THE RELATIONSHIP BETWEEN BOVINE MASTITIS AND SOMATIC CELL COUNTS IN DAIRY HERDS IN SCOTLAND.

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ABSTRACT

Thirty-five dairy herds were investigated in a three-year study of high Bulk Tank Somatic Cell Count (BTSCC). *Streptococcus agalactiae* was isolated from 19 (83%) of 23 herds selected initially as being representative of all those in Scotland with such a milk quality problem. In this group it accounted for 57% of all isolates of the major mastitis-causing pathogens with *Staphylococcus aureus* (29%) the second most frequent isolate. All these herds were selected for investigation using an "MQFILE" personal computer database which allowed the first scientific analysis of the national epidemiology of SCC in Scotland. Previously herd BTSCC data was retained on a mainframe computer for only a rolling 12 month period and was not subjected to detailed analysis. High BTSCC herds in Scotland were found to make a major contribution to national production and thus could not be ignored. An almost linear relationship was demonstrated between their annual mean BTSCC and the number of months over the 400,000 EC threshold. This indicated that an annual mean BTSCC target of less than 250,000 was required to avoid exceeding this threshold throughout the year and particularly in the autumn. A new database ("CCGM") format was established to store and analyse Individual Cow SCC (ICSCC) data from successive herd-tests. These herd investigations were the first in the UK to use a "Linear Score" (LS) 5+ (over 283,000) ICSCC threshold calculated by "CCGM" to select infected cows and thus reduce the cost of bacteriological examination. This threshold was selected by the analysis of historical data from whole herd bacteriological examinations conducted by SAC Aberdeen. This analysis revealed that a significant isolate was recovered from only 27.4% of all composite samples but that infection by any of the major mastitis pathogens was the most important cause of raised SCC in both Individual Cow (IC) and Quarter (Q) samples. This SCC increase caused by infection was very significant ($P<0.001$) irrespective of stage of lactation or lactation number and thus allowed the identification of carrier cows. Herd-specific mastitis control advice was then formulated using the CCGM-ICSCC and bacteriological profiles from each investigation. A large questionnaire study which examined the relationship between management practices and BTSCC illustrated the very significant ($P<0.001$) advantages of the "five point" mastitis control plan and membership of the Scottish Milk Records Association. The more comprehensive data from project herds showed that the adoption of paper-towels in premilking udder preparation was associated with a very significant ($P<0.001$) reduction in BTSCC. The group mean of these "assisted" project herds was very significantly ($P<0.001$) less than their contemporaries. Thus the adoption of the mastitis control recommendations had successfully achieved control of subclinical mastitis which was economically worthwhile. It is estimated that these assisted herds actually gained £33/cow/year in gross margin. All producers in Scotland have received advisory literature developed from this study by direct mailing. The study has allowed the development of an integrated system for the investigation and control of high BTSCC problem herds in Scotland.

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The man who had received the five talents brought the other five.
"Master" he said "you entrusted me with five talents.
See, I have gained five more".

Matthew 25:20

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DECLARATION

The studies described in this thesis were carried out in the Dairy Health Unit of Scottish Agricultural College Veterinary Services between March 1991 and September 1995. The author was responsible for all results except where it is stated otherwise.

No part of this thesis has been presented to any other university but it has been reproduced in parts in the following scientific publications and abstracts:

Logue, D N; Gunn, J; Fenlon, D. 1993. Results of our approach to mastitis control in Scotland. *Proceedings British Mastitis Conference 1993*, p33-49

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DEFINITIONS

A&DMMB	Aberdeen & District Milk Marketing Board
ACR	Automatic Cluster Removers
Board SCC	Board Somatic Cell Count (Monthly group mean BTSCC)
BRH	Breed Replacement Heifers
BTSCC	Bulk Tank Somatic Cell Count (cells/ml)
DAISY	Dairy Information System
DCT	Dry Cow Therapy
DHI	Dairy Herd Improvement
E.coli	*Escherichia coli* (ESCO)
EC	European Community
F pr	Statistical significance
ICSCC	Individual Cow Somatic Cell Count (udder composite sample, cells/ml)
IDF	International Dairy Federation
IMI	Intramammary infection
LS	Linear Score
MFE	Mastitis Field Experiment (NIRD)
MMT	Milking Machine Test
NIRD	National Institute for Research in Dairying
NMR	National Milk Records
NOSMMB	North of Scotland Milk Marketing Board
NSI	No significant isolate
OMMB	Ontario Milk Marketing Board
PapT	Paper towels (used in udder preparation)
PMTD	Post-milking teat dipping
PMN	polymorphonuclear leucocytes
QSCC	Quarter Somatic Cell Count (cells/ml)
S.aureus	*Staphylococcus aureus* (SFAU)
S.agalactiae	*Streptococcus agalactiae* (SPAG)
S.bovis	*Streptococcus bovis*
S.dysgalactiae	*Streptococcus dysgalactiae* (SPDY)
S.faecalis	*Streptococcus faecalis*
S.pyogenes	*Staphylococcus pyogenes*
S.uberis	*Streptococcus uberis* (SPUB)
spp	species (bacteria)

SAC	Scottish Agricultural College
SCC	Somatic Cell Count (cells/ml)
Se	Selenium
SMMB	Scottish Milk Marketing Board
SMRA	Scottish Milk Records Association
TBC	Total Bacterial Count (bacteria/ml)
UK	United Kingdom
USA	United States of America
VIDA	Veterinary Investigation Diagnosis Analysis

INTRODUCTION

Milk produced on dairy farms is intended for human consumption and the European Community (EC) has adopted standards for the hygienic quality of such milk. These maximum thresholds were set in EC Directive 92/46 as 400,000 cells/ml for Somatic Cell Count (SCC) and 100,000 bacteria/ml for Total Bacterial Count (TBC). Although the current derogation to measure SCC on the tanker load will continue until 1 July 1997, both upper limits will be fully enforced in the UK at the farm gate after 1 January 1998. Thereafter if these are exceeded the milk will be deemed unfit for human consumption.

The available information indicated that in 1991 20% of Scottish producers were unable to meet the 400,000 SCC quality criterion (Anon, 1991). In contrast, virtually all herds were consistently below the EC limit of 100,000 bacteria per millilitre (TBC). TBC has been used in the UK as a direct measurement of the final bacterial load of milk and thus its quality since 1982 (Booth, 1988b). In fact mastitis rather than contaminant bacteria was found to be the most frequent cause of high (in excess of 45,000) TBC (Jeffrey & Wilson, 1987). By contrast SCC provided a quantitative measurement of the udder's inflammatory response to infection by these mastitis bacteria (Brolund, 1985). The Bulk Tank SCC (BTSCC) can provide a measure of the prevalence of infection in the herd (Pearson & Greer, 1974). Thus SCC had two main advantages to recommend its adoption as an infection-specific measure of milk quality. Firstly the electronic automation of BTSCC measurement made economic its widespread use in quality payment schemes (Tolle et al., 1971). Secondly Individual Cow SCC (ICSCC) data could indicate the contribution of each cow to the herd BTSCC and was thus a valuable tool for mastitis detection and control (Reneau, 1986). Furthermore, loss of tissue function is a recognised consequence of inflammation. With respect to the udder, ICSCC has also provided a quantitative measure not only of this inflammation but also the consequent reduction in yield and compositional quality (Shook, 1982; Saeman et al., 1988).

This project sought to investigate the factors which influenced raised BTSCC. Two distinct groups of dairy herds in Scotland were available for this analysis. The first group comprised only herds in the Aberdeen & District Board area. The relevant information consisted of an historical collection of data from investigations into milk quality problems by the local Scottish Agricultural College (SAC). In contrast the second group was composed of all contemporary producers in Scotland organised on a regional basis (Scottish, Aberdeen & District and North of Scotland) as Milk Marketing Boards. These Board structures were later disbanded in November 1994. Their information comprised monthly quality records for bulk milk from June 1990 onward. In addition ICSCC data was available for those members of the Scottish Milk Records Association (SMRA) who had opted for this additional information. Three major objectives were identified in the analysis of this information.

The first objective was to obtain working experience in the manipulation and analysis of individual herd records of SCC, bacteriology and cow data. Although the Aberdeen herds were not selected on the basis of high SCC, this database would allow an assessment of the pathogens present in herds in Scotland which cause subclinical mastitis, their prevalence and their effect on SCC in the individual cow. In addition it was hoped that this database would allow an assessment of ICSCC thresholds appropriate for the cost-effective bacteriological investigation of high BTSCC herds.

The second objective was to identify the contemporary causes of high BTSCC and the factors influencing them in Scottish dairy herds. This section of the project would itself have two parts, the epidemiological analysis of national BTSCC movements and influences and the bacteriological investigation of a small number of herds selected as representative of the spectrum of high BTSCC herds. However this would require the establishment of a new personal computer database of BTSCC data from all SMMB herds.

The third objective was to assimilate and analyse herd management information relevant to mastitis control. This was particularly in view of the primary source of ICSCC data in this study, namely the SMRA statement data. At the outset of the project only a single hard-copy of ICSCC herd-test data was ever produced and this was distributed to the producer in isolation from any previous results. The collation of these records was intended to form the basis of an efficient strategy for the investigation and subsequent control of a herd SCC problem. Such a strategy would be based on the presentation of SCC data in a pragmatic format.

The findings of this study are presented in this thesis as five chapters, namely a literature review (1), analysis of historic records from SAC Aberdeen (2), the investigation of contemporary individual herds (3), a census of parameters affecting BTSCC in all Scottish herds (4) and a general discussion and conclusions (5). Furthermore educational leaflets were designed which presented mastitis control advice targeted towards a rapid reduction of SCC. Their distribution throughout Scotland attempted to ensure the maximum technology transfer from this research project. This was reinforced by a series of mastitis subject-days at SAC farms to which only those producers with a SCC problem were invited.

CHAPTER 1. REVIEW OF THE LITERATURE.

1.1 Introduction

A review of the current literature on the relationship of Somatic Cell Count (SCC) and bovine mastitis was undertaken. This was in response to European Community (EC) Directive 92/46 which adopted SCC as an indirect measurement of the hygienic quality of milk intended for human consumption. This review had two objectives.

The first objective was to establish the relative importance of the factors which raised the BTSCC of the individual production holding. This involved an assessment of the factors which affected SCC in not only bulk tank milk (BTSCC) but also Individual Cow (ICSCC) and Quarter (QSCC) samples. ICSCC data was required to determine the contribution of each cow to the overall BTSCC. Thus the prevalence of high ICSCC was an important component rather than the absolute cause of raised BTSCC. Barnum (1990) reported that abnormally large numbers of neutrophils and macrophages migrated from the general circulation into infected quarters in an attempt to phagocytose mastitis pathogens. This was the pathological basis for the indirect measurement of milk hygienic quality by SCC data. Therefore reaction to infection was the main cause of high ICSCC and thus raised BTSCC. However quarter data provided the most accurate assessment of the herd prevalence of infection since QSCC was not affected by the dilution of low SCC milk from non-infected quarters as was the case for ICSCC (Reneau, 1986). Factors other than infection contributed to variation in both quarter and individual cow SCC. The most important of these were stage of lactation and lactation number (Brolund, 1985). Nevertheless Pearson & Greer (1974) were able to demonstrate from herd investigations that low BTSCC was definitely associated with a reduced prevalence of cow infection.

The second objective of the literature review was to collate information on mastitis control programmes which was contemporary. The traditional strategy reported by Dodd & Neave (1970) incorporated "five points" of practical mastitis control. However these were in fact based on two principles, namely milking hygiene especially post-milking teat dipping and antibiotic treatment of all cows at the end of their lactation (so called "Dry Cow Therapy"). Recently premilking teat dipping has been advocated to improve the control of environmental (*E. coli* & *S. uberis*) mastitis (Galton et al., 1982). As well as prevention of new infections, improvements in the elimination of existing infections have also been investigated. McDermott et al. (1983) examined the use of increased lactating cow treatments while Cummins & McCaskey (1987) investigated repeated dry cow treatments. The recent availability of ICSCC data to Scottish producers has given them access to a valuable source of information on which to base their mastitis control.

1.2 The Somatic Cell Count

The Bulk Tank Somatic Cell Count (BTSCC) is one measure of quality recorded by the Milk Marketing Boards and is now the subject of EC legislation. It represents the number of somatic

cells present per ml of milk collected from the farm and therefore applies to all the milk collected from all lactating cows for purchase during the period of collection i.e. between tanker visits. It therefore generally represents the product of more than one milking from all cows. In the recent Council Directive 92/46 the EC has adopted the Somatic Cell Count (SCC) as one of the basic measurements of the hygienic quality of milk for inter-community trade (**Figure 1:1**) since infection by mastitis-causing bacteria is the main reason for increased SCC.

Cell Count (per ml) (SCC)	≤400,000[1]
Plate Count (per ml) @ 30°C (TBC)	≤100,000[2]

[1] 3 month geometric mean [2] 2 month geometric mean

Figure 1:1. EC Directive 92/46 minimum standards for milk.

The SCC analysis is performed on logarithm-transformed data but the interpretation is based on the antilogarithm (geometric mean) of the results. These standards became enshrined in UK legislation in May 1995 such that by 1 January 1998 the buyers of milk will be obliged to regard the individual herd whose BTSCC is in excess of 400,000 as producing a product unfit for human consumption. The Individual Cow SCC (ICSCC), measured in a composite milk sample from all lactating quarters, can indicate the contribution of each cow to the herd BTSCC. However the SCC of quarter milk samples (QSCC) is the only way to positively identify a highly inflamed quarter since the composite production from the other non-inflamed quarters may result in a low ICSCC (Reneau, 1986). Consequently with increasing numbers of mastitic quarters the use of ICSCC becomes increasingly more accurate in predicting whether a cow has mastitis (Meek et al., 1980).

1.2.1 Quarter Somatic Cell Count

The QSCC is the most accurate assessment of udder pathology since the count is not affected by the dilution of low SCC milk from the other non-infected quarters in the case of an ICSCC (Reneau, 1986). The overall correlation between the cell counts in quarter foremilk and quarter total milk is in the order of 0.70 - 0.86 (Reichmuth, 1975; Mijnen et al., 1983). It is generally accepted that variation in bacteriological status is the most important cause of variation in cell count (Brolund, 1985). The presence of pathogenic bacteria and the quantitative cell count are used for diagnosis of subclinical mastitis on a quarter basis by the International Dairy Federation (IDF) (Kastli, 1967). Daley et al. (1991) emphasised the importance of the phagocytic and bactericidal activity of the polymorphonuclear cells which constitute a cell count response.

Mastitis ought to be interpreted as a continuous variable as accurate discrimination between

presence or absence of pathogens, or indeed different pathogens, in a quarter as estimated by a single QSCC is impossible to obtain (Poutrel & Rainard, 1982). With increasing cell count there is a gradual change in the composition and characteristics of milk (Reichmuth, 1975; Schultz, 1977; Kitchen, 1981) as well as a reduction in yield (Janzen, 1970).

The difficulty in interpreting QSCC can be partly overcome by expressing the cell counts as intra-udder deviations from the healthiest quarter within the udder. This method was employed by Mijnen et al. (1983) and has the advantage of comparing the quarters at the same level of influence by non-bacterial factors.

Despite these difficulties a QSCC threshold of 500,000 was suggested as indicating an abnormal cell count and thus a diagnosis of subclinical mastitis (Kastli, 1967; Anon, 1971). This choice of threshold of 500,000 corresponds to twice the standard deviation from the modal value of about 2,300 quarters studied (Tolle et al., 1971; Tolle, 1975) and was further supported by the relationship between quarter foremilk cell count and changes in milk yield and in the concentration of milk constituents. The decision by Renner (1975) to use a threshold of 400,000 shows that there is no unanimity in adopting a threshold although 500,000 is now generally accepted.

1.2.2 Individual Cow Somatic Cell Count

The need to maintain low BTSCC values coupled with the advent of automated SCC testing equipment and regular monthly collection of individual milk samples from cows for yield recording purposes has led to the logical extension of examining the SCC from individual cow samples (ICSCC). The ICSCC data has two primary applications: firstly the identification of individuals affected by subclinical mastitis and secondly, as a consequence of this, as a tool in the reduction of BTSCC with the consequent improvement of milk quality (Thurmond, 1986).

Barnum (1990) and others have considered the level of an ICSCC threshold used for the diagnosis of subclinical mastitis. Jones et al. (1984) investigated the relationship between SCC threshold and prevalence of infection. He reported that of 26,739 composite milk samples cultured from 29 herds monitored in Virginia 12,206 (46%) were free of major or minor mastitis pathogens. The most prevalent major mastitis pathogens were the streptococci *S. uberis* and *S. dysgalactiae* 6.6% of samples, *S. aureus* 5.3% and *S. agalactiae* 0.6%. *S. aureus* was isolated from every herd. Only 5.9% of the samples with less than 100,000 SCC contained major mastitis pathogens. As SCC increased, percentage of major pathogens increased to 11.7% between 100 and 200,000, 17 to 19% between 200 and 400,000 and 23% in milk samples exceeding 400,000.

According to Bodoh et al. (1981) Type I errors (false positive), and Type II errors (false negative) are related inversely in any screening programme. As the critical score (the test score above which cows are infected) is raised, false positives decrease and false negatives increase. For a mastitis screening test, false negative interpretations are less desirable than are false positive (Barnum, 1990). For any given ICSCC, the probability that the cow is infected varies according

to the prevalence of subclinical mastitis in the herd. The coefficient of correlation for the general relationship between lactation mean $\log_{10}$(ICSCC) and subclinical infectious mastitis was in the order of 0.84 (David & Jackson, 1984). When the relationship between single samplings was estimated the coefficients of correlation was only about 0.6 (Brolund, 1985). This difference based on single samples and lactation records illustrates the greater reliability when estimates are based on longer periods. The most important sources of QSCC and ICSCC variation were bacteriological status, lactation number and daily milk yield. These sources of variation, defined in statistical models, accounted for 40% of the variation in $\log_{10}$(QSCC) and 36-45% of the variation in $\log_{10}$(ICSCC) (Brolund, 1985). Obviously considerable variation remained unidentified.

There are several dimensions of information included in the lactation mean cell count. Although the lactation mean $\log_{10}$(ICSCC) increases with the duration of subclinical infectious mastitis and the number of affected quarters per udder (Meek et al., 1980) it does give a satisfactory estimate of the udder health status on a lactation basis (David & Jackson, 1984). Ali & Shook (1980) have shown that a logarithmic transformation of ICSCC to "Somatic Cell Score" achieved nearly normal distribution and higher heritability. The United States National Cooperative Dairy Herd Improvement Programme thus adopted a logarithm base 2 ($\log_2$) scale for reporting ICSCC to dairy producers. This "Linear Score" (LS) scale was developed by Shook (1982) and adjusted so that nearly all SCC are in the interval 0 to 9 with the advantage of a more linear relationship to losses in milk yield than the ICSCC figure itself (Meijering et al., 1978; Raubertas & Shook, 1982; Miller et al., 1983; Jones et al., 1984; Fox et al., 1985).

However the assertion by Shook (1982) that production loss changes as a logarithm-linear function of SCC may be an over-simplification (Thurmond, 1990). Meijering et al. (1978), Raubertas & Shook (1982) and Jones et al. (1984) found that milk losses at the same cell count were twice as high in later than in first lactations. Furthermore it has generally been assumed there is no compensatory increase in milk production in the non-inflamed quarters and that milk production from each of the other three quarters was equal (Meijering et al., 1978).

Setting thresholds facilitates the analysis and interpretation of results. Levels of ICSCC previously proposed as a threshold above which milk production is adversely affected range from 148,000 to 283,000 (Reneau, 1986). However it cannot be determined from the ICSCC of a composite sample whether the QSCC is the same in all quarters or whether the QSCC in a highly inflamed quarter with a low milk production is diluted by the higher production of non-inflamed quarters, resulting in a seemingly low composite ICSCC (Reneau, 1986). Andrews et al. (1983) suggested a lactation arithmetic mean cell count of 250,000 as a threshold to discriminate between non-infected and infected cows in second and later lactations. This classification correctly identified 77% of 295 lactations, while 2% were classified as false negatives and 21% as false positives. Lindstrom et al. (1981) reported that 90% of positive and 50% of negative samples were correctly diagnosed at a threshold value of 250,000. Dohoo & Leslie (1991) found that the critical

cell count thresholds varied from 183,000 for cows younger than 4 years to 269,000 for cows nine years or older. The pooled threshold value was 228,000 and 86% of the samples were correctly classified. Thus the use of fixed ICSCC thresholds around 250,000 underestimates the prevalence of mastitis in earlier lactations and overestimates it in later ones (Thurmond, 1990). Brolund (1985) found 90% of udder infectious mastitis negative quarter samples were below, and 60% of udder infectious mastitis positive samples above the relative thresholds set within each lactation number. These thresholds were calculated as 98% statistical confidence limits for use within Sweden as the Geometric Mean QSCC plus 2 standard deviations of all bacteriologically negative quarter samples.

Hoblet et al. (1988) conducted a total herd (individual cow composite sample) bacteriological culture survey of a low SCC herd experiencing an outbreak of clinical mastitis. Despite 87% of the cows in the herd having ICSCC less than 283,000 (LS 0-4) during 1985, 11.3% of cows had quarter composite milk samples from which coagulase-positive *Staphylococcus* spp. were isolated and 81% of coagulase-positive *Staphylococcus* spp., including *S. aureus*, were cultured from cows with low SCC (less than 200,000).

In summary, in most herds a fixed ICSCC threshold of 250,000 should detect about 80% of infections and correctly classify about 80% of non-infected cows.

In addition the ICSCC can be used to assess the dynamics of infection within the herd (Barnum, 1990). Changes in the prevalence of infection (as indicated by high ICSCC) across different stages of lactation and age groups were useful indicators of when most new infections occurred. Where subclinical infections with major pathogens like *S. aureus* were not well controlled it was possible to see a gradual increase in BTSCC over a period of time, rather than a sudden explosive increase. A sudden spectacular rise in BTSCC where the levels had been consistently less than 400,000 could indicate an outbreak with mastitic milk reaching the bulk tank due to inadequate mastitis detection.

1.2.3 Bulk Tank Somatic Cell Count

Although the development of an automated method of counting SCC in milk was originally used to serve as a research tool, it has become a method of monitoring milk for the purpose of quality standards. The various types of tests for counting somatic cells have now been standardised by adoption of IDF recommendations (Heeschen, 1975). Even though BTSCC requires careful interpretation, it remains the most effective cheap measure currently available for monitoring the subclinical mastitis status of herds (David & Jackson, 1984).

The 1985 EC Directive 85/397 set out hygiene rules for the dairy industry. Various modifications have been announced and were finalised as the EC Milk Hygiene Directive 92/46. The relevant thresholds for BTSCC and TBC are detailed in **Figure 1.1**. There are a number of derogations and in summary these allow individual producers until 1 January 1998 before their milk

must carry a health mark and thus comply with the BTSCC limit measured at their production holding. In using BTSCC as a component of the hygienic quality of milk in member countries, the EC recognises the relationship of SCC and mastitis.

The BTSCC (or any other milk quality index) is not a random measurement: farms exhibit behaviour in certain recognisable patterns (Schukken et al., 1990). Thus the Ontario Milk Marketing Board BTSCC data showed a significant seasonal pattern: the lowest mean BTSCC occurred in April, and the highest mean SCC occurred in October (Schukken et al., 1992a). Although percentage of fat and lactose increased significantly with decreasing BTSCC there was very little effect on protein percentage. In herds that produced milk of lower BTSCC, TBC was significantly lower. Several studies have shown a negative correlation between ICSCC and milk fat, lactose and casein production (Shook, 1982; Bartlett et al., 1990). Lowering the ICSCC in the population should have a beneficial effect on the productivity of dairy cows (Bartlett et al., 1990). This is usually achieved following the introduction of a cell count scheme in which high BTSCC is discouraged by payment penalties. A BTSCC decrease of approximately 58,000 per annum was attributed to the Ontario Bulk Milk Somatic Cell Count Reduction Programme (Schukken et al., 1992a).

The prevalence of infection is usually considered as the factor which has the greatest effect on BTSCC (Pearson & Greer, 1974). Barnum (1990) reported that the correlation of BTSCC with the mean ICSCC was good (0.8 - 0.9). According to Pearson & Greer (1974), BTSCC less than 500,000, 500 to 800,000 and in excess of 1,000,000 corresponded with average infection levels in quarters of about 10%, 20% and 30% respectively. Although infection status had the greatest impact on QSCC, the correlation of BTSCC with the percentage of infected quarters was not higher than 0.5 (Westgarth, 1975). Similarly a correlation of 0.43 has been found between a single BTSCC taken at the day of individual quarter sampling and the proportion of infected quarters (Wilson & Richards, 1980). In summary BTSCC is not a good predictor of quarter infection rate but is a good indicator of the overall udder health of the herd. However while a single BTSCC is not a reliable measure of herd infection it can be improved by averaging a number of counts (Wilson & Richards, 1980). Thus a number of monthly BTSCC figures or the annual rolling mean BTSCC must be examined to detect trends over a period of time rather than relying on the figure for just a single month (David & Jackson, 1984). BTSCC trends are therefore a good means of evaluating the overall performance of mastitis control programmes.

Approximately 85% of all milk produced in England & Wales comes from herds with BTSCC below 400,000 (Booth, 1994). The national cell count in England and Wales has shown two periods of marked fall, in 1975-1976 and in 1983 (Booth, 1988a). The first reflected a time of severe economic pressures on dairy farmers in the UK, compounded by drought conditions, and the rate at which farmers left the industry tripled during those 2 years. Surveys showed that the herds ceasing production tended to have higher than average cell counts (Booth, 1988b). The

second period of rapid decline in 1983 followed immediately on the introduction of the payment system for bulk tank Total Bacterial Count (TBC) in late 1982.

Low BTSCC is associated with low prevalence of infection with major mastitis pathogens (Erskine et al., 1988). In order to evaluate the contribution of individual Ontario farms to the overall number of somatic cells in the milk supply, Schukken et al. (1992b) calculated their SCC contribution. This novel parameter was a measure of the number of somatic cells produced by each farm in excess of an arbitrary 250,000 upper limit of normality. This contribution parameter was a product of the adjusted monthly mean BTSCC and the volume of milk produced in that month so that for example a farm with an annual production at exactly the Ontario mean and a BTSCC of 251,000 has an SCC contribution of 1. Most Ontario farms with very high BTSCC (greater than 750,000) did not have high SCC contributions since they produced low volumes of milk. They therefore concluded that the most effective way to keep the Ontario mean BTSCC low was to target financial encouragement at farms already with low BTSCC. However this ignored the fact that milk of poorer hygienic quality was still being utilised, merely diluted by the large volumes of low BTSCC.

1.2.4 Economic significance of Somatic Cell Count

Blosser (1979) reported that mastitis caused more loss to the dairy industry in the United States of America (USA) than any other disease. Esslemont & Peeler (1993) in the UK agree that mastitis is one of the most expensive diseases affecting cattle. They estimated the cost of lost production in herds with penalty levels of BTSCC (over 400,000) at £10/cow/year and the total cost of a high rate of subclinical mastitis in a 100 cow herd at £5,000 per year (Esslemont & Peeler, 1993).

Evidence from surveys has also shown an association between BTSCC and milk yield. Jones et al. (1984) analyzed the relationship between 67,707 observations of a Dairy Herd Improvement (DHI) programme test-day milk yield and ICSCC in 34 dairy herds over 3 years. The decrease of milk yield for second and later lactations, as ICSCC increased, was greater than for first lactations. In herds averaging less than 7,700 kg milk per lactation, as the ICSCC doubled, milk production fell by 0.36 - 0.72 kg per day per cow. Reduced milk yield has been estimated to be 69-80% of total mastitis cost (Janzen, 1970; Dobbins, 1977; Blosser, 1979).

Economic losses associated with mastitis are not limited to the farm. Losses also occur for the processor (Everson, 1984). Although the relationship between BTSCC and milk composition is indirect, low BTSCC milk has a higher total solids content (Asby et al., 1977). Furthermore udder infections cause major alterations in milk composition (King, 1969; Schultz, 1977). This affects its use in manufacturing dairy products (Richter, 1976; Everson, 1984). The manufacturer is also interested in the effect of mastitis on proteolytic enzyme activity in milk (Saeman et al., 1988). Proteolytic enzymes cause time and temperature-dependent breakdown of casein, the major milk protein. Proteolysis of casein results in decreased cheese yields, off-flavour development, and

decreased shelf-life of dairy products (Ali et al., 1980; Everson, 1984; Senyk et al., 1985). The relative proportion of the native milk proteolytic activity that originates from plasmin and non-plasmin enzymes is important, because these enzymes may have different characteristics (e.g. heat resistance) that will affect dairy product manufacturers. Milk proteolytic enzyme activity increases as SCC increases (Saeman et al., 1988). Native milk proteolytic activity can be separated into 2 categories: plasmin activity and non-plasmin activity. In high SCC mastitic milk, somatic cell (non-plasmin) proteases contribute to the total milk proteolytic activity. Fresh milk samples with high SCC had significant casein proteolysis indicating that some damage to casein had already occurred in the udder. After infections had been eliminated and milk SCC had dropped to preinfection values, proteolytic activity remained higher than preinfection (Saeman et al., 1988). Thus, detrimental effects of mastitis on milk quality continue even after intramammary infections have been eliminated and the SCC returns to normal.

1.3 Factors affecting Somatic Cell Count

Several factors make the interpretation and comparison of results concerning cell counts difficult. The relationships between cell counts determined by various direct methods like direct microscopic cell counting, electronic Coulter Counter and fluoro-opto-electronic Fossomatic are, among other things dependent on the levels of the cell counts (Szijarto & Barnum, 1984). Thus for example these authors found that the Coulter Counter instrument showed a higher count in low level cell count milk than the Fossomatic instrument while at higher levels the instruments yielded similar figures.

The somatic cell count of milk is influenced both by pathological and physiological conditions (David & Jackson, 1984). Investigations of environmental factors affecting SCC (Bodoh et al., 1976; Kennedy et al., 1982; Miller et al., 1983; Emanuelson, 1985) have shown herd, cow, year, lactation number and stage (Blackburn 1966; Cullen 1968; Bodoh et al., 1976; Bakken, 1981), methods of sampling (Cullen, 1967; Smith & Schultze, 1967) and management (Eberhart, 1986; Jones, 1986; Reneau, 1986) are important sources of variation. In Sweden corrective factors for these causes have been established and are used in their national milk recording service (Barnum, 1990).

1.3.1 Mastitis

The major factor causing high SCC figures is mastitis, whether clinical or subclinical, though of course only milk from cows with the latter should be included in bulk milk. Identification of the causative pathogen(s) involved is fundamental to the investigation of any mastitis problem. This information will help to identify the predisposing factors and thus the aspects of control to be concentrated upon (David & Jackson, 1984). It has been customary to classify mastitis pathogens by their origin and the term "environmental" bacteria has been widely used to indicate the types

of organism which are derived from the environment, e.g. *E. coli* and *S. uberis*. This distinguishes them from the contagious "parlour" micro-organisms which primarily exist on or in the mammary gland of the host and are most readily spread at milking time from infected to uninfected quarters, e.g. *S. aureus*, *S. dysgalactiae* and *S. agalactiae*. David & Jackson (1984) reported that they were prepared to interpret the bacteriology results from all of the 65% of clinical milk samples which yielded a single pathogen in pure culture, including *E. coli*. A further 5% of positive samples yielded a combination of pathogens. Some of the samples which failed to give an interpretable result were contaminated by extraneous bacteria which gained entry due to faulty sampling procedures. Approximately 15% of samples gave a 'no isolate' result on aerobic culture. The reasons given for this included mastitis not present; mastitis present but viable micro-organisms not present in sufficient numbers to be detected due to rapid elimination by the host; presence of inhibitory substances and finally that unsuitable laboratory methods were being used.

The SAC Veterinary Investigation Diagnosis Analysis (VIDA) mastitis diagnoses presented in **Table 1:1** were the only indication of the national prevalence of mastitis pathogens available at the beginning of this study. Although the number of submissions did show a marked increase in 1990 compared to the previous year, the number of specimens did not increase in the same proportion. It was therefore likely that these submissions continued to originate predominantly from clinical cases rather than batches of specimens from groups of high ICSCC cows with subclinical mastitis.

The identification of the aetiological agents involved in elevated SCC depends on bacteriological examination of appropriate samples which may be from the quarter, individual cow or herd bulk tank milk. Erskine & Eberhart (1988) analyzed the results of bacteriological culture of 5426 pairs of duplicate quarter milk samples for agreement. Overall the percentage of agreeing pairs was 98.1%. The repeatability of culture measured as the percentage agreement by infection type (as percentage of duplicate pairs yielding that organism in one or both samples) was greater for the contagious pathogens *S. agalactiae* (96.4%) and *S. aureus* (94.2%) than for other *Streptococcus* spp. (81.6%) and coliform organisms (55.6%). 96.4% of the *S. agalactiae*-yielding sample pairs were in agreement. With an error rate of less than 2%, it can be estimated that single quarter samples would identify more than 98% of the *S. agalactiae*-infected quarters identified by duplicate quarter samples. Thus single quarter samples may be adequate for determining the status of quarter infection with *S. agalactiae*. In contrast the 20% calculated disagreement between duplicate pairs yielding organisms of environmental origin suggests that neither single nor duplicate pairs would offer a high degree of accuracy in identifying intramammary infection with these organisms. Without considering age or stage in lactation, ICSCC of composite (all four quarters) samples from which a pathogen was recovered were significantly higher ($p < 0.001$) than those for samples from which no pathogens were isolated or those from which no bacteria were recovered (Brooks et al., 1982).

	1989	1990	1991	1992
Specimens (sp)	1762	2202	3080	3139
Submissions (sb)	692	979	952	811
Sp/Sb	2.5	2.2	3.2	3.9
Number (%) of diagnoses	604 (34)	964 (44)	904 (31)	797 (26)
S. aureus	15.2%	16.2%	21.4%	23.8%
S. agalactiae	9.6%	10.7%	11.5%	6.15%
S. dysgalactiae	11.4%	10.7%	15.0%	16.6%
S. uberis	15.9%	14.9%	11.0%	10.8%
E. coli	20.9%	24.2%	21.9%	22.6%

Table 1:1. SAC Veterinary Investigation Diagnosis Analysis 1989 - 1992: Mastitis diagnoses.

The "parlour" bacteria *S. aureus*, *S. dysgalactiae* and *S. agalactiae* have been incriminated as the major cause of intramammary infections in most dairy herds (Natzke, 1981; Smith, 1983; Dodd, 1983). These bacteria, which usually cause chronic subclinical infection are a major cause of increased BTSCC (Jain, 1979). Ward & Schultz (1972) found higher somatic cell counts and California Mastitis Test reaction were associated with the major pathogens *S. aureus* and *S. agalactiae* than with less pathogenic organisms. *S. aureus*, *S. agalactiae* and other streptococcal species were found to be the most frequent isolates from the 500 herds examined by Wilson & Richards (1980) to determine the prevalence of subclinical mastitis in the British dairy herd. They reported that the quarter prevalences of the various infections were *S. aureus* 8.1%, *S. agalactiae* 3.4%, *S. uberis* 1.5% and *S. dysgalactiae* 1.1%. Dodd & Neave (1970) reported that about 20% of staphylococcal and streptococcal infections were spontaneously eliminated. Griffin et al. (1977) reported that about 11% of quarter infections disappeared spontaneously. The IDF (Kastli, 1967) stated that where the milk and udder are macroscopically normal a QSCC of more than 500,000 together with the presence of pathogenic bacteria signifies subclinical mastitis. Using these criteria Wilson & Richards (1980) reported that the national prevalence of subclinical mastitis was 9.6% of all quarters. A subsequent survey of 300 herds in England and Wales showed the average incidence of clinical mastitis for 1982 to be 33 cases/100 cows/year with 21% of cows being affected at least once (David & Jackson, 1984). Decreased BTSCC was associated with a lowered prevalence of infection by contagious pathogens (Schukken et al., 1990). 2.4% of herds in England & Wales had an average BTSCC of less than 200,000 (Wilson & Richards, 1980). Unfortunately control of *S. agalactiae* and *S. aureus* did not result in the elimination of mastitis as a significant problem (Eberhart & Buckalew, 1972; Eberhart & Buckalew, 1977; Dodd, 1983; Smith, 1983; Oliver & Mitchell, 1984). The clinical mastitis cases on low BTSCC farms are generally caused by environmental pathogens. Paape et al. (1988) showed that a threshold level of somatic cells was necessary to prevent infection of the mammary gland after challenge with mastitis pathogens, notwithstanding that diapedesis, phagocytosis and bacterial killing also play a major role in the pathogenesis of mastitis (Hill, 1981; Oliver & Sordillo, 1988; Hill, 1988). These findings support the concept that a low QSCC may put a quarter at risk of infection. Consequently some field studies indicate that the incidence of clinical mastitis has not decreased in herds with a low BTSCC (Erskine et al., 1988; Hogan et al., 1989).

