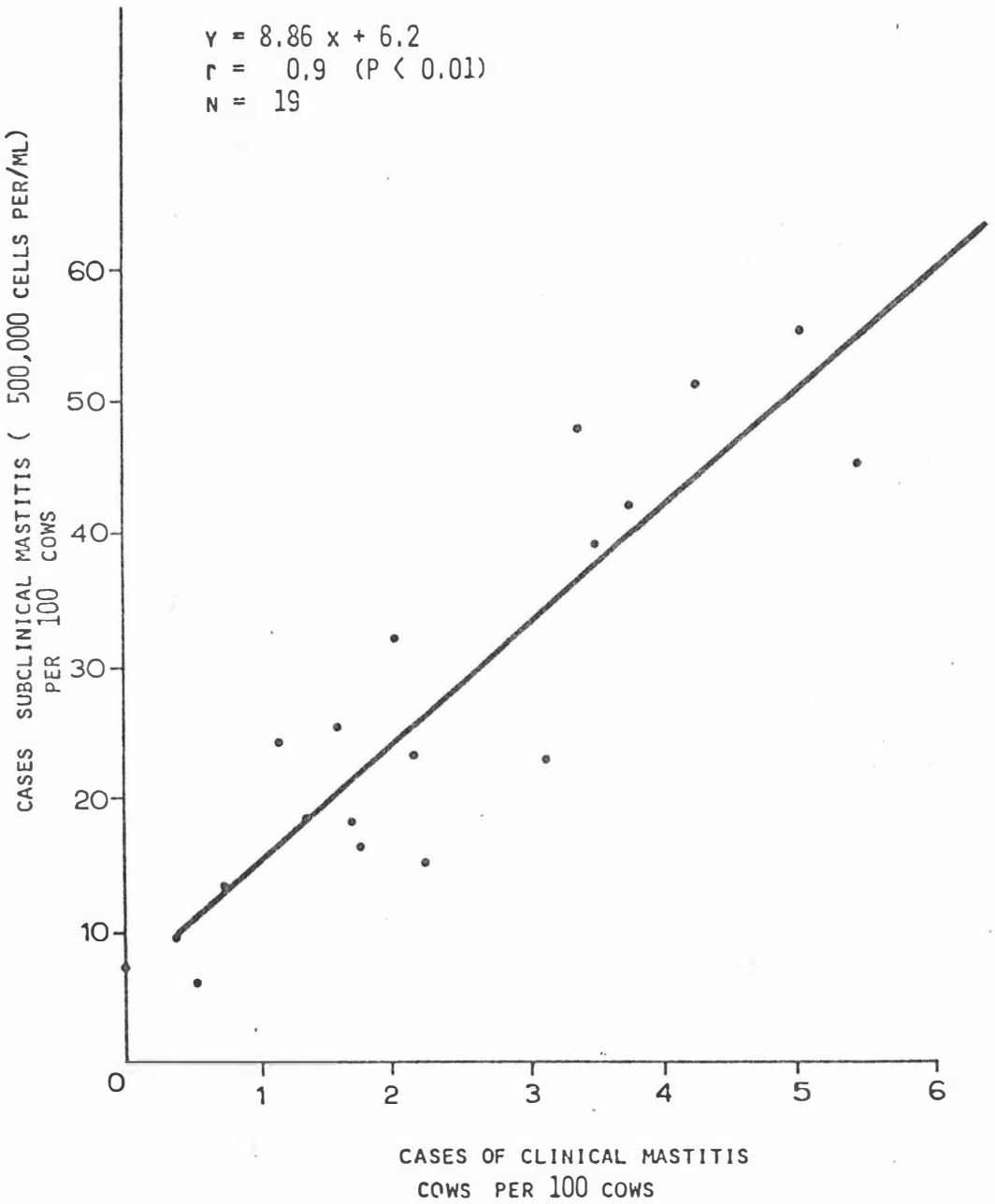


TABLE 4.8: Summary of linear regression analyses showing the relation between mean cell counts (X) (arithmetic and geometric) and production index (Y) of individual cows.

Herd No.	No. of cows	ARITHMETIC MEAN				GEOMETRIC MEAN			
		Correlation coefficient	Regression coefficient (10^{-4})	Y-Intercept	'F' Test for significance of regression coefficient	Correlation coefficient	Regression coefficient (10^{-4})	Y-Intercept	'F' Test for significance of regression coefficient
1	145	-0.01	-0.004	101	N.S.	-0.004	-0.004	101	N.S.
2	89	-0.14	-0.092	105	N.S.	-0.13	-0.095	105	< 0.25
3	114	-0.16	-0.093	102	< 0.1	-0.14	-0.089	101	< 0.25
4	203	-0.03	-0.029	102	N.S.	-0.05	-0.046	102	N.S.
5	77	-0.37	-0.153	113	< 0.01	-0.34	-0.144	112	< 0.01
6	98	-0.17	-0.110	106	< 0.1	-0.20	-0.147	107	< 0.05
7	139	-0.26	-0.139	109	< 0.01	-0.25	-0.149	109	< 0.01
8	129	-0.05	-0.028	103	N.S.	-0.15	-0.099	105	< 0.1
9	117	-0.18	-0.062	101	< 0.05	-0.20	-0.074	101	< 0.05
10	102	-0.15	-0.040	103	< 0.25	-0.14	-0.043	103	< 0.25
11	82	-0.18	-0.227	107	< 0.1	-0.11	-0.216	106	N.S.
12	104	-0.16	-0.088	104	< 0.1	-0.19	-0.119	104	< 0.05
13	91	-0.4	-0.178	116	< 0.01	-0.14	-0.063	106	< 0.25
14	160	-0.20	-0.093	103	< 0.01	-0.22	-0.111	103	< 0.01
15	71	-0.26	-0.174	103	< 0.05	-0.32	-0.250	105	< 0.01
Total of all herds	1721	-0.18	-0.090	105	< 0.01	-0.16	-0.092	105	< 0.01