In situations of high herd infection prevalence, the presence of mastitis may contribute significantly to the TBC. All commercial dairy herds in the UK are subject to weekly testing of bulk milk supplies for TBC as a measure of milk quality: bonus and penalty payments are applied according to the level. This bacterial count refers to all organisms in the milk. Thus it will include saprophytic and thermoduric bacteria, faecal organisms such as *E. coli* as well as other major udder pathogens. Since milk from mastitic quarters can contain 10^5-10^6 bacteria/ml, two litres of such milk may raise the TBC by 10^5 bacteria/ml (David & Jackson, 1984). Herds with

satisfactory average TBC sometimes experience occasional wild fluctuations. This may indicate that mastitis cases were not detected promptly and mastitic milk thus entered the bulk tank. It is therefore essential for mastitis detection to be as thorough as possible especially where the milk is sent direct to the pipeline. Since mastitis-causing bacteria do not multiply rapidly in milk, relatively high numbers of bacteria associated with mastitis isolated from herd bulk milk with high somatic cell counts indicate that high numbers of those bacteria enter the bulk tank at each milking. Therefore the bacteria isolated from such samples may be considered the major species of bacteria involved in the herd's mastitis problem (Oz et al., 1986). The number of bacteria isolated from bulk milk samples collected by the milk haulier at the time of pickup was approximately 20% higher than the number of bacteria that entered the bulk tank at each milking. This percentage was slightly, but not significantly, lower in herd bulk tank milk with a very high somatic cell count, e.g. mastitic milk. Cultures of three or more consecutive bulk tank milk samples were recommended to evaluate or monitor the mastitis status of dairy herds (Oz et al., 1986). Isolation of *S. agalactiae* and *S. aureus* from bulk tank milk was considered strong evidence that intramammary infections caused by these bacteria existed in a herd (Gonzalez et al., 1986).

BTSCC epidemiology may be analyzed by the pattern of infection in high and low BTSCC herds. Oliver & Mitchell (1984) used an alternative approach by characterisation of herd type according to the predominant subclinical mastitis pathogen. They found that the predominant mastitis organisms in *S. agalactiae*-positive herds were *S. agalactiae* and *S. aureus* (**Tables 1:2-4**).

These results were to be expected as mastitis control procedures were poorly applied in these herds. The bacteriological results in *S. agalactiae*-negative herds differed markedly. The most frequently isolated pathogens in these herds were other streptococci, *S. aureus* and coliforms. The frequency of *S. aureus* isolation from quarter samples was markedly lower in *S. agalactiae*-negative herds compared with *S. agalactiae*-positive herds but still accounted for 25.2% of bacteria isolated. Environmental organisms (other streptococci and coliforms) accounted for over 50% of the bacteria isolated in *S. agalactiae*-negative herds despite the fact that all these herds were using the standard mastitis control procedures of post-milking teat dipping (PMTD) and dry cow therapy (DCT). This finding agrees with a number of other groups (Eberhart & Buckalew, 1972; Eberhart & Buckalew, 1977; Oliver & Mitchell, 1983; Smith, 1983) and suggests that infection in *S. agalactiae*-negative herds will not be lowered until procedures are developed for controlling environmental pathogens.

32 dairy herds, 16 with 12-month mean BTSCC less than 150,000 ("Low") and 16 with 12-month mean herd BTSCC greater than 700,000 ("High") were evaluated, by a single herd visit, to determine the relationship between the prevalence of mastitis and each mastitis control and management practice (Erskine et al., 1987). Duplicate quarter milk samples were collected from lactating cows and a survey of herd mastitis control, milking hygiene and management practices was performed and milking machine function evaluated.

Herd type	Cow	Quarter
S. agalactiae-positive	58.5%	37.0%
S. agalactiae-negative	26.3%	10.2%

Table 1:2. Prevalence of major pathogen infection by herd-type.
(Adapted from Oliver & Mitchell, 1984).

	Quarter Prevalence	All isolates
S. agalactiae	25.5%	69.0%
S. aureus	6.6%	17.8%

Table 1:3. Prevalence of mastitis pathogens in *S. agalactiae*-positive herds.
(Adapted from Oliver & Mitchell, 1984).

	Quarter Prevalence	All isolates
S. agalactiae	0.02%	0.20%
S. aureus	2.56%	25.2%

Table 1:4. Prevalence of mastitis pathogens in *S. agalactiae*-negative herds.
(Adapted from Oliver & Mitchell, 1984).

A significantly ($P<0.01$) higher prevalence of intramammary infection with *S. agalactiae* and *S. aureus* was observed in the high BTSCC group (**Table 1:5**). Only 2 of the 2696 quarters cultured bacteriologically in the low BTSCC group yielded *S. agalactiae*. In both groups, the quarters infected with major pathogens had the highest mean QSCC. Non-infected quarters had the lowest mean QSCC in both groups. For each type of infection, the mean QSCC tended to be lower in the low BTSCC group than in the high BTSCC group. The geometric mean QSCC in "non-infected" quarters with no isolate was threefold higher in the high BTSCC group than in the low BTSCC group. The use of post-milking teat dip and treatment of all cows in the herd at the start of the non-lactating period, so-called Dry Cow Therapy (DCT), was practised significantly more frequently in the low BTSCC group than in the high BTSCC group ($P<0.05$).

Major differences were not found between the two groups of herds in the maintenance and functional characteristics of the milking equipment (Erskine et al., 1987). Operating characteristics of the milking system or frequency of liner replacement or of regularly scheduled service of the milking system did not differ significantly between groups. The largest single difference in herd mastitis management practices between the two groups was the combined use of teat dipping and dry cow therapy. This is in agreement with previous reports (Kingwill et al., 1970; Eberhart & Buckalew, 1972; Natzke, 1981; Oliver & Mitchell, 1984). Schukken et al., (1989) also examined a large number (125) of low BTSCC (less than 150,000) herds but in this case with a high incidence of clinical mastitis The average size (lactating and dry cows) of farms selected was 50.9 cows providing a total of 6,369 cows with an average production 6,416 kg/cow in 306 days. A total of 1,140 clinical cases of mastitis, with at least one inflamed quarter, were reported from 946 cows. The average annual incidence was 17.9 cases per 100 cows (17.9%) and ranged from 0 to 80 clinical cases per 100 cows. Erb et al. (1984) recorded an incidence of 9.3%, Dohoo et al. (1982) recorded an incidence of 16.8% and Wilesmith et al. (1986) recorded an incidence of 28.2%. It can be concluded that mastitis is still a major concern in a large proportion of low BTSCC herds. Schukken et al. (1989) reported that the microorganisms most frequently isolated were *E. coli* (16.2%), coagulase negative staphylococci (13.0%), *S. aureus* (9.6%) and *S. uberis* (8.0%). Only 2 cases of *S. agalactiae* were found. Case studies on low BTSCC farms have reported that *E. coli* was the major cause of clinical mastitis (Jasper et al., 1975). A high incidence of clinical mastitis due to *S. aureus* mastitis was also reported in a case study by Hoblet et al. (1988). Although *S. aureus* was regarded as an important mastitis pathogen on high BTSCC farms, it may also be a significant problem in herds with a low cell count. *S. uberis* was present in 8% and *S. dysgalactiae* in 4.8% of the cases. The relative importance of these non-agalactiae *Streptococci* has also been shown by their high incidence in other surveys. Wilesmith et al. (1986) reported 22%, Robinson et al. (1983) reported 26.2%, and Erskine et al. (1988) 12.3%. *S. agalactiae* was cultured in only two cases (0.2%) by Schukken et al. (1989) and the low incidence of this pathogen in the majority of low BTSCC herds suggests that it can be eradicated from herds.

Variable	BTSCC <150,000 (n=16)	BTSCC >700,000 (n=16)
S. aureus	44% of herds 0.7% quarters	100% of herds 22.2% cows 7.6% quarters
S. agalactiae	12.5% herds 0% quarters	100% herds 50.9% cows 25.7% quarters
Other Streptococci	1.9% quarters	3.7% quarters
Post-milking teat dipping & Dry Cow Therapy	81.3% of herds	37.5% of herds

Table 1:5. Mastitis pathogen prevalence and control in herds with high or low BTSCC. (Adapted from Erskine et al., 1987)

Most of the clinical cases of mastitis occurred in early lactation. After correction for the number of calvings per month, the incidence of mastitis was highest in the early summer when the predominant isolates were those associated with bedding material, *E. coli* and *S. uberis*, although only 14 of the 125 herds practised zero-grazing. Similar results were found by Smith et al. (1985) in a herd with mainly environmental mastitis. Dohoo et al. (1982) found no evidence of seasonality, but their study included herds with much higher cell counts.

In summary, BTSCC is correlated with the prevalence of subclinical intramammary infection in dairy cows (Eberhart et al., 1982). Intramammary infection with major pathogens, particularly with streptococci and *S. aureus*, is the single most important factor associated with high BTSCC (Eberhart et al., 1982). Similarly low BTSCC herds have a low prevalence of infection with these organisms (Oliver & Mitchell, 1984; Schukken et al., 1989).

1.3.2 Other factors

Poutrel & Rainard (1982) considered the age-related increase in ICSCC to be mainly due to the higher incidence of infection as the cow gets older. The percentage of cows (composite samples) from which a bacterial pathogen was isolated increased significantly with age (Brooks et al., 1982). Wilton et al. (1972) reported that the incidence of clinical mastitis increased from the first to the fourth and later lactations and this was confirmed by Pearson & Mackie (1979). However Brooks et al. (1982) observed no change in the percentage of pathogens isolated throughout each lactation. Clearly there is a slight conflict between these observations. It would seem that the prevalence of infection increased more during the dry period than within each lactation (Dodd & Neave, 1970).

The somatic cell count of milk also depends upon the stage of lactation. Uninfected cows have high ICSCC in the first week of lactation, thereafter falling to a low level before rising again prior to drying off (Cullen, 1968). Increase in ICSCC with stage of lactation can be caused by response to infection as well as increased concentration due to declining yield or physiological effects associated with lactation that are independent of infection (Wiggans & Shook, 1987). A BTSCC rise can occur in herds with a well defined seasonal calving pattern in that part of the year with a large number of cows in late lactation as a physiological phenomenon, not related to mastitis. Bodoh et al. (1976) reported a significant ICSCC increase only when milk yield was below 4 kg/day in late lactation.

The oestrus cycle has been suggested as a factor which may influence the occurrence of clinical mastitis and selected biochemical and cytologic characteristics of milk (Anderson et al., 1983). Guidry et al. (1975) studied the effects of oestrus on circulating neutrophils, SCC, neutrophil phagocytosis, and occurrence of clinical mastitis. Oestrus did not significantly influence any factor studied. However King (1977) reported that an increase in SCC occurred at oestrus. The observation that SCC did not vary significantly during days of the oestrous cycle was consistent with the results of Guidry et al. (1975).

There are relatively few reports of any interaction between SCC and nutrition. However Erskine et al. (1990) reported that Selenium (Se) status did not affect the percentage of challenge exposures resulting in infection, duration, or severity of experimentally induced *S. aureus* mastitis. This conclusion differs from that of an earlier study in which Se supplementation enhanced mammary resistance to experimental *E. coli* infection (Erskine et al., 1989).

1.4 Mastitis control programmes

The objective of mastitis prevention programmes is to reduce intramammary infection (Bushnell, 1980; McDonald, 1984; Grommers et al., 1985). Three large-scale Mastitis Field Experiments (MFE) were conducted at the National Institute for Research in Dairying (NIRD) in England during the 1960's (Dodd & Neave, 1970). The first 2 studies, MFE1 and MFE2, involved several commercial dairy farms and compared various milking hygiene routines for reducing incidence of new intramammary infection (IMI). In MFE1 three hygiene levels were compared on 14 farms: "complete" (operators wore rubber gloves; teat-cup clusters pasteurised between cows; udders washed with single-service paper towels; teats dipped in sanitizer after every milking); "partial" (as complete minus pasteurization of teat-cup clusters); none (no hygiene practices). An average of 2 IMI developed per cow per year in the "no hygiene" group. An average of 1 IMI developed per cow per year in the "complete" and "partial" hygiene groups. The rate of new IMI was significantly reduced by the hygienic procedures, but the prevalence (% quarters infected) within herds did not decrease appreciably. Therapy and of course culling was required to shorten the duration of existing IMI in order to reduce the prevalence of mastitis. Dry cow therapy was evaluated in combination with hygiene practices in subsequent studies (Brander et al., 1975). A further 3-year field trial (MFE3) was conducted on 32 dairy farms near the NIRD. The "partial" hygiene programme was used in half the herds, and teat dipping alone was used in the others. Additionally, all cows received intramammary infusion with a specially formulated antibiotic preparation at drying-off. The level of infection decreased approximately 75% in all herds within 3 years. The benefits of teat dipping and dry cow therapy were not determined separately though Pankey et al. (1984) claimed that post-milking teat dipping with a germicidal agent was the single most effective method for reducing the rate of new IMI.

With the MFE data Dodd et al. (1969) calculated that the probability of a cow having the same infection state after 12 months was 70%. In spite of the large number of infections contracted and eliminated, 55% of the cows did not change their infection state at any time in the year, 39% were infected throughout and only 16% were never found to be infected. Mastitis control requires attention to multiple factors involving host, agents and environment (Hueston et al., 1987). BTSCC, and consequently mastitis status, can be maintained at an acceptable level by the use of established control techniques (David & Jackson, 1984). In an on-farm interview questionnaire survey of 498 randomly selected producers, Wilson & Richards (1980) reported that

63.5% used post-milking teat dip while 59.6% also used dry cow therapy. These mastitis control measures were responsible for a considerable reduction in the proportion of quarters infected with major pathogens. However, mastitis control measures effective in reducing infections caused by *S. agalactiae* and *S. aureus* were less so in preventing infections caused by environmental bacteria (Bramley & Neave, 1975; Eberhart, 1977; Smith, 1983; Bramley, 1984). Wilson & Richards (1980) reported that udder infections were less prevalent in herds where mastitis control measures were used. The prevalence of infection also declined as the size of herd increased. However as the uptake of mastitis control measures was greater in the larger herds it seems likely that the widespread use of control measures, including culling, was the most important factor responsible for their relatively low prevalence of subclinical mastitis (Wilson & Richards, 1980).

Hueston et al. (1987) used the variation in the number of high ICSCC cows within herds as a measure of mastitis prevalence in dairy herds. High ICSCC prevalence was calculated as the 12-month rolling herd average percentage of lactating cows with ICSCC in excess of 283,000. The presence of either coagulase-positive staphylococci or *S. agalactiae* in bulk-tank milk samples was associated with significantly higher high ICSCC prevalence. Three of the variables examined were associated with significant decreases in high ICSCC prevalence: absence of *S. agalactiae* in the bulk tank milk; adoption of post-milking teat dipping; the practice of dry-cow antibiotic therapy of all cows.

1.4.1 Pre-milking preparation

Previous studies have suggested that milking system (Bodoh et al., 1976) and method of udder preparation (Moxley et al., 1978) were significant sources of BTSCC variation. The effectiveness of udder preparation techniques have been studied in terms of milk quality, raw milk TBC and reduction of udder infection (Edwards & Smith, 1970; McKinnon et al., 1983; Galton et al., 1986a&b). Pre-milking udder preparation affects the number of bacteria on teats and in milk (Galton et al., 1982; Bushnell, 1985). Galton et al. (1984) reported that the TBC increased when teat surfaces were wetted and not adequately dried before milking and that procedures that wetted both udder and teat surfaces resulted in higher TBC in milk than those that wetted only the teats. Galton et al. (1986a&b) stated that manual drying of teats was an essential part of any procedure to achieve effective reduction of bacterial counts of milk. Thus as Pankey (1989) reported, milk bacteriological quality was clearly improved by effective udder preparation. In addition the incidence of mastitis can be reduced by effective pre-milking udder sanitation. Moreover, cases of clinical mastitis can be increased when ineffective techniques of pre-milking preparation are used (Pankey, 1989).

Disinfection of the teat cup liner may aid the control of *S. aureus* but the benefits were small and uneconomic. This procedure did not control environmental mastitis. Smith et al. (1985) concluded that the results did not justify the use of backflushing milking clusters in a herd with a

low prevalence of contagious pathogens.

The failure of conventional methods to control environmental bacteria led to the development and testing of other control measures such as dipping teats in disinfectant before milking (Galton et al., 1982; Pankey & Wildman, 1985). Pankey & Wildman (1985) reported preliminary data from a herd trial that revealed a 61% reduction in new infection. The effect of pre-milking teat dipping has been evaluated using both experimental challenge (Galton et al., 1988) and by natural infection (Pankey et al., 1987). Compared to no preparation, washing and drying significantly reduced new infection by 43% and predipping and drying by 66%. Both these reductions were significant and the predipping treatment was significantly superior to washing and drying. Galton et al. (1988) reported that pre-milking teat dipping plus drying further reduced IMI by 41.0% compared with the use of wet towels plus drying. Pankey et al. (1987) reported predipping significantly reduced udder infections with environmental pathogens by more than 50%. In a trial of an iodine pre-milking teat dip Blowey & Collis (1992) reported that the mean incidence of clinical mastitis was reduced by 57%, the total bacterial count by 70% and the count of thermoduric organisms by 32%. There was no effect on somatic cell count, milk production or milk iodine residues. Effects of udder hygiene practices on iodine residues in milk were studied by Galton et al. (1986b). The data suggest that iodophor post-milking teat dipping may be a more important contributor to milk residues than pre-milking teat dipping. Several factors are related to iodine in milk. The major source of iodine in milk was the feed rather than iodine teat dips or sanitizers (Bruhn & Franke, 1978; Hemken, 1979; Blowey & Collis, 1992). High iodine levels in milk (6,000 μg iodine/l) were not high enough to have any bacteriostatic effects on organisms in the udder (Ruegsegger et al., 1983).

1.4.2 Post-milking dips

Numerous studies, reviewed by Pankey et al. (1984), have demonstrated the merits of teat dipping or teat spraying in the control of contagious mastitis pathogens such as *S. aureus* or *S. agalactiae*. Post-milking teat dipping has been consistently identified as a significant herd determinant of low BTSCC (Pearson et al., 1972), and ICSCC (Bodoh et al., 1976). Moak first advocated teat dipping in 1916 (Moak, 1916; Pankey, 1984). A dilute pine oil solution was used to reduce the spread of *S. agalactiae*. The practice was not adopted widely because products were ineffective and supportive data were not conclusive. In the late 1950's interest was revived in teat dipping by the demonstration of a reduced staphylococcal population on milking machine liners following use of germicidal teat dips (Newbould & Barnum, 1958 & 1960; Pankey, 1983).

Pankey (1984) defined a good teat dip as one which will reduce new IMI 50 to 90% as measured within the controlled trials of the IDF teat dip evaluation protocols. The effectiveness of teat dipping with a germicidal solution post-milking to control new IMI resulting from staphylococcal and streptococcal pathogens has been shown (Eberhart & Buckalew, 1972; Pankey

et al., 1983; Nickerson et al., 1986). However, the majority of post-milking teat dips provide little or no protection against infection by coliform bacteria (Eberhart & Buckalew, 1972; Eberhart, 1984). The post-milking use of an acrylic latex teat dip without germicide proved effective in reducing coliform infections by 76% and *S. aureus* and coagulase-negative staphylococci (CNS) by 28% and 33% respectively (Farnsworth et al., 1980). Latex teat dips were designed to form a physical barrier over the teat end to prevent environmental bacterial contamination between milkings (Matthews et al., 1988). The 50% reduction in IMI by contagious pathogens was because transfer at milking time and growth on teat skin and lesions were crucial in the pathogenesis of infections by the major mastitis pathogens *S. aureus* and *S. agalactiae*. They were less effective in the control of mastitis caused by environmental pathogens because of the difference in the pathogenesis of these forms of mastitis (King, 1981a&b).

Pankey (1984) reported that teat dipping prevented many new infections, but the duration of existing infections was not shortened. Teat dipping, practised alone, required several months before the level of infection in a herd was reduced substantially. The impact of teat dipping on the level of mastitis was enhanced by simultaneous use of dry cow therapy and culling, measures designed to reduce the duration of existing infections. Pankey (1984) considered that although there are several main classes of post-milking teat sanitizers, the management practices on individual dairy farms had more effect on reduction of rate of infections than did small differences in product efficacy. Francis (1984) reported that an average figure for teat disinfectant use was 1 litre diluted chemical/100 cows/day or 3.3 (1.4 - 6.0) litres/cow/year. Teat disinfecting sprays used twice these average quantities (Francis, 1984).

1.4.3 Management

Several authors have examined the relationship between management practices and the effectiveness of mastitis control (Hueston & Heider, 1986). Fox & Hancock (1989) found that milking cows infected with *S. aureus* last at each milking did not reduce the prevalence of infected cows. Hutton et al. (1990) analyzed the effects of differences in herd mastitis control management in maintaining low herd average BTSCC. In contrast they found that cows which had *S. aureus* clinical mastitis were milked last in approximately half of the low BTSCC herds as contrasted to only 13% of the high BTSCC herds. 96% of the low BTSCC herds routinely disinfected teat ends prior to intramammary infusion, whereas significantly fewer (67%) of the high BTSCC herds adopted this practice. Automatic milking unit detachers were used on more low than high BTSCC herds. Number of cows per dairy and milking parlour size and efficiency (cows milked/man/hr) were greater in low than high BTSCC herds. A greater percentage of low SCC herds culled cows because they had mastitis. Pearson et al. (1979) reported a higher frequency of post-milking teat dip use in low than high BTSCC herds. In contrast, individual paper towels were used to prepare udders before milking as frequently in the high BTSCC group as in the low BTSCC group. This

is in agreement with the findings of Moxley et al. (1978) and Pearson et al. (1972) that there was no significant relationship between the use of individual paper towels and lower BTSCC.

1.4.4 Treatment

The probability of infection increases and milk production decreases with increasing ICSCC (Jones et al., 1984). Schultz et al. (1978) attributed approximately 70% of the economic loss to reduced milk production caused by subclinical mastitis. Dodd & Neave (1970) considered that antibiotic therapy was essential to reduce the duration of infection and thus the incidence of mastitis. Dodd et al. (1969) stated that clinical mastitis was nearly always preceded by subclinical infection. Approximately 33-50% of the subclinical infections postpartum resulted in clinical symptoms of mastitis during lactation (Philpot, 1969; Neave et al., 1969). It has been suggested (Dodd et al., 1969; McDermott et al., 1983) that treatment of subclinical infections may reduce both the rate and duration of new infections. However McDermott et al. (1983) administered antibiotic therapy to cows with subclinical mastitis, based on elevated SCC, and found no advantage in milk production over control cows treated only for clinical cases of mastitis. Timms & Schultz (1984) reported no significant decrease of SCC (either composite or quarter) following intramammary treatment after a single high ICSCC (in excess of 400,000). Seymour et al. (1989) also found treating cows for subclinical mastitis based on elevated SCC did not significantly improve milk production. Thus treatment of animals after high SCC (subclinical infections) is difficult to justify economically except in the eradication of *S. agalactiae* (Edmondson, 1989; Erskine & Eberhart, 1990; Kirk et al., 1994).

While antibiotic treatment of mastitis infections during lactation generally will eliminate less than 60% of the pathogenic infections, therapy at drying-off eliminated over 90% of the *S. agalactiae* and 40-70% of the *S. aureus* infections (Natzke, 1971). Philpot (1979) reported success rates for therapy of clinical mastitis of 24.8% for *S. aureus*, 51.6% for *S. agalactiae*, 36.0% for other streptococci, and 71.4% for coliforms. McDermott et al. (1983) reported that treating subclinically infected cows did not decrease the SCC significantly and produced a bacteriological cure rate for major and minor pathogens combined of only 23.3%. They concluded that it was generally more economical to defer this treatment until drying off when treatment would likely be more effective. Timms & Schultz (1984) reported that the bacteriological cure rate for cows with clinical mastitis was 21.6% for major and minor pathogens combined. Composite and QSCC decreased significantly following treatment. They concluded that lactation therapy in a herd that has been on a teat dipping and dry cow therapy programme for some time was relatively ineffective.

Several authors have examined the relationship between *in-vitro* and *in-vivo* antibiotic sensitivity. Craven & Anderson (1980 & 1983) concluded that host factors, such as intracellular sequestration of bacteria and impairment of antibiotic distribution in diseased mammary tissue,

must be considered as well as the known bacterial resistance mechanisms where therapeutic failures occur in *S. aureus* mastitis. Mackie et al. (1988) tested eight hundred and forty-eight strains of *S. aureus* and coliforms isolated from milk samples taken from cows with clinical mastitis or subclinical mastitis for their sensitivity to a range of antibiotics, comparing strains isolated in 1984, 1985, 1986 and 1987. Their finding that all antibiotics had small fluctuations from year to year in their effectiveness against the different pathogens is similar to that of Davidson (1980) who conducted a similar five year study in the USA and supports the view that antibiotic resistance in bacteria has not increased during the last 20 years (Craven et al., 1986; Walton, 1988).

1.4.4.1 Dry Cow Therapy

The advantages of dry period therapy over lactation treatment are well recognized (Philpot, 1969; Dodd & Neave, 1970; Natzke, 1971; Dodd & Griffin, 1975; Philpot, 1979; Natzke, 1981). Advantages include higher cure rate than with lactational therapy, prevention of new dry period infections, allowance for regeneration of damaged tissue, reduction of clinical mastitis at parturition, and elimination of drug residues in saleable milk (Philpot, 1969 & 1979). Field trials which measured the effects of post-milking teat dipping and dry period therapy programme showed a reduction of 50-70% within 1 - 3 years in the percentage of cows or quarters with intramammary infections (Wilson & Kingwill, 1975; Harmon et al., 1986). Results from controlled studies on the efficacy of specific dry period therapeutic preparations support the findings of the field trials with overall cure rates for staphylococci generally above 50% and for most streptococci above 80% (Dodd & Griffin, 1975; Philpot, 1979).

Neave et al. (1950) found that staphylococci and streptococci invaded 24% of previously uninfected quarters during the dry period, principally during the first few weeks. About half of these infections persisted into the next lactation and about half of the persistent infections became clinical. This rate of infection during early involution was over six times that observed during lactation, though Oliver (1988) reported only a threefold increase. Natzke (1971) reported that without dry cow therapy the rate of new infection was 10-15%. It is now generally accepted that without dry cow therapy, approximately 8-12% of quarters in herds with average infection levels will become infected during the dry period despite the presence of natural protective factors such as lactoferrin (Breau & Oliver, 1986; Bushe & Oliver, 1987). These infections reduced milk production by 36% during the first month of the subsequent lactation (Smith et al., 1968).

1.4.4.2 Modifications of Dry Cow Therapy

Poutrel & Rainard (1981) used California mastitis test scores and bacteriological analysis of quarter foremilk samples to determine which quarters or cows to treat in a selective dry cow therapy programme. Selective treatment of all cows that had one or more positive quarters led to treatment of twice as many quarters than if only positive quarters were treated. The most discriminating,

simplest and economic method of selective treatment was one test on quarter foremilk samples collected 8 weeks before the expected dry-off day and dry treatment of all positive mammary quarters. However the risk of new infection should be less in herds with low infection. Incidence of mastitis following the dry period was less with complete therapy (4.6% of the quarters) compared to selective therapy (7.8% of the quarters) (Rindsig et al., 1978). Selective therapy was as effective in eliminating existing infections. While the difference in rate of new infection between complete (3.1%) and selective therapy (6.5%) looked small, it was important in terms of the overall herd infection because it was a percentage of all quarters rather than just those infected. The rate of new infection under a selective therapy programme was affected by the proportion of quarters infected at drying-off, the proportion of cows in the herd selected for therapy and the efficacy of the intramammary infusion product. In any type of selective therapy some quarters will be missed and any quarter not treated would be more susceptible to new infections. Complete therapy would be the choice in situations where new infections in the dry period are of concern. In a study of herd factors associated with ICSCC, selective dry cow therapy was associated with lower SCC than complete dry-cow therapy (Bodoh et al., 1976). In a 100 cow herd, the production gain from preventing nine new IMI would pay for the cost of treating all cows at drying off (Nickerson, 1990). A typical infected quarter treated at drying-off and cured at calving produced 90% of its potential during the next lactation. However cure rates depended on the organism since the overall cure rate for *S. aureus* was over 50% and that for streptococci greater than 80% (Nickerson, 1990). Alternatively, quarters that became infected during involution or were infected at drying-off and remained infected at calving produced 30-40% less milk (Natzke, 1982). In general it would appear that treatment of all quarters of all cows at drying-off was a safer option than selective treatment in all but very low BTSCC herds.

Natzke (1982) reported the results of 2 field trials that indicated no benefit from multiple sequential infusions of antibiotic in the dry period. Natzke (1982) suggested that multiple antibiotic infusions in the dry period had the potential to actually increase the risk of introducing pathogens into the mammary gland. The β-lactam antibiotics do not enter the intracellular space and, therefore, the phagocytosed staphylococci are not affected by these antibiotics. Dry cow antibiotic therapy with cloxacillin is well established as a means of controlling and eliminating new and existing mammary infections (Philpot, 1969; Dodd & Griffin, 1975; Natzke, 1981). Previous studies indicated that 70-98% of infections were eliminated by therapy with cloxacillin (Clegg et al., 1975; Harmon et al., 1986). Smith et al. (1975) found that cloxacillin in a slow-release base infused at drying off and 2 weeks later was more effective than either a quick or slow-release cloxacillin preparation infused only at drying off. Multiple infusions of cloxacillin did not improve efficacy but decreased the occurrence of new infections (Cummins & McCaskey, 1987). Based on covariant analysis of the data, multiple dry cow therapy with cloxacillin had no effect on SCC. The number of infected quarters per cow detected at the start of lactation was decreased

significantly by multiple dry cow therapy relative to the negative control (Cummins & McCaskey, 1987). Logue et al. (1993) also reported a slight advantage to two dry cow treatments during the dry period compared to one. However they doubted if this was economic in anything other than exceptional circumstances.

1.5 Summary

The literature revealed that infection by recognised mastitis bacteria was the main cause of raised QSCC and hence of elevated ICSCC (Brolund, 1985). This ICSCC elevation in response to infection provided a useful management tool for the detection of subclinically infected cows (Reneau, 1986) especially since only 40% of subclinical infections ever became clinically apparent (Dodd & Neave, 1970). In most herds a fixed ICSCC threshold of 250,000 both detected about 80% of infections and also correctly classified about 80% of non-infected cows (Andrews et al., 1983). Infection by those mastitis bacteria particularly associated with the "parlour" (*S. aureus*, *S. dysgalactiae* and *S. agalactiae*) rather than the "environment" (*S. uberis* and *E. coli*) was the most common cause of raised SCC (Dodd, 1983). However the prevalence of all types of subclinical infection was the most important cause of raised BTSCC at the individual herd level (Eberhart et al., 1982) and, by extrapolation, raised "Board SCC" of the national herd (Schukken et al. 1992a). However the identification of cows subclinically infected by *S. aureus* yet with low ICSCC posed two particular problems (Hoblet et al., 1988). Firstly the ICSCC threshold required to identify all such infected cows on the basis of a single test would lower the test specificity unacceptably. Secondly the accuracy with which BTSCC could estimate herd prevalence of all types of infection was reduced. The seasonal variation in national Board SCC and thus, by inference, in herd BTSCC would have a similar effect on such estimates of herd infection prevalence (Schukken et al., 1992a). Thus the last comprehensive UK survey used quarter milk samples to accurately establish the prevalence of subclinical mastitis at 9.6% of all such samples (Wilson & Richards, 1980).

In addition to infection *per se* two further sources of QSCC and ICSCC statistical variation were recognised (Brolund, 1985). These were firstly stage of lactation (Blackburn, 1966) and secondly lactation number (Bodoh et al., 1976). Physiological variation explained the two normal periods of high ICSCC in uninfected animals which were related to stage of lactation. These occurred just after calving due to high SCC colostral milk and prior to drying-off, this time as a dilution effect from reduced milk yield (Schultz, 1977). By contrast, it was actually infection which caused the increased prevalence of high ICSCC with lactation number. More particularly there was a significant increase in the proportion of subclinically infected cows with lactation number (Brooks et al., 1982).

The identification of the main cause of raised BTSCC as the prevalence of cow infection by "parlour" pathogens fulfilled the first objective of the review of the literature. Furthermore

stage of lactation, lactation number and season were demonstrated to influence SCC. However the review did not reveal a report in which all these recognised components of SCC variation were brought together in a herd investigation strategy. This made it virtually impossible for the practising veterinarian to assimilate the information and merely compounded their difficulty in having any sort of regular access to their clients' SCC data let alone having an advisory input on milk quality.

Mastitis control was a key element of quality milk production (Senyk et al., 1985). In contrast to the foregoing review on SCC, the literature on mastitis control did include a comprehensive single report which collated the individual components into a practical strategy (Wilson & Kingwill, 1975). Although not completely successful, it reduced the herd prevalence of subclinically infected cows or quarters by up to 70% within 1 - 3 years of its adoption. The strategy was based on two principles. Firstly milking hygiene and in particular post-milking teat dipping. This was a significant herd determinant of low SCC (Pearson et al., 1972; Bodoh et al., 1976) because it could reduce new intramammary infections by up to 90% (Pankey, 1984). Secondly antibiotic treatment but particularly dry period rather than lactation therapy. This itself had two main advantages (Natzke, 1981). Firstly the production gain in the subsequent lactation from the prevention of new dry period infections. In a 100 cow herd, this gain in just nine cows paid for the cost of treating the whole herd at drying off (Nickerson, 1990). Secondly a higher bacteriological cure rate than with lactational therapy (Philpot, 1969 & 1979). While the bacteriological cure rate for antibiotic treatment of mastitis infections during lactation was only between 23 and 60%, therapy at drying-off eliminated over 90% of *S. agalactiae* and 40-70% of *S. aureus* infections (Nickerson, 1990). Furthermore the treatment of lactating cows after high SCC (subclinical infections) was difficult to justify economically except in herd-eradication of *S. agalactiae* (Edmondson, 1989; Erskine & Eberhart, 1990). This was because there was no advantage in terms of quarter and individual cow SCC or milk production (Seymour et al., 1989).

However this strategy failed to adequately control "environmental" (*S. uberis* and *E. coli*) mastitis (Oliver & Mitchell, 1984). This failure focused attention on pre-milking udder preparation particularly since Pankey (1989) reported that cases of clinical environmental mastitis increased when ineffective techniques of pre-milking udder preparation were used. The use of individual paper towels to prepare udders before milking did not seem to be associated with lower BTSCC (Moxley et al., 1978). Pre-milking teat dips were therefore developed (Pankey & Wildman, 1985) and reduced new environmental infections by more than 50% (Pankey et al., 1987).

The disparate reports in the literature demonstrated the potential use of SCC data within a milk quality advisory service for both field investigation and education. An appropriate ICSCC threshold could clearly identify infected cows for bacteriological investigation economically. The production and quality benefits of low SCC could be quantified and appropriate presentations made to veterinarians and their clients.

CHAPTER 2. THE DEVELOPMENT OF A SYSTEM FOR ANALYSIS OF MASTITIS RECORDS.

2.1 Introduction

One of the main sources of data about mastitis in the UK is the submission records of the Veterinary Investigation Services. However the information from each submission is limited to the SCC and bacteriology for each sample since production details such as age and stage of lactation are generally not provided. In 1992 each submission was restricted to an average of 3.87 specimens, suggesting as in 1989 and 1990 that they were from clinical cases rather than the investigation of a group of high SCC cows (Anon, SAC 1992). The relative proportion of the major mastitis pathogens in all submissions was calculated for Scotland. This showed that *S. aureus* accounted for 23.8% of all mastitis diagnoses and thus was easily the most common "parlour" isolate compared to 16.6% by *S. dysgalactiae* and 6.15% by *S. agalactiae*. *E. coli* accounted for 22.6% of "environmental" mastitis diagnoses and *S. uberis* a further 10.8%.

The last comprehensive survey of mastitis in the UK was reported in 1980 (Wilson & Richards, 1980). They reported that the quarter prevalence of the various mastitis pathogens was *S. aureus* 8.1%, *S. agalactiae* 3.4%, *S. uberis* 1.5% and *S. dysgalactiae* 1.1%. The collation of ICSCC data within the "DAISY" system is the only contemporary information available on the national incidence of subclinical mastitis (Esslemont & Peeler, 1993).

Once the link between infection, mastitis and cell count was made the SCC became an important parameter for estimating mastitis prevalence in cattle populations. While estimates based on BTSCC can be quite inaccurate nevertheless Pearson & Greer (1974) estimated that 42% of cows had subclinical mastitis when the herd BTSCC was between 500,000 and 800,000. However ICSCC, now readily accessible to producers, does have a fairly good correlation with infection and thus allows one to initiate an investigation of the problem without the need for a costly visit (Cassel et al., 1994; Peters et al., 1994a&b).

Levels of ICSCC previously proposed as a threshold above which milk production is adversely affected range from 148-283,000 (Reneau, 1986; Thurmond, 1990). However the difficulty of employing an ICSCC threshold for the diagnosis of subclinical mastitis has already been discussed. Using the "Linear Score" (LS) system developed by Shook (1982) for reporting ICSCC to dairy producers as a scale of 0-9 then 283,000 is the start of LS5. The difficulty of using this ICSCC threshold was illustrated by Hoblet et al. (1988) who conducted a total herd bacteriological culture survey of a low SCC herd experiencing an outbreak of clinical mastitis. Despite 87% of the cows in the herd having ICSCC below 283,000, coagulase-positive *Staphylococcus* spp. were isolated from 11.3% of the individual cow samples.

This chapter describes the development of a system for analysis of the bacteriological and SCC data from selected Scottish mastitis investigation records. The data used was that obtained by SAC Aberdeen over the period 11/02/1974 to 15/05/1990 and included bacteriological

information on the whole herd, QSCC or ICSCC data and details of the age and stage of lactation of the individual cow. The data was used to establish the prevalence of the major subclinical mastitis pathogens in a defined area of Scotland and to develop guidelines for the analysis of SCC for use in the studies described in Chapter 3.

2.2 Materials & Methods

2.2.1 Herds

A series of herds were referred to SAC Aberdeen in the period 11/02/1974 to 15/05/1990 for investigation of an elevated bulk tank Total Bacterial Count (TBC). Scientific staff undertook a whole herd bacteriological survey to identify infected cows and the aetiological agent(s) causing subclinical mastitis. The general description of the investigation protocol presented below was furnished by Dr David Fenlon, SAC Aberdeen. Milk samples were collected from all lactating cows at a farm visit during afternoon milking. The udders were prepared by the dairyman using the herd routine, which in most instances involved washing the teats. SAC Aberdeen scientific staff sterilised each teat end by vigorous wiping with a cotton wool plug soaked in 70% v/v ethanol (British Drug House, England).