4.2.3 Production Index and Cell Count for Individual Cows:

The relationship between production index and mean cell count for each cow within each of the 15 herds was calculated by regression analyses; the results are shown in Table 4.8 for each herd calculated from both the arithmetic and geometric mean cell count. In all of the herds the correlation coefficients and regression coefficients between individual cow cell counts and production index are negative. The total correlation and regression coefficient for cell count on production index in the 15 herds using Arithmetic mean was - 0.18 and - 0.09 x 10^{-4} respectively and highly significant $P < 0.01$; the corresponding values for analyses using the geometric mean cell count were - 0.16 and - 0.052 x 10^{-4} ($P < 0.01$).

4.2.4 Age and Cell Count:

A series of chi-square analyses was carried out to examine the relation between age of cows and level of cell count (Table 4.9). The values for cell counts were divided into two groups, low (<300,000) and high(>300,000)and the relation was significant in 14 of the 15 herds. The mean cell count from cows of each age were calculated from the actual data recorded in each herd and the results are shown in Table 4.10; this shows that an increase in age was associated with an increase in the mean somatic cell count of cows.

TABLE 4.9: Relation between age and cell count of cows and somatic cell counts using individual cows (Chi-square distribution analyses.)

Herd No.	Somatic Cell Count	Number of Cows in Herd				X^2 value	Significance
		Total	2-3yrs	4-6yrs	7yrs		
1	Low	103	32	49	22	14.7	0.005
	High	42	3	19	20		
2	Low	65	32	-	33	7.7	0.05
	High	24	4	-	20		
3	Low	105	43	37	25	8.217	0.05
	High	9	1	2	6		
4	Low	169	80	57	32	18.51	0.005
	High	34	5	12	17		
5	Low	25	17	7	1	7.19	0.05
	High	52	19	24	9		
6	Low	70	28	33	9	11.56	0.005
	High	28	5	11	12		
7	Low	92	40	43	9	31.12	0.005
	High	47	11	11	25		
8	Low	106	50	41	15	30.7	0.005
	High	23	1	7	15		
9	Low	65	19	38	8	30.38	0.005
	High	52	4	17	31		
10	Low	38	26	10	2	17.03	0.005
	High	64	18	29	17		
11	Low	69	17	34	18	1.046	N.S.
	High	13	1	6	5		
12	Low	58	29	19	10	10.75	0.005
	High	46	9	21	16		
13	Low	18	12	6	0	24.07	0.005
	High	73	10	34	29		
14	Low	103	46	39	18	29.04	0.005
	High	57	9	15	33		
15	Low	54	36	17	1	19.5	0.005
	High	17	2	11	4		

Low $\leq$ 300,000 cells/ml

High $\Rightarrow$ 300,000 cells/ml

TABLE 4.10: Average Somatic Cell Count of cows at different ages.

Herd No.	No. of cows	Average Somatic Cell Count (10^3cells/ml) 2 yrs.	3 yrs.	4 yrs.	5-6 yrs.	7 yrs as above
1	145	101	139	294	238	354
2	89	131	154	-	-	324
3	114	98	97	114	151	254
4	203	104	137	157	204	308
5	77	315	484	595	926	670
6	98	166	214	217	329	435
7	139	181	204	183	240	518
8	129	107	104	130	190	412
9	117	-	142	150	356	556
10	102	323	363	590	606	949
11	82	-	118	160	244	222
12	104	271	208	282	491	453
13	91	380	460	884	781	989
14	160	182	197	206	337	534
15	71	102	153	289	293	756
Mean	1721	189	212	304	385	516

4.2.5 Daily values for cell counts in May '75 and Feb '76:-

Bulk milk cell counts were measured daily for the 19 herds in May 1975 and February 1976; these results are given in Table 4.11. In May 1975, Bulk Milk Cell Counts means of the herds ranged from 110,000 cells per ml to 745,000 cells per ml; in February 1976 the range was 90,000 to 980,000 cells per ml. The coefficient of variation values for the mean values of each of the herds are also shown in Table 4.11. The mean coefficient of variation for all 19 herds in May 1975 was 21% and in February 1976 was 20%.