Two protocols were used based on herd size. In smaller herds (mean 47 cows) quarter samples were taken into sterile 10ml McCartney bottles for all cows in the herd. In larger herds (mean 98 cows) a composite sample of all lactating quarters was collected from each cow to fill an individual bottle. The samples were refrigerated at 4°C overnight at the laboratory prior to examination. The sample was divided and a portion fixed for SCC determination using a Coulter TAI Counter (Coulter Electronics, Luton, Bedfordshire). This composite sample was taken as the "equivalent" of an ICSCC.

2.2.2 Examination

Determinative bacteriology was performed by inoculation of 0.1μl of the remaining unfixed milk sample on to sheep blood agar (Oxoid blood agar base No. 2 with 5% defibrinated sheep blood) which was then aerobically incubated for 24 hours at 37°C. Significant mastitis bacteria (**Table 2:1**) were presumptively identified by colony morphology and subcultured. White or yellow 2-4mm diameter circular convex colonies with a zone of haemolysis (α, β or both) typical of *S. aureus* were subcultured on to sheep blood agar to achieve a pure growth. *S. aureus* was identified by the presence of coagulase using commercially available rabbit plasma (Biomerieux, France) until 1988, when hyperimmune serum (Staphaurex, Wellcome Diagnostics, Dartford, England) became available. Colonies typical of mastitis streptococci were subcultured from the primary sheep blood agar after 24 hours incubation on to Edward's crystal violet medium and incubated aerobically for a further 24 hours at 37°C. *S. agalactiae* was presumptively identified by a zone of blue complete (β) haemolysis and confirmed as such by a positive CAMP (Darling, 1975) test. *S. dysgalactiae*

Code	Pathogen	Abbreviation
-	No significant isolate	NSI
1	*S. aureus*	SFAU
2	*S. agalactiae*	SPAG
3	*S. dysgalactiae*	SPDY
4	*S. uberis*	SPUB
8	*E. coli*	ESCO

Table 2:1 **Numerical coding of mastitis pathogens used in SAC Aberdeen and other databases.**

colonies were surrounded by a zone of green, incomplete (α) haemolysis. Colonies of *S. uberis* were surrounded by a brown zone of aesculin hydrolysis, a characteristic shared by *S. bovis* and *S. faecalis*. From 1988 the identity of mastitis streptococci was confirmed by determination of the Lancefield grouping using a slide agglutination test (Streptex, Wellcome Diagnostics, Dartford, England). In addition, *S. uberis* was positively identified by its sugar fermentation pattern (Anon, MAFF 1984).

2.2.3 Data analysis

The data was in the form of printed records of the results of investigations on either an individual quarter sample or a composite of all quarters designated an individual cow sample. Samples were identified by the individual herd and cow number and, where appropriate, quarter. Date of sampling, lactation number and month of lactation were also recorded. The SCC data was recorded with the bacteriology results as individual herd reports of quarter samples (**Table 2:2**) or composite samples (**Table 2:3**). **Table 2:1** shows the numerical codes used to record the bacteriology results.

All computing tasks undertaken within this thesis were performed on a 486DX, 250Mb personal computer (Datalink Computers, Edinburgh) running Version 6.0 of the MS-DOS (Microsoft, Redmond, USA) operating system. The data was entered manually into an ASCII-format computer file to create 2 separate databases, one for individual quarter samples and one for composite samples (**Table 2:4**). "Minitab 8" (Minitab 8 Committee, 1991) statistical software was used for the initial quantitative analysis of both databases. The lactation number of 52 cows (208 samples) in database 1 (quarter) and 145 cows in database 2 (composite) recorded as 8 to 22 was re-coded and incorporated in a "7+" group in the respective databases. Month of lactation above 10 was treated as a missing value so that the results of the data analysis could be interpreted using the standard 305-day lactation. This applied to 92 cows (326 samples) in the quarter database and 348 cows in the composite database. Statistical analysis of SCC was performed by Mr A. Sword on logarithm-transformed data following the recommendations of Shook (1982). The limits of Linear Score 5 (283,000 to 566,000) were chosen for investigation because this was a recognised scale for the interpretation of SCC data and provided a margin round the EC limit of 400,000 (Logue et al., 1994). Genstat (Genstat 5 Committee, 1987) statistical software was used for this analysis. The entire SCC dataset was first analysed and then restricted to LS5 samples (in excess of 283,000). Chi-square and T-test methods of statistical significance were applied as appropriate (Mead & Curnow, 1983). Logistic regression (Collett, 1991) was used to examine the relationship between lactation number and prevalence of infection.

Herd	Lactation		QSCC in thousands (Mastitis Pathogen Code*)			
	Month	Number	LF	LH	RF	RH
034	4	3	320(-)	230(-)	4800(-)	2940(3)
	10	2	770(-)	900(-)	1930(1)	1960(1)
	6	1	420(-)	7100(1)	590(-)	1040(-)

* See Table 2:1 for numerical coding of mastitis pathogens

Table 2:2. Format of SAC Aberdeen Quarter SCC (QSCC) data.

Case Number	Lactation		ICSCC(Isolate Code)
	Month	Number	
028	9	1	150(1)
	6	4	1440(-)
	7	4	480(2)

Table 2:3 Format of SAC Aberdeen Individual Cow SCC (ICSCC) data.

Database	Sample type	Number of Herds
1 (Quarter)	Quarter	31
2 (Cow)	Composite	55

Table 2:4. SAC Aberdeen Investigations 1974-1990: Number of herds and sample type.

2.3 Results

2.3.1 Quarter Somatic Cell Count Data

The results from a total of 5,860 quarter milk samples from 1,465 cows in 24 different herds (31 herd tests) was recorded as database 1 (**Table 2:4**). Data analysis was performed on 5,807 quarter bacteriology results which recorded either the isolation of a single significant mastitis pathogen (*S. aureus*, *S. agalactiae*, *S. dysgalactiae*, *S. uberis*, *E. coli*) or no significant isolate (NSI). **Table 2:5** shows a summary of database 1.

The complete bacteriology results were not available for 53 (1%) quarters in 41 (3%) cows (**Table 2:6**). The data in **Table 2:6** include isolations of different bacteria from the same cow. The results presented include those cows from which more than one type of significant mastitis pathogen was recovered and thus allow direct comparison of cow and quarter infection prevalence.

A significant mastitis pathogen was isolated from 493 (35%) of the 1424 cows from which quarter milk samples were collected for bacteriological examination and a significant mastitis pathogen was isolated from 805 (14%) of the 5807 quarter milk samples examined. *S. aureus* was the most common major mastitis pathogen and was isolated from 504 (8.68%) of all quarter samples (**Table 2:6**). *S. agalactiae* was the next most frequent isolate and was found in 171 (2.94%) quarter samples. **Figure 2:1** presents the number and proportion of significant isolates graphically. In relation to the epidemiology of the pathogens, 753 (94%) isolates were regarded as "parlour" organisms (*S. aureus*, *S. agalactiae* and *S. dysgalactiae*) while only 52 (6%) were of "environmental" (*S. uberis* and *E. coli*) origin.

The SCC of quarter samples was significantly raised by the presence of a mastitis pathogen. The data relating to *S. aureus*, the most common isolate, are presented in **Tables 2:7 & 2:8** to illustrate this. The $\log_{10}$(QSCC) of quarter samples from the first month of lactation not yielding a significant mastitis pathogen was 2.347 (222,000) (**Figure 2:2**). In contrast the $\log_{10}$(QSCC) of quarter samples from the first month of lactation from which *S. aureus* was isolated was 2.931 (853,000). The results presented in **Table 2:8** also show that infection caused a significant ($P < 0.001$) increase in QSCC irrespective of the age of cow (**Figure 2:3**).

A considerable number of cows were infected in 1 or more quarters by the major mastitis pathogens (**Table 2:9**). The majority (411, 51%) of the 805 isolates came from 200 cows infected in 2 or more quarters. *S. agalactiae* and *S. aureus* were particularly associated with multiple quarter infections. 100 (58%) of the 171 *S. agalactiae* isolates were from cows infected in at least 2 quarters. Similarly 274 (54%) of the 504 *S. aureus* isolates were from multiple quarter infections.

The distribution of the significant pathogens isolated from samples with QSCC of less than 283,000, 283 to 566,000 and greater than 566,000 was examined to detect differences in the proportions of the pathogens isolated. The chi-square statistic of 14.375 on 6 degrees of freedom indicated that the distribution of isolates based on the QSCC was significantly different ($P < 0.05$).

Information		Cows	Quarter Samples
Herd Number	Total	1,465	5,860
	Mean (s.d.)	47 (23)	189 (92)
	Median	49	196
	Range	1-94	4-376
Lactation Month Number		1,334 1,394	5,492 5,652
QSCC		1,465	5,860
Bacteriology		1,465	5,807

Table 2:5. SAC Aberdeen Quarter Sample Database.

	Cows	Quarters			
		All	SCC < 283	SCC 283-566	SCC > 566
Total	1,465	5,860	3,325	929	1,606
Missing bacteriology	41	53	16	7	30
No significant isolate	931	5,002	3,209	827	966
Significant isolate	493	805	100	95	610
S. aureus	344	504	69	71	364
S. agalactiae	111	171	13	14	144
S. dysgalactiae	63	78	9	5	64
S. uberis	44	49	9	5	35
E. coli	3	3	0	0	3

Table 2:6. Significant mastitis pathogens isolated in SAC Aberdeen investigations: Quarter Sample Database (1974-1990).

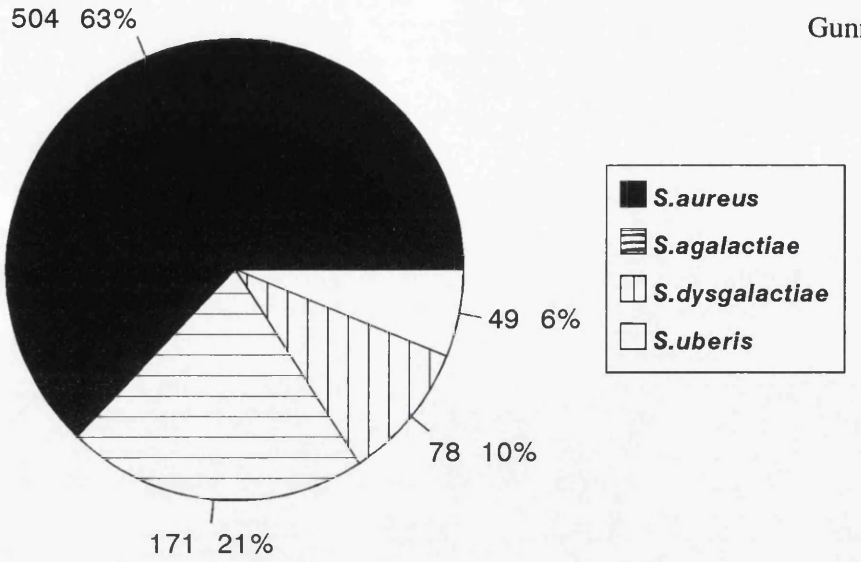


Figure 2:1
Isolates from SAC Aberdeen Quarter Milk Samples
(n=5860)

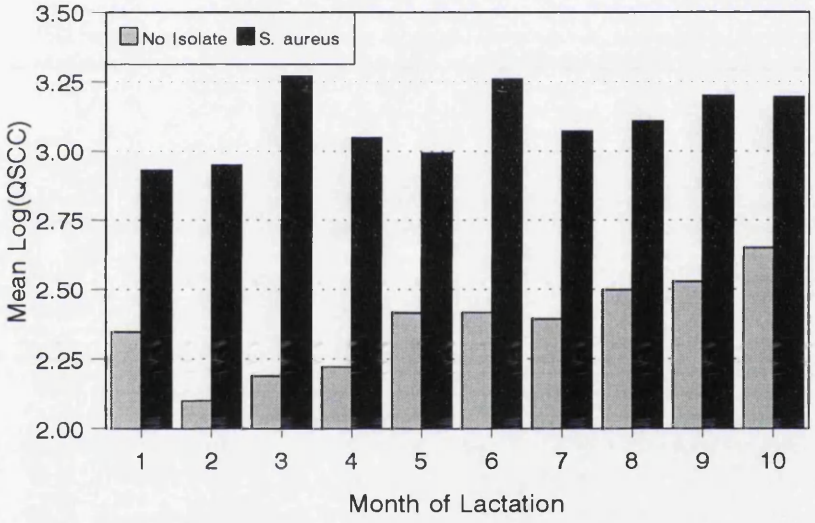


Figure 2:2
Relationship between Month of Lactation, QSCC and isolation of *S. aureus*
SAC Aberdeen Data

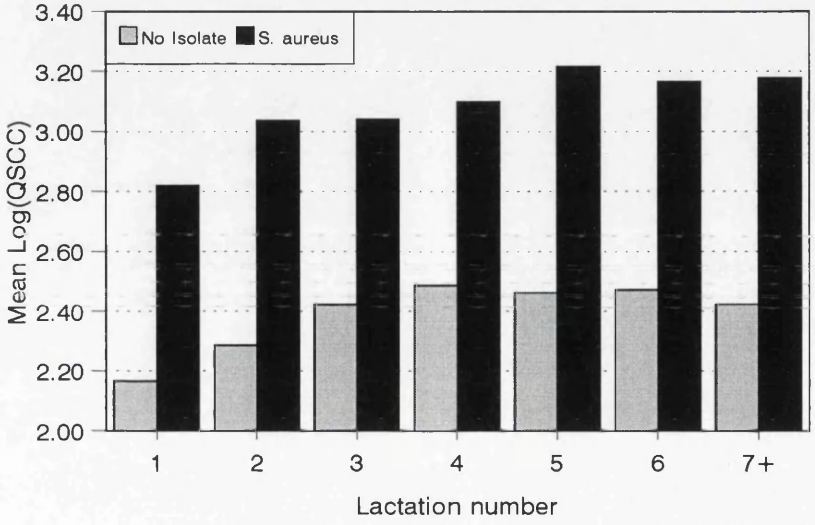


Figure 2:3
Relationship between Lactation number, QSCC & isolation of *S. aureus*
SAC Aberdeen Data

Month of lactation		No significant isolate	*S. aureus*	sed	F pr
1	log10(QSCC)	2.347	2.931	0.0904	<0.001
	QSCC(000)	222	853		
2	log10(QSCC)	2.101	2.95	0.0588	<0.001
	QSCC(000)	126	891		
3	log10(QSCC)	2.189	3.271	0.0921	<0.001
	QSCC(000)	154	1866		
4	log10(QSCC)	2.222	3.048	0.0629	<0.001
	QSCC(000)	168	1117		
5	log10(QSCC)	2.416	2.994	0.0751	<0.001
	QSCC(000)	261	986		
6	log10(QSCC)	2.417	3.259	0.0678	<0.001
	QSCC(000)	261	1815		
7	log10(QSCC)	2.393	3.072	0.0681	<0.001
	QSCC(000)	247	1180		
8	log10(QSCC)	2.498	3.109	0.09	<0.001
	QSCC(000)	315	1285		
9	log10(QSCC)	2.528	3.199	0.0941	<0.001
	QSCC(000)	337	1581		
10	log10(QSCC)	2.65	3.195	0.1111	<0.001
	QSCC(000)	447	1567		

Table 2:7. Effect of *S. aureus* infection and stage of lactation on QSCC (SAC Aberdeen).

Lactation Number		No significant isolate	*S. aureus*	sed	F pr
1	log10(QSCC)	2.166	2.820	0.0556	<0.001
	QSCC(000)	147	661		
2	log10(QSCC)	2.285	3.037	0.0530	<0.001
	QSCC(000)	193	1089		
3	log10(QSCC)	2.422	3.040	0.0753	<0.001
	QSCC(000)	264	1096		
4	log10(QSCC)	2.487	3.099	0.0659	<0.001
	QSCC(000)	307	1256		
5	log10(QSCC)	2.462	3.216	0.0632	<0.001
	QSCC(000)	290	1644		
6	log10(QSCC)	2.472	3.160	0.1005	<0.001
	QSCC(000)	296	1445		
7	log10(QSCC)	2.423	3.178	0.0683	<0.001
	QSCC(000)	265	1507		

Table 2:8. Effect of *S. aureus* infection and lactation number on QSCC (SAC Aberdeen).

Cows		Number(%) of quarters infected				Total
	0	1	2	3	4	
All	931	293(59.4)	109(22.1)	70(14.2)	21(4.3)	493(100)
By isolate	*S. aureus*	230	75	32	7	504
	S. agalactiae	71	24	12	4	171
	S. dysgalactiae	51	9	3	0	78
	S. uberis	39	5	0	0	49
	E. coli	3	0	0	0	3
	All Isolates(%)	394(48.9)	226(28.1)	141(17.5)	44(5.5)	805(100)

Table 2:9. Prevalence of multiple quarter infections: SAC Aberdeen quarter samples.

The cell with the largest contribution (3.2) to the total chi-square was the low number of *S. agalactiae* isolates from quarters with a SCC less than 283,000. However there were still a substantial number of isolates from this "group". A significant isolate (SI) was recovered from 100 (12.4%) quarter samples with SCC less than 283,000 (LS5) (**Table 2:6**).

2.3.2 Individual Cow (Composite) Somatic Cell Count Data

The results from 5416 composite milk samples in 45 different herds (55 herd tests) was recorded as database 2 (**Table 2:10**). Data analysis was performed on 5212 bacteriology results which recorded either the isolation of a single significant mastitis pathogen (*S. aureus*, *S. agalactiae*, *S. dysgalactiae*, *S. uberis*, *E. coli*) or no significant isolate (NSI) (**Table 2:10**). For the purposes of this analysis multiple isolations in 204 cows were ignored since these accounted for less than 5% of the total.

A two-sample T-test was performed on the data for "no significant isolate" and either "all significant isolates" or each isolate in turn. Presence of a significant pathogen (demonstrated by *S. aureus* in **Tables 2:11 & 2:12** and illustrated in **Figures 2:4 & 2:5**) was associated with a highly significantly elevated ICSCC ($P<0.001$) irrespective of lactation month or age respectively.

The proportion of all samples which yielded a significant mastitis pathogen increased with the age of the cow (**Figure 2:6 & Table 2:13**). Logistic regression was used to examine this relationship as binary dataset of the total number of composite samples and the number from which a major mastitis pathogen was recovered. The logistic regression (logit) equation showed that a significant ($P<0.05$) positive relationship existed between lactation number (L) and prevalence of infection (P):

$$\text{logit}(P) = 0.2614(L) - 1.813$$

Examination of the type of pathogen isolated by age showed that 1130 (96%) of the 1179 samples yielded a "parlour" mastitis pathogen (*S. aureus*, *S. agalactiae* or *S. dysgalactiae*), of which 312 (27%) were *S. agalactiae* (**Table 2:14**). *S. agalactiae* was most common in the youngest and oldest cows (**Figure 2:7**). The highest proportion of *S. agalactiae* isolates, a peak of 49 (37%) from a total of 133, came from animals in lactation 7 or greater.

Figure 2:8 shows how the ICSCC was related to the proportion of samples from which *S. aureus* was recovered. The proportion of composite milk samples yielding *S. aureus* increased with ICSCC up to 500,000. At higher counts 25% of the composite samples yielded an isolate of *S. aureus*. However 31.06% of all the *S. aureus* isolates were recovered from composite milk samples with ICSCC of less than 400,000.

The effect of restricting bacteriological examination to cows with ICSCC above a given threshold was examined (**Tables 2:15&16**). **Table 2:15** shows that a significant isolate (SI) was found in 1,427 (27.4%) of all 5,212 samples but in a much higher proportion (1,134 47%) of the 2,414 samples with SCC in excess of 283,000. *S. aureus* was the most common isolate from all

Information	Cows	
Herd Number	Total	5416
	Mean (s.d.)	98 (35)
	Median	95
	Range	40-192
Month of lactation Lactation number	4420 4600	
ICSCC	5416	
Bacteriology	5212	

Table 2:10. Description of SAC Aberdeen ICSCC database.

Month of lactation		No significant isolate	*S. aureus*	sed	F pr
1	log10(ICSCC)	2.235	2.669	0.075	<0.001
	ICSCC(000)	172	467		
2	log10(ICSCC)	2.077	2.744	0.0588	<0.001
	ICSCC(000)	119	555		
3	log10(ICSCC)	2.14	2.78	0.0501	<0.001
	ICSCC(000)	138	603		
4	log10(ICSCC)	2.179	2.861	0.0604	<0.001
	ICSCC(000)	151	726		
5	log10(ICSCC)	2.279	2.875	0.0497	<0.001
	ICSCC(000)	190	750		
6	log10(ICSCC)	2.378	2.936	0.0574	<0.001
	ICSCC(000)	239	863		
7	log10(ICSCC)	2.487	3.055	0.0592	<0.001
	ICSCC(000)	307	1135		
8	log10(ICSCC)	2.465	3.005	0.0577	<0.001
	ICSCC(000)	292	1012		
9	log10(ICSCC)	2.422	3.028	0.0715	<0.001
	ICSCC(000)	264	1067		
10	log10(ICSCC)	2.572	3.072	0.0816	<0.001
	ICSCC(000)	373	1180		

Table 2:11. Effect of *S. aureus* infection and stage of lactation on ICSCC (SAC Aberdeen).

Lactation Number		No significant isolate	*S. aureus*	sed	F pr
1	log10(ICSCC)	2.185	2.699	0.0392	<0.001
	ICSCC(000)	153	500		
2	log10(ICSCC)	2.288	2.892	0.0464	<0.001
	ICSCC(000)	194	780		
3	log10(ICSCC)	2.362	2.934	0.0484	<0.001
	ICSCC(000)	230	859		
4	log10(ICSCC)	2.423	2.896	0.0538	<0.001
	ICSCC(000)	265	787		
5	log10(ICSCC)	2.413	3.023	0.065	<0.001
	ICSCC(000)	259	1054		
6	log10(ICSCC)	2.439	2.934	0.0843	<0.001
	ICSCC(000)	275	859		
7+	log10(ICSCC)	2.521	3.011	0.0859	<0.001
	ICSCC(000)	332	1026		

Table 2:12. Effect of *S. aureus* infection and lactation number on ICSCC (SAC Aberdeen).

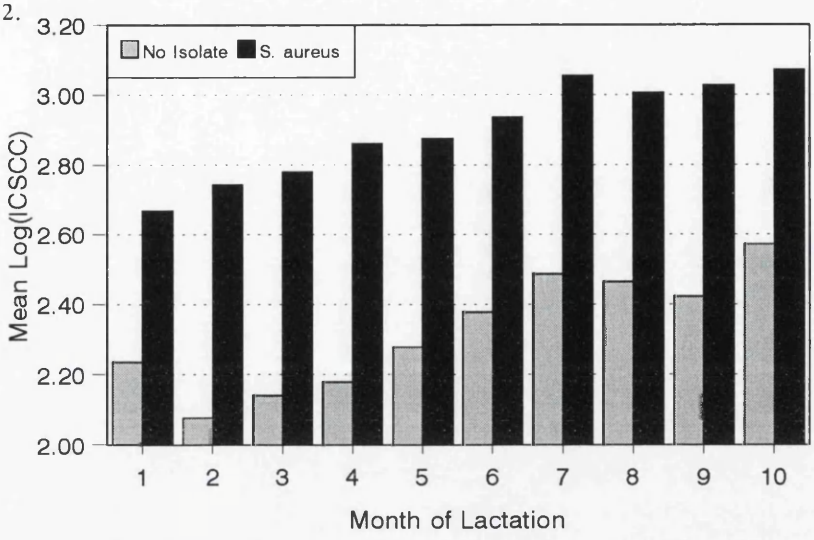


Figure 2:4
Relationship between Month of Lactation, ICSCC & isolation of *S. aureus*
SAC Aberdeen Data

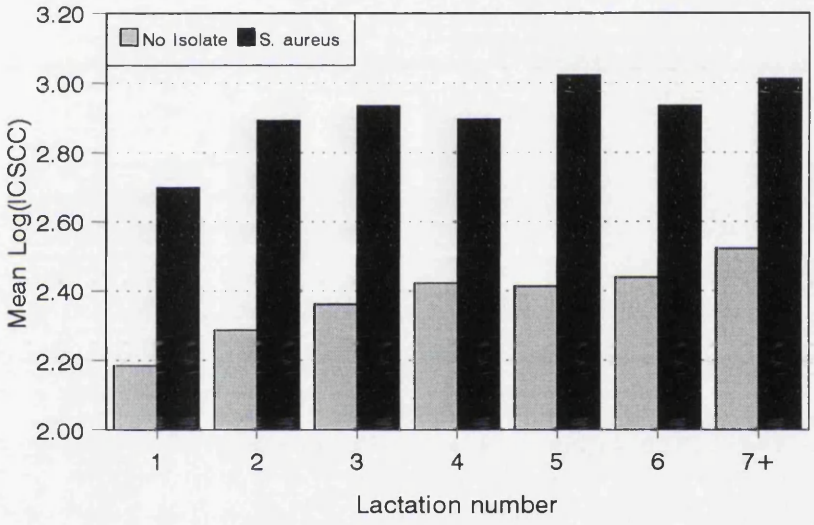


Figure 2:5
Relationship between Lactation number, ICSCC & isolation of *S. aureus*
SAC Aberdeen Data

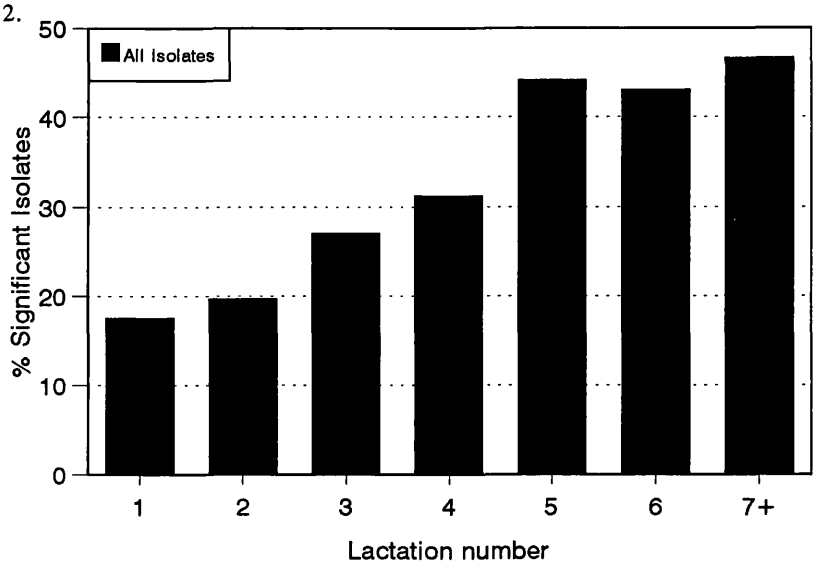


Figure 2:6
Proportion of significant isolates by lactation number

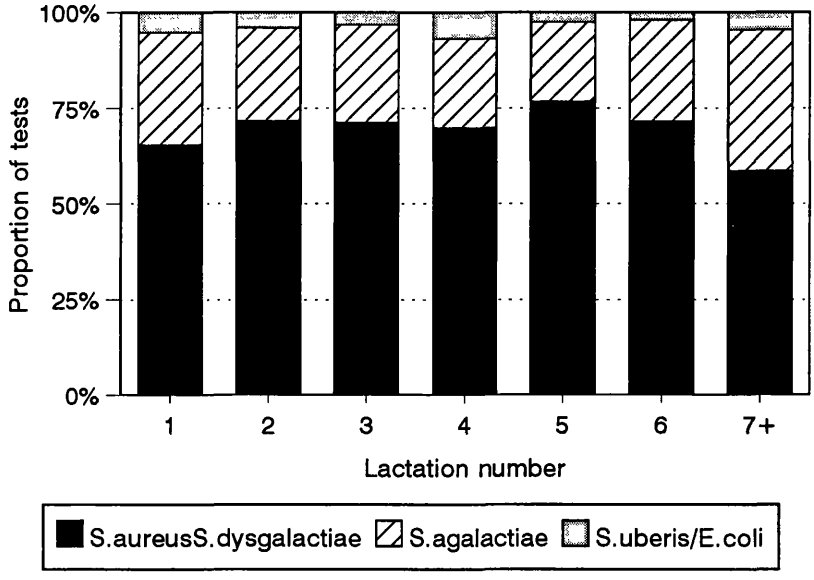


Figure 2:7
Proportion of isolate groups by lactation number

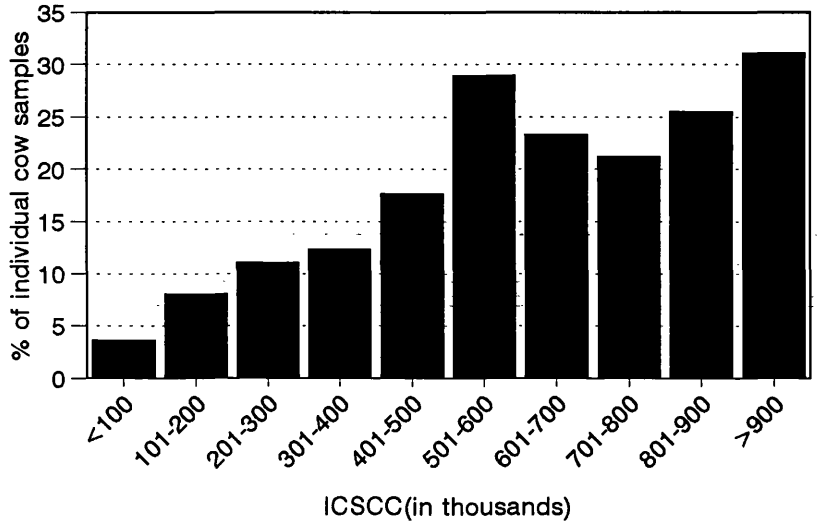


Figure 2:8
Proportion of infected samples by ICSCC
S. aureus

Lactation Number	Number of Composite Samples			
	TOTAL	NSI	SI	%SI
1	1312	1083	229	17
2	900	723	177	20
3	812	593	219	27
4	513	353	160	31
5	360	201	159	44
6	237	135	102	43
7+	285	152	133	47
TOTAL	4419	3240	1179	27

Table 2:13. Effect of lactation number on the isolation of significant mastitis pathogens from 4419 composite milk samples (SAC Aberdeen).

Lactation Number	Number of Composite Samples (%)					
	SFAU	SPDY	SPAG	SPUB	ESCO	TOTAL
1	139 (60.7)	11 (4.8)	67 (29.26)	9 (3.93)	3 (1.31)	229
2	117 (29.26)	10 (5.65)	43 (24.29)	4 (2.26)	3 (1.69)	177
3	144 (65.75)	12 (5.48)	56 (25.57)	5 (2.28)	2 (0.91)	219
4	105 (65.63)	7 (4.38)	37 (23.12)	10 (6.25)	1 (0.62)	160
5	111 (69.81)	11 (6.92)	33 (20.75)	4 (2.52)	0	159
6	65 (63.73)	8 (7.84)	27 (26.47)	2 (1.96)	0	102
7+	70 (52.63)	8 (6.02)	49 (36.84)	5 (3.76)	1 (0.75)	133
TOTAL	751 (63.7)	67 (5.68)	312 (26.46)	39 (3.31)	10 (0.85)	1179

Table 2:14. Bacteriology data from composite samples with a significant mastitis pathogen (SAC Aberdeen).

	Samples (%)			
	All	SCC <283	SCC 283-566	SCC >566
Total	5416 (100)	2836 (100)	928 (100)	1652 (100)
Missing bacteriology	204 (3.77)	38 (1.34)	46 (4.96)	120 (7.26)
No significant isolate	3785 (69.89)	2505 (88.33)	632 (68.1)	648 (39.23)
Significant isolate	1427 (26.34)	293 (10.33)	250 (26.94)	884 (53.51)
S. aureus	837 (15.46)	202 (7.12)	158 (17.03)	477 (28.87)
S. agalactiae	453 (8.36)	55 (1.94)	75 (8.08)	323 (19.55)
S. dysgalactiae	75 (1.38)	15 (0.53)	10 (1.08)	50 (3.03)
S. uberis	51 (0.94)	19 (0.67)	7 (0.75)	25 (1.51)
E. coli	11 (0.20)	2 (0.07)	0	9 (0.55)

Table 2:15. Significant mastitis pathogens isolated in composite samples by SAC Aberdeen (1974-1990).

Lactation Number	Number of Composite Samples (%)					
	SFAU	SPDY	SPAG	SPUB	ESCO	TOTAL
1	88 (58.7)	7 (4.67)	52 (34.67)	1 (0.67)	2 (1.33)	150
2	89 (63.57)	8 (5.71)	37 (26.43)	3 (2.14)	3 (2.14)	140
3	144 (62.64)	10 (5.49)	52 (28.57)	5 (2.75)	1 (0.55)	182
4	83 (62.88)	7 (5.3)	34 (25.76)	7 (5.3)	1 (0.76)	132
5	97 (72.39)	7 (5.22)	28 (20.9)	2 (1.49)	0	134
6	53 (60.92)	7 (8.05)	26 (29.89)	1 (1.15)	0	87
7+	59 (50.43)	7 (5.98)	45 (38.46)	5 (4.27)	1 (0.85)	117
TOTAL	583 (61.89)	53 (5.63)	274 (29.09)	24 (2.55)	8 (0.85)	942

Table 2:16. Bacteriological data from 1134 composite samples with SCC in excess of 283,000 yielding a significant mastitis pathogen (SAC Aberdeen).

samples, accounting for 837 (59%) of 1,427 significant isolations (**Figure 2:9**). *S. agalactiae* (SPAG) was recovered from a further 453 (32%) of all samples.

A significant mastitis pathogen was isolated from only 293 (10%) of 2,798 individual cow samples with SCC less than 283,000. *S. aureus* was recovered from 202 (69%) and *S. agalactiae* from a further 55 (19%) of these 293 samples.

S. aureus accounted for 635 (56%) and *S. agalactiae* for 398 (35%) of the 1,134 significant isolates in samples with SCC in excess of 283,000 (**Table 2:15**). **Figure 2:10** summarises this data for samples with SCC of 283,000 or greater to allow a simple comparison with the results for samples from all ranges of ICSCC (**Figure 2:9**). A significant mastitis pathogen was isolated from 884 (58%) of the 1,532 samples with ICSCC over 566,000. *S. aureus* accounted for 477 (54%) of these 884 isolates.

The relative proportions of the significant pathogens constituted a bacteriological profile of the cause of subclinical mastitis. The bacteriology profile which resulted from samples above and below the 283,000 ICSCC threshold was statistically examined for similarity. The largest contribution (15.6) to the chi-square statistic of 41.4 on 6 degrees of freedom came from the much lower rate of isolation of *S. agalactiae* from samples with SCC less than 283,000. This indicated that the distribution of isolates based on ICSCC were very significantly different ($P < 0.001$). Thus a minimum ICSCC threshold of 283,000 produced a representative bacteriology profile: the presence of *S. agalactiae* in the herd was disclosed without a significant reduction in the detection of *S. aureus* individuals. Detailed bacteriology results are presented in **Table 2:16** for the 4,600 cows for which lactation number was available (**Table 2:10**).

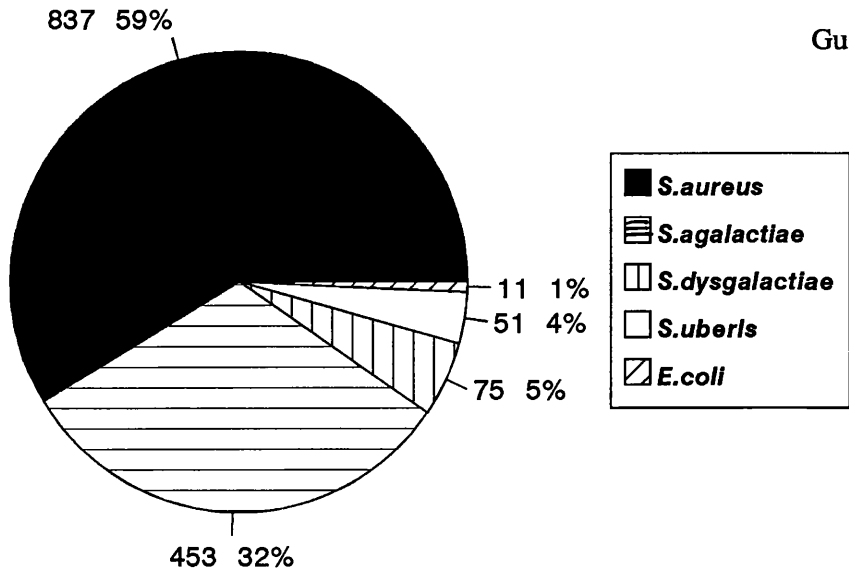


Figure 2:9
SAC Aberdeen Individual Cow Isolates
Entire Dataset

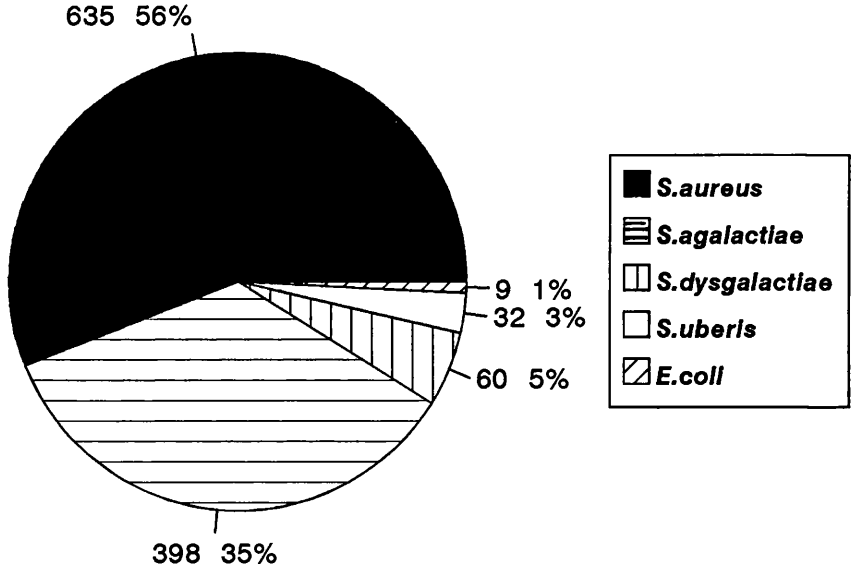


Figure 2:10
SAC Aberdeen Individual Cow Isolates
Samples with SCC over 283,000

2.4 Discussion

EC Directive 92/46 adopted SCC as a measure of milk hygienic quality in addition to TBC. The original Aberdeen herd investigations were mostly conducted in herds with an elevated bulk tank Total Bacterial Count (TBC). These were herds in which study of a single bulk tank sample revealed a preponderance of mastitis bacteria, identified using the quantitative bacteriology techniques reported by Jeffrey & Wilson (1987). This database was unique in Scotland since it recorded both bacteriology and SCC data from a large number of milk samples, collected as either quarter (5,860) or composite udder (5,416) samples. Quarter samples are preferred for the bacteriological examination of problem cows since the count is not affected by the dilution of low SCC milk from other non-infected quarters in the case of ICSCC (Reneau, 1986). The statistical analysis of all SCC data in this project used logarithm-transformed data in accordance with the recommendations of Shook (1982) but since daily milk yield data were not recorded it was not possible to study the relationship of SCC and yield depression.