The means of the daily values for bulk milk cell counts measured in May 1975 were calculated for each herd; the relations between these values and the bulk milk cell count received on one day in May (from samples collected at the milk processing station) referred to as FACTORY BULK MILK SAMPLES and those collected during individual farm visits, referred to as FARM BULK MILK SAMPLES were subjected to linear regression analyses. This procedure was repeated for the data collected in February 1976. Table No. 4.12 shows the mean of daily bulk milk cell counts (BMCC) and the one day values of factory and farm bulk milk cell counts for May 1975 and February 1976.

TABLE 4.11: Somatic cell counts of bulk milk collected daily during May 1975 and February 1976 from Milk Processing Station.

Herd No.	10^3 cells/ml			Coefficient of variation (%)	10^3 cells/ml			Coefficient of variation (%)
	Mean	Std.error	Std.deviation		Mean	Std.error	Std.deviation	
1	250	14	55	22	110	5	15	14
2	195	10	40	21	215	8	30	14
3	100	9	20	20	90	5	20	22
4	130	9	40	31	125	12	40	32
5	470	14	70	15	845	54	200	24
6	140	11	40	29	255	16	60	24
7	180	11	50	28	170	12	50	30
8	120	11	40	33	180	17	60	33
9	285	11	50	18	290	13	50	17
10	745	11	30	4	755	24	85	11
11	80	9	20	25	90	6	20	22
12	180	11	30	17	400	35	115	29
13	730	43	140	19	980	53	195	20
14	320	11	50	16	365	18	65	18
15	110	13	50	46	245	14	50	20
16	400	15	60	15	880	60	215	24
17	445	13	50	11	590	14	50	9
18	500	13	55	11	450	25	95	21
19	745	10	40	5	840	25	85	10

TABLE 4.12: Mean of Daily Bulk Milk Cell Counts (BMCC) collected in May 1975 and February 1976 and the corresponding values for Factory and Farm Bulk Milk Cell Counts collected on one day.

Herd No.	MAY			FEBRUARY		
	Bulk Milk cell count (Daily)	Bulk Milk cell count for milk factory (1 day)	Bulk Milk cell count from farm when visited (1 day)	Bulk Milk cell count (Daily)	Bulk Milk cell count for milk factory (1 day)	Bulk Milk cell count from farm when visited (1 day)
	10^3 cells per ml			10^3 cells per ml		
1	250	210	120	110	100	140
2	195	210	270	215	210	210
3	100	120	210	90	100	120
4	130	100	120	125	120	100
5	470	380	380	845	1000	1280
6	140	210	210	255	230	300
7	180	230	230	170	210	210
8	120	180	180	180	200	210
9	285	380	580	290	300	320
10	745	710	530	755	710	920
11	80	80	120	90	80	120
12	180	210	140	400	380	380
13	730	1000	1000	980	1100	1100
14	320	640	640	365	400	380
15	110	210	100	245	300	370
16	400	420	530	880	1480	1000
17	445	580	530	590	580	580
18	500	380	640	450	640	470
19	745	780	780	840	830	920

The regression analyses results were:-

(1) MAY

(i) Mean of daily B.M.C.C.= 27.3 + 0.8 x Factory BMCC
($r = 0.91$; $P < 0.01$)

(ii) Mean of daily B.M.C.C.= 109 + 0.49 x Farm BMCC
($r = 0.75$; $P < 0.01$)

(2) FEBRUARY

(i) Mean of daily B.M.C.C. = 71.3 + 0.73 x Factory BMCC
($r = 0.95$; $P < 0.01$)

(ii) Mean of daily B.M.C.C. = 41.4 + 0.79 Farm BMCC
($r = 0.94$; $P < 0.01$)

These results show that the Daily Bulk Milk cell counts are significantly related to the Factory Bulk Milk cell and Farm Bulk Milk cell counts in May 1975 and February 1976.

CHAPTER FIVE

DISCUSSIONS AND CONCLUSIONS:

The present study was carried out over 12 months with 72 Town Milk Supply herds in the Palmerston North area which supply milk for liquid consumption; it was based on examination of herd bulk milk samples by the Wisconsin Mastitis Test and showed that mean somatic cell counts for herds varied from 82,000 to 1,105,000 cells per ml. The results indicated a large range in cell counts between the suppliers of the Milk Treatment Station (Town Milk Supply). The average cell count for all herds for 12 months was 430,000 cells per ml and 32.5% of the herds had cell counts of greater than 500,000 cells per ml and would therefore be considered as mastitis problem herds. Holmes (per comm. 1977) reported that the cell counts obtained from April 1976 to March 1977 showed similar overall results to those reported from April 1975 to March 1976 for the individual herds, however the total mean of all the herds was 480,000 cells per ml which was slightly higher. Earlier Holmes (unpublished 1975) monitored the cell counts of 80 seasonal supply herds of the Manawatu Co-operative from September 1974 to February 1975 and showed that the average cell count for these herds was 460,000 cells per ml and 35% of the herds had cell counts exceeding 500,000 cells per ml.