A major objective of the work described in this chapter was the assessment of Individual Cow SCC (ICSCC) as a screening tool in bacteriological investigation of herds with a milk quality problem. In doing so it was necessary to develop a database suitable for the handling and analysis of records of mastitis investigations which proved of great value in later investigations.

The isolation of a significant mastitis pathogen from 14% of all Aberdeen quarter samples would appear to be in very close agreement with the 14.1% of all the quarters examined by Wilson & Richards (1980) in their study of the national prevalence of subclinical mastitis throughout the British dairy industry. However their diagnosis of subclinical mastitis was restricted in accordance with International Dairy Federation guidelines to the 9.6% of quarters which also had SCC over 500,000. On this basis, the incidence of subclinical mastitis in the Aberdeen data was much higher at 35.8%.

The results of quarter sample examinations clearly demonstrated that infection was the most important cause of high SCC. Infection caused a significant ($P<0.001$) elevation of QSCC irrespective of stage of lactation or lactation number. The data relating to *S. aureus* was presented to illustrate these findings since it was the most common recognised mastitis pathogen. The quarter database gave an impression of the dynamics of subclinical mastitis in that a significant pathogen was recovered from at least 2 quarters in 200 (41%) of the 493 infected cows. This would appear to agree closely with the report by Natzke (1982) that 45 to 55% of all new infections were the result of spread from another infected quarter within the udder. This spread could be due to cross-infection within the udder or mechanical transfer between quarters at milking time (Buddle et al., 1987). An important corollary to the high proportion of cows with several infected quarters was the implied agreement with Meek et al. (1980) that with increased numbers of mastitic quarters, ICSCC became a more accurate predictor of subclinical mastitis. This current study provided a more detailed analysis of the effect of SCC thresholds on mastitis diagnosis than the presentation

by Wilson & Richards (1980) because they used only the IDF recommendation of 500,000.

Analysis of the Aberdeen ICSCC data similarly demonstrated that infection caused a significant SCC rise irrespective of stage of lactation or lactation number. Examination of the composite sample database showed that the prevalence of infection increased with the age of the cow. Examination of the effect of age on the prevalence of infection by pathogens grouped by either "parlour" or "environmental" origin revealed that the proportion of "parlour" isolates reached a peak by 5th lactation (**Figure 2:7**). This is likely to be a function of the prolonged exposure to infection within the herd rather than an increased susceptibility to infection with age. Indeed the QSCC data for "no significant isolate" did not show a significant rise with age which is in agreement with Eberhart et al. (1979).

This Aberdeen database also provided an opportunity to examine the effect of restricting bacteriological investigation to cows with ICSCC above a given threshold. There was no simple answer to this although the Linear Score system developed by Shook (1982) provided a recognised scale for the interpretation of SCC data. The limits of Linear Score 5 band of 283 to 566,000 were chosen for investigation because they provided a margin around the EC limit of 400,000. An ICSCC threshold of 283,000 was found to increase the efficiency of bacteriological examination from the isolation of a significant pathogen in 27% of all composite samples to 47% of composite samples with SCC over 283,000. A significant pathogen was isolated in 58% of composite samples with ICSCC in excess of 566,000 i.e. LS6. The effect on bacteriological recovery rates of sampling individual cows was therefore quantified for the ICSCC thresholds of 283,000 (LS5 and over) and 566,000 (LS6 and over). They showed the potential to increase the efficiency of bacteriological examination by factors of 72% and 112% respectively based on the isolation rate achieved by examination of all cows in the Aberdeen herds. It also gave two reasonable ICSCC thresholds for herd investigations limited by finance. Furthermore it demonstrated that the bacteriological profiles differed significantly ($P < 0.001$) because *S. agalactiae* was not recovered from as many samples with ICSCC below 283,000 as predicted. In other words this was statistical evidence of the strong SCC reaction associated with *S. agalactiae* infection. In particular, although expensive bacteriological examinations could be reduced by the use of the higher Linear Score 6 (566,000) threshold, the value of the resulting bacteriological profile will depend on the predominant mastitis pathogens in the herd. The isolation of *S. agalactiae* indicates that effective action has the potential to eradicate it from the herd. Indeed the majority (71%) of *S. agalactiae* isolates were recovered from composite samples with ICSCC in excess of 566,000. In contrast the strategy in dealing with a *S. aureus* herd problem centres on the identification of all infected individuals. Examination of the Aberdeen database showed 24% of these cows had ICSCC less than 283,000. The bacteriological profile of such low ICSCC cows differed significantly ($P < 0.001$) from that of the entire database because of the relative under-representation of *S. agalactiae* isolations. The isolation of *S. aureus* from low ICSCC samples is consistent with the

cyclic SCC increase and decrease reported by Daley et al. (1991) in subclinical infection by *S. aureus*.

In conclusion, the analysis of SAC Aberdeen data from both quarter and composite samples clearly demonstrated that infection was the main cause of high SCC, the elevation of which was significant ($P<0.001$) throughout lactation. *S. aureus* and *S. agalactiae* were the most common mastitis pathogens and were frequently demonstrated to infect several quarters of the same cow. Prevalence of infection was shown to have a significant ($P<0.05$) positive statistical relationship with lactation number. The use of ICSCC thresholds for herd investigations was demonstrated to produce a statistically representative bacteriological herd profile and to be cost-effective. The exact threshold employed would depend on the extent of the subclinical mastitis problem in the herd and the financial limitations imposed on the bacteriological investigation. Applying the lower limits of the American Linear Score system as an ICSCC threshold, LS5 (in excess of 283,000) detected 79% of infections by significant mastitis pathogens while the figure associated with LS6 (in excess of 566,000) was 62%. These thresholds increased the efficiency of bacteriological examination by factors of 72% and 112% respectively. Chapter 3 describes the application of the LS5 ICSCC threshold in the bacteriological investigation of high BTSCC herds.

CHAPTER 3. INVESTIGATION OF HIGH SCC HERDS.

3.1 Introduction

In 1975 Booth warned that there was an increasing acceptance in Europe that the bulk tank somatic cell count (BTSCC) was a measure of the quality of milk: EC 92/46 has now made this a fact. Unfortunately a single BTSCC is not a reliable measure of herd infection though it can be improved by averaging a number of counts (Wilson & Richards, 1980; David & Jackson, 1984). The national cell count in the UK has shown two periods of marked fall, in 1975/6 and in 1983, both attributable to increased culling of cows (Booth, 1988b). At present 85% by volume of all UK milk is produced by herds with BTSCC lower than 400,000 (Booth, 1994).

The Milk Marketing Board of England and Wales started measuring BTSCC monthly on 10,000 randomly selected herds in early 1971 (Booth, 1988a). In the following year monthly BTSCC were provided on a commercial basis to dairy farmers in England & Wales who wished to avail themselves of this service. Eventually in 1977 it was decided to provide monthly BTSCC as a service to all producers in the Milk Marketing Board of England & Wales.

BTSCC was first provided to all SMMB producers in 1972 on the basis of a single test each month. The SMMB adopted the Cell Count Scheme in April 1990 at which time the frequency of testing bulk milk samples was increased to once per week. The July 1990 issue of the "SMMB Milk Bulletin" notified producers of the proposed introduction of a SCC-based component of payment in April 1991. Individual weekly results, monthly arithmetic mean and 3-month geometric mean figures appeared on the monthly milk statement at this time also. The SMMB cell count scheme combined a premium and penalty payment structure in April 1991, details of which were published in January 1991 (Anon, 1991) (**Table 3:1**). Despite over 12 months of advance publicity, insufficient progress was made in reducing the number of producers with BTSCC in excess of 400,000 and on March 15 1991 the SMMB wrote to all producers notifying them that introduction of the entire Cell Count Scheme had been deferred by 9 months until January 1992. However there was a significant groundswell of pressure from producers with BTSCC below 400,000, especially those who had reduced their BTSCC in line with the April 1991 timetable, and on April 9 1991 the SMMB notified all producers to this effect and of their decision to introduce the premium part of the Cell Count Scheme immediately but penalties remained deferred until January 1992.

The two other Milk Marketing Boards in Scotland, Aberdeen & District (A&DMMB) and North of Scotland (NOSMMB), also adopted cell count schemes, though differing from that of the SMMB (**Table 3:1**). The NOSMMB began testing four bulk tank samples each month in November 1990 (NOSMMB, personal communication). The NOSMMB announced in the April 1991 issue of their monthly "Milk Bulletin" newsletter that the introduction of their entire SCC premium/penalty scheme had been delayed until April 1992 to allow producers greater time to adjust. Both the premium and penalty components of the NOSMMB cell count scheme were

BTSCC(000) Band	Payment Premium/Penalty (ppl)		
	SMMB[a]	A&DMMB[b]	NOSMMB[a]
Less than 250	+0.1	+0.1	+0.1*
250-400	0	0	0
401-600	-0.2	-0.2	-0.2
More than 600	-0.5	-0.5	
601-1,000			-1.5
More than 1,000			-2.0

[a] 3-month Geometric Mean; [b] 6-month Geometric Mean

Table 3:1. SCC payments by the three Scottish Milk Marketing Boards (December 1993).

introduced together in April 1992 (**Table 3:1**). The A&DMMB introduced a penalty cell count scheme in April 1992 which was amended in September 1993 to that outlined in **Table 3:1**. Thus by 1992 the three Milk Marketing Boards of Scotland had incorporated SCC as a component of milk price in response to EC Directive 92/46 which adopted a bulk tank SCC of 400,000 as the upper limit of the hygienic quality standard of milk for human consumption. Individual Cow SCC (ICSCC) testing had been available since June 1989 on the samples collected as part of milk recording by the Scottish Milk Records Association (SMRA).

Most studies of SCC epidemiology have concentrated on the relationship between cow infection prevalence and BTSCC (Pearson & Greer, 1974; Erskine et al., 1988). There has been relatively little published information on the epidemiology of SCC at the national herd level. Booth (1988a&b) described a sharp fall in the overall national cell count in England and Wales in 1983 following the introduction of the payment system for total bacterial count in late 1982. In Canada the Ontario Milk Marketing Board (OMMB) adopted a SCC penalty programme in 1989 (Schukken et al., 1992a). Starting at 800,000, this programme incorporated an annual reduction of 50,000 in the maximum BTSCC so as to achieve the target penalty threshold of 500,000 by 1995. Producers paid a penalty when their BTSCC exceeded the threshold for three out of four consecutive months. It is unfortunate that this slow progressive approach was not taken in the United Kingdom. In one of the few reports on the effect of differential payments based on SCC, Schukken et al. (1992a) analysed OMMB milk quality data for the period January 1985 to September 1991 and attributed a decrease of 58,000 to the SCC control program. A follow-up study showed that the reduction in the overall OMMB SCC was mainly due to improved SCC performance of farms within the BTSCC band 300 to 599,000. Conversely when the OMMB SCC rose, the increased SCC of farms with BTSCC less than 299,000 was mostly responsible (Schukken et al., 1992b).

The analysis of SAC Aberdeen data in Chapter 2 established that the significant mastitis pathogens most frequently isolated in a group of "problem" Scottish herds were *S. aureus* and *S. agalactiae*. However these herds were largely identified on the basis of a raised TBC. The inclusion of SCC in the proposed EC regulations (92/46) meant there was a need to study the epidemiology of Scottish herds with a SCC problem. This would identify the major infectious causes contributing to their subclinical mastitis problem.

This chapter firstly records the establishment in May 1991 of the MQFILE personal computer database of BTSCC and TBC of each dairy herd in Scotland, secondly how this was used to study the performance of all herds in the SMMB region and to select high SCC herds for field investigation and finally discusses the results of these investigations.

3.2 Materials & Methods

3.2.1 Analysis of BTSCC data

3.2.1.1 SMMB MQFILE

In May 1991 the SMMB Computer & Information Technology (IT) Department created a 3.5", 1.4Mb computer disc which contained the BTSCC and TBC data for all 2147 SMMB producers for the 11 month period from June 1990 to April 1991. The files were named using the convention MQLA***.9*, where *** represented the 3 letter abbreviation of the month name and 9* represented 1990 or 1991. The format of MQLAJUN.90 is illustrated in **Table 3:2** and described in **Table 3:3**. The top "reference" line in **Table 3:2** is a 10-digit repeated sequence 0123456789 and thus illustrates the precise location of each character string in the "file". Unless otherwise stated, BTSCC refers to a 3-month rolling geometric mean since this was the mode of calculation described by EC Directive 92/46. A sample line from these files is presented in **Table 3:4** and fully interpreted in **Table 3:5**.

A personal computer database of this BTSCC and TBC data was established using MQFILE software written by Mr D. Arnot, SAC Auchincruive Computing & IT Department. An explanation of the final format of the MQFILE software is presented in **Table 3:6**. Subsequent SMMB files of the same format containing the data for the previous month were routinely created by the SMMB and incorporated into the MQFILE database using the "data to file" command (**Table 3:6**). The SMMB mainframe computer SCC could only accommodate BTSCC information from the previous 11 months and thus data was routinely discarded. In addition, the information that was maintained on the mainframe computer was accessible only with detailed programming knowledge.

3.2.1.2 A&DMMB MQFILE

The A&DMMB mainframe computer was accessed in April 1993 to create monthly files beginning April 1990 of the BTSCC and TBC data of all 156 producers. The A&DMMB supplied BTSCC data as the monthly arithmetic average figures (based on four weekly measurements), in contrast to the 3-month Geometric Mean figure from the SMMB. The A&DMMB TBC data was also supplied as the monthly arithmetic mean. The SAC***9*.TXT filename convention was adopted where *** represented month of year and 9* year (90/91/92/93). These files were used to create an MQAB MQFILE database after minor modification of the MQFILE software since the SAC***9*.TXT format (**Table 3:7**) differed from that created by the SMMB. The A&DMMB file format is explained in **Table 3:8**.

```
Ref   12345678901234567890123456789012345678901234567
File  ************** **********                060249006
```

Table 3:2. Format of SMMB MQLAJUN.90 computer file

Reference point	Character options	Data in Source File
1	0 1 2 3	Not milk recording SMRA member Simplified scheme Resigned from SMRA
2-7	000000-999999	O.S. map reference acting as producer code
8	0-9	Check digit
9-38	Text	Surname & Farm name
39-40	01-12	Month of year
41-44	01-9999	3-month Geometric Mean BTSCC(000)
45-47	01-999	2-month Geometric Mean TBC(000)

Table 3:3. Structure of SMMB MQLAJUN.90 computer file.

```
Ref   12345678901234567890123456789012345678901234567
File  13812330SCOT AGRIC COLL AUCHINCRUIVE  060246007
```

Table 3:4. Sample line from SMMB MQLAJUN.90

Reference	Character	Interpretation of data in file
1	1	Producer is a SMRA member
2-7	381233	Producer code
8	0	Producer code check digit
9-38	Text	Producer = Scottish Agricultural College, Auchincruive
39-40	06	Month of year = June
41-44	0246	3-month Geometric Mean Bulk Tank SCC = 246,000
45-47	007	2-month Geometric Mean Total Bacterial Count = 7,000

Table 3:5. Explanation of Table 3:4 (SMMB MQLAJUN.90)

Onscreen Menu	Subcommands	Function
Data to file	**A**&DMMB or **S**MMB data Data file name	Identify file format Load file into database
Report on farm	Grid Reference **D**ata or **G**raph format	Access individual herd records **D**isplay information in on-screen format or output **G**raph to printer
Multiple farm	Start Month End Month **D**ata or **G**raph format	Access herd records listed in MQFARM.DAT file of MQFILE subdirectory for period. Create MQHERDT.REP file of on-screen format and MQHERDT2.REP file in MINITAB format
Location data to file	Data file name	Update Producer surname and farm name of associated producer code
Overall report (to file)	Start Month End Month	Create output file MQHERDT.REP of all producers
Change minimum months over limit - currently 1	New limit	Specify minimum number of months in penalty (>400) for farms to appear in MQHERDT.REP created by the **O**verall Report option. Default is 1 month, 0 reports all herds on file
Quit		Exit MQFILE and return to DOS

Table 3:6. Operation of the MQFILE software.

```
Ref   12345678901234567890123456789012345678901234567890123456789012345678901
File  1, *****,0,**************                , 6,  21961, 489, 10
```

Table 3:7. Format of A&DMMB SACJUN93.TXT computer file.

Reference point	Character options	Data in file
1	1	SMRA member
2-9		Producer code
10-11	0	
12-42	Text	Surname & farm name
43-44	01-12	Month of year
46-52		Milk sales (litres/month)
54-57	0-9999	BTSCC(000)
59-61	0-999	TBC(000)

Table 3:8. Structure of MQAB computer file.

3.2.1.3 NOSMMB MQFILE

Computer printouts of BTSCC and TBC data from each producer beginning in January 1990 were supplied by the NOSMMB in July 1993. Both the BTSCC and TBC data were calculated as simple monthly arithmetic averages. These were manually entered to create ASCII-format MQNB***.9* monthly files as illustrated in **Table 3:9** and described in **Table 3:10**. A separate MQNB MQFILE database was created following minor modification of the MQFILE software.

3.2.1.4 MQFILE Data Analysis

Initially the individual herd BTSCC and TBC were available from MQFILE only as an on-screen display (**Table 3:11**) accessed by the "report on farm" facility (**Table 3:6**). The interpretation of the on-screen display is presented in **Table 3:12**.

Having established the SMMB MQFILE database, the programme was developed to analyse the BTSCC data from all producers. In order to obtain summary information there were two sets of output files. Firstly, the output file MQHERDT.REP contained the various components of the MQFILE data analysis (**Tables 3:13-16**). The individual herd information is illustrated in **Table 3:13** and described in **Table 3:14**. An arithmetic mean of the monthly BTSCC data was presented in **Table 3:13**. This constituted either an average of the SMMB rolling 3-month geometric mean or an average of the respective A&DMMB and NOSMMB arithmetic monthly mean. It was not mathematically possible to calculate the original monthly SMMB BTSCC data since the data was only supplied as the rolling 3-month geometric mean.

An analysis of this individual data from all producers for a specified period of months was also available within the "overall report" option (**Table 3:6**). The number of herds whose BTSCC exceeded 400,000 was presented in the form illustrated in **Table 3:15**.

The number of herds within each 100,000 BTSCC band (**Table 3:16**) was computed to provide a more precise analysis of the performance of all producers.

```
Ref   12345678901234567890123456789012345678901234567
File  0000***0******,**********                000392015
```

Table 3:9. Format of NOSMMB MQNB*.9* computer file.**

Cursor position	Character options	Data field
1-4	0	Blank
5-7	3 Digit	Producer code
8	0	Blank
9-38	Text	Surname & farm name
39-40	00	Blank
41-44	0-9999	BTSCC(000)
45-47	0-999	TBC(000)

Table 3:10. Structure of MQNB*.9* computer file.**

Grid ref ****** Farm ****** ****					
	1990	1991	1992	1993	1994
jan	-1(0)	429(17)	467(13)	298(16)	501(46)
feb	-1(0)	406(18)	336(7)	272(14)	606(58)
mar	-1(0)	457(35)	297(14)	298(15)	709(63)
apr	-1(0)	385(11)	308(19)	343(14)	739(50)
may	-1(0)	380(17)	335(15)	397(27)	676(15)
jun	415[a](13[b])	416(44)	354(14)	433(40)	606(19)
jul	518(26)	475(35)	360(14)	389(20)	636(32)
aug	652(24)	567(17)	419(17)	425(11)	678(30)
sep	743(12)	593(15)	480(18)	399(13)	791(28)
oct	650(14)	573(16)	439(14)	424(27)	828(43)
nov	543(15)	575(56)	390(23)	407(39)	
dec	441(16)	524(55)	319(27)	442(29)	

[a] =BTSCC, [b] =TBC & see Table 3:12

Table 3:11. Report on farm option of MQFILE programme.

Parameter	No value available	Result	
		Display	Interpretation
BTSCC	-1	415	415,000
TBC	(0)	(13)	13,000

Table 3:12 Interpretation of the on-screen display of the MQFILE option "report on farm" (Table 3:11).

```
Ref   12345678901234567890123456789012345678901234567890123456789012345678901234567890
File  5 00011111000000000000000000000000 ****** 8 396.9  53.6 ******** ****
```

Table 3:13. MQFILE report (MQHERDT.REP) for an 8-month period (March to October 1994).

File	Interpretation
5	BTSCC over 400,000 5/8 months
00011111...	Chronological pattern of BTSCC penalty
******	Producer code
8	No. months data available for herd
396.9	8-month arithmetic mean BTSCC
53.6	8-month arithmetic mean TBC
******** ****	Producer surname & Farm name

Table 3:14. Interpretation of MQHERDT.REP report.

Month	Number of herds in penalty					
	Number of months in penalty					Total
	1	2	3		8	
Mar/94	0	24	29	...	241	459
Apr/94	0	3	29	...	241	454
May/94	0	7	30	...	241	464
Jun/94	0	8	21	...	241	479
Jul/94	0	11	21	...	241	481
Aug/94	0	9	28	...	241	457
Sep/94	0	6	26	...	241	451
Oct/94	0	45	29	...	241	488
Total	1487	113	71	...	241	
Mean CC	218	329	404	...	638	

Table 3:15. MQFILE report: Chronological pattern of number months BTSCC over 400,000.

Month	Number of herds within BTSCC (000) range				
	1-99	...	400-499	...	1200-1299
Mar/94	61	...	209	...	6
Apr/94	69	...	190	...	9
May/94	67	...	206	...	11
Jun/94	51	...	220	...	9
Jul/94	36	...	236	...	4
Aug/94	29	...	231	...	2
Sep/94	23	...	243	...	4
Oct/94	27	...	271	...	3

Table 3:16. Analysis of SCC data into number of producers in 100,000 BTSCC bands.

Data from specific subgroups of producers was computed by the "multiple farm report" option of the MQFILE software (**Table 3:6**). Using this command two reports were created based on the list of herds in the MQFARM.DAT input file of the MQFILE computer subdirectory. The MQHERDT.REP file had the same format as the onscreen display (**Table 3:11**). The format of the second output file, MQHERDT2.REP (**Table 3:17**), was designed so as to be compatible with "Minitab 8" (Minitab 8 Committee, 1991) statistical software and thus allow flexible analysis of the BTSCC data.

"Harvard Graphics 3.0" (Software Publishing Corporation, California) software was used to create the profiles of **Appendix I**, originally by the manual entry of the MQHERDT.REP file data (**Table 3:11**). The development within MQFILE of a "graph format" option within "multiple farm report" (**Table 3:6**) created a macro command format of the MQHERDT2.REP file which operated Harvard Graphics automatically to produce the profiles.

The "overall" report format (**Table 3:6**) was used to analyse information on the monthly BTSCC distribution of all producers (**Tables 3:15 & 3:16**). Interrogation of the MQFILE database using the "data" option of "multiple" report format (**Table 3:6**) created a MINITAB-compatible file (**Table 3:17**) of BTSCC data for the analysis of defined subgroups of producers.

3.2.1.5 Contribution Index

The relative proportion of the Board cell count supplied by each individual herd, the Contribution Index (Schukken et al., 1992b), was calculated for each month in 1993 (**Table 3:18**). The mean annual milk sales of all 2149 SMMB producers was calculated to be 451,619 litres. This was used as the quotient in the calculation of the Month Volume Ratio (MVR) which related the level of production in each herd to that of the average herd. The Index Cell Count (ICC) represented the herd SCC performance above the arbitrary threshold of 250,000 (premium) and was calculated using the available 3-month Geometric Mean BTSCC data. The total number of cells contributed by each producer (Month Contribution) was then calculated as the product of MVR and ICC. The herd Contribution Index was then calculated as the annual total of Month Contributions and was used to rank herds relative to each other.

```
******    *    *    *    *    * 165 176 162 158 149 144 125 126 119 130 151 161 170
202 220 210 169 145 147 170 180 184 178 196& 219 249 254 262 234 201 166 159
179 168 187 200 247 249 303   *    *    *    *    *    *    *    *    *    *  25  27  30  24
13   5   8&  10   9  13  18  12  13   4   4   4  14  15   5   9   9   8  11  17  14  10   8  12  14
 8   7   8   6   4  10  11  11   9   8   *   *   *   *   *   *&
 *************
```

Table 3:17. MINITAB compatible MQHERDT2.REP format.

	BTSCC (000)	Volume (l)	Index Cell Count (ICC) (BTSCC-250)	Month Volume Ratio (MVR) (Vol/451619)	Month Contribution (MC) (ICC x MVR)
Jan	507	61558.7	257	0.136	34.952
Feb	472	54104.6	222	0.120	26.640
Mar	476	55151.9	226	0.122	27.572
Apr	499	58656.8	249	0.130	32.370
May	511	75311.1	261	0.167	43.587
Jun	470	67273.4	220	0.149	32.780
Jul	498	70092.6	248	0.155	38.440
Aug	519	71581.3	269	0.158	42.502
Sep	498	67873.6	248	0.150	37.200
Oct	447	61157.8	197	0.135	26.595
Nov	419	65793.3	169	0.146	24.674
Dec	467	71741.8	217	0.159	34.503
Contribution Index ($\sum$MC)					401.815

Table 3:18. Worked calculation of a herd Contribution Index.

3.2.2 Field investigations

3.2.2.1 Criteria for selection of Phase-1a herds

Figure 3:1 illustrates the type of information available from **Table 3:15** to show the number of months a total of 1050 SMMB herds recorded BTSCC in excess of 400,000 in the 11 month period June 1990 to April 1991 (data prior to June 1990 was not available from the SMMB database). The distribution of 247 of these herds by the arithmetic average for the 11 months they were continuously in penalty (using the information shown in **Table 3:13**) is presented in **Figure 3:2**. A total of 25 SMRA herds within the SMMB region which were continuously in penalty were selected in proportion to the overall distribution shown in **Figure 3:2**. The information presented in the MQHERDT.REP output file (**Table 3:13**) was used to actually identify the producers.

These 25 herds represented the first group of investigations conducted within the project. Unfortunately two did not participate fully. One herd did not cooperate at all and the other left milk production 6 months after the start of the project. Details of the 23 remaining SMMB project herds (Phase-1a, **Figure 3:3**) are presented in **Table 3:19**.

3.2.2.2 Criteria for selection of Phase-1b herds

Three SAC herds within the SMMB region (Auchincruive, Acrehead and Crichton Royal Farm) and one within the A&DMMB region (Craibstone) with consistently low BTSCC were monitored as controls (**Figure 3:3**). The Craibstone data was not included in the control group analysis because the BTSCC data was a simple monthly arithmetic average. The BTSCC data from the other three herds was supplied as a 3-month Geometric Mean.

3.2.2.3 Criteria for selection of Phase-1c herds

Six of the 23 Phase-1a herds were selected because their BTSCC remained in excess of 400,000 one year after their original investigation (**Figure 3:3**). They were revisited and investigated for a second time using the protocol developed in this study.

3.2.2.4 Criteria for selection of Phase-2 herds

After 12 months of the project a further 8 SMRA herds (**Table 3:20**) were selected for bacteriological examination from the original 247 herds with average BTSCC in excess of 400,000 for the entire 11 month period June 1990 to April 1991 (**Figure 3:1**), again as near in proportion with **Figure 3:2** as possible.

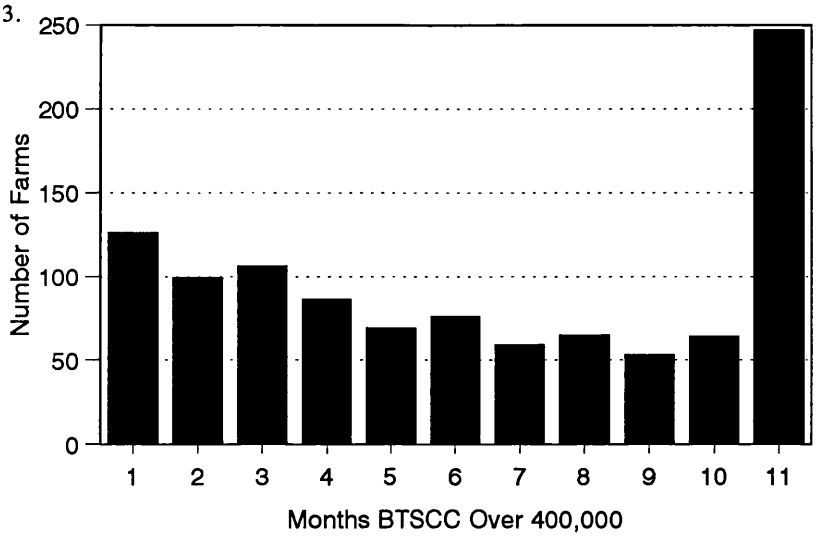


Figure 3:1
Identification of Phase-1a Herds
SCC Penalty June/90-April/91

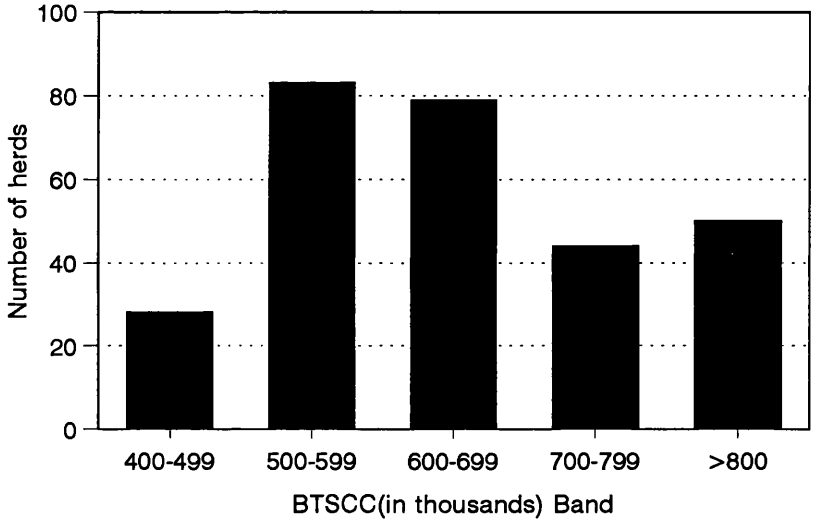


Figure 3:2
Selection of Phase-1a Herds
(n=23)

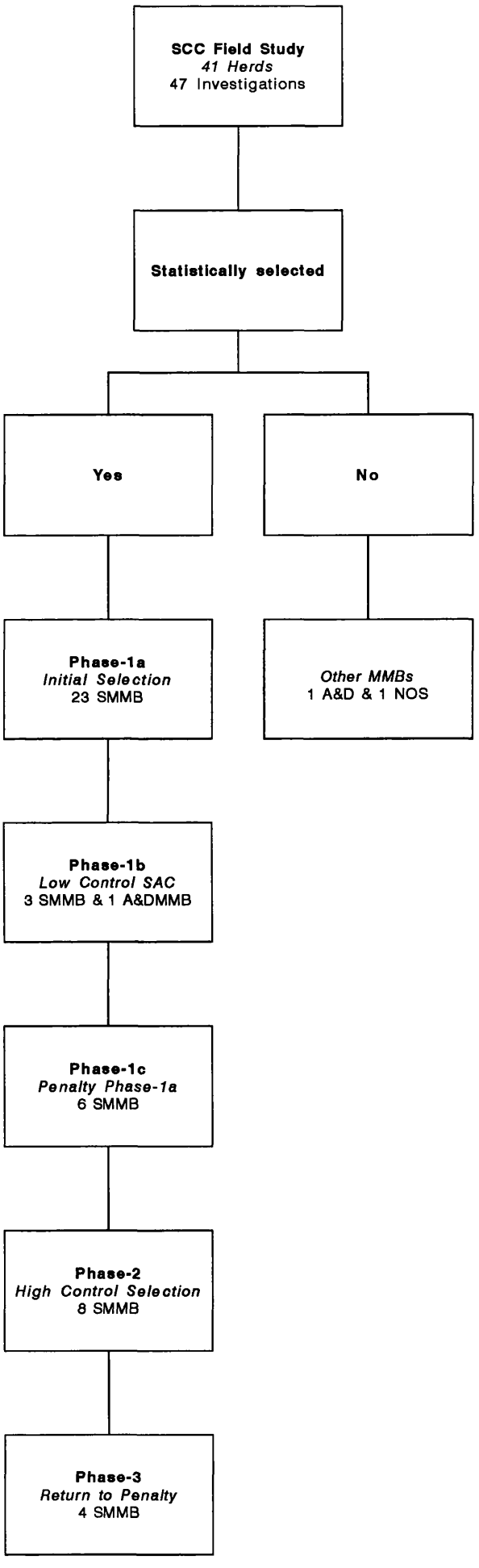


Figure 3:3
Plan/Flow diagram of herd investigations

BTSCC range (000)	Herd	Mean BTSCC(000) June/90-April/91
400-499	1	482
	2	487
	3	499
500-599	4	513
	5	517
	6	524
	7	530
	8	549
	9	553
	10	554
	11	571
	12	587
	13	589
600-699	14	615
	15	616
	16	621
	17	631
	18	635
	19	642
	20	651
>700	21	766
	22	772
	23	788
	24	804
	25	953

Table 3:19. Selection criteria for SCC project herds.

BTSCC (000)	Herd	Mean BTSCC(000) June/90-April/91
400-499	1	429
	2	464
	3	471
500-599	4	524
	5	542
	6	585
600-699	7	619
>700	8	737

Table 3:20. Selection criteria for Phase-2 project (high control) herds.

3.2.2.5 Criteria for selection of Phase-3 herds

A further group of 4 SMRA producers (**Figure 3:3**) were selected because their BTSCC moved below 400,000 following the introduction of premium payments in April 1991 but then moved back into penalty after January 1992 (**Table 3:21**). Initially the search looked for the pattern 10:0:12 (**Table 3:21**). The four SMRA herds nearest to this pattern are detailed in this table and these were the herds investigated.

3.2.2.6 Other herds

A further 2 high SCC herds, 1 in the A&DMMB region (herd 25 **Table 3:19**) and 1 in the NOSMMB region (herd 12 **Table 3:19**) also agreed to participate in the project. The latter 2 herds were not statistically selected but were identified by the respective local SAC Veterinary Investigation Centres as suffering from a serious subclinical mastitis problem. Since these herds were not statistically selected, their bacteriology results have not been included in the analysis. SAC Craibstone in the A&DMMB region was also investigated as low SCC herd. However the A&DMMB MQFILE database consisted of simple monthly arithmetic BTSCC data rather than the 3-month Geometric Mean SMMB data. This precluded analysis of SAC Craibstone BTSCC data with that of the 3 other low SCC SMMB herds.

3.2.3 Identification of problem cows

A protocol was established to select individual cows for bacteriological examination within the herds under investigation. An individual herd computer spreadsheet was created using "CA-Supercalc 5.1" (Computer Associates International Inc., California) software (**Table 3:22**). This contained the latest calving date, current lactation number and all ICSCC data for each lactating cow. Thus for example the Julian format (34178) of days after the base date of 01/01/1900 was equivalent to the standard calendar format (9/26/1993) for September 26, 1993. "CCGM" software was written to analyse the ASCII-format output of the "CA-Supercalc" individual farm file. The format of this analysis is presented in **Tables 3:23-26**. The "CCGM" programme calculated days in milk (**Table 3:23**) as the difference in the Julian-format dates (days from 1/1/1900) (**Table 3:22**) for the latest calving date and the date on which the analysis was performed. The Julian-format dates for the latest calving date and each ICSCC test (**Table 3:22**) were also used in calculating whether the ICSCC data referred to the previous or current lactation (**Table 3:23**). This data was then presented in cow number order (**Table 3:23**). A further table (**Table 3:24**) showing the mean ICSCC and cow number in ascending ICSCC order for the current lactation was also computed. The "CCGM" software analysed the number of cows in each 100,000 ICSCC band (**Table 3:25**). A more sophisticated analysis of the mean ICSCC of cows grouped by age and stage of lactation (**Table 3:26**) was also computed using the difference in the Julian date-format for the latest calving date and the date of ICSCC data analysis.

	Phase-3 Herd	No. of months BTSCC over 400,000 in period Jun/90-Mar/91	Apr-Dec/91	Jan-Dec/92
Target pattern		10	0	12
Actual pattern	1	9	3	12
	2	6	3	12
	3	6	2	9
	4	9	1	2

Table 3:21. Details of SMRA herds selected for Phase-3.