The monthly counting of bulk milk cell counts is important in educating the dairyman about mastitis and creating awareness amongst them so that they will adopt some sort of mastitis control programme. Pearson et. al.(1972) reported a

successful testing and awareness campaign in which suppliers' milk were subjected to an electronic cell count and sent the results with an explanatory note. Hoare (1976) suggests that awareness, interest and knowledge of mastitis precede, rather than follow the adoption of control procedures. Mein et. al., (1977) in a study in Victoria (Australia) reported that when dairymen were associated with greater knowledge and awareness of mastitis it resulted in significantly higher levels of adoption of control measures in their herds. Hence it is reasonable to conclude that greater awareness and knowledge about mastitis will result in faster and greater adoption of control procedures and this awareness is best accomplished by reporting the bulk milk cell counts to the dairyman regularly.

There was considerable variation in the average cell count for all the suppliers between months during the experimental period from April 1975 to March 1976. The highest average cell numbers were in November and December and the lowest in July. The percentage of suppliers averaging cell counts greater than 500,000 cells per ml increased from 13% in July to 51% and 46% in November and December respectively. (Fig. 4.2). Similar monthly variations in cell counts have been reported in Wisconsin by Bodoh et. al., (1976) who used the Filter - DNA method and showed that cell counts were highest in July and August and lowest in March; July and August are the summer months in the Northern Hemisphere and hence correspond to November and December in the Southern Hemisphere. Several studies have reported that this seasonal trend is due to a positive relationship between ambient temperature and milk somatic cell concentration (Nelson et. al., 1967 and Roussel, 1972) while others (Roussel et.al.

1969) have reported no such relationship. In many instances a total lack of bacteriological data has made the interpretation of results difficult, however Paape et. al. (1973) showed that unaffected cows under thermal stress had significantly less milk yield and did not have increases in milk somatic cells during the period of high ambient temperature.

The results of the monthly trend in cell counts of the 19 herds of the present study (Fig. 4.9), showed that in May when the cell count was 370,000 cells per ml the number of cases of clinical mastitis was 1.9% and in November when the cell counts were 520,000 cells per ml the percentage of clinical mastitis for the month was 2.6%. In this connection Paape et. al.(1973) reported that there was an increase in the number of cases of clinical mastitis during the warmer months. Furthermore Bodoh et. al. (1976) concluded that the monthly variation in somatic cell counts in their Wisconsin study was similar to the season -al trend in the incidence of clinical mastitis as shown by Paape et. al. (1973). Thus the monthly trend in cell counts data from the present study and that of Paape et. al. (1973) and Bodoh et. al.(1976) indicate an association between monthly cell counts and clinical mastitis.

Fig. (4.3) showed that higher cell count herds reported greater variation in their cell counts between months than herds with lower cell counts. It would seem reasonable that the difference in cell count between the two groups are a reflection of the differences in mastitis status and probably the higher cell count herds having greater number of cows with previously infected quarters or existing chronic,latent and subclinical infections, hence stresses in the form of weather dietary change etc., will result in greater variation in

cell counts compared with the lower cell count herds. Whittlestone et. al. (1970) reported that cows showing a cell response to stress were found to be infected.

The results from Fig. 4.1 & Fig. 4.3 indicate the importance of regular monthly monitoring of herd cell counts and show that only one month's value could give false results. In order to minimise the variations in bulk milk cell counts it is important to examine trends in the results over a period of time. Use of the rolling mean over a period of 12 months as in the present study compensates for seasonal variations. However this will require waiting for 12 months to get the first mean and Reichmuth (1975) considers a 6 month rolling mean as a satisfactory compromise as he showed that a longer period does not increase the reliability of the information. Hence the month's test result with an arithmetic or geometric mean of the last 6 month's cell counts will be a reasonable way to allow for a more accurate and meaningful interpretation of milk somatic cell count data. Pearson et. al. (1971) have confirmed that monthly cell counts and rolling mean values give a realistic reflection of the mastitis situation in a herd.

The relation between milk yield and bulk milk cell count for individual suppliers was shown in Fig. 4.4. The regression of average daily milk yield on bulk milk cell counts was highly significant. This demonstrated that the milk yield decreased as the cell counts increased in the milk. The regression analysis indicated that of the total variation among herd means for milk yield per cow only 12.5% was associated with the differences in the 12 months mean herd bulk milk cell counts.

This results not only from the poor relationship between bulk milk cell count and mastitis infection for an individual farm (Postle et. al., 1971 and Westgarth, 1971) but also because a number of other factors other than mastitis which influence milk yield, e.g. feeding, genetic characteristics etc. However a high bulk milk cell count does seem to ensure that a high milk yield cannot be obtained.

From Fig. 4.4. the average milk yield was calculated and this is shown in Table 5.1.

TABLE 5.1: Relationship between annual mean values for milk yield; milk fat yield and total milk protein yield and bulk milk cell count from 56 herds. Results calculated from Fig. 4.4; 4.5 and 4.6.

	Bulk Milk Cell Counts x 10^3 cells per ml.			
	250	500	750	1000
Daily milk yield (litres per cow)	12.4	11.8	11.2	10.5
Daily milk fat (kg. per cow)	0.53	0.50	0.47	0.44
Daily total milk protein yield (kg. per cow)	0.42	0.40	0.38	0.36

The results show that at bulk milk cell counts of 500,000 cells per ml the percentage loss in production is 5% compared to that at 250,000 cells per ml; at 750,000 cells per ml the loss is 10% and at 1,000,000 cells per ml it is 16%. These results are similar to those reported by Booth (1970) and Pearson (1970) who reported losses of milk yield of 18% and 24% respectively at bulk milk cell counts greater than 1,000,000 cells per ml.

At an average bulk milk cell count of 430,000 cells per ml for the 72 herds for the Manawatu Milk Processing Station (Town Milk Supply); this cell count can be calculated to be costing these suppliers an estimated loss in milk production of $131,000 a year compared with herds with a bulk milk cell count of 250,000 cells per ml.

For the 1975/76 season, the total milk production for New Zealand was 6359 million litres and using 430,000 cells per ml as an average for the country, the total cost through loss in milk production can be calculated as $35,000,000 compared with bulk milk cell count of 250,000 cells per ml. This does not include the loss from clinical cases and losses for the cost of therapy and cows culled.

The linear regression analysis also showed a drop in milk fat yield with an increase in bulk milk cell counts. This regression analysis indicated that 20% of the total variation in mean fat yield per cow could be accounted for by the 12 months arithmetic mean cell count of the bulk milk from each herd. However in the study of Mein et. al.(1977) the corresponding figure was 8%. A possible explanation for the poorer correlation found by Mein et. al. (1977) could be that in their study measurements were made over a 6 month period whereas the present study was for 12 months.

The present results indicate a loss of 0.03 kg of milk fat per cow per day or 8.1 kg a year for each 250,000 cells per ml increase in bulk milk cell count. Mein et. al. (1977) report a loss of 6 kg of milkfat per cow per year for each 250,000

cells per ml increase in bulk milk cell count. Table 5.1 shows the average milk fat yield at different cell counts calculated from Fig. 4.5.

The linear regression analysis for fat percentage and bulk milk cell count though significant only at 0.1 level did show that an increase in bulk milk cell count resulted in decreases in milkfat percentage; indicating a loss of 0.02% of milkfat percent for each 250,000 cells per ml increase in bulk milk cell count.

The relationship between the bulk milk cell count and total protein yield of the milk measured by amido black dye binding was shown in Fig. 4.6. From this figure the average protein yield was calculated and is shown in Table 5.1.

This data shows that with an increase in bulk milk cell count there was a decrease in the total protein yield.

From the above discussion it can be seen that the dairy herds which maintain low bulk milk cell counts will in general have higher milk production values.

The data from the present study showed that the herd bulk milk cell count and the somatic cell count of individual cows in the herd were closely related. (Fig.4.10). Using this relationship the percentage of cows having cell counts less than 250,000 cells per ml and those with greater than 500,000 cells per ml can be predicted from the value for the herds whose bulk milk cell count is known (Table 5.2).

TABLE 5.2: Bulk Milk Cell Counts compared to percentage of cows with less than 250,000 cells; greater than 500,000 cells per ml and incidence of clinical mastitis. Data compiled from 19 herds which had 1721 cows.

	Farm Bulk Milk Cell Count				
	100	250	500	750	1000
		10^3 cells per ml			
% of cows less than 250,000 cells per ml	86	71	61	50.5	40
% of cows greater than 500,000 cells per ml	12	17.5	26.5	35.5	45.0
% of cows with incidence of clinical mastitis	-	1.35	2.25	3.15	4.10

The results show that as the herd bulk milk cell count increase the percentage of cows having cell counts greater than 500,000 cells per ml increase in each herd and if 500,000 cells per ml and above cell count is used as an index for cows having sub-clinical mastitis it is seen that the cows in herd with sub-clinical mastitis increase with an increase in cell count. In order that the relations discussed above are valid it is essential that cell counts be measured on several occasions over a period of some months. Using only one bulk milk cell count will not be able to indicate the number of cows affected by mastitis.

Similar results were shown by Pearson et. al.(1971) in a study using C.M.T. and involving 31 herds, they reported that if bulk milk cell counts are consistently over 1,000,000 cells per ml then 60 to 80% of the cows are affected with mastitis.

At 750,000 cells per ml 40% may be affected and at 500,000 cells per ml 20% may have mastitis.

They reported correlations between bulk milk cell count and C.M.T. 2 & 3 of 0.88 in 31 herds and 0.96 in 28 herds. Earlier Kleinschroth et. al. (1969) showed that the corresponding correlation relating milk cell counts with mastitic cows was 0.88 and with mastitic quarter samples it was 0.72. Gray and Schalm (1962) using C.M.T. on the bulk milk from 126 commercial herds and the reactions related to the bucket milk of individual cows showed that at 300,000 cells per ml bulk milk cell count, 30.7 percent of cows were positive to the C.M.T. At 900,000 cells per ml and 2,700,000 cells per ml the corresponding percentage were 39.6 and 58.5 respectively.