Cow No.	Lact No.	Calving Date	Julian calving date	ICSCC test date Julian ICSCC date
2	4	9/26/93	34178	0.201
3	10	11/16/93	34229	1.226
4	4	6/1/93	34061	1.757

Table 3:22. "CA-Supercalc" spreadsheet of herd ICSCC data.

Cow	Lact No.	Calving Date	Days in milk	Mean ICSCC Last lact.	This lact.	Last 3 ICSCC
1	6	931017	108	0.315	0.534	0.990
3	4	931223	41	0.344	0.038	0.038

Table 3:23. "CCGM" ICSCC data analysis presented in cow order for previous and current lactations.

Cow	Mean ICSCC
538	0.680
114	0.706
108	0.742

Table 3:24. "CCGM" ICSCC data analysis presented in mean ICSCC order for the current lactation.

ICSCC Band	No. of Cows
< 100	28
101-200	29
201-300	13
. .	. .
801-900	1
> 900	9

Table 3:25. "CCGM" ICSCC data analysis presented as number of cows in 100,000 ICSCC bands.

Lact No.	Days in lactation					
	less than d140		d140 - d280		d280	
	Records	ICSCC	Records	ICSCC	Records	ICSCC
1	82	0.065	45	0.132	18	0.147
2-5	208	0.137	131	0.301	25	0.512
5+	66	0.535	60	0.642	15	1.084

Table 3:26. "CCGM" ICSCC analysis presented by age and stage of lactation.

3.2.4 Assessment of mastitis control

During the herd visit at which quarter milk samples were collected from high ICSCC cows, the mastitis control practices of participating herds were noted. In addition to bacteriology results, the subsequent investigation report included herd-specific advice on implementation of the mastitis control recommendations outlined in **Table 3:27**. Subsequently the uptake of these recommendations was recorded in a questionnaire format (**Appendix II**). This was completed either at a revisit or by a telephone call on Thursday November 25 1993 in some cases up to 2 years after the first visit. The arithmetic mean of the 6 months BTSCC figures May to October 1993 was used as a medium-term measure of mastitis control to access the SCC reduction associated with each measure. This study utilized some more complex statistical analyses and the prepared data was analysed by Mr A Sword of Scottish Agricultural Statistics Service (SASS).

3.2.5 Examination of the subclinical mastitis infection

3.2.5.1 Collection of milk samples

The CCGM analysis illustrated in **Table 3:24** was used to identify individual cows for bacteriological investigation with arithmetic mean ICSCC LS5 (in excess of 283,000) for the current lactation. The cows for sampling were presented as a group in the milking shed at the start of afternoon milking and the dairyman was instructed to remove gross contamination from the teats by washing. The teats were then dried using individual paper towels irrespective of whether this was the routine herd practice or not. The skin of each teat was then sprayed with 70% ethanol and the teat end scrubbed with an individual paper towel. The teat was again sprayed with alcohol and allowed to evaporate to dry before sampling. Milk sampling was conducted wearing an arm-length plastic rectal glove over which latex surgical gloves were worn. The latter were sprayed with alcohol and wiped with a paper towel between cows and were discarded after sampling 10 cows. The first few streams of foremilk were discarded from each teat prior to collection of a sample from each milking quarter. One capped, sterile plastic bottle (Sterilin) which had previously been labelled with the cow identification number and quarter (LF/LH/RF/RH) was filled with up to 20 ml of milk. The bottle was held at 45° to the horizontal during sampling to minimise entry of contaminating debris. It was then capped immediately and placed in a box with internal divisions designed to hold each bottle upright. On return to the Veterinary Investigation Centre the quarter samples were placed in a cold storage room (4°C) within 2 hours of sampling where they remained overnight.

3.2.5.2 Bacteriological examination of milk samples

Identification of the major mastitis pathogens was undertaken by SAC Veterinary Services scientific staff using an adaptation of the standard Veterinary Investigation Service regime (Anon, MAFF 1984).

Principle	**Traditional Advice**	**Contemporary Adjunct**
Reduce level of infection	Dry Cow Therapy	Sensitivity results
	Treat clinical cases	Early dry cow therapy
		Treat high ICSCC cows
	Cull	Cull known *S.aureus* carriers
Reduce transfer of infection	Post-milking teat dipping	Pre-milking teat dip
		Use paper towels in udder preparation
		Breed own replacements
		Milk high ICSCC cows last
Limit predisposing factors	Annual machine test	Modern cluster liners
		Automatic cluster removal[1]

[1] not applicable to 10 byre-milking herds.

Table 3:27. Factors considered in mastitis control advice to project herds.

In summary, 0.1 ml of agitated milk from each quarter milk sample bottle was spread on each of a sheep blood agar and Edward's medium bacteriology plates. Presumptive macroscopic identification of colonies was first undertaken after 24 hours incubation at 37°C (**Table 3:28**). A Gram-stained smear of each colony type was then prepared for microscopic examination. *S. aureus* appeared as pairs and clusters while the mastitis streptococci tended to form chains. Staphylococci were differentiated from streptococci on the basis of a positive catalase test while a positive coagulase test confirmed the isolate as *S. aureus*. *S. uberis* was presumptively identified on the basis of a darkened zone of aesculin hydrolysis surrounding the colony on Edward's medium. Biochemical tests were used to differentiate *S. uberis* from other organisms which exhibited aesculin hydrolysis such as *S. faecalis* and *S. bovis* (**Table 3:29**).

Lancefield serogrouping by slide co-agglutination (Phadebact Streptococcus Test, Karo Bio Diagnostics, Sweden) was also used to identify *S. agalactiae* and *S. dysgalactiae*. This system utilises antibody specific against Group A (*agalactiae*) or Group B (*dysgalactiae*) streptococci, which are bound to Protein A on the surface of non-viable staphylococci. The interaction of the group-specific reagent with the streptococcus forms a co-agglutination lattice visible to the naked eye.

Large, grey, shiny, haemolytic or non-haemolytic colonies of Gram-negative rods were biochemically identified as *E. coli* by a catalase-positive, oxidase-negative profile. Further biochemical tests were used to identify occasional isolates of other gram-negative mastitis bacilli such as *Pseudomonas aeruginosa* or *Klebsiella aerogenes*.

3.3 Results

3.3.1 Analysis of BTSCC results

The MQFILE had 3 parts representing the three co-operating Milk Marketing Boards in Scotland. **Table 3:30** shows the length of time the data has been accumulated, the number of producers and highest and lowest figures while **Table 3:31** summarises the percentage of producers who had a geometric BTSCC figure (on which premiums and penalties are based) greater than 400,000. In all cases the percentage of herds in penalty followed the overall SCC figure for the Board (see **Figures 3:4-6**).

3.3.1.1 SMMB MQFILE

This database extended for 53 months from June 1990 to October 1994. **Figure 3:4** shows that peaks of the mean or overall 'Board' SCC figure (BSCC) were apparent every year for the months of August to October. The maximum value for this 'Board' SCC of 354,000 was recorded in September 1990. The minimum of 278,000 was recorded 46 months later in July 1994. Premium payments for a geometric mean BTSCC below 250,000 were introduced by the SMMB in April

	Sheep Blood Hydrolysis	Aesculin Hydrolysis	Serological Group	CAMP test
S. agalactiae	β	-	B	+
S. dysgalactiae	α	-	C	-
S. uberis	α/Non Haem	+	E	+/-
S. faecalis	α/Non Haem	+	A	-
S. bovis	α	+	A	-

Table 3:28 Cultural profile used in the identification of streptococci.

	Streptococcus				
	agalactiae	*dysgalactiae*	*uberis*	*faecalis*	*bovis*
Glucose	+	+	+	+	+
Lactose	+	+/-	+	+	+
Sucrose	+	+	+	+/-	+
Maltose	+	+	+	+	+
Trehalose	+	+/-	+	+	+/-
Salacin	+/-	+/-	+	+	+
Sorbitol	-	+/-	+	+	+/-
Inulin	-	-	+	-	+/-
Mannitol	-	-	+	+	+/-
Rhaffinose	-	-	-	-	+
Aesculin	-	+/-	+	+	+
Methblue milk	-	-	-	+	-
Ox Bile Agar	-	-	-	+	-

Table 3:29 Biochemical profile used in the identification of streptococci.

		SMMB	NOSMMB[1]	A&DMMB[1]
Size	Months	53	58	55
	Producers	2203	99	153
Board SCC	Minimum	278	250	279
	Maximum	354	427	461
	Oct/94	293	294	293

[1] Calculated using Arithmetic Mean SCC (BTSCC) producer data

Table 3:30. Constituents of MQFILE database.

	Producers (%) SCC over 400,000		
	SMMB	A&DMMB	NOSMMB
Maximum	31	54	47
Minimum	15	12	8

Table 3:31. Percentage of producers with BTSCC over 400,000 (1990 - 1994).

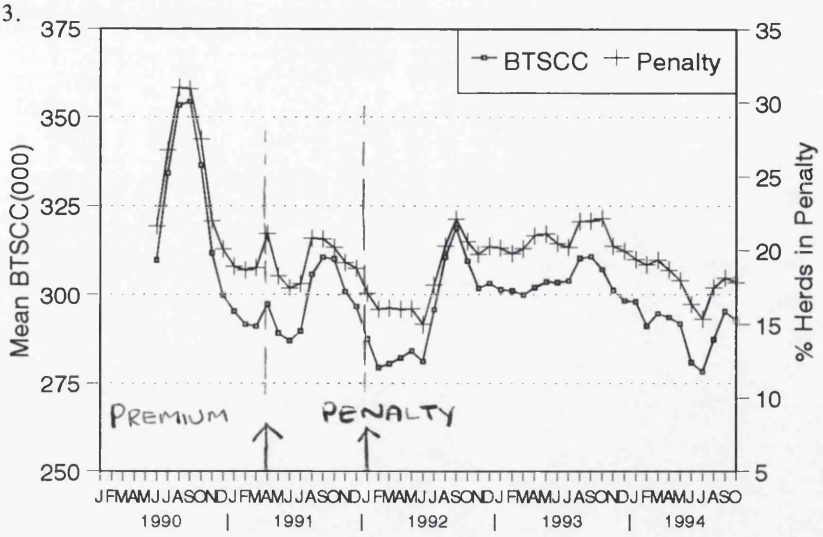


Figure 3:4
Somatic Cell Count Profile 1990-1994
SMMB

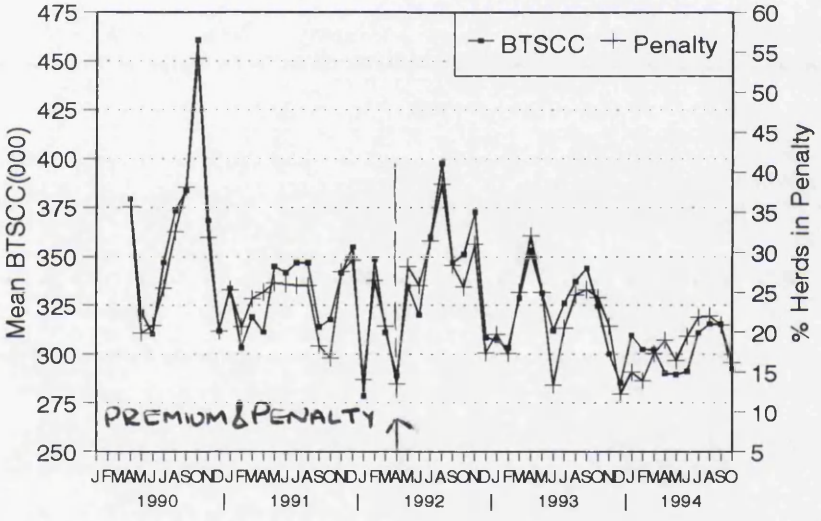


Figure 3:5
Somatic Cell Count Profile 1990-1994
A&DMMB

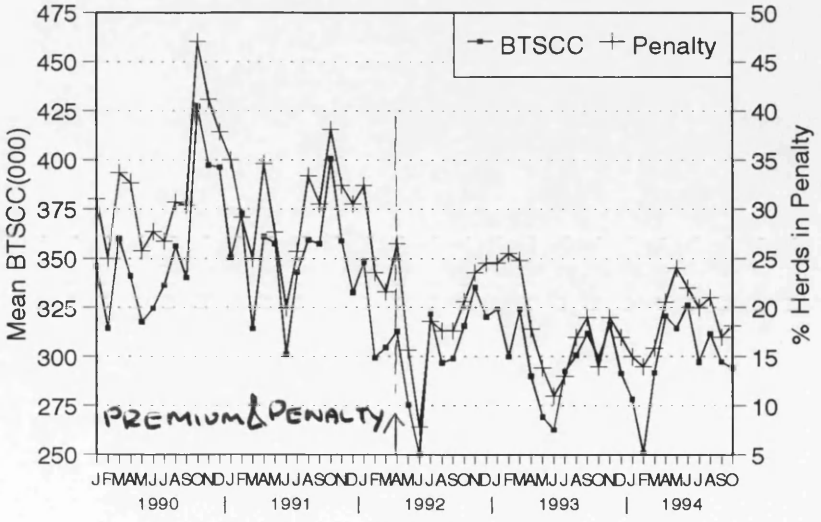


Figure 3:6
Somatic Cell Count Profile 1990-1994
NOSMMB

1991 and were followed in January 1992 by the introduction of payment penalties for 3-month Geometric Mean BTSCC in excess of 400,000. In October 1994 the average was 293,000. The profile of the percentage of all SMMB producers whose geometric mean BTSCC was above 400,000 (i.e. in penalty after January 1992) is also presented in **Figure 3:4** and both the introduction of premiums and penalties were related to falls in the SCC.

3.3.1.2 A&DMMB MQFILE

Figure 3:5 shows the profile for A&DMMB producers which, as mentioned in the Materials and Methods, was calculated from the monthly arithmetic average data rather than the 3-month Geometric Mean data used in **Figure 3:4**. **Figure 3:5** shows a maximum value of 461,000 was recorded in October 1990 and peaks occurred in the months of August to September in the following years. There was a marked reduction in the number of producers with high BTSCC after April 1992 when penalties and premiums were introduced. The Board SCC minimum of 279,000 was recorded 15 months later in January 1992 while the latest available figure for October 1994 was 293,000. The percentage of herds with monthly BTSCC in excess of 400,000 showed a close relationship with the monthly BTSCC (**Figure 3:5**). A maximum of 54% of producers had BTSCC in excess of 400,000 in October 1990 while a minimum of 12% of producers recorded a BTSCC in excess of 400,000 in December 1993.

3.3.1.3 NOSMMB MQFILE

The monthly BTSCC data illustrated in **Figure 3:6** was also supplied by NOSMMB as a simple arithmetic average. The general BTSCC trend was the same with a peak value in autumn 1990 (**Figure 3:6**). In particular, prominent peaks were apparent every year for the months of November to December. The profile of the percentage of all NOSMMB producers whose monthly BTSCC was above 400,000 (also presented in **Figure 3:6**) showed a maximum of 47% of producers in October 1990. The simultaneous introduction of premiums and penalties in April 1992 was related to a fall in the BTSCC. The close relationship between the percentage of herds with BTSCC in excess of 400,000 and BTSCC showed a minimum of 8% of producers were in penalty in June 1992. In 1994 a minimum of 14% of producers recorded BTSCC in excess of 400,000 although by October 1994, the last figure available, this figure had risen to 18%.

3.3.1.4 Variation around penalty

Figure 3:7 shows a histogram of the number of SMMB herds with a geometric mean BTSCC in a particular range during September for the four year period 1990 to 1993. The September 1990 profile shows the distribution of all herds when the maximum 31% of SMMB producers had a 3-month Geometric Mean BTSCC in excess of 400,000 and the overall SMMB SCC was a maximum of 355,000. A quite consistent pattern has been apparent within producers grouped on the basis

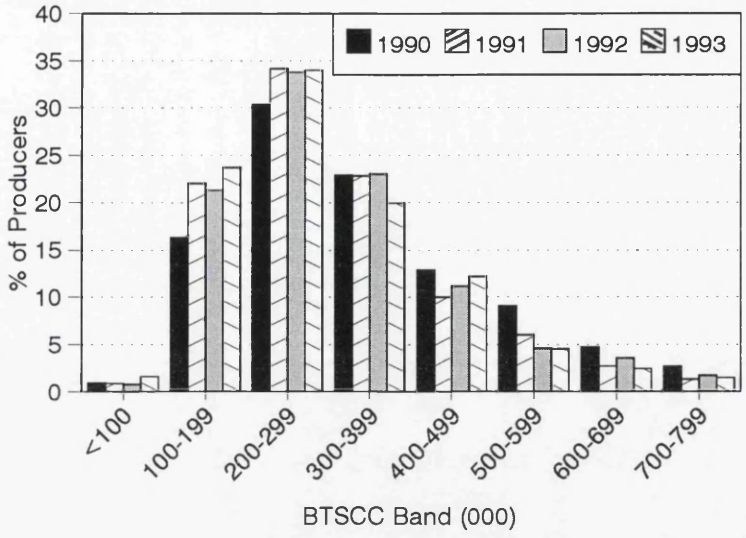


Figure 3:7
Movement of SMMB herds based on annual BTSCC
September

of the SCC ranges shown in **Figure 3:7**. The number of producers with an annual mean SCC figure between 500,000 and 799,000 displayed a steady downward trend and there was a concomitant increase in the number of producers below 300,000 particularly in the group 100,000 to 199,000. **Figure 3:8** shows that most (269, 53%) of the 503 producers within the 101 to 200,000 BTSCC band for 1993 had entered this band as a result of their improved performance on the previous 12 months. However only 36 (7%) producers had reduced their annual mean BTSCC from above 300,000 and all these were from the 301 to 400,000 group. More recently with an overall rise in the mean BTSCC for SMMB producers there was a regressive interchange of producers between the 300,000 to 399,000 and 400,000 to 499,000 groups. **Figure 3:9** shows which mean SCC band producers were in during 1992 before 'entry' into the 401 to 500,000 band in 1993. **Figure 3:9** shows that the BTSCC of 56 (30%) producers had increased from 1992, but only 35 (19%) producers had improved their mean BTSCC. Equally out of the 385 herds which recorded a 1993 mean BTSCC of 301 to 400,000 (**Figure 3:10**) only 13 (3%) producers had improved their mean BTSCC and 130 (34%) producers had suffered an increase over the 1992 figure.

3.3.1.5 Seasonality

Examination of these bands also quantified the influence of season. The seasonal variation noted in the progress of the 3 Boards (**Figures 3:4-6**) was best demonstrated in **Figure 3:11** which shows the profile of the number of SMMB producers which recorded a geometric mean BTSCC for that month of 100,000 to 199,000. This figure illustrated a regular seasonal pattern in which the maximum number of producers with a figure within this range was recorded in March (mean 30.9% of all producers) and the minimum in September (mean 20.4% of all producers). These were months already noted as low and high respectively for the mean Board SCC figures (**Figure 3:4**). This seasonal variation became less distinct as the mean annual SCC figure rose (**Figure 3:12**) although a minimum still occurred between January and April (mean 8.0% of all producers) and a maximum occurred in August and September (mean 11.8% of all producers).

3.3.1.6 Effect of milk recording and use of ICSCC service

Figure 3:13 illustrates the proportion of SMRA and non-recording SMMB herds which recorded a geometric mean BTSCC over 400,000 and thus "in penalty" after January 1992. Both groups consistently recorded a seasonal peak in September and a minimum in June (**Table 3:32**).

The number of SMMB producers who used the ICSCC service each month since it was introduced in May 1990 is illustrated in **Figure 3:14**. These were virtually all SMRA herds since, for example, only 4 non-recording herds undertook an ICSCC herd test in December 1993 when 373 herds (41% of SMRA herds; 16.9% of all producers) undertook such a test. Of these herds, 57 were in penalty in December 1993 and thus represented only 13% of the herds in penalty

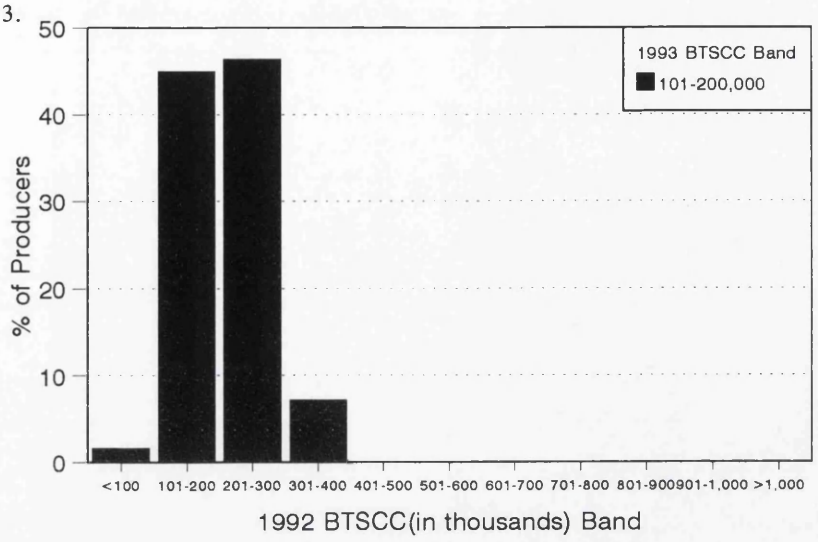


Figure 3:8
1992/3 Change in annual BTSCC bands
SMMB Herds in 101-200,000 band in 1993

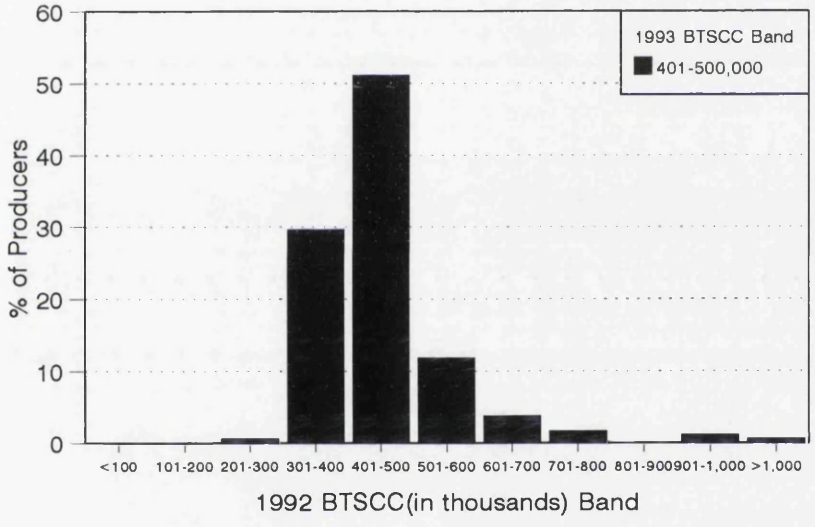


Figure 3:9
1992/3 Change in annual BTSCC bands
SMMB Herds in 401-500,000 band in 1993

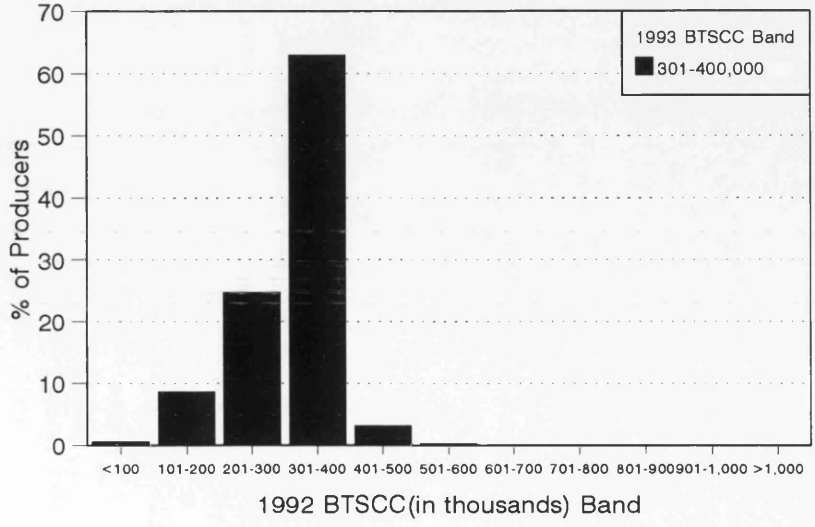


Figure 3:10
1992/3 Change in annual BTSCC bands
SMMB Herds in 301-400,000 band in 1993

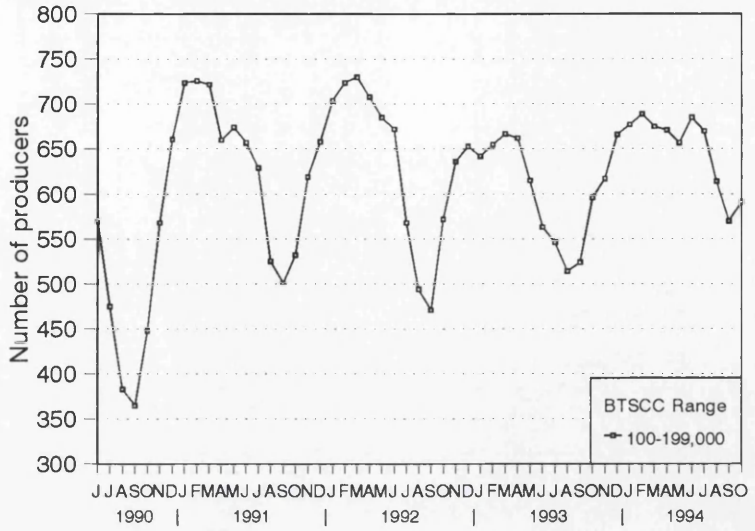


Figure 3:11
Number of SMMB herds in BTSCC band each month
100-199,000

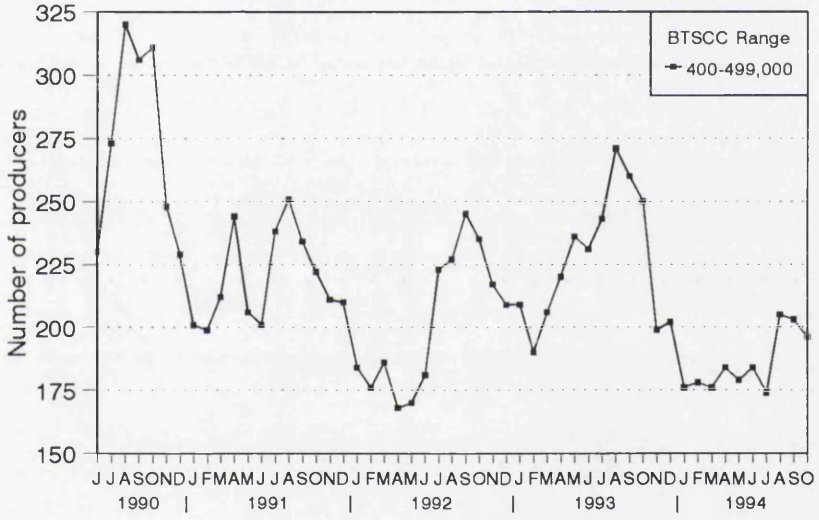


Figure 3:12
Number of SMMB herds in BTSCC band each month
400-499,000

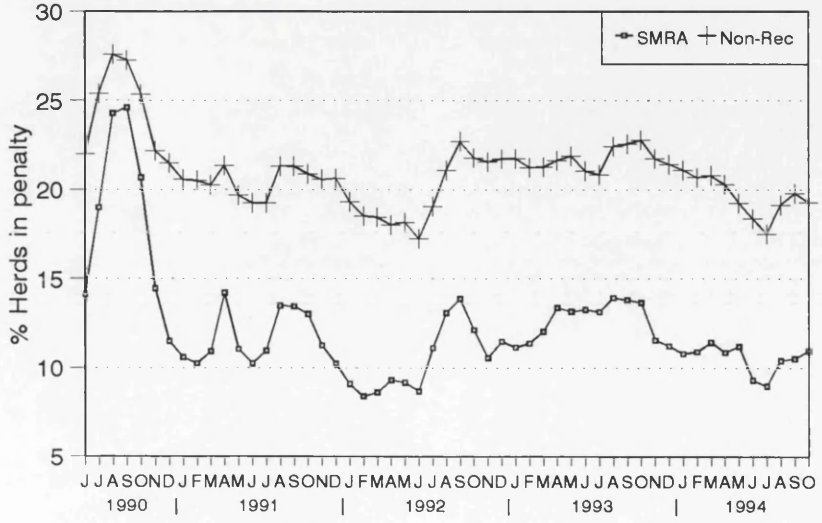


Figure 3:13
Proportion of SMRA and non-recorded herds in penalty each month
June/90 - October/94

Year	Percentage of Producers with BTSCC over 400,000			
	SMRA		Non-Recorded	
	Jun	Sep	Jun	Sep
1990	14	25	28	36
1991	10	13	23	26
1992	8	14	20	28
1993	13	14	26	28

Table 3:32. Influence of season upon the proportion of SMRA and non-recording herds with BTSCC in excess of 400,000.

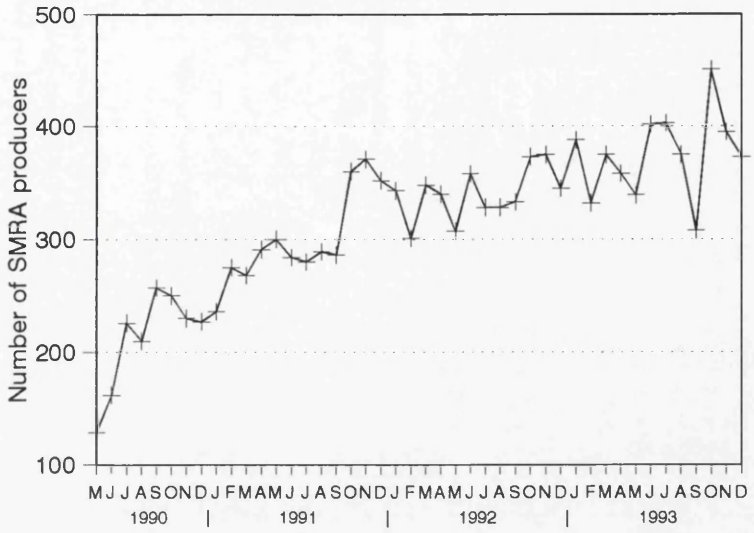


Figure 3:14
Number of SMRA producers using ICSCC service
May 1990 - December 1993

that month. **Figure 3:15** presents a histogram showing the frequency of use of the ICSCC service in the 18 month period July 1992 to December 1993. 11.5% of the herds undertook only 1 test in this period while 11.8% undertook a test each month. The majority of the remaining 76.7% of participating herds undertook tests on an irregular basis. Of the small number of herds in penalty which were using the ICSCC service, 37 (65%) tested regularly (**Figure 3:16**).

Figure 3:17 shows an almost linear relationship existed between the number of months in penalty and mean BTSCC for all SMMB producers in the 24 month period following the January 1992 introduction of penalties. The mean BTSCC of producers who were not penalised in this period was 207,000 while, for example, the mean BTSCC of the 46 herds who were in penalty for 8 months between January 1992 and December 1993 was 375,000. **Figure 3:18** shows that the BTSCC of most of the 100 SMRA producers in penalty in December 1993 was 401 to 500,000 BTSCC band in contrast to the much wider distribution of non-recording herds.

3.3.1.7 Contribution Index

Figure 3:19 shows the number of months herds with "adequate" (<100) and "high" (>100) Contribution Indices which had BTSCC figures in excess of the 400,000 EC threshold (in penalty) in 1993. The majority of high Contribution Index herds which were in penalty remained so for at least 3 months and as can be seen the 26% of herds which were continuously in penalty have a considerable input.

3.3.2 Field investigations

3.3.2.1 Phase-1a herd investigations

A total of 2240 quarter samples were collected from 572 cows in the 23 Phase-1a SMMB project herds over the period 11/10/91 to 15/12/92. The 23 individual herd computer spreadsheets contained a total of approximately 2,500 cows. A database containing the bacteriology and QSCC data from each herd was constructed in a similar manner to the QSCC database for the Aberdeen data (Chapter 2) and analysed. The prevalence of cows infected by a major pathogen (*S. aureus*, *S. agalactiae*, *S. dysgalactiae*, *S. uberis* and *E. coli*) is presented in **Table 3:33**. Significant mastitis pathogens were isolated from 406 (71%) cows, of which 372 (92%) were infected by a single pathogen.

The quarter infection prevalence, broken down by major isolate, is presented in **Table 3:34**. Allowing for 48 non-lactating quarters, significant mastitis pathogens were isolated from 828 (37%) quarter samples, of which 47 (6%) were infected by two significant bacteria. *S. agalactiae* was the most common significant mastitis pathogen, recovered from 19 (83%) of the 23 herds. *S. agalactiae* was found in 496 (57%) of all significant isolations (**Figure 3:20**) and was the only pathogen isolated from 470 (95%) of these quarters. *S. aureus* (SFAU) was isolated from 250 (29%) infected quarters and was therefore the second most common mastitis pathogen. It was the

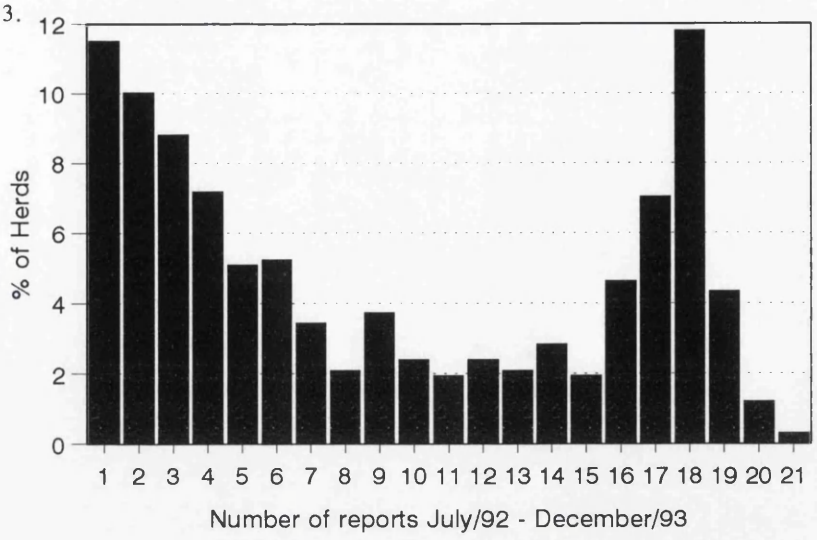


Figure 3:15
Herds using ICSCC service (n=669)
Frequency of use July/92-December/93

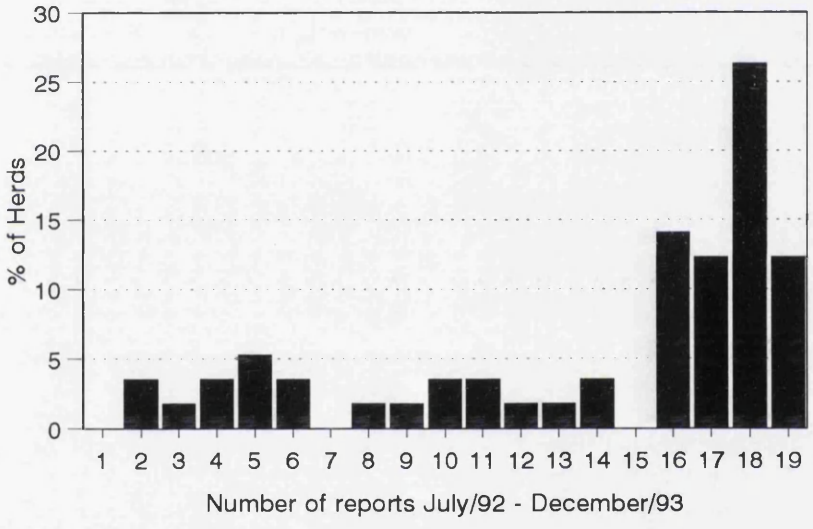


Figure 3:16
Frequency of use of ICSCC service
Herds in penalty December/93 (n=57)

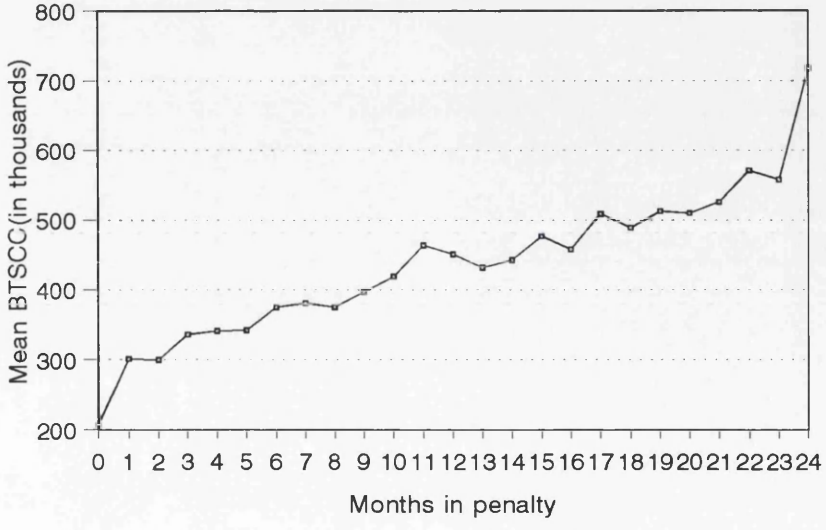


Figure 3:17
Cumulative months in penalty of SMMB producers with high SCC
January/92 - December/93

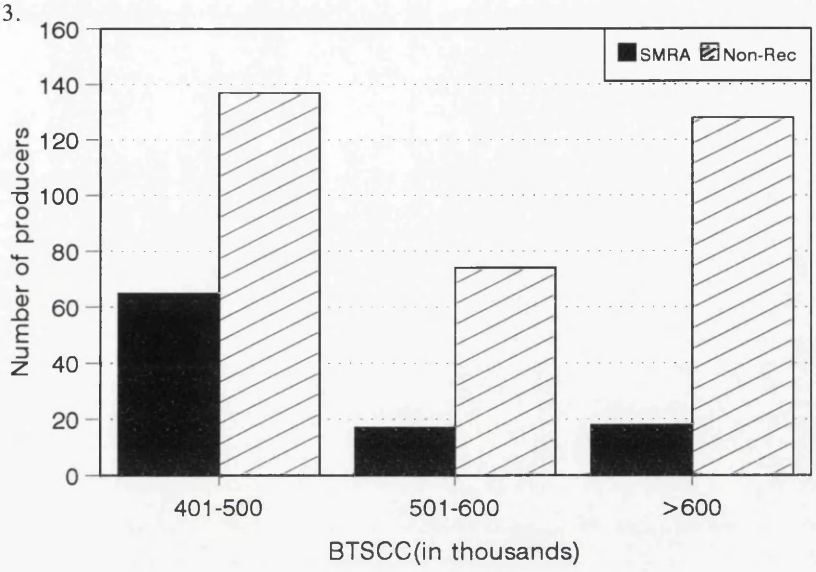


Figure 3:18
December/93 BTSCC performance of SMRA and non-recorded herds in penalty

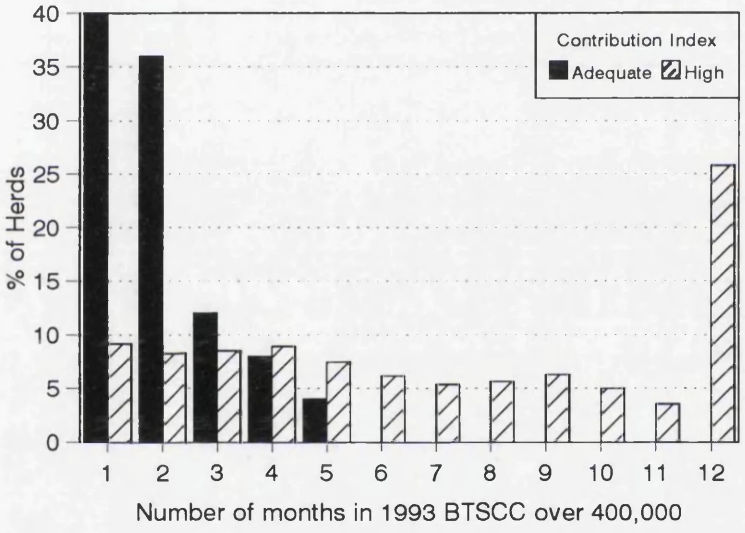


Figure 3:19
SCC Contribution Index of herds in penalty
Relationship to months in penalty in 1993

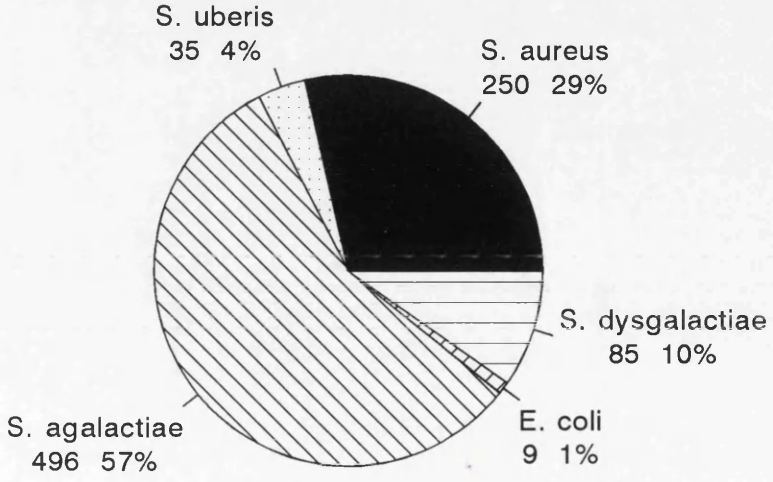


Figure 3:20
Isolates from Quarter Milk Samples
Phase-1a herds (n=23)

No. of Quarters	No.(%) of cows with infection	
	Single	Dual
1	139 (34.2)	14 (3.4)
2	124 (30.5)	7 (1.7)
3	62 (15.3)	13 (3.2)
4	47 (11.5)	

Table 3:33. Prevalence of cows infected by a significant mastitis pathogen: Phase-1a herds.