The linear regression analyses (Fig 4.11) showed a strong correlation relation of 0.92 between Farm Bulk Milk Cell count and the percentage of cows showing clinical mastitis and Table 5.2 shows the percentage of clinical cases at different bulk milk cell counts. If bulk milk cell counts are 1,000,000 cells per ml then clinical mastitis incidence of the herd is 4.1 cases / 100 cows. The percentage of cows having cell counts greater than 500,000 cells per ml were similarly related to the cases of clinical mastitis and indicated that for 1 case of clinical mastitis there were 12 cases of subclinical mastitis (using above 500,000 cells per ml as subclinical mastitis). The relationship between the incidence of clinical mastitis and herd bulk milk cell count was also shown by Pearson et. al. (1971) and the correlation co-efficient obtained was 0.65 when the individual quarter was used

and with cows it was 0.7. The ratio of clinical cases to subclinical cases reported by Pearson et. al. (1971) was 1:14 in cows and 1:32 in quarters.

When considering data in which clinical assessment is the criterion used, care must be taken in interpreting the results because there will be instances where inflammatory changes in the udder or actual bacterial infections will not always be present. However the correlation coefficient of the present study were highly significant to indicate its importance. Neave (1975) showed that 96% of all quarters showing clinical mastitis had cell counts greater than 500,000 cells per ml.

Though the present study has shown a strong relation between Bulk Milk Cell Count and individual cow cell counts and clinical mastitis, this relation is not always close when bacterial infection of individual cows or quarters is considered.

Kleinschroth et. al (1969) when relating bulk milk cell counts to infection in both cows and quarters obtained correlations of 0.58 to 0.63. Postle et. al. (1971) and Westgarth (1971) basing their definitions of infection on bacterial isolation with no reference to cellular components reported correlation coefficients of 0.50 and 0.52 respectively. Despite this Pearson et. al. (1974) basing the definition of infection on CMT reaction and bacterial isolations reported correlations of 0.71 to 0.88 between Herd Bulk milk cell count and the incidence of mastitis infection in individual quarters.

It is important to note that relating herd bulk milk cell count with bacterial infections may not hold true in all cases. Pearson et. al. (1974) states that mastitis can and

often does occur before bacteria enter the udder and in some cases the mechanical factors e.g. machine, housing, husbandry and milking routine may be among the first items which require attention. In addition the problem of teat canal infections in the pathogenesis of mastitis suggest that a number of false positive quarters could in fact be teat canal infections rather than udder infections and hence these infections would be of some importance when relating herd bulk milk cell counts to bacterial infections. Giesecke et. al. (1968) and Black et. al. (1972) have shown that the presence of bacteria in aseptically collected samples via teat canal may frequently be associated with infections of the teat canal rather than udder infections which suggest that bacterial isolation from the teat canal may not be associated with infection of udder. This would probably have some bearing on the results of Postle et.al (1971) and Westgarth (1971) in obtaining poor correlations between herd bulk milk cell counts and bacterial infections in individual cows.

Increased somatic cell counts from individual cows have been shown to be related to reduced milk yield and changes in milk composition (Heeschen, 1975, Reichmuth 1975; Reichmuth et. al., 1976, Linzel & Peaker, 1975) with no reference to whether or not high cell counts were caused by infective organisms or not. This indicates that there is some disturbances in the milk secretory tissue and cell numbers in milk should therefore be considered as a measure of udder damage. The present study did show a relation between Herd Bulk Milk cell count and milk yield, milk fat and milk protein together with the cell counts of individual cows. This would indicate that herd bulk milk cell

counts should be considered as an indicator of individual cow cell counts and probably mastitis will be the most important factor to influence cell counts provided that cell counts are taken regularly. However in very small herds 1 or 2 cows with high cell counts can have a marked influence on the bulk milk cell count. On the other hand, milk from several cows with high cell counts in a large herd could be diluted out to acceptable levels in the bulk tank. Similarly herds in which cows exhibiting clinical symptoms of mastitis are milked into the bulk tank are likely to show different relation between Bulk Milk cell counts and the incidence of mastitis in individual cows when compared with herds in which clinical cases are isolated from the herd milk.

All the 15 herds involved in the study showed a negative relation between somatic cell count and Production Index (PI) of individual cows within herds. The Production Index (PI) provides an estimate of the productive merit of the cow in relation to other cows in the industry after taking into account the breed, age and stage of lactation of the cow and the genetic ability of the herd, and is calculated from the amount of milk that is obtained from the cow up to the 240th day following calving date (Livestock Improvement Association Herd Testing Manual, 1977). When using the Arithmetic Mean Cell Count, 11 of the 15 herds showed a significant relation between PI and cell count of individual cows ($P < 0.01$) and when the Geometric Mean cell counts were used, 9 of the 15 showed the significant relation ($P < 0.01$). When all the cows in the 15 herds were considered together the relationship was highly significant ($P < 0.01$) for both the Arithmetic and Geometric Mean Cell Counts.

This relationship can be used to estimate the loss of milk production resulting from an increase in cell counts. Assuming 1 unit of Production Index to be approximately equivalent to 1.5 kg of milk fat; the average fat content of the milk to be 4.0% and the length of lactation to be 270 days; it can be calculated that the milk yield decreases by 0.14 litres per cow per day in association with an increase of 100,000 cells per ml in somatic cell count. Similar results were reported by Daniel & Fielden (1972) with 22 seasonal supply herds in the Manawatu using Wisconsin Mastitis Test; they reported for every 100,000 cells per ml increase in cell count there was a decrease of 0.2 litres of milk per cow per day. Gray & Schalm (1962) and Daniel *et. al.* (1966) have also reported similar decreases in milk yield using the California Mastitis Test as an indicator of cell counts. Recently Bodoh *et. al.* (1976) in U.S.A. using the Filter DNA method to determine cell numbers reported a correlation of - 0.165 in 16 herds between cell numbers and test day milk yield, adjusted for effects of herd, year, season,age and stage of lactation in 16 herds with 752 cows. Hence most of the available results indicate that there is a decrease in milk yield of cows with an increase in cell counts within herds. However the decrease of 0.14 litres per cow per day for 100,000 cells per ml within herds is different from that reported earlier in the study between herds showing a decrease of 0.24 litres per cow per day for 100,000 cells per ml. The reason for this discrepancy is not clear.

The results also showed that the total regression co-

efficient for all the 19 herds of Arithmetic mean with production index was 2.2% higher than that of Geometric mean, thus showing that when cell counts are taken regularly over the year there is little difference between the 2 types of means. Similar results were shown by Reichmuth (1975) in his analysis of 21,000 herds where the regression coefficient for the Arithmetic mean was only 4.3% higher than that for the Geometric mean. As the results show that the Arithmetic mean is more convenient to use and more readily understood by the farmer the results above indicate that working with it is not always wrong provided cell counts are taken regularly and one is not dealing with very small herds.

The study showed that there was a highly significant relation (demonstrated by the Chi-square test) between the age of the cow and somatic cell counts in 14 of the 15 herds. As the age of the cow increased, the cell count increased. This is well documented in literature and has been shown by a number of workers (Van Resburg 1947, Blackburn 1966, Cullen 1966, Blackburn 1968, Bodoh et.al., 1976). This increase in cell count is probably a reflection of the past infection history of the udder since Natzke et. al. (1972) reported that uninfected cows showed no upward trend over 5 years lactations. However it is noticed from Table 4.10 that the effect of age on cell counts differ between herds. The data from Herd 3 indicated that the 5 and 6 year olds had a somatic cell count of 151,000 cells per ml (low incidence of mastitis) whereas the 5 and 6 year olds in Herd 13 had somatic cell counts of 781,000 cells per ml (hence high incidence cows) and the 2 year olds in this herd had a cell

count of 380,000 cells per ml. This indicates that there are differences between herds and that a high percentage of older cows in a herd is not necessarily associated with high cell counts in either bulk milk or in the individual cows. The retaining of older animals can be economic as their milk yields are usually greater than those of heifers and maintaining older animals with a lower incidence of mastitis can be financially rewarding.

The normal daily variation in bulk milk cell count in the 19 herds for May 1975 and February 1976 was shown in Table 4.11. The coefficient of variation ranged from 4% to 46% in May and from 9.4% to 33% in February. The mean coefficient of variation for all 19 herds in May was 21% and in February it was 20%, which are lower than that reported by Westgarth (1975) of 24% in 10 herds.

The Mean and Standard deviation for the daily cell counts for both months for each herd were graphed and from this it was seen that the standard deviation increased as the mean cell count increased for each. The range of standard deviation was 20,000 to 140,000 cells per ml in May and for February it was from 15,000 to 215,000 cells per ml. Up to 500,000 cells per ml mean cell count the range of standard deviation did not change much but at 700,000 - 800,000 cell count in May the standard deviation ranged from 30 to 140,000 cells per ml and in February it ranged from 75,000 to 215,000 cells per ml. However as there were only 3 herds too much cannot be made of the results. The relationship of standard deviation and mean was linear and it passed near the

origin and hence the use of coefficient of variation was justified.

Despite the high coefficient of variation there was a close relation between the mean of daily cell count for the two months and the cell count on one sample day from the milk factory and from the bulk milk tank when the farms were visited. This means that the one day milk sample in the bulk tank is a good indicator of the average bulk milk cell count for the month, but there is variation and by doing more frequent sampling and averaging the results within herds the variation will be reduced.

As pointed out earlier monthly cell count results are a valuable tool in stimulating the farmer to mastitis control routine. The main objective of mastitis control is economic, to reduce the losses of milk to the producer and processor which is caused by this udder disease.

Cell counts should provide farmers with an incentive when informed of its significance in terms of economic loss. Once the farmer realises that cell counts can be an economic problem as well as a disease problem in his herd with economic effects the planning and execution of a control programme becomes much easier.

A number of organised programmes from different countries where bulk milk cell counts are carried out regularly consider herds whose average somatic cell count exceeds 500,000 cells per ml as 'mastitis' affected herds. Some of the countries are Australia (Hoare et. al.,1975), England (Booth 1975), Ireland (Pearson et. al.,1972) and Italy (Nardelli et. al.,1975). The National Milk Quality Advisory

Service of Ministry of Agriculture and Fisheries of New Zealand regards herds with 500,000 cells and above as problem herds.