	Quarter Sample Infections		All Isolates (%)
	Single (%)	Dual (%)	
TOTAL	2240 (100)		875 (100)
No Significant Isolate	1412 (65.1)		
Significant Isolate	781 (34.9)	47 (100)	
S. aureus	207 (9.2)	43 (46)	250 (28.6)
S. agalactiae	470 (21.0)	26 (28)	496 (56.7)
S. dysgalactiae	68 (3.0)	17 (18)	85 (9.7)
S. uberis	28 (1.3)	7 (7)	35 (4.0)
E. coli	8 (0.4)	1 (1)	9 (1.0)

Table 3:34. Quarter sample results from investigation of 23 high BTSCC SMMB herds.

only mastitis pathogen isolated from 207 (83%) of these quarters.

These findings were clearly different from the SAC Aberdeen data (**Table 3:35**). The datasets containing the bacteriology profile of those samples with SCC over 283,000 from SAC Aberdeen and Phase-1a herds were cross-classified by mastitis pathogen since that was the threshold used here. The main difference was that the majority of SAC Aberdeen isolates were *S. aureus* but in contrast *S. agalactiae* was the most prevalent isolate from Phase-1a herds.

Table 3:36 shows the frequency with which significant mastitis pathogens infected multiple quarters of the same cow in the 23 Phase-1a herds. This indicates a trend towards multiple quarter infection by both *S. aureus* and *S. agalactiae*. In order to further investigate this the data presented in **Table 3:36** was "collapsed" for statistical analysis into 2 datasets: either animals infected in only 1 quarter or in 2 or more quarters. The prevalence of *S. agalactiae* in multiple infected quarters made the largest contribution to the chi-square statistic of 51.8 on 3 degrees of freedom and caused the significant ($P<0.001$) difference between the 2 collapsed datasets.

The frequency of isolation of *S. aureus* and *S. agalactiae* by month of lactation is presented in **Table 3:37**. **Figure 3:21** illustrates this data, showing that the proportion of quarters infected with *S. agalactiae* increased throughout lactation. Analysis of this dataset revealed a chi-square statistic of 35.6 on 9 degrees of freedom. This indicated that the prevalence of infection by *S. agalactiae* and *S. aureus* throughout lactation was significantly different ($P<0.001$). The chi-square contributions were interpreted as showing that *S. aureus* was more prevalent in early lactation but *S. agalactiae* tended to predominate in late lactation. Further analysis of this data by the complex technique of Logarithm Linear Modelling was not done.

Figure 3:22 shows the median SCC of quarter samples from which no significant isolate (NSI), *S. aureus* and *S. agalactiae* were isolated respectively. The SCC of quarter samples from cows selected on the basis of average ICSCC did demonstrate a clear relationship with the presence of a significant mastitis pathogen as illustrated in **Figure 3:22** by *S. agalactiae* and *S. aureus*. The SCC of quarter samples which were not infected by a significant mastitis pathogen did not increase with the age of the cow.

3.3.2.2 Phase-1c herd investigations

The profile of mastitis bacteria in these six herds had changed markedly compared to their first investigation. The proportion of *S. aureus* isolates had increased and it had become the most prevalent pathogen (**Figures 3:23 & 3:24**). However *S. agalactiae* was still present in these herds and accounted for 22% of the significant isolates recovered at the follow-up herd investigations (**Figure 3:24**). **Table 3:38** presents the prevalence of multiple quarter infections for each pathogen at the second herd visit. **Table 3:38** was collapsed for statistical analysis into 2 datasets of either animals infected in only 1 quarter or in 2 or more quarters. Chi-square analysis was used to establish that multiple quarter infections were no more common at the follow-up herd test compared

	Aberdeen	Phase-1a
Total	5807	2193
NSI	5002	1412
S. aureus	504	207
S. agalactiae	171	470
S. dysgalactiae	78	68
S. uberis	49	28
E. coli	3	8

Table 3:35. Comparison of quarter sample bacteriology (single significant isolates).

	No. of quarters infected			
	1	2	3	4
S. aureus	108	28	9	4
S. agalactiae	101	68	39	29
S. dysgalactiae	47	9	1	0
S. uberis	16	6	0	0
E. coli	8	0	0	0

Table 3:36. Prevalence of multiple quarter infections: Phase-1a project herds.

Month of Lactation	Quarter Samples		
	Total	*S. aureus*	*S. agalactiae*
1	168	20	14
2	188	23	20
3	224	20	47
4	124	9	21
5	120	20	20
6	76	13	24
7	124	13	36
8	108	6	28
9	120	7	42
10	56	4	6

Table 3:37. Prevalence of significant pathogen infection in Phase-1a herds.

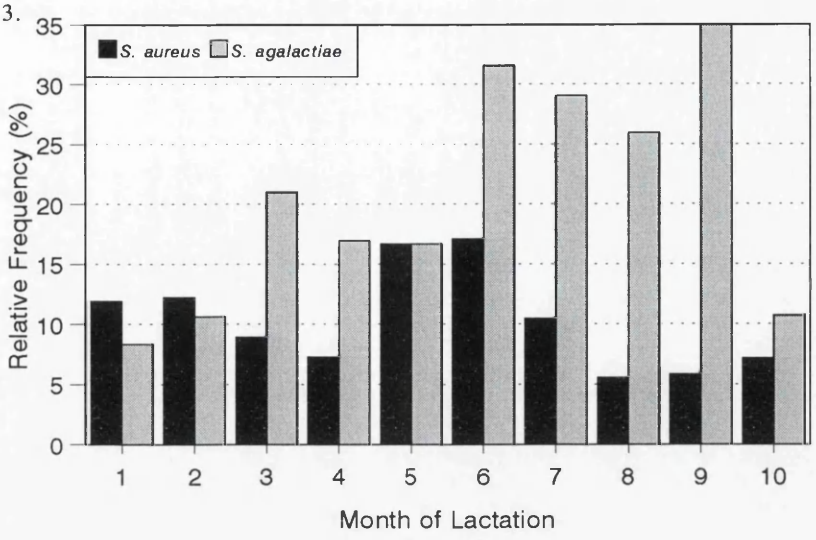


Figure 3:21
Isolation of *S. aureus* & *S. agalactiae*
Stage of lactation effect in Phase-1a herds (n=23)

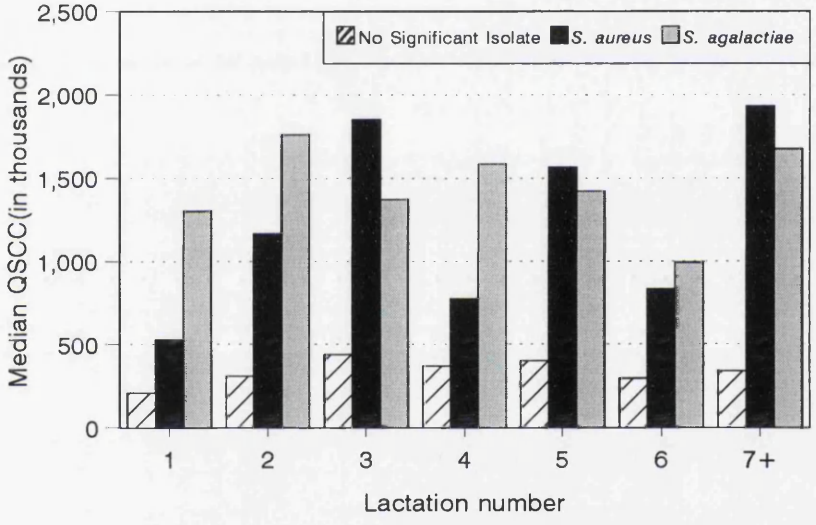


Figure 3:22
Effect of mastitis pathogen on QSCC
Variation with lactation number in Phase-1a herds (n=23)

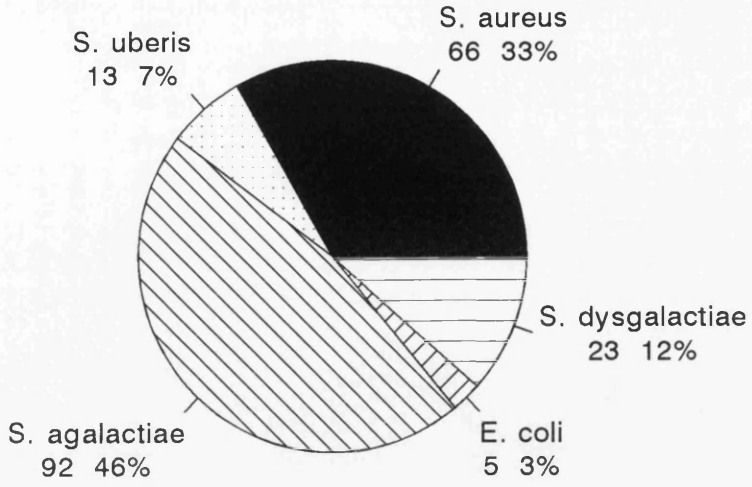


Figure 3:23
Investigation of 6 herds in penalty
Visit 1 (Phase-1a)

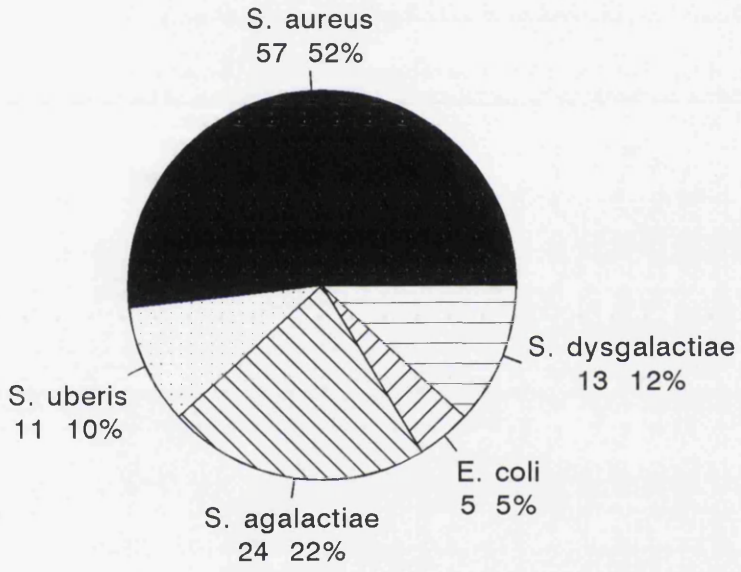


Figure 3:24
Investigation of 6 herds in penalty
Visit 2 (Phase-1c)

		No. of quarters infected			
		1	2	3	4
S. aureus	Test 1	25	14	3	0
	Test 2	18	8	4	1
S. agalactiae	Test 1	20	13	5	5
	Test 2	6	3	1	0
S. dysgalactiae	Test 1	14	4	0	0
	Test 2	8	0	0	1
S. uberis	Test 1	5	3	0	0
	Test 2	4	2	1	0
E. coli	Test 1	4	0	0	0
	Test 2	5	0	0	0

Table 3:38. Prevalence of multiple quarter infections: Phase-1c project herds.

to the first investigation and thus did not skew the distribution of isolates illustrated in **Figures 3:23 & 3:24.**

3.3.2.3 Phase-2 herd investigations

These 8 SMRA herds were selected 12 months into the project from the original 247 herds with average BTSCC in excess of 400,000 for the entire 11 month period June/1990 to April/1991. The results of the bacteriological investigation of 151 cows in these 8 herds with ICSCC over 283,000 are presented in **Table 3:39**. Allowing for the 23 non-lactating quarters, overall 35.3% of the quarters were carrying a significant mastitis pathogen. *S. agalactiae* was the most common mastitis pathogen, followed by *S. aureus* (**Figure 3:25**). It can be seen that the pattern of pathogen isolation is very similar to that of Phase-1a (**Figure 3:20**).

3.3.2.4 Phase-3 herd investigations

These 4 SMRA herds were selected because their BTSCC moved below 400,000 following the introduction of premium payments in April 1991 but then moved back into penalty after January 1992. The results of the bacteriological investigation of 98 cows in these 4 herds with ICSCC over 283,000 are presented in **Table 3:40**. Allowing for 12 non-lactating quarters, overall 47.5% of the quarters were carrying a significant mastitis pathogen (**Figure 3:26**). Again this pattern is very similar to all the initial investigations above.

3.3.3 Analysis of BTSCC performance following advisory input

3.3.3.1 Phase-1a herds

Figure 3:27 illustrates the 53-month BTSCC profile for the 23 Phase-1a project herds (**Figure 3:3**) since June 1990. The peak geometric BTSCC of 670,000 was recorded in August 1990, but by July 1992 the BTSCC had fallen by almost 41% to 398,000. The BTSCC of 280 producers from the original high SCC group were analysed for comparison (**Figure 3:27**). The July 1994 minimum BTSCC of 490,000 for the non-project herds occurred 47 months after the August 1990 peak of 690,000. By October 1994 the group mean BTSCC for the 229 herds still in production was 511,000.

After an initial rapid reduction in 1991/2 the mean BTSCC of these 23 herds showed little or no overall progress. The BTSCC/TBC profiles for the individual project herds constitute **Appendix I.** The mean BTSCC for 1993 of the 23 Phase-1a herds is presented in **Table 3:41**.

Six herds did not incur any penalty throughout 1993, 12 had some months with penalty but most without any but 5 herds remained continuously in penalty throughout 1993. The progress of the 23 Phase-1a herds is presented in **Table 3:42**, which compared their 1993 mean BTSCC result with the 1990/1 figure on which herd selection was based (**Table 3:19**). Analysis of variance (ANOVA) was used to demonstrate a highly significant reduction in the group mean BTSCC of

	Samples	% of Samples	% of positive samples
No isolate	379	64.7	
S. aureus	60	10.2	29.0
S. agalactiae	101	17.2	48.8
S. dysgalactiae	15	2.6	7.2
S. uberis	29	5.0	14.0
E. coli	2	0.3	1.0
TOTAL	586	100.0	35.3

Table 3:39. Results from investigation of 8 high BTSCC control (Phase-2) herds.

	No. of isolates	% of Quarters	% of positive quarters
No isolate	207	52.5	
S. aureus	60	15.2	32.1
S. agalactiae	87	22.1	46.5
S. dysgalactiae	21	5.3	11.2
S. uberis	12	3.1	6.4
E. coli	7	1.8	3.7
TOTAL	394	100.0	47.5

Table 3:40. Results from investigation of 4 Phase-3 high BTSCC herds.

1993 mean BTSCC(000)	No. of Phase-1a herds
<250	1
250-400	11
401-600	8
>600	3

Table 3:41. 1993 BTSCC Performance of 23 Phase-1a herds.

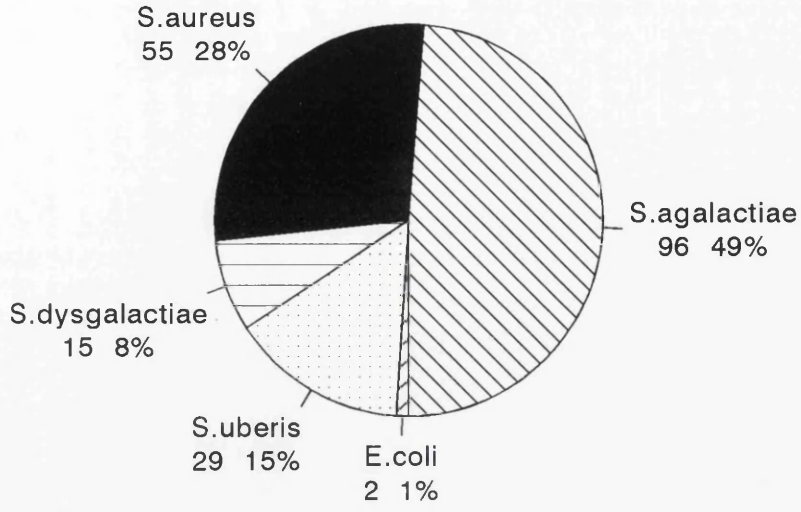


Figure 3:25
Investigation of 8 herds in penalty
Phase-2 (High Control)

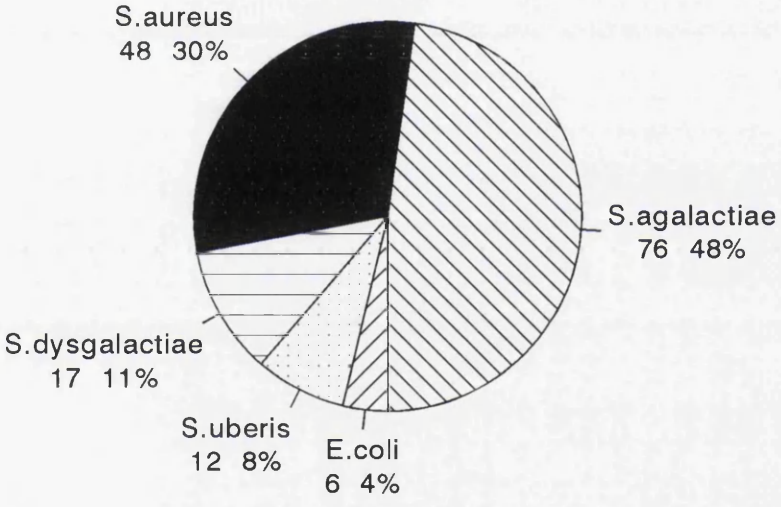


Figure 3:26
Investigation of 4 "return to penalty" herds
Phase-3

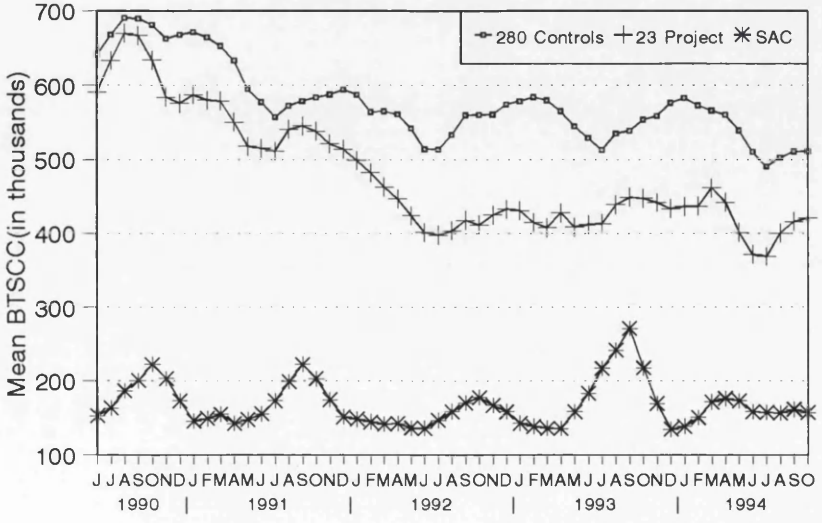


Figure 3:27
SCC Project Herds' progress
Mean BTSCC profile June/90-October/94

Project herd	Mean BTSCC		No. months BTSCC >400,000 (1993)
	1990/91	1993	
1	482	367	4
2	487	341	3
3	499	373	3
4	513	377	5
5	517	336	0
6	524	680	12
7	530	401	9
8	549	380	3
9	553	410	3
10	554	294	0
11	571	488	10
13	589	254	0
14	615	510	9
15	616	290	0
16	621	532	12
17	631	603	10
18	635	495	9
19	642	350	4
20	651	671	12
21	766	339	0
22	772	496	12
23	788	249	0
24	804	587	12

Table 3:42. Progress of BTSCC reduction in 23 Phase-1a project herds.

the 23 collaborating herds from 605,000 in 1990/91 to 414,000 in 1993 ($P<0.001$).

Figure 3:28 shows that all 3 of the project herds originally from the "400 to 499,000" group had reduced their BTSCC. Similarly 5 of the 9 "500 to 599,000" herds, 2 of the 7 "600 to 699,000" herds and 2 of the 4 "700,000+" herds had controlled their subclinical mastitis problem in the *medium term* as measured by the reduction of the *annual* BTSCC below the EC standard. Despite clear progress in most cases, 11 herds remained firmly in penalty.

3.3.3.2 Phase-1b herds

The BTSCC performance of the 3 low BTSCC SAC control herds within the SMMB region is also presented in **Figure 3:27** for comparison with that of the 23 Phase-1a commercial project herds. The BTSCC of these 3 SAC herds showed an annual peak between September to October. This annual variation illustrated the seasonal changes in BTSCC experienced by low BTSCC herds. It also coincided with their main calving period when the number of late and early lactation cows was at an annual maximum.

3.3.4 Assessment of mastitis control

Appendix II contains the individual herd records of mastitis control following the herd-specific advisory input, of which a regularly updated profile of BTSCC and TBC (**Appendix I**) from the "MQFILE" database was an important element. The average size of these co-operating herds was 88 cows (range 24 to 260). Of the 10 byre systems, 5 herds were housed in the byre, 2 were housed in cubicles and a further 3 were kept in a straw yard. Both last mentioned groups were put into the byre to milk. An estimate of the proportion of the herd which had been culled for mastitis within the previous 18 months and thus since the on-farm investigation indicated a group mean of 13% of the herd (range 0 to 33%). The May to October 1993 BTSCC arithmetic mean for the 23 herds was 431,000/ml (range 230 to 746,000). The mean BTSCC was calculated for fixed herd characteristics (**Table 3:43**) and the management elements of mastitis control in **Table 3:44**. Statistical analysis of each element of the latter revealed that only one parameter was associated with a significant BTSCC reduction. The average SCC figure for the 16 farms who had adopted the use of paper towels (373,000/ml) as part of their udder preparation technique, either simply as a dry wipe or to dry teats after washing, was very significantly ($P<0.001$) lower than in herds which had not (564,000/ml). The small sample size precluded a comprehensive quantitative analysis of the BTSCC associated with the individual elements of a mastitis control programme. However most parameters did show a positive influence on reducing the BTSCC figure.

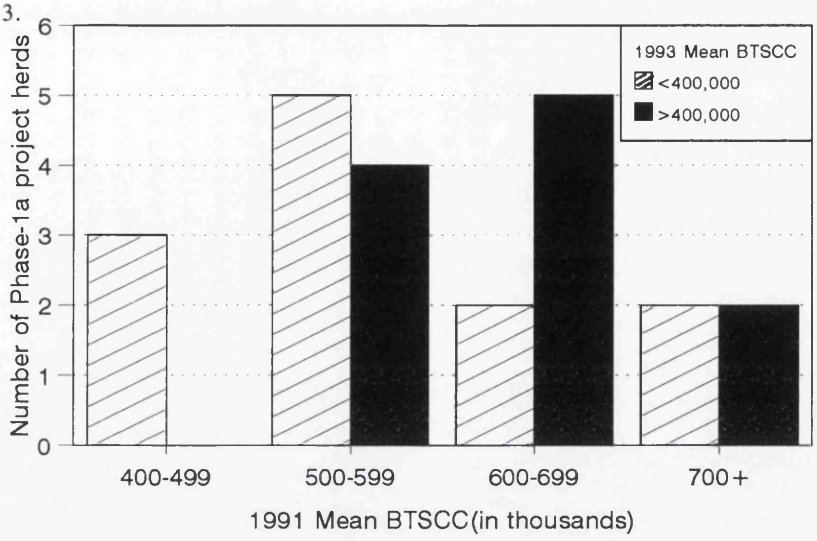


Figure 3:28
Movement of project herds from SCC band at selection
Phase-1a Herds (n=23)

Parameter		No. of herds	BTSCC (May-Oct/93)
Herd Size	0-49	4	410
	50-99	11	476
	100-149	5	380
	150+	3	378
Milking System	Byre	10	483
	Parlour	13	391
Udder preparation	Wet	11	468
	Dry	12	398

Table 3:43. Herd size and milking system of 23 SCC project herds (SMMB).

Factor	Yes No. of herds (6-month BTSCC)	No No. of herds (6-month BTSCC)
Dry cow therapy (DCT)	22(435)	1(339)
Post-milking teat dipping (PMTD)	13(438)	10(422)
Treat lactating high SCC cows	7(428)	16(432)
Cull for high SCC (Cull)	20(420)	3(503)
Milking machine test (MMT)	18(410)	5(508)
Breed replacement heifers (BRH)	17(427)	6(443)
Automatic cluster removal[1] (ACR)	7(415)	6(347)
Use individual paper towels (PapT)	16(373)	7(564)

[1] not applicable to 10 byre-milking herds.

Table 3:44. Mastitis control questionnaire: relationship of management to mean BTSCC in 23 project herds.

3.4 Discussion

The two major objectives of this chapter were firstly to study the epidemiology of high BTSCC in all Scottish dairy herds by the analysis of existing BTSCC data and secondly to study a small number of high BTSCC herds in depth. An initial target, determined by financial resources, was the selection of 25 herds for bacteriological investigation which could be reliably described as representative of all high SCC herds. The establishment of the MQFILE database meant that for the first time BTSCC data for all Scottish producers was stored on a personal computer and in a single accessible database with the flexibility to both analyse the BTSCC trends of all producers and produce an individual herd profile.

Variation in the mean monthly BTSCC of all producers, the "Board" SCC, was demonstrated to have a distinct seasonal component and was also influenced by the introduction of payment penalties (**Figures 3:4-6**). This was best demonstrated by the number of herds with BTSCC 100 to 199,000 which tended to be highest in March and lowest in September (**Figure 3:11**). The seasonal variation of this elite group was interpreted as reflecting the underlying seasonal variation since the low BTSCC figure implied a low subclinical mastitis incidence (Pearson & Greer, 1974) and thus the BTSCC increase of late lactation was likely to be physiological due to increased drying-off and calving August to October. This finding is broadly in line with Schukken et al. (1990) who reported that the Ontario Board SCC showed a seasonal minimum in April and a peak in October for the same reason.

As data was available prior to April 1991, the monthly distribution of all producers permitted a qualitative analysis of the effect of premiums and penalties. Important landmarks in the SMMB SCC profile were the introduction of premium payments in April 1991 and penalties in January 1992. The SMMB SCC decreased in the period of advance publicity prior to the introduction of premiums but rose following the actual introduction of penalties in January 1992 (**Figure 3:4**). This was interpreted as indicating that the threat of penalties was worse than the reality and did not provide sufficient financial motivation for problem herds to reduce their BTSCC. A quantitative assessment of the trends underlying this rise showed that the BTSCC of 34% of herds with 1993 annual mean BTSCC 300 to 400,000 (**Figure 3:10**) showed an increase on the 1992 figure. Indeed the underlying trend in 1993 has been a rise in Board SCC. Without use of the complex mathematical techniques used by Schukken et al. (1992a&b), it was not possible to quantify the change in SMMB SCC caused by premiums and penalties. These authors calculated that the Ontario mean monthly BTSCC decreased by 58,000 when the regulatory limit was reduced from 800,000 to 750,000 in August 1990.

SMRA herds were demonstrated to have a monthly mean BTSCC 50,000 lower than their non-recording contemporaries and be less likely to incur penalties (**Figure 3:13**). This was interpreted to stem directly from firstly the benefit of good herd records ensuring efficient dry cow management and secondly a higher uptake of ICSCC testing (**Figure 3:14**). However the use of

ICSCC was quite complex. A distinct pattern was evident within the group of SMRA producers who did use the ICSCC service (**Figure 3:15**). The group mean BTSCC for those herds (36%) who used the service infrequently was higher than that of herds (12%) using the service every month. This suggested that the herds with high BTSCC only used the ICSCC service as a snap-shot of the situation, possibly in conjunction with misguided culling. In contrast the premium BTSCC performance of SMRA herds which consistently used the ICSCC service clearly suggested the data was effectively used as a management tool. However this conclusion needs to be tempered as recently more "penalty" herds have been using ICSCC and here 37 (65%) of the 57 herds were actually using regular ICSCC service regularly (**Figure 3:16**). However it appears they were not using this information since most had remained in excess of 400,000 for some time.

Non-recording herds were 2.5 times more likely to be in penalty than SMRA herds (**Figure 3:13**). Very few non-recording herds carried out regular ICSCC testing. The need for these herds to generally improve their recording systems and in particular to manage their BTSCC more effectively is obvious. The main reasons for lack of ICSCC investigation were disinterest because of the low level of SCC penalties, ignorance of the existence of the ICSCC service, "inconvenience" because the non-recording producer was required to collect the individual cow samples himself and, of course, cost. However since as part of this study free bacteriology of clinical cases was offered and very few samples were received it would appear inconvenience and apathy prevailed.

An analysis of the MQFILE database demonstrated that high BTSCC was a widespread problem with approximately 10% of producers consistently in penalty (**Figure 3:1**). The analysis of the entire MQFILE database showed an almost linear relationship between the annual mean BTSCC and the number of months continuously in penalty (**Figure 3:17**) with two important sequelae. Firstly, premium BTSCC performance (less than 250,000) was required to completely avoid penalty throughout the year, especially where a tight calving pattern emphasised the physiological SCC rise of early and late lactation milk. Secondly, a minority of producers was not sufficiently motivated by payment penalties to resolve their persistent subclinical mastitis problem and thus remained continuously in penalty. Study using the "Contribution Index" developed by Schukken et al. (1992b) showed that persistently high SCC herds in Scotland were found to make a major contribution to the Board SCC and thus could not be ignored (**Figure 3:19**). This is in contrast to the findings of Schukken et al. (1992b) in Ontario who concluded that most farms with high BTSCC did not have high SCC contributions. They therefore concluded that in order to keep the Ontario Board SCC low an incentive should be offered to farms with low SCC.

In total 35 SMMB high SCC herds were the subject of 41 herd investigations. In conjunction with the observations of the farm visit to these high SCC herds, bacteriological examination of quarter milk samples permitted herd specific mastitis control recommendations. Their implementation was monitored using the BTSCC data of MQFILE and reported to each

individual producer at periodic intervals (approximately quarterly) as a graphical profile of the BTSCC and TBC data (**Appendix I**). This simple presentation of individual herd performance was considered a very useful part of the herd support strategy developed in this chapter because it gave producers a long-term appreciation of the BTSCC performance in addition to presenting the current position.

S. agalactiae and *S. aureus* were identified as the most prevalent major mastitis pathogens in high BTSCC herds (**Figures 3:20, 3:25 & 3:26**). *S. agalactiae* was found in 83% of the original herds and accounted for 57% of the significant mastitis pathogens isolated from cows with lactation mean ICSCC in excess of 283,000 while 29% of isolates were *S. aureus* (**Figure 3:20**). In this respect these findings differed substantially from those of SAC Aberdeen data (**Figures 2:1 & 2:10**). The finding of a tendency to multiple quarter infections (**Table 3:36**) was explained by the highly contagious nature of *S. agalactiae* while infections by *S. aureus* tended to be more chronic in nature and thus overlap with more recent infections in adjacent quarters.

The second investigation of 6 herds which remained in penalty after 12 months (Phase-1c) showed that *S. aureus* had become the most prevalent mastitis pathogen (**Figures 3:23 & 3:24**). However *S.agalactiae* was still far too frequent an isolate given that most herds had treated all *S. agalactiae* carrier cows during lactation and claimed to have treated all cows with dry cow therapy. Thus despite the reduction in the level of *S. agalactiae* the deficiencies identified in the mastitis control programme even at the second visit, particularly attention to detail in milking routine, resulted in a failure to contain spread from those animals which were not cured. This meant a continuation of the *S. agalactiae* infection and a relative increase in *S. aureus* which is less responsive to antibiotic (**Figure 3:24**).

The mastitis pathogen profile of the group of 8 Phase-2 high BTSCC herds was not significantly different from that of the 23 Phase-1a contemporary herds (**Figure 3:25**). Both Phase-1a and Phase-2 groups were selected from the same original list of 247 herds continuously in penalty for the 11 month period June/1990 to April/1991 (**Figure 3:1**). This Phase-2 group could therefore be considered to be representative of the remaining unselected or "control" herds since they had been subjected to a barrage of press, veterinary and other advice on SCC and subclinical mastitis control during that year. This group therefore served as a further comparison with the initial 23 herds which were investigated (**Figure 3:3**).

The BTSCC profile of herds which moved below 400,000 when premiums were introduced but exceeded this figure after January 1992 to incur penalties allowed the investigation of what factors, if any, caused this short-term improvement (**Table 3:21 & Appendix IIc**). *S. agalactiae* was the most prevalent mastitis pathogen found in these 4 herds (Phase-3) and would suggest they were typical high BTSCC herds (**Figure 3:26**). However their attempt to reduce BTSCC was limited to culling high ICSCC cows (**Appendix IIc**) and only produced a transient improvement because the underlying infectious problem was not addressed. The 41 herd investigations failed

to find any veterinary reason for the BTSCC of some of the herds remaining high apart from their inability to apply the recommended mastitis control programme (**Table 3:27**). The continued presence of *S. agalactiae* in these herds reinforced this view (**Figure 3:24**). The increase with stage of lactation in the proportion of cows infected by *S. agalactiae* (**Figure 3:21**) was interpreted as reflecting the length of exposure in a heavily infected high BTSCC herd throughout lactation. However the finding using DNA fingerprinting techniques of different bacterial genotypes within the *S. aureus* isolates from the same farm has confirmed the potential of an underlying difference in pathogenicity (Platt et al., 1994). The possibility of explaining the observed differences in herd *S. aureus* prevalence on the basis of bacterial genomic variation is currently the subject of further investigation. The potential for food poisoning was confirmed by the detection of enterotoxins in 28% of all *S. aureus* isolates. The most common was Enterotoxin C which was found in 65% of all isolates in Scotland (Platt et al., 1994).