Olsen (1975) showed that in Denmark where a national control programme is established the number of herds with more than 500,000 cells per ml dropped from 38.8 in 1968 to 19.6% in 1973. During the period the number of herds with 3 consecutive counts above 500,000 cells per ml fell from 20% to 6%.

Brander et.al. (1975) reported the results of a mastitis control programme in south west England for 3 years in which monthly bulk milk cell counts were an important factor in motivating the farmers to participate in the programme. They reported increased yields of 36 litres after one year and 182 litres per cow after the 2 years which were attributable to control measures and hence demonstrated the value of applying control measures.

Hoare et. al. (1975) reported the results of a pilot programme in New South Wales involving 35 herds where herds were tested at monthly intervals using the C.M.T. They showed that after the 1st year, milk production increased by 9% and by 15% in 2nd and 3rd years. Fat production increased by 10% and 17% respectively and net benefit per cow over costs was $4.59 the 1st year, $14.50 the 2nd and $16.08 the 3rd year based on improved butterfat production.

The most effective means of demonstrating to farmers that they have an udder health problem appears to be regular monitoring of bulk milk cell counts with an average of at least

six tests and making them aware of the economic importance of these cell counts. Losses in milk production that result from high cell counts as shown in this study can be quite convincing evidence to persuade the farmer to attempt to overcome the disease of mastitis.

In concluding this discussion the results do not represent cause and effect relationships for there are other factors which can affect somatic cell counts but the general association does exist in the field as this study has shown.

APPENDIX 1

WISCONSIN MASTITIS TEST

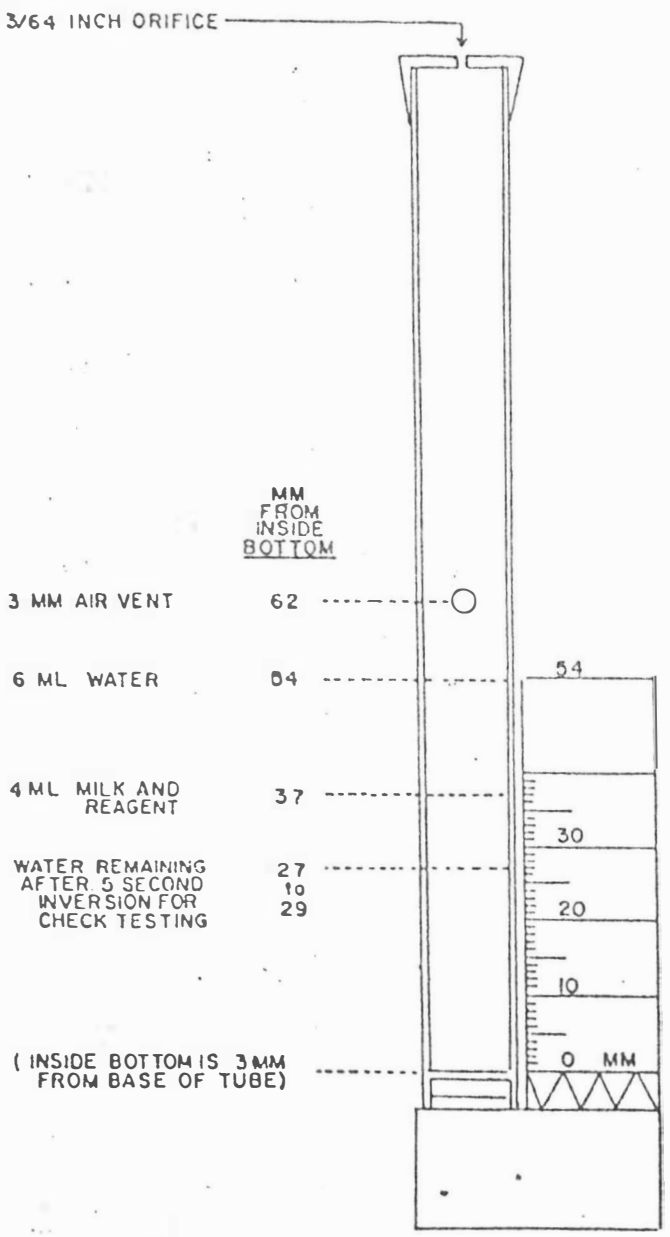


Figure 1. Diagram showing 12.5 x 125 mm test tube and measuring square. Test tube has air vent in side of tube and is equipped with a cap having a 3/64 in. orifice in its center. Measuring square is calibrated in mm. Data for checking accuracy of caps and tubes are shown.

The tests as described by Thompson & Postle (1964) and Daniel et.al. (1971). It consists of the addition of 2 ml of reagent to 2 ml of a well mixed milk sample in a 12.5 x 130 mm (outside diameter) plastic tube. Ten samples are tested at a time and the rack of 10 tubes is gently rocked after addition of the reagent to mix the contents. The tubes are then inverted for exactly 15 sec and the mixture allowed to run through the hole in the calibrated WMT cap which fits on the plastic tube. The amount of mixture remaining in the tube after 15 sec depends on the degree of gelling present and is measured by using a 1-35 mm scale.

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