In summary, the implementation of EC 92/46 will cause difficulty for a minority of Scottish producers, particularly those 10% with BTSCC consistently over the accepted threshold of 400,000 (**Figure 3:1**). The MQFILE database proved invaluable in establishing the extent and contributing factors of the problem of high BTSCC among Scottish producers. The evolving producer response to differential BTSCC payments (**Figures 3:4-6**) was interpreted to show insufficient direct financial incentive for many of the producers with a consistently high count. Study of the "Contribution Index" developed by Schukken et al. (1992b) showed that persistently high BTSCC herds in Scotland were found to make a significant contribution to the national SCC (**Figure 3:19**) and thus, in contrast to the findings of Schukken et al. (1992b) in Ontario, could not be ignored. Further analysis also showed that an annual mean BTSCC performance of less than 250,000 was required to completely avoid being in excess of 400,000 at some time in the year (**Figure 3:17**).

The field studies described in this chapter have shown that the main cause of high BTSCC in herds in Scotland was due to subclinical mastitis and that the most common cause in 83% of herds and 57% of the significant isolations was *S. agalactiae* (**Figure 3:20**). Although this pathogen responds well to the major elements of mastitis control (**Table 3:27**) its high prevalence in multiple quarter infections (**Table 3:36**) and late lactation cows (**Figure 3:21**) reflects a highly contagious epidemiology. *S. aureus*, the other common pathogen associated with high BTSCC and 29% of isolations (**Figure 3:20**), was more difficult to control but nevertheless the application of herd specific advice resulted in significant progress being made. *S. aureus* was found to have become the most prevalent mastitis pathogen (**Figure 3:24**) when those herds which remained over 400,000 after approximately 1 year were re-examined. Deficiencies in mastitis control permitted continuation of the *S. agalactiae* infection and a relative increase in *S. aureus*. A separate field investigation of herds which moved below 400,000 when premiums were introduced in April 1991 but then exceeded this figure after January 1992 (**Table 3:21**) revealed culling high ICSCC cows without adoption of a herd-specific mastitis control strategy (**Appendix IIc**) as the cause of their

unsustained BTSCC improvement.

The work of Chapter 4 to undertake a statistical analysis of individual mastitis control procedures in terms of BTSCC reduction was inspired by a preliminary attempt using herd records (**Appendix II**) constructed within the work of this chapter. The MQFILE facility to examine the BTSCC data of specific subgroups of producers allowed such a correlation of BTSCC data with information from another but significantly more extensive database of herd management.

CHAPTER 4. MANAGEMENT SURVEY.

4.1 Introduction

During the field studies of this thesis it became evident that there was considerable scepticism amongst producers, especially those in penalty, about the value of the major elements of mastitis control, this despite the evidence from the field study reported in Chapter 3. The primary objective of mastitis control is to reduce the level of intramammary infection (Grommers et al., 1985). Mastitis status, and consequently BTSCC, can be maintained at an acceptable level by the use of established control techniques such as post-milking teat dipping, dry cow therapy and milking machine maintenance (David & Jackson, 1984).

Post-milking teat dipping has been consistently identified as a significant herd determinant of BTSCC (Pearson et al., 1972; Mein et al., 1977; Hoare et al., 1979). Pearson et al. (1979) reported a higher frequency of post-milking teat dip use in low SCC herds than in high SCC herds. In the last major survey of mastitis in England and Wales it was reported that 63.5% of herds used post-milking teat dipping only, while 59.6% of all herds used both post-milking teat dipping and dry cow therapy.

The value of dry period therapy over lactation treatment is also well recognised (Dodd & Neave, 1970; Dodd & Griffin, 1975; Philpot, 1979; Natzke, 1981). Field trials by Harmon et al. (1986) showed a marked reduction within 1 to 3 years in the percentage of cows or quarters with intramammary infections resulting from a programme of post-milking teat dipping and dry cow therapy. Natzke (1971) reported that, in general, while antibiotic treatment of mastitis during lactation cured less than 60% of the pathogenic infections, therapy at drying-off eliminated over 90% of *S. agalactiae* and 40 to 70% of *S. aureus* infections. Thus the major advantages of dry cow therapy include prevention of new dry period infections which would otherwise overcome the cow's natural defences and a higher bacteriological cure rate than that achieved by treatment of the lactating cow (Philpot, 1969; Philpot, 1979). However McDermott et al. (1983) administered lactating antibiotic therapy to cows with subclinical mastitis diagnosed by ICSCC above 400,000 and found no advantage in milk production over control cows treated only for clinical mastitis. Seymour et al. (1989) used LS5 (ICSCC over 283,000) to select cows with subclinical mastitis for treatment. They could not find a significant advantage for either subsequent monthly ICSCC or milk production.

The scientific literature also contains several reports which have focused on the effectiveness of various management aspects in the control of mastitis. The report by Hutton et al. (1990) was based on the efforts Washington State Dairy Herd Improvement producers made to maintain low average BTSCC. High ICSCC cows were milked last in half of the low BTSCC herds in contrast to only 13% of the high BTSCC herds. Hutton et al. (1990) also found a greater percentage of low BTSCC herds culled cows because they had been treated for clinical mastitis. Pearson et al.

(1979) reported that individual paper towels were used to prepare udders before milking as frequently in high as in low BTSCC herds. Wilson & Richards (1980) also reported that the prevalence of udder infections declined as herd size increased due to greater use of mastitis control measures especially culling.

The objective of the work described in this chapter was to provide contemporary evidence using Scottish dairy herds that the basic tenants of the mastitis control strategy developed by Dodd et al. (1969) were effective in reducing BTSCC figures. The presentation of information in this format from all Scottish dairy herds was intended to reinforce the findings of the field study reported in Chapter 3 concerning the value of a committed approach to the application of mastitis control as a means of reducing somatic cell count. The effect of udder preparation technique, especially the use of individual paper towels, on BTSCC was also investigated in this chapter since the scientific literature either only documented an effect on TBC or rarely discussed a relationship with BTSCC.

4.2 Materials and Methods

4.2.1 The "EPIDEM" database

An 11-question confidential census form of their farm as at May 1 1993 was returned to their own Milk Marketing Board by all dairy producers in Scotland since this was a statutory obligation. All this information was then numerically coded and used to establish a computer database for statistical analysis by the SMMB Commercial Department. All information relating to an individual herd was recorded on a single line of this spreadsheet format and confidentiality was assured by identification only on the basis of producer code. An ASCII-format file was supplied on computer disc by Mr David Young, SMMB Commercial Department on Wednesday February 16, 1994 of selected, coded census information (**Table 4:1**). Only the datafields which related to mastitis control (**Table 4:2**) were extracted for each herd and the file was arranged in order of increasing producer numerical code. Only the information on SMMB herds was further analysed because this was by far the largest distinct group of producers whose 3-month geometric mean BTSCC for the period was conveniently available as the MQLAMAY.93 file (**Table 3:2**). This file was then also arranged by increasing producer numerical code. Finally the census and BTSCC files were merged for the creation of a new "EPIDEM" database of 2,187 SMMB producers. Genstat (Genstat 5 Committee, 1987) statistical software was used for the analysis of this database. The effect of milking system, milking hygiene (udder preparation, post-milking teat dipping), dry cow therapy, milking machine testing, milk recording and overall implementation were investigated.

Column	1	2	3	4	5	6
Factor	Milking system	Post-milking teat dipping	Dry cow therapy	Udder preparation	Milking machine test	Milk recording
Code	2	1	1	1	2	3
Description	Byre pipeline	Yes	Yes	Water & cloth	No	No

Table 4:1. Mastitis control information from May 1993 Census of 2187 SMMB herds: Arrangement of computer-coded ASCII file.

Information	Response options		Code
Milking system	Byre	Buckets	1
		Pipeline	2
	Parlour	Herringbone	3
		Rotary	4
		Other	5
Post-milking teat dip	Yes/No		1/2
Dry cow therapy	Yes/No		1/2
Udder preparation	Water & cloth		1
	Continuous water flow		2
	None		3
Milking machine test	Yes/No		1/2
Milk recording	With SMRA		1
	With another company		2
	No		3

Table 4:2. Mastitis control information from May 1993 Census of 2187 SMMB herds: Computer coded response options.

4.2.2 SMMB Milking Machine Test Data

The SMMB Producer Services Department provided an ASCII-format computer file of all SMMB herds who had an annual milking machine test contract with SMMB itself on and as at Thursday February 3 1994. The then latest available 3-month geometric mean BTSCC for December 1993 was used for analysis of this factor. The MQLADEC.93 file was also used to define how many SMMB producers did not have an annual milking machine testing contract with SMMB itself.

4.3 Results

4.3.1 The "EPIDEM" database

The "EPIDEM" database examined consisted of selected information from 2187 SMMB producers. All producers fulfilled their legal obligation of making a census return but 57 producers elected to supply the minimum information which was to answer only questions 2 (dairy herd breeds) and 7 (milking system). **Table 4:3** shows the non-response rate of the remaining 2130 producers to other selected questions. In summary less than 0.5% of producers failed to give a response to any question.

Analysis of the remainder of the database was restricted to the information from 2,114 producers which was complete. The initial analysis of the raw MQLAMAY.93 BTSCC data indicated a distribution skewed by some very large BTSCC values. When all MQLAMAY.93 BTSCC values over 1,000,000 were disregarded and the statistical analysis repeated, the relationship between milk recording and BTSCC was found to differ fundamentally from that calculated using the complete dataset. This indicated that the high raw BTSCC results made a significant contribution to the statistical analysis, for which it was more appropriate to use logarithm-transformed data. The statistical significance of the results were calculated on the logarithm-transformed dataset.

4.3.2 Effect of milking system

An analysis of milking systems showed that the mean BTSCC of 1486 (70.3%) herds milking in a parlour (252,000) was very significantly ($P<0.001$) lower than 628 (29.7%) herds milking in a byre (307,000) (**Tables 4:4 & 4:5**). Results from the field study supported this trend.

4.3.3 Milking Hygiene

4.3.3.1 Udder preparation

The relationship between the method of udder preparation, milking system and BTSCC is presented in **Table 4:6**. The majority of producers with a byre system (543, 86%) used an udder cloth and this was associated with a higher BTSCC (309,000) than the 67 herds who did no preparation (297,000) but this was not statistically significant. Within byre systems, the lowest mean BTSCC

Information	Number of producers not responding
Teat Dip	10
Dry Cow Therapy	5
Udder Preparation	6
Milking Machine Test	5

Table 4:3. Non-Response rate in SMMB Census Data (May 1993).

Milking System	Producers(%)
BYRE: with buckets BYRE with pipeline	32 (1.51) 596 (28.19)
PARLOUR: herringbone PARLOUR: rotary PARLOUR: other	1393 (65.89) 9 (0.43) 84 (3.97)

Table 4:4. Milking systems within SMMB region (May 1993).

		BYRE	PARLOUR	s.e.d
Census	$\log_{10}$(BTSCC)	2.4878	2.4008	0.01012
	BTSCC(000)	307	252	
Field study	BTSCC(000)	483	391	

Table 4:5. Effect of milking system on BTSCC.

		UDDER PREPARATION			F Pr
		Water & Cloth	Continuous Flow	None	
BYRE	No. (%)	543 (86)	18 (3)	67 (11)	
	$\log_{10}$(BTSCC)	2.4904	2.4660	2.4728	0.771
	BTSCC(000)	309	292	297	
PARLOUR	No. (%)	460 (31)	698 (47)	328 (22)	
	$\log_{10}$(BTSCC)	2.4378	2.4072	2.3353	<0.001
	BTSCC(000)	274	255	216	
ALL HERDS	No. (%)	1003 (47)	716 (34)	395 (19)	
	$\log_{10}$(BTSCC)	2.4663	2.4087	2.3586	<0.001
	BTSCC(000)	293	256	228	

Table 4:6. Effect of udder preparation protocol on cell count.

of 292,000 was recorded by the 18 herds who used continuous water flow udder preparation technique. In sharp contrast, the proportion of parlour systems who did not undertake udder preparation was double that of byre systems (22% v 11%). The mean BTSCC (216,000) of 328 (22%) parlour herds who did not wet the udder was very significantly lower than that of either the wet preparation protocols ($P<0.001$). A majority of 698 (47%) parlour systems performing udder preparation used a continuous water flow system and recorded a group mean BTSCC of 255,000. This was very significantly lower ($P<0.001$) than the 460 (31%) parlour systems using water and a common udder cloth (274,000). Unfortunately in view of the field data there was no record of the use of individual paper towels but the mean BTSCC (228,000) of 395 (19%) producers who did not wet the udder before milking was very significantly ($P<0.001$) lower than that of the 716 (34%) producers who used a continuous water flow wash system.

4.3.3.2 Post-milking teat dipping

The mean cell count of the 1489 (70%) herds who used a post-milking teat dip (255,000) at some time during the year was very significantly lower ($P<0.001$) than those 625 (30%) herds which did not (298,000). The census indicated that almost half the byre systems did not teat dip,and these herds also recorded a higher mean BTSCC (**Figure 4:1** & **Table 4:7**).

4.3.4 Dry Cow Therapy

The mean cell count of 1902 (90%) herds who used Dry Cow Therapy (DCT) (260,000) was very significantly lower ($P<0.001$) than those 212 (10%) herds who did not (339,000) (**Figure 4:2** & **Table 4:8**). Since all but one of the field study herds reported using dry cow therapy it was difficult to compare the two data sets.

4.3.5 Milking Machine Test

The mean cell count (258,000) of the 1469 (69%) herds who had a milking machine test or maintenance contract in May 1993 was very significantly ($P<0.001$) lower than the 645 (31%) herds who did not (289,000) (**Table 4:9** & **Figure 4:3**). This gave a difference between testing and not of 31,000. The difference for the MQFILE study of 25,000 was very similar. **Figure 4:4** illustrates the BTSCC performance of the 1230 SMMB producers who had a milking machine test contract with the 931 producers who did not. There was no indication in the census data as to either the frequency or timing of the machine inspection. The December 1993 group mean rolling BTSCC for those herds holding a contract was 288,000 and was lower than the figure of 313,000 for non-contracted herds. There was a consistent difference of approximately 25,000 in the mean BTSCC performance of both groups (**Figure 4:4**).

Table 4:10 shows that the December 1993 BTSCC of non-recording herds holding an SMMB machine test-contract was 328,000 compared to 346,000 for non-recording herds not

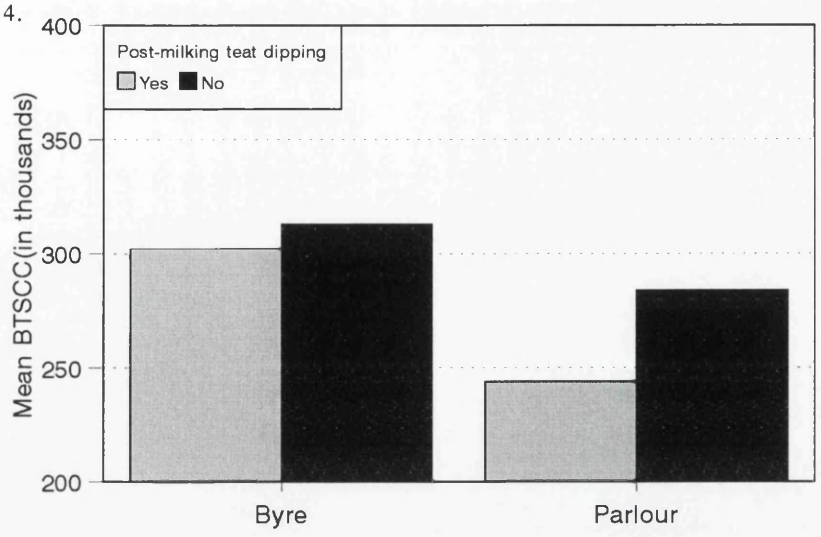


Figure 4:1
Effect of post-milking teat dipping on herd BTSCC
May 1993 census data

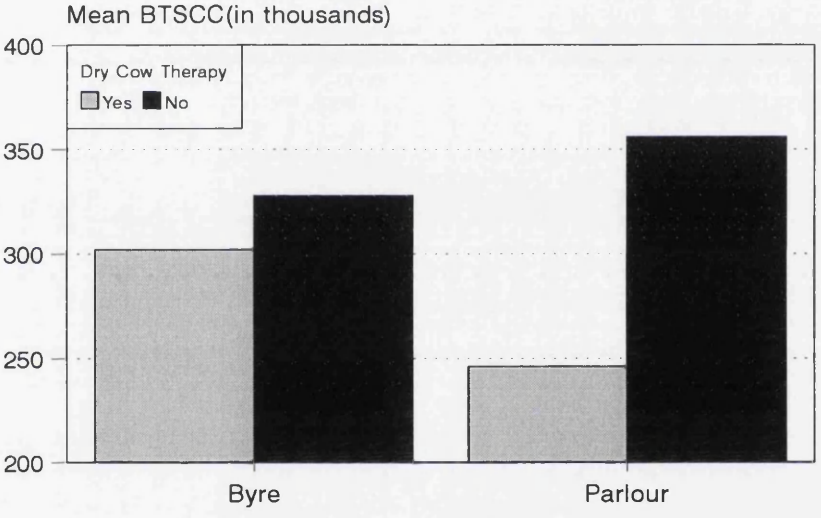


Figure 4:2
Effect of Dry Cow Therapy on herd BTSCC
May 1993 census data

		Post-milking teat dipping			
		Yes	No	s.e.d	F pr
BYRE	log_{10}(BTSCC)	2.4807	2.4950	0.01817	0.433
	BTSCC(000)	302	313		
PARLOUR	log_{10}(BTSCC)	2.3866	2.4537	0.01297	<0.001
	BTSCC(000)	244	284		
ALL HERDS	log_{10}(BTSCC)	2.4067	2.4742	0.01020	<0.001
	BTSCC(000)	255	298		

Table 4:7. Effect of post-milking teat dipping on cell count.

		Dry Cow Therapy			
		Yes	No	s.e.d	F pr
BYRE	log_{10}(BTSCC)	2.4807	2.5155	0.02253	0.123
	BTSCC(000)	302	328		
PARLOUR	log_{10}(BTSCC)	2.3918	2.5517	0.02277	<0.001
	BTSCC(000)	246	356		
ALL HERDS	log_{10}(BTSCC)	2.4151	2.5298	0.01546	<0.001
	BTSCC(000)	260	339		

Table 4:8. Effect of dry cow therapy on cell count.

		Yes	No	s.e.d	F pr
BYRE	log_{10}(BTSCC)	2.4812	2.4961	0.01829	0.416
	BTSCC(000)	303	313		
PARLOUR	log_{10}(BTSCC)	2.3903	2.4329	0.01235	<0.001
	BTSCC(000)	246	271		
ALL HERDS					
Census	log_{10}(BTSCC)	2.4119	2.4602	0.01016	<0.001
	BTSCC(000)	258	289		
SMMB	BTSCC(000)	288	313		

Table 4:9. Effect of milking machine testing on cell count.

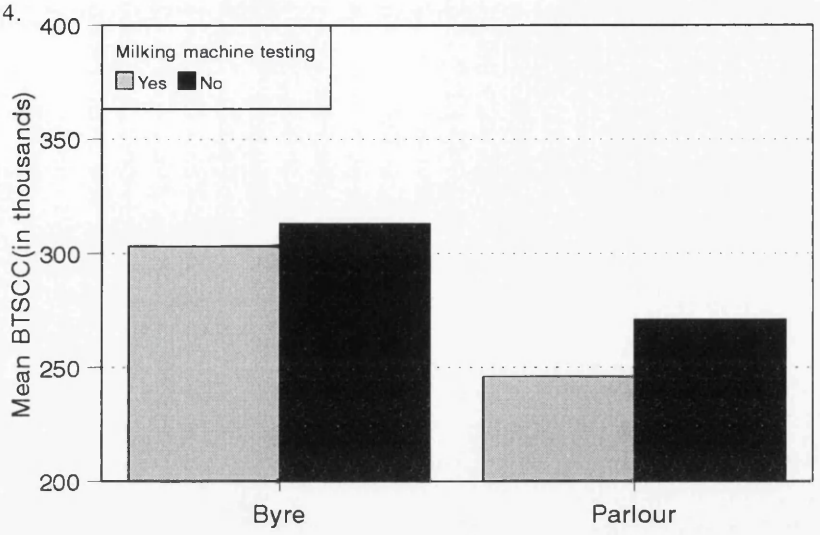


Figure 4:3
Effect of milking machine testing on herd BTSCC
May 1993 census data

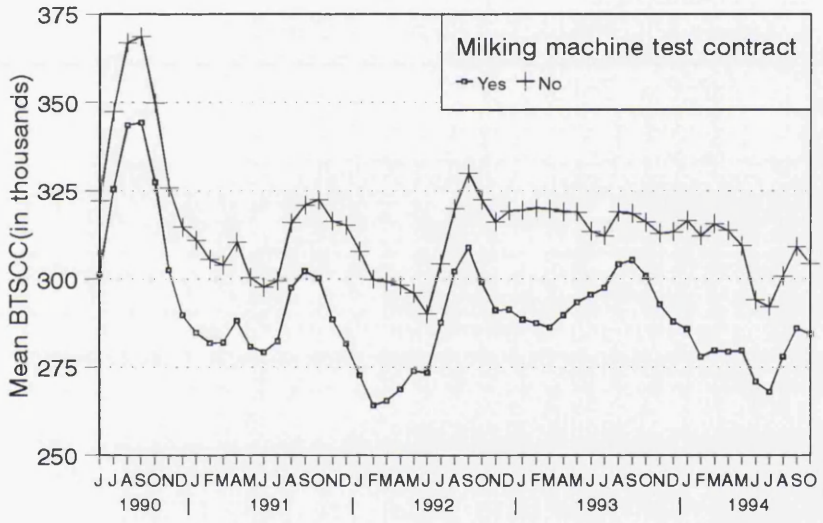


Figure 4:4
BTSCC levels in SMMB herds with Board milking machine test contract
December 1993

		December 1993	
		% in Penalty	Mean BTSCC(000)
Milking machine test	SMRA	10.18	246
	Non-Rec	24.44	328
	Total	17.49	288
No milking machine test	SMRA	12.87	244
	Non-Rec	28.05	346
	Total	23.18	313

Table 4:10. Effect of milking machine testing on December 1993 BTSCC.

holding an SMMB machine test-contract. A smaller proportion of non-recording herds holding an SMMB machine test contract were in penalty in December 1993 (24.44%) compared to non-recording herds without a contract (28.05%). No advantage was observed for SMRA herds in this analysis (see **Table 4:10**).

4.3.6 Effect of milk recording

Table 4:11 shows that the mean cell count of the 890 (42%) SMRA herds (237,000) was very significantly ($P<0.001$) less than that of 1125 (53%) non-recording herds (295,000) and of 99 (5%) herds recording with various private organisations (261,000).

4.3.7 Overall implementation

The producers who used the three types of udder preparation technique recorded in the 1993 census (**Table 4:2**) were further grouped by the application of dry cow therapy, milking machine testing and post-milking teat dipping (**Table 4:12**). A majority (224, 56%) of herds who did not use a wet udder preparation did use all three of these control measures and this increased (293, 74.2%) when the further 69 dry-preparation herds who used any two of these three measures were included.

The proportion of producers from the 1993 census who used various elements of a milk hygiene strategy are presented in **Table 4:13** where the level of producer compliance is compared with the previous census taken in 1990. The proportion of producers using a post-milking teat-dip has increased by 13.7%, while 8.9% more producers used DCT. The number of producers who do not undertake any type of udder preparation has increased by 4.8%, the majority of which are herds who previously used an udder cloth.

4.4 Discussion

The information presented in this chapter was extracted from the latest 3-yearly census of all Scottish milk producing herds, collected in May 1993. The correlation of census information with BTSCC data was performed using only a numerical producer code thereby maintaining the anonymity of the information. The 3-month geometric mean BTSCC for May 1993 was used in the statistical analysis since it corresponded with the period March, April and May 1993 just before the census was taken. This census was part of a UK dairy industry census taken by the various Milk Marketing Boards at the request of government. In addition, Milk Board staff personally collected forms which were not returned by post and were thus available to assist in the completion of the forms where necessary. It is quite possible that given the reorganisation of the UK dairy industry this will be the last such comprehensive survey. Even allowing for the official nature of the census the response rate of producers was remarkable. Thus the availability of reliable information from 97% of all SMMB herds made possible a comprehensive analysis of the

		Milk Recording			
		SMRA	Private	Not	F pr
BYRE	$\log_{10}$(BTSCC)	2.4510	2.5137	2.4983	0.084
	BTSCC(000)	282	326	315	
PARLOUR	$\log_{10}$(BTSCC)	2.3592	2.4001	2.4483	<0.001
	BTSCC(000)	229	251	281	
ALL HERDS	$\log_{10}$(BTSCC)	2.3740	2.4173	2.4691	<0.001
	BTSCC(000)	237	261	295	

Table 4:11. Cell Count of milk recording and non-recording herds.

Dry Cow Therapy	Milking Machine Test	Post-Milking Teat Dip	Udder Preparation		
			Wet		None (Dry)
			Bucket	Pipe	
Yes	Yes	Yes	367	442	224
		No	184	79	61
	No	Yes	179	127	60
		No	122	36	21
No	Yes	Yes	31	11	9
		No	42	12	7
	No	Yes	30	6	3
		No	48	3	10

Table 4:12 Mean BTSCC associated with udder preparation technique and mastitis control programme.

Mastitis Control	Producer implementation(%)		% change 1990-1993
	1990	1993	
Udder Preparation:			
Bucket & cloth	50.0	47	-3.0
Running water	35.8	34	-1.8
None	14.2	19	4.8
Teat Dip	56.3	70	13.7
Dry Cow Therapy	81.1	90	8.9
Milking Machine Test	Not available	69	*

Table 4:13. Implementation of a milk hygiene strategy by SMMB producers.

effectiveness of the recorded milking hygiene techniques. By contrast, analysis of a database created by a 25% response rate to a postal questionnaire is considered acceptable. The previously available national information from MAFF (Anon, 1992) reported there were 31577 agricultural and horticultural holdings in Scotland of which 2649 were mainly in dairying: 2400 in the SMMB area, 150 in the A&DMMB area and 99 in the NOSMMB region (Anon, The three Milk Marketing Boards in Scotland, 1990). The average herd size was 91 cows, most herds (68%) milked in parlours and nationally 61% of their cows were Friesian, 15% Holstein and 13% Ayrshire. Logue et al. (1993) commented that the number of cows milked by one person had risen from 55 in 1978 to 69 in 1990. This was closely related to the one-third reduction in the proportion (46% & 31%) of herds milking in a byre over the same period. The Scottish Milk Recording Association (SMRA) (Anon, 1992) reported an average production of 6086 kg per lactation based on membership by 42.1% of all Scottish producers milking 44.1% of the dairy cows in Scotland.

The statistical analysis of the "EPIDEM" database provided conclusive quantitative evidence of the BTSCC advantage from dry cow therapy and post-milking teat dipping in the control of subclinical mastitis and thus BTSCC in Scottish herds. Erskine et al. (1987) confirmed that both post-milking teat dipping and dry cow therapy were used by only 37.5% of high SCC herds but in 81.3% of low SCC herds. Further verification came from Hueston et al. (1987), who reported that the percentage of low SCC cows was significantly increased by either of these standard mastitis control measures. It is now accepted that an effective teat dip, correctly used, will reduce incidence of new IMI by 50 to 90% (Pankey, 1984). Major "parlour" mastitis pathogens such as *S. aureus* and *S. agalactiae* are controlled largely by post-milking teat dipping (Pankey et al., 1984). Neave et al. (1950) and Natzke (1971) reported that without dry cow therapy 8 to 24% of quarters became infected, especially in the first few weeks, of which half persisted into the next lactation and about half of these persistent infections went on to became clinical.

Milk bacteriological quality is improved by effective udder preparation (Pankey, 1989). This is consistent with the report of lower BTSCC in parlour systems by Bodoh et al. (1976). It is noteworthy that more than one third of the high BTSCC field study herds of Chapter 3 milked in a byre (**Table 3:43 & Appendix II**). Mein et al. (1977) and Hoare et al. (1979) reported that the method of udder preparation was a significant source of BTSCC variation. This is however in contrast to the findings of Pearson et al. (1972) and Moxley et al. (1978) who reported there no significant relationship between the use of individual paper towels and lower BTSCC. More recently the effectiveness of udder preparation techniques have been studied in terms of milk quality, total bacterial counts (TBC) in raw milk, and reduction of udder infection (Edwards & Smith, 1970; McKinnon et al., 1983; Galton et al., 1984; Galton et al., 1986a; Galton et al., 1986b). Such studies show that TBC increases when teat surfaces are wetted and not adequately dried before milking. Galton (1986a&b) stated that manual drying of teats was an essential part of any procedure to achieve effective reduction of bacterial counts of milk. Pre-milking udder

preparation affects the number of bacteria on the teats and in the bulk milk (Galton et al., 1982). Galton et al., (1984) reported preparation procedures that involved wetting both the udder surface and teats resulted in the highest bacterial counts in milk compared with methods that wetted teats only. The "EPIDEM" database unfortunately was ambiguous on the format of udder preparation before milking and in particular it did not explicitly record if not washing was a conscious decision. This was indirectly confirmed by the calculation that 74.2% of these herds did have a committed approach to mastitis control as indexed by the practice of at least two of the three fundamentals i.e. dry cow therapy, milking machine testing and post-milking teat dipping. The value of a future census could be further enhanced by the inclusion of such a direct question. The analysis of census data presented in this chapter was undertaken specifically to convince producers in penalty of the necessity and value of standard milking hygiene techniques as the first step in BTSCC reduction. The findings are however of interest to all Scottish producers, including those below 400,000. Dutch workers already assist low BTSCC herds by the investigation of risk factors associated with the sometimes unacceptably high incidence of clinical mastitis (Schukken et al., 1989). Their study unearthed a paradoxical association of increased BTSCC with post-milking teat dipping which they explained on the basis of uptake of this technique in the face of a problem. Additional census questions about the incidence of clinical mastitis would help define the extent of this problem.

In summary, reliable mastitis control information expressed in terms of BTSCC advantage was not previously available for Scotland. The advantage for milking machine testing was very similar when calculated independently from the producers own census reply and from the SMMB contract records. The slight difference could be accounted for by those herds who also benefited from a milking machine test but undertaken by another organisation.

CHAPTER 5. GENERAL DISCUSSION.

The 1992 EC Milk Hygiene Directive (92/46) governing the production of liquid and manufactured milk products imposed a maximum for Somatic Cell Count (SCC) of 400,000. The objective of the work described in this thesis was to investigate the relationship between SCC and mastitis. This study was conducted in accordance with the principles of veterinary epidemiology described by Thrusfield (1986). These were firstly the determination of the extent of the problem, secondly identification of the causes, thirdly a description of their ecology and fourthly an assessment of appropriate control measures.

At the outset of this study the national extent of the problem of high SCC in Scotland was poorly quantified because the relevant information was inaccessible. Little more was known in January 1991 than the fact that the Bulk Tank SCC (BTSCC) of over a fifth of all Scottish producers failed to meet the 400,000 EC standard. Prior to this, for instance, the SMMB only maintained BTSCC data for all herds for a rolling 12-month period, discarding earlier information, and even this national information was not readily accessible from their mainframe computer.

The national extent of high SCC in Scotland was scientifically investigated for the first time by the development of a new personal computer database ("MQFILE") (**Table 3:6**). This made available milk quality (BTSCC and TBC) information from all Scottish dairy herds, identified only by an anonymous producer code, for analysis as a national dataset (**Table 3:17**). Information from the largest Milk Marketing Board, Scottish (SMMB), which comprised 90% of all producers, was used for this analysis unless otherwise stated.

The relative contribution of all herds to the overall "Board" SCC was examined to establish which section of SMMB producers was primarily responsible for elevating the Board SCC. This concept of a "Contribution Index" was developed by Schukken et al. (1992b). When calculated for SMMB herds (**Table 3:18**) it showed that it was those herds with high BTSCC (over 400,000) which made a considerable contribution to the Board SCC because their yield was at least the national average. This was in marked contrast to the original report by Schukken et al. (1992b) which found that high BTSCC herds did not contribute significantly to the overall Ontario Board SCC because they had below average yield. The concept of relative contribution was developed within the current study by demonstration that the herds which made a major contribution to the extent of the high Board SCC problem in Scotland were those consistently over 400,000 (**Figure 3:19**). The Ontario Board applied Schukken's findings by providing premiums to low BTSCC herds in order to produce a further reduction of the Board SCC. This strategy nonetheless effectively relied on the dilution of milk from high BTSCC herds. However, in contrast, the SMMB contribution data clearly indicated the need to apply penalties to herds with BTSCC consistently over the 400,000 EC threshold.

At the individual herd level the extent of an SCC problem can be determined by the use of

Individual Cow SCC (ICSCC) data. However ICSCC data only became available to producers in Scotland in 1989. At first its availability was strictly limited to members of the Scottish Milk Records Association (SMRA) on the same milk samples taken for fat and protein estimation at milk recording. Careful interpretation of ICSCC data can assist the identification of infected cows. However Brolund (1985) reported that factors other than infection, such as stage of lactation and lactation number, also contributed to ICSCC variation. Their influence was reflected in the fact that despite infection being the most important cause of such variation, the correlation of $\log_{10}$(ICSCC) with subclinical mastitis was only 0.6 for a single sample. Unfortunately the ICSCC service was offered to SMRA producers before a coordinated advisory support system was in place and this presented two problems in particular. Firstly these ICSCC results were distributed to the producer in isolation of BTSCC data. Secondly, any previous ICSCC herd-test results were inaccessible since only a single hard-copy was ever created. Against this background of lack of advice on the application of SCC data the three Milk Marketing Boards (MMB) in Scotland (Scottish, Aberdeen & District and North of Scotland) commissioned the research on SCC in Scottish dairy herds reported in this study. In addition they introduced differential SCC payments in April 1991 to encourage producers to meet the 400,000 EC standard.

The first analysis of the SMRA ICSCC service, reported in Chapter 3, was undertaken to determine the extent of its uptake. This was found to be only limited. The data for December 1993 indicated that only 17% of all SMMB producers used the service (**Figure 3:14**). Further analysis indicated a complete dichotomy of the monthly use of the ICSCC data (**Figure 3:15**). One group of users (12%) ICSCC-tested every month and maintained their BTSCC consistently below 250,000. They clearly demonstrated effective use of the ICSCC data. In contrast another quite distinct minority (12%) of users who had a BTSCC problem believed that identification of individual high SCC cows by a single herd-test was a complete investigation. The failure of this latter group to use their ICSCC data properly was one indication of the need to investigate how best to apply ICSCC data in controlling subclinical mastitis and then to educate producers accordingly. This need was further supported by an apparently contradictory finding. The majority (37, 65%) of 57 herds with BTSCC over the 400,000 EC threshold in December 1993 who had ICSCC-tested in that month had done such testing on a regular basis (**Figure 3:16**). Thus it would appear that despite regular testing their ICSCC data was not used as part of an effective mastitis control programme. Had such producers been aware that reduced milk yield rather than direct payment penalties represented up to 80% of the total cost of mastitis (Janzen, 1970; Dobbins, 1977; Blosser, 1979) they might have acted more effectively. Presentation of ICSCC data as a "Linear Score" (Ali & Shook, 1980) and thus directly related to yield reduction would have disclosed the production and therefore true financial loss caused by high SCC. However Scottish producers preferred to see the raw ICSCC figures and it proved very difficult to convince them that the relationship with yield reduction was actually logarithmic.

This part of the study confirmed that high BTSCC was a widespread problem throughout Scotland and revealed that attention should be focused on those herds continuously over the 400,000 EC threshold. It also identified a need to educate producers in the use of ICSCC data.

The causes of high SCC were identified by review of the literature, analysis of mastitis investigation records and field investigation of a representative group of problem herds. Brolund (1985) was one of many authors (**Chapter 1**) to report that infection by the recognised mastitis pathogens was the main cause of high SCC. However at the outset of this study there were only two recent large databases of mastitis bacteriology available in the literature to access the relative prevalence of these pathogens in the UK. These were the Veterinary Investigation Diagnosis Analysis (VIDA) annual reports and the earlier MAFF-sponsored study by Wilson & Richards (1980). The VIDA database included a record of every submission to, and diagnosis by, the laboratories of both the Veterinary Investigation Service of England & Wales and SAC Veterinary Services (see the section on diseases of the reproductive and mammary system). However although VIDA provided an indication of the relative prevalence of pathogens, the majority of submissions were single specimens (**Table 1:1**) and thus presumably only from clinical cases. According to Dodd & Neave (1970) only 40% of all subclinical infections ever became clinically apparent. Thus the fraction of mastitis incidents which eventually appeared in VIDA introduced a bias particular to its mastitis data. This was considered in the interpretation of both figures for individual pathogens and apparent trends of their ecological groupings. Nevertheless the 1990 VIDA report for Scotland, available at the outset of this project, indicated that *S. aureus* (16.2%) was a more frequent finding than other isolates (*S. agalactiae*, 10.7% or *S. dysgalactiae*, 10.7%) which could also occur as subclinical infection (**Table 1:1**). This trend was in agreement with the last comprehensive survey of subclinical mastitis in the UK (Wilson & Richards, 1980) which also reported that *S. aureus* was the most prevalent pathogen and affected 8.1% of all quarters.

Chapter 2 examined a previously unanalysed body of mastitis investigation records from SAC Aberdeen and thus created two new databases of mastitis bacteriology (**Table 2:4**). They were unique for the UK since they recorded both bacteriology and SCC data from a large number of milk samples, collected from either individual quarters (**Table 2:5**) or as composite udder samples (**Table 2:10**). The analysis of this data was undertaken firstly as a background to investigation of herds with a milk quality problem and secondly to assess the use of ICSCC as a screening tool in bacteriological investigation of herds.

The Aberdeen data analysis confirmed that infection by the recognised mastitis pathogens was the most important cause of SCC elevation. Two aspects within this part of the analysis were of particular note. Firstly the SCC increase caused by infection remained significant ($P<0.001$) despite the physiological increase reported at both the start and end of lactation (**Tables 2:7 & 2:11**). Secondly Logistic Regression analysis clarified several reports of an age-related increase in ICSCC (Brooks et al., 1982; Poutrel & Rainard, 1982). It showed that the significant ($P<0.05$)

positive relationship was actually between the prevalence of infection and age (**Section 2.3.2** & **Figure 2:6**). Thus the increased prevalence of high ICSCC was a direct consequence of infection. The veterinary interpretation of this statistical finding was that it reflected the repeated mastitis challenge experienced by cows within a herd with a high level of subclinical infection.

The extent of herd infection was estimated from BTSCC data with reasonable accuracy. A significant mastitis pathogen was isolated from 35% of the cows which were quarter sampled in the Aberdeen study (**Table 2:6**). Although a bulk tank sample was not examined at the time of the original Aberdeen investigation the "BTSCC" was estimated by taking the median ICSCC of each herd. This showed the cow infection prevalence for the "BTSCC" range less than 500,000 was 26%, 500,000 to 1,000,000 was 33% and greater than 1,000,000 was 65%. This was in close agreement with Pearson & Greer (1974) who reported average cow infection prevalences of 25.8%, 42% and 54.4% respectively for the same BTSCC ranges. Thus the Aberdeen herds were in fact typical of those with a subclinical mastitis problem despite the fact that the herd investigations were in response to high TBC. In other words BTSCC was high as well as high TBC. This was consistent with the report by David & Jackson (1984) that mastitic milk could contain 100,000,000 bacteria/ml. Two litres of such milk could raise the TBC of 2000 litres of bulk milk by 100,000.

S. aureus was found to be the most common major pathogen (**Figure 2:1**) in agreement with the reports by Wilson & Richards (1980) and VIDA records (Anon, 1994). It was isolated from 8.68% of all the Aberdeen quarter samples (**Table 2:6**). The Aberdeen data revealed that it was frequently possible to isolate *S. aureus* from cows when their ICSCC was low since 31.06% of all such isolates were from composite samples with SCC of less than 400,000 (**Figure 2:8**). This was consistent with the cyclic pattern of *S. aureus* shedding and inverse SCC variation reported by Daley et al. (1991). A particular concern this indicated was the impossibility of identifying all *S. aureus* carriers using only ICSCC data. Furthermore the Aberdeen quarter data also showed that a substantial proportion (40.6%) of infected cows were infected in 2 or more quarters (**Table 2:9**) which was in agreement with that (61%) reported by Meek et al. (1980). In particular multiple quarter infections by *S. aureus* were common and comprised 54% of all such isolations (**Table 2:9**). Natzke (1982) reported that 45 to 55% of all new quarter infections were actually the result of cross-infection within the udder and several reports suggested that this had occurred by mechanical cross-contamination at milking time (Bodoh et al., 1981; Buddle et al., 1987). However neither of these hypotheses could be tested using the single samples recorded in the Aberdeen quarter database. The DNA fingerprinting techniques now under development (Platt et al., 1994) could identify the *S. aureus* genotype and thus determine whether subsequent quarter isolates were actually identical.

The Aberdeen cow database provided an opportunity to investigate the use of an ICSCC threshold as a technique to make the bacteriological investigation of a herd subclinical mastitis problem more cost-efficient. Dohoo & Leslie (1991) reported that a low ICSCC threshold

(200,000) was required to correctly identify 85% of all infections by major pathogens. Although their report did not specifically investigate which major pathogen required such a low threshold, the evidence from the Aberdeen cow database would clearly implicate *S. aureus*. However a low ICSCC threshold had the benefit of minimising false negative diagnoses. This was an important facet of any mastitis screening test (Bodoh et al., 1981; Barnum, 1990). Two ICSCC values from the recognised Linear Score (LS) scale were evaluated in the analysis of Aberdeen cow data. The margins of LS5 (283,000 and 566,000) encompassed the 400,000 EC BTSCC limit yet reflected the lower thresholds advised by several authors (Griffin et al., 1977; Dohoo & Leslie, 1991). The analysis (**Table 2:15**) showed that these thresholds increased the efficiency of bacteriological examination and still produced a representative profile of the infection in the herd under investigation. A significant isolate was found in 47% of composite samples with SCC over 283,000 compared to 27.4% of all samples. This threshold therefore increased the efficiency of bacteriological examination by a factor of 72%. This Aberdeen cow data (**Table 2:15**) indicated that a single ICSCC test above 283,000 had 79% sensitivity and 66% specificity for the detection of subclinical infection. This compared with the lower (62%) sensitivity and higher (83%) specificity achieved by the use of a LS6 (over 566,000) threshold. Only 20.5% of infected samples were diagnosed false negative using a 283,000 ICSCC threshold compared to 38% with a 566,000 threshold. The positive predictive value of an ICSCC over 283,000 for infection was 47% but 58% for ICSCC over 566,000. Both these estimates for predictive value were obtained from the Aberdeen cow database and thus in a population with the same prevalence of infection.

Further validation of a 283,000 ICSCC threshold was provided by the veterinary interpretation of an apparently contradictory finding. Statistical analysis found a very significant (Chi-square=41.4, 6DF, $P<0.001$) difference in the bacteriological profiles from those composite samples with SCC less or greater than 283,000 (**Table 2:15**) because there were fewer *S. agalactiae* isolates in low SCC samples. Thus although the LS5+ sampling threshold was biased against *S. agalactiae* the presence of the pathogen within the herd was disclosed without disadvantaging the estimation of *S. aureus* prevalence. This is consistent with the report by Wilson & Richards (1980) in which *S. agalactiae* demonstrated the strongest relationship between QSCC and the presence of a major pathogen.

A high prevalence of infection by *S. agalactiae* was found to be the most common cause of high BTSCC in a group of representative SMMB herds (**Figure 3:20 & Table 3:34**). The new MQFILE database of milk quality information from all SMMB producers allowed the selection of herds for investigation which where accurately representative of all those with such a milk quality problem. Financial constraint limited this in-depth investigation to 25 herds each with a consistently high BTSCC. While ideally one would have examined all cows, the herd investigation protocol selected cows for bacteriology with ICSCC of LS5+ (in excess of 283,000), calculated as a lactation mean. As described in Chapter 3 the new "CCGM" software stored and analysed

successive ICSCC results (**Table 3:22**). Thus for the first time in Scotland a mechanism was available to collate the SMRA ICSCC data from successive herd tests and present the data as an action list sorted in order of mean ICSCC (**Table 3:24**). Similar schemes have since been adopted by DAISY and National Milk Records (NMR). These were the first herd investigations in Scotland to use LS5 as a threshold to increase the efficiency of bacteriological examination. Indeed the isolation of a significant pathogen from 71% of the cows compared very favourably with the small proportion (35%) of quarter-sampled Aberdeen cows which were actually infected. The inefficiency of whole-herd bacteriological examination was even more strikingly illustrated by the larger Aberdeen composite-sampled database where, as previously mentioned, only 27.4% of all bacteriologically examinations revealed an infection.

The cause of most of these infections was in fact *S. agalactiae*. Its isolation in 19 (83%) of the 23 herds which cooperated in Phase-1a of the study (**Figure 3:3**) demonstrated that *S. agalactiae* rather than *S. aureus,* was the major problem in high SCC herds. In contrast Wilson & Richards (1980) reported that only 38% of herds were infected by *S. agalactiae*. Similarly Pearson et al. (1972) isolated *S. agalactiae* from 48% of high (annual mean in excess of 1,000,000) BTSCC herds and not at all in low (less than 300,000) BTSCC herds. Fenlon et al. (1995) provided contemporary evidence from another Board (Aberdeen & District) region within Scotland confirming *S. agalactiae* was indeed the most prevalent pathogen in herds with a subclinical mastitis problem. It was the predominant pathogen in 13 (42%) of 31 bulk milk samples with total mastitis bacteria exceeding 10,000 cfu/ml in which the mastitis pathogens could be identified. Furthermore their quantitative assessment of TBC found that the correlation between BTSCC and streptococcal count (r^2 = 0.827) was higher than for *S. aureus* (r^2=0.686). This clearly indicated that subclinical streptococcal mastitis could affect TBC as well as BTSCC, the two statutory measures of milk quality adopted by EC Directive 92/46, simultaneously. Marshall (1991) also considered that the excretion of mastitis bacteria from a herd with subclinical mastitis was actually a more important source of bulk milk bacterial contamination than either the teat surface or an inadequately cleaned milking machine. He therefore agreed with Jeffrey & Wilson (1987) that mastitis was the main problem in over 40% of bulk tank milk samples with high (over 45,000) TBC. Thus a subclinical mastitis problem could be manifest as either raised BTSCC, TBC or both.

Although prevalence of infection was the most important cause of high BTSCC in the individual herd, the "Board SCC" was also affected, though to a lesser degree, by season and payment penalties. The Ontario Board SCC (Schukken et al., 1990) showed a seasonal peak in October whereas the maximum Board SCC occurred from August to October in Scotland (**Figure 3:4**). The Scottish observation was considered a reflection of the known national calving-pattern. Thus the volume of late and early lactation milk with physiologically-elevated SCC (Blackburn, 1966) was at an annual maximum in this period. This interpretation was corroborated by

examination of the number of producers each month with BTSCC of 100-199,000 (**Figure 3:11**) who had therefore achieved excellent control of subclinical mastitis (Pearson & Greer, 1974). Many such producers were unable to remain within this band around the month of September. This was considered due to the tight calving pattern typical of many such well-managed herds. With this in mind, further examination of the SMMB data revealed that a target annual mean BTSCC less than 207,000 (**Figure 3:17**) was actually required to completely avoid high BTSCC. Thus annual SCC performance at this level would accommodate physiological increase within the 400,000 EC threshold. Likewise it would provide a level so that when significant subclinical mastitis was identified there was time for its control before the 400,000 EC threshold was exceeded. This target was considerably less than the 250,000 SCC limit described as "premium" within the SCC payment structure. Excluding these seasonal influences, a modest but distinct downward trend in the Board SCC was apparent for the latter part of 1991 (**Figure 3:4**). This was believed to be the effect of advance publicity about financial penalties for BTSCC over the 400,000 EC threshold which were actually introduced in January 1992. Schukken et al. (1992a&b) also reported the success of differential payments based on BTSCC. They attributed a decrease of 58,000 in the Ontario Board SCC to the first year of their SCC Control Program.

In summary, infection by *S. agalactiae* was the most important cause of high BTSCC at the individual herd level and thus, by extrapolation, at the Board level also. The use of a LS5 (over 283,000) ICSCC threshold increased the efficiency of a bacteriological herd investigation by a factor of 72% without significantly disadvantaging the detection of cows subclinically infected by *S. aureus*.

The ecology of the mastitis pathogens must be considered in the design of a mastitis control strategy. Thus as David & Jackson (1984) agreed, identification of the subclinical pathogens is an essential step in a herd mastitis investigation. The known ecological preferences of these pathogens thus helped determine the predisposing herd factors and thus the aspects of control to be concentrated upon. In this respect it has been customary to classify mastitis pathogens by their origin such that the contagious "parlour" bacteria (*S. agalactiae*, *S aureus* and *S. dysgalactiae*) primarily exist in or on the mammary gland. Therefore they are most readily spread from infected to uninfected quarters at milking time. In contrast the "environmental" pathogens *E. coli* and *S. uberis* have, by definition, a more ubiquitous distribution. This explained the difficulty noted by Schukken et al. (1989) which low BTSCC herds experienced in reducing their incidence of clinical environmental mastitis.

The fourth aspect of this study was the assessment, by SCC data, of appropriate mastitis control measures. Dodd & Neave (1970) reported the success of a mastitis control strategy which could reduce the prevalence of subclinical infection by about 70% within a year. Although implemented as a "five-point" practical plan, the strategy was based on two principles. These were firstly milking hygiene especially post-milking teat dipping and secondly antibiotic treatment

especially at the start of the dry period. However their work was conducted before electronic automation made the measurement of SCC in large numbers of samples economic (Tolle et al., 1971). Dodd & Neave (1970) suggested that the physiological elevation in SCC after calving and towards the end of lactation would limit the application of such an indirect test for subclinical mastitis. Furthermore they were unhappy that, by their own calculations, even a very high BTSCC (over 1,000,000) could only put the quarter infection prevalence somewhere within the range 10 to 48% of quarters. The subsequent report by Pearson & Greer (1974) confirmed that although a single BTSCC over 1,000,000, 800 to 500,000 and between 500 and 200,000 was associated with an overlapping range of quarter infection prevalences (21 to 44%, 8.5 to 26.3% and 4 to 14.6% respectively) a definite trend of reduced subclinical infection existed. The EC Directive 92/46 has adopted a logarithmic method of BTSCC calculation and used data averaged over three successive months to maximise the relationship with herd infection prevalence. This is consistent with the report by Brolund (1985) of a higher correlation between infection and ICSCC when the latter is calculated as a logarithm and on the basis of all the available ICSCC data in the lactation.

The new MQFILE and CCGM databases developed within this study provided the mechanisms necessary to analyse Scottish BTSCC and ICSCC information in accordance with internationally accepted techniques. Furthermore although historical data on both these SCC parameters had previously been routinely discarded, this project retained such information and used it in two main ways. It was first incorporated as part of the new mastitis control strategy (**Table 3:27**) offered to the project herds and then used to monitor their progress (**Figure 3:27**). This new strategy successfully reduced the problem in most of the project herds and was economically worthwhile. This was demonstrated by the very significant ($P<0.001$) fall in the group average BTSCC of 23 "project" herds compared to 280 of their peers (**Figure 3:27**). Although described as "control" herds this latter group of herds did have the opportunity to avail themselves of assistance from their own veterinarian and other advisers within the dairy industry during the course of this study. However the coordinated advisory input to the project herds enabled them to make and consolidate rapid progress. Furthermore their BTSCC reduction was made in relation to a more recent overall rise in BTSCC figures for the SMMB as a whole (**Figure 3:4**). The economic benefit which accrued from this SCC reduction was calculated from information presented in this thesis and additional data on milk sales. Logue et al. (1993) reported that the difference in milk quality payments between the "assisted" herds and their contemporaries amounted to over £3/cow (in the herd) per year. They estimated however that these herds actually gained in the order of £33/cow when figures calculated by Beck & Dodd (1988) for the increased efficiency of milk production as a result of less mastitis were also considered. This was subsequently confirmed by a case study (Treacey, 1994) in a Scottish herd identified by this project.

The reasons for lack of progress in mastitis control of some herds were assessed in two

distinct groups of herds. Firstly, 6 project herds (Phase-1c, **Figure 3:3**) which still had BTSCC over 400,000 approximately 1 year after their first herd investigation were re-examined. This showed that the bacteriological profile had changed with *S. aureus* becoming the most prevalent (52%) mastitis pathogen (**Figures 3:23 & 3:24**). However infection by *S. agalactiae* (22%) was only reduced rather than eliminated despite dry cow treatment. This was because physical transfer of bacteria at milking time continued to propagate infection within these herds. This resulted in a failure to control fully the *S. agalactiae* infection and a relative increase in *S. aureus* which was less responsive to antibiotic. *S. aureus* presents particular problems because this organism is very difficult to treat effectively either during lactation or in the dry period (Logue et al., 1993). Secondly, a group of 4 herds (Phase-3, **Figure 3:3**) which moved below 400,000 when premiums were introduced in April 1991 but then exceeded this figure after January 1992 allowed a small scale investigation of the factors, if any, which caused their short-term but unconsolidated improvement. *S. agalactiae* was identified as the most prevalent mastitis pathogen at the herd investigation and as such this was consistent with other high BTSCC herds. The proportion of isolates (**Figure 3:26**) were not significantly different from the initial project group (Phase-1a) and there was no evidence that either their mastitis control or herd management practices were different. It would appear that initially these herds merely culled "problem" cows (**Appendix IIc**) and thus succeeded in temporarily lowering their BTSCC. However they failed to alter their inadequate mastitis control measures and thus maintain low BTSCC.

Education of the producer in the ecology of these subclinical mastitis pathogens was found to be an essential component in ensuring diligent long-term application of the standard mastitis control recommendations. This was because many producers simply did not appreciate that their inadequate application of the five point plan contributed to both the origin and persistence of their high BTSCC problem. This educational requirement was fulfilled in two novel ways. Firstly a new series of advisory leaflets (**Appendix IV**) funded by the EC was designed to fulfil this educational requirement. Their illustration of the use of ICSCC data provided mastitis control advice that was contemporary (**Table 3:27**), based on the research reported in this current study, and as such they did not rely on mere reiteration of standard advice (Dodd & Neave, 1970). These leaflets were subsequently distributed to all 2400 producers throughout Scotland. In addition, a series of meetings were held throughout Scotland specifically for producers in excess of the 400,000 EC threshold. A second unique approach to education in quality milk production was developed in this study. This centred on the provision to Scottish producers of Scottish evidence which was derived from two sources. One was the small group of project herds (Phase-1a, **Figure 3:3**). In particular 10 (43%) of these herds did not routinely teat dip post-milking. The very fact that these herds had a subclinical mastitis problem was because they did not adopt such critically important control measures (Pearson et al., 1979; Pankey, 1984). However the use of paper towels in the udder preparation routine of 16 of the project herds (**Table 3:44**) was the only mastitis

control factor associated with a significantly ($P<0.05$) lower BTSCC. This was in contrast to previous reports (Pearson et al., 1972; Moxley et al., 1978; Pearson et al., 1979) that paper towels offered no significant BTSCC advantage. The second much larger source of mastitis control information was derived from question 8 of the May 1993 census of all Scottish dairy herds (**Appendix III & Table 4:2**) and the corresponding BTSCC data from the MQFILE system. This showed that a dry udder preparation technique was associated with significantly ($P<0.001$) lower BTSCC and thus corroborated the earlier evidence on paper towels from the small group of project herds. Previous reports by Galton (1986a&b) and Pankey (1989) only reported an association with the TBC of milk. The veterinary interpretation of this statistical BTSCC advantage advocated a dry wipe with single-service paper towels only where clean cows were presented for milking. Otherwise such paper towels should be used to dry the teats after they have been washed which Galton et al. (1986a&b) reported was an essential to minimise TBC.

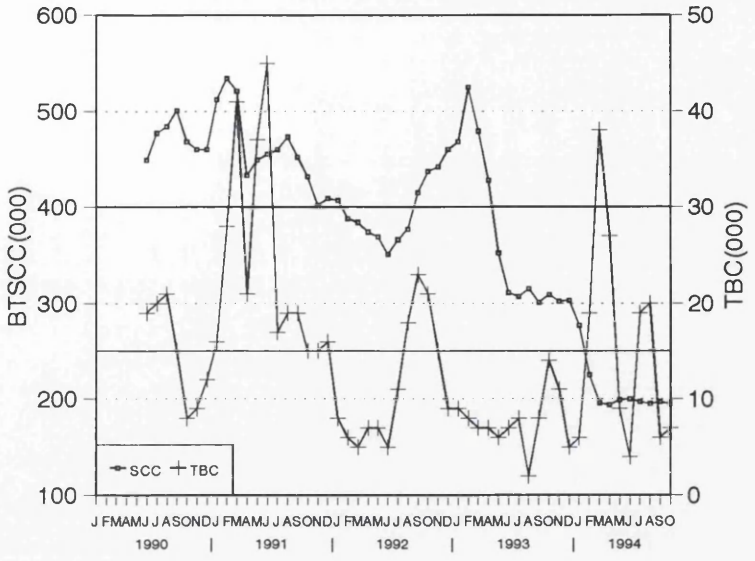
In summary, infection was found to be the most important cause of high SCC at both the quarter and individual cow level. Consequently the prevalence of this infection caused high SCC at both the individual (BTSCC) and national (Board SCC) herd levels. *Streptococcus agalactiae* was the most common cause of subclinical mastitis. This was at once surprising and disappointing since it is possible to eradicate this organism from dairy herds by antibiotic treatment and the application of standard mastitis control measures (Bramley & Dodd, 1984). The advice provided by this study, although in large part based on traditional control techniques, was able to produce a rapid consolidated BTSCC reduction rather than merely provide a general recommendation to apply the five fundamental points of practical mastitis control. The success of this advisory strategy was based on the effective integration of ICSCC data both in the production of herd-specific recommendations and routine management thereafter. The project demonstrated that mastitis control required attention to multiple factors in agreement with Hueston et al. (1987) and that a single instant panacea, such as culling, did not exist. It also countered the notion that high BTSCC was inevitable in some herds, especially those milked in a byre. However the ecological grouping of the mastitis pathogens highlighted the most important predisposing factors which required attention in a herd-specific mastitis control strategy. Finally, at the Board level the study has shown that the emphasis of mastitis control in Scotland must remain targeted on reducing the number of herds with persistently high BTSCC. This will require more severe penalties to encourage producers who have thus far ignored demands for high hygienic quality milk to seek and heed appropriate advice.

The major innovative conclusions of this project were:

1. Infection by *S. agalactiae* was demonstrated to be very extensive. It was the cause of high SCC in 83% of a representative sample of Scottish dairy herds which was a much higher prevalence than anticipated from the previous report of 38% of herds (Wilson & Richards, 1980). This clearly indicated inadequate application of standard mastitis control techniques which would eliminate *S. agalactiae* from infected herds.
2. The mechanism for a nationally coordinated mastitis control strategy for Scotland was developed. Central to this was new computer software (MQFILE) which made available the milk quality data (BTSCC & TBC) of all Scottish producers. This allowed the epidemiological potential of such national data to be fully exploited for the first time. An important innovation was the presentation of this data as a graph of the individual herd.
3. Although ICSCC data required careful interpretation it was a valuable mastitis control tool. In particular a threshold of LS5+ (over 283,000) reduced the cost of bacteriological herd investigations without compromising the detection of cows subclinically infected by *S. aureus*.
4. The prevalence of infection increased significantly ($P < 0.05$) with lactation number. There were two aspects of the veterinary interpretation of this statistical finding. Firstly, it reflected the repeated mastitis challenge experienced by cows within a herd with a high level of subclinical infection. Secondly, the reported increase in prevalence of high ICSCC with lactation number should be attributed to infection rather than age *per se*.
5. The use of paper towels in the udder preparation routine was associated with a significantly lower BTSCC in the 23 "project" herds which were the subject of field investigation. Subsequent analysis of a much larger database of census information from all Scottish dairy herds confirmed the statistical BTSCC advantage of a dry udder preparation technique. The veterinary interpretation of these findings advocated a dry wipe with single-service paper towels only where clean cows were presented for milking. Otherwise such paper towels should be used to dry the teats after they have been washed.
6. A substantial minority of Scottish producers required further encouragement to resolve their persistent high BTSCC problem. The desired effect could be achieved by the complementary strategies of higher payment penalties and mastitis control education. The census data was used to convince such producers of the significant ($P < 0.001$) BTSCC reduction achieved by standard mastitis control and management measures.

APPENDIX I

Individual herd BTSCC profile produced by MQFILE.



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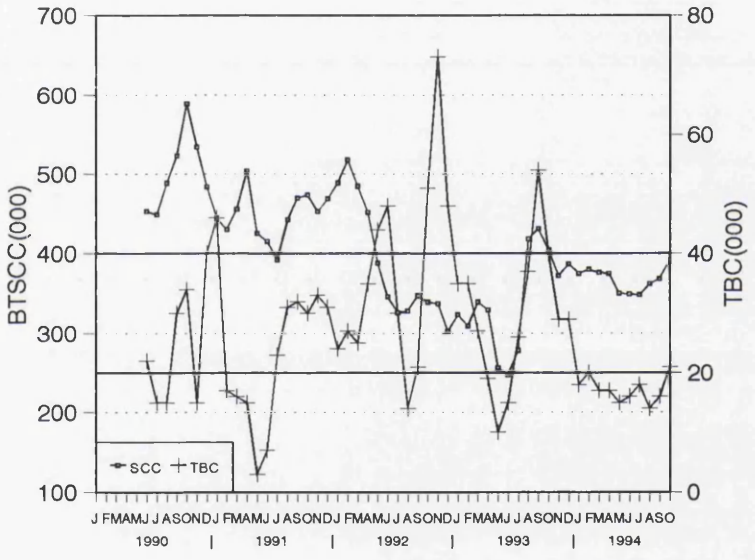
Phase-1a



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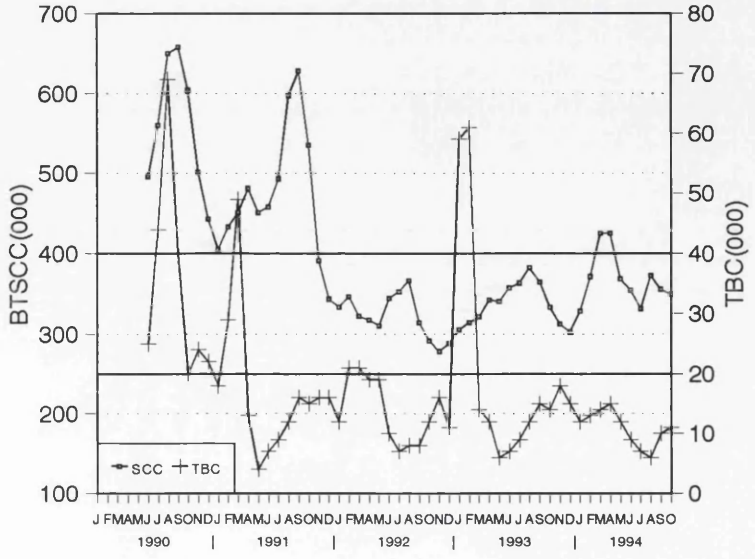

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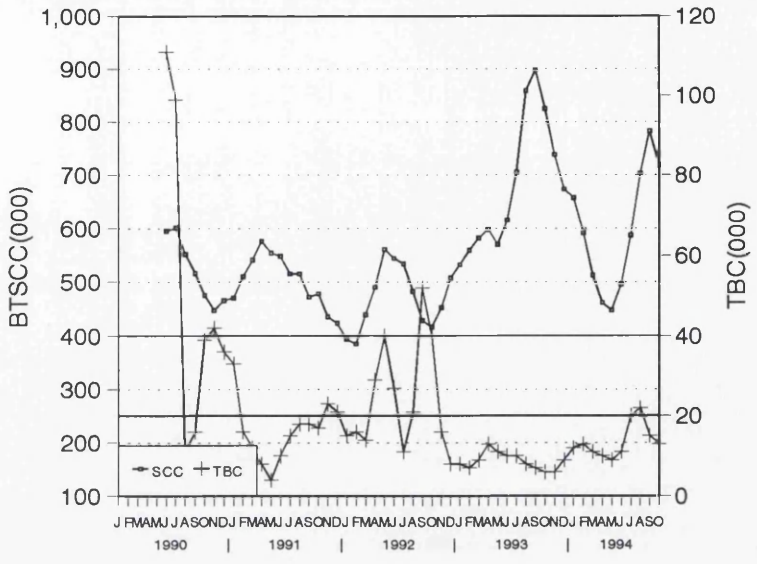

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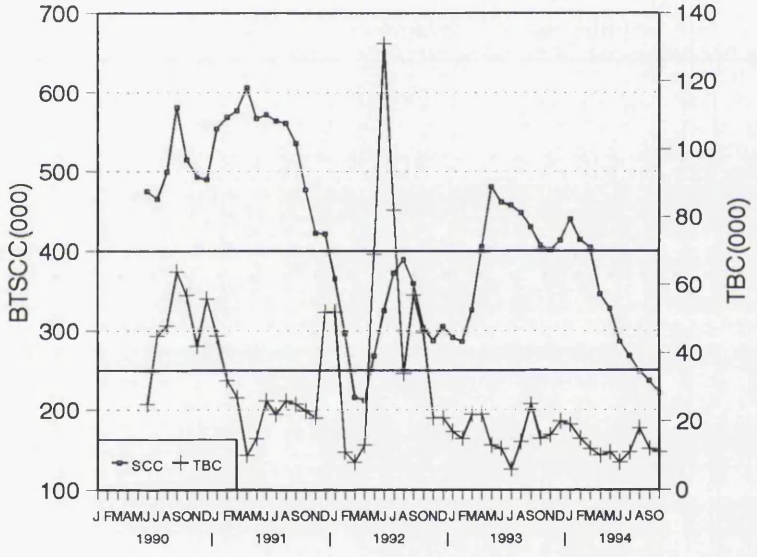

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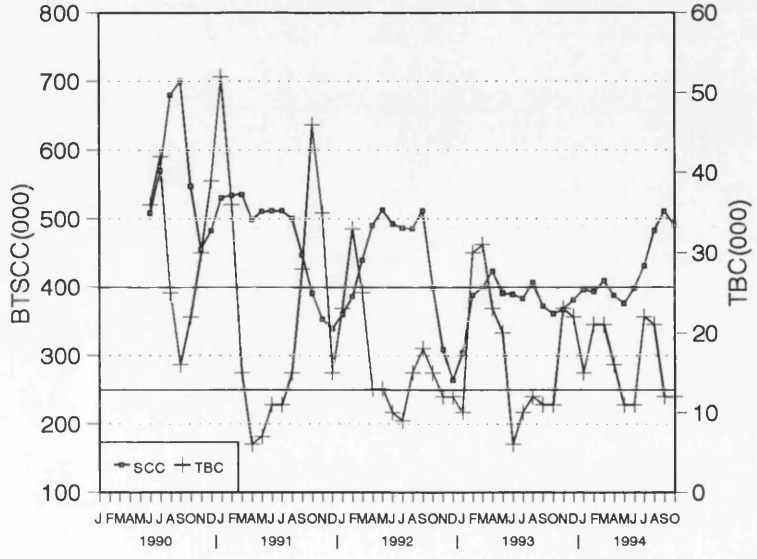

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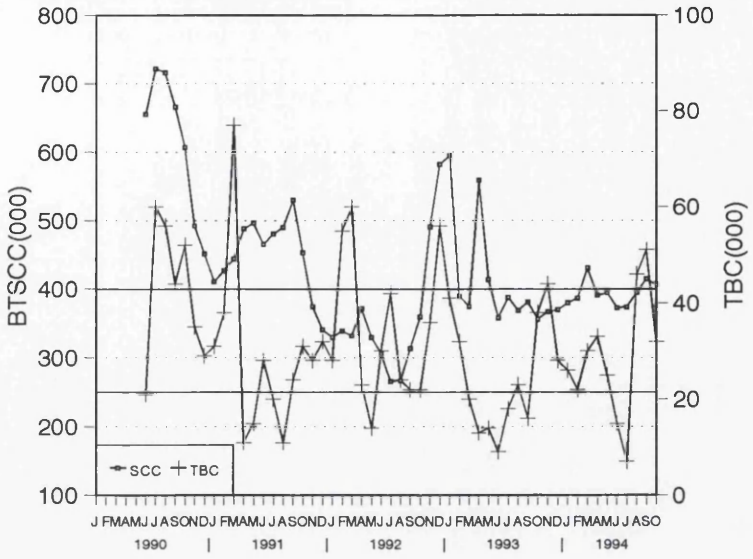

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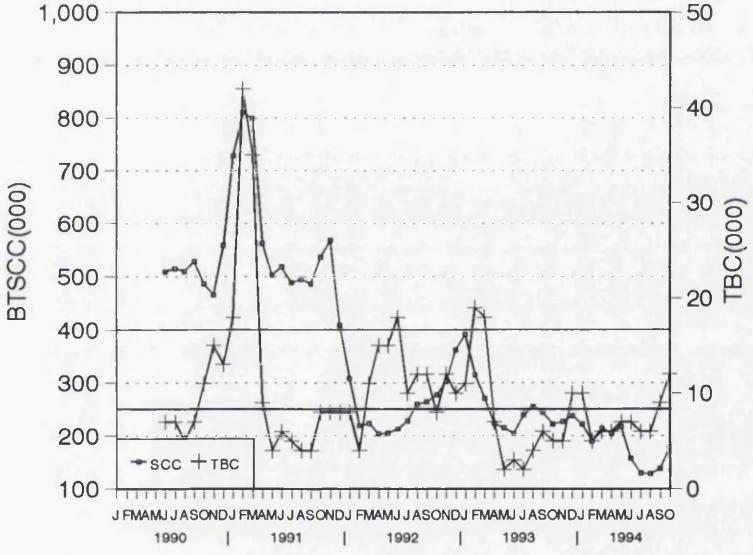

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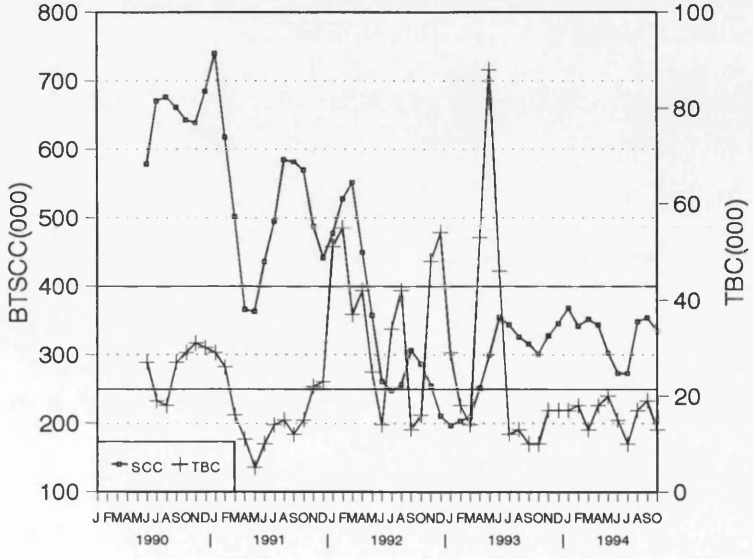

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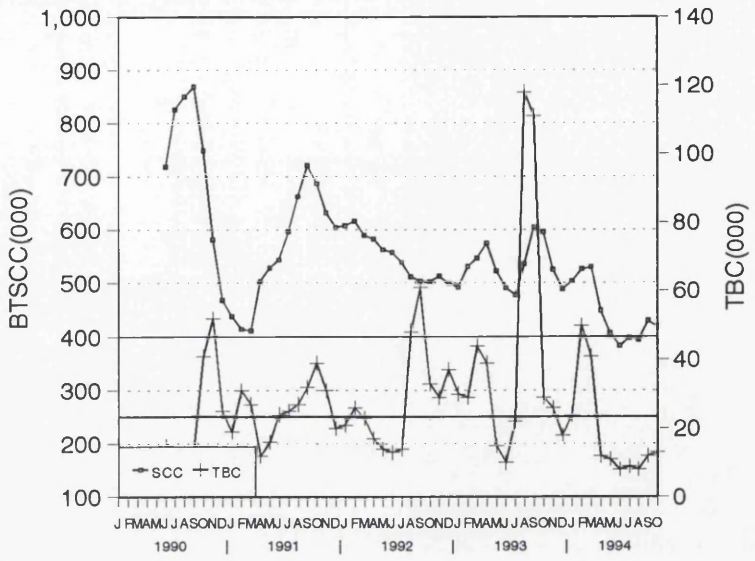

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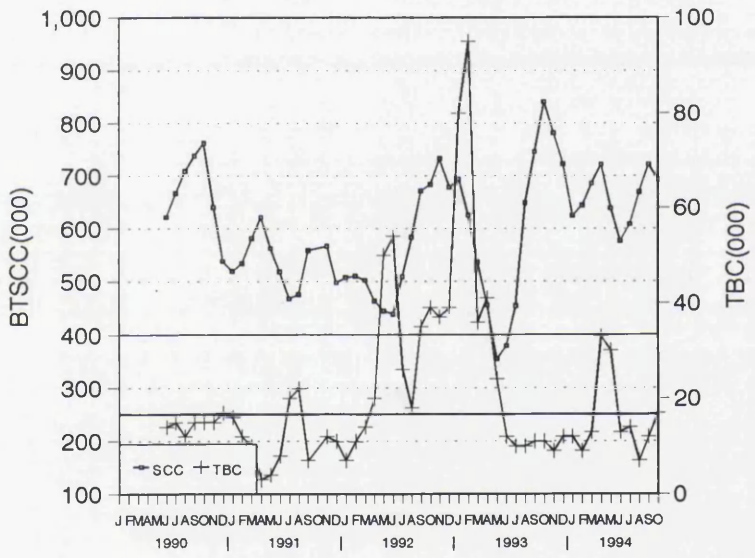

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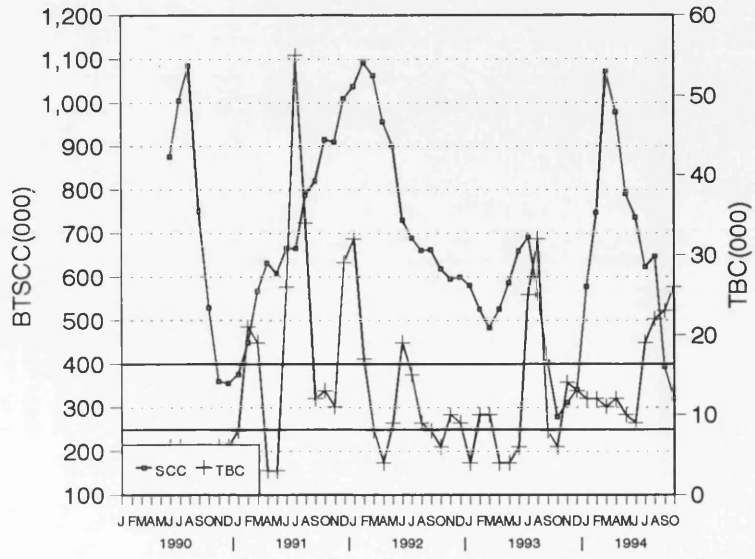

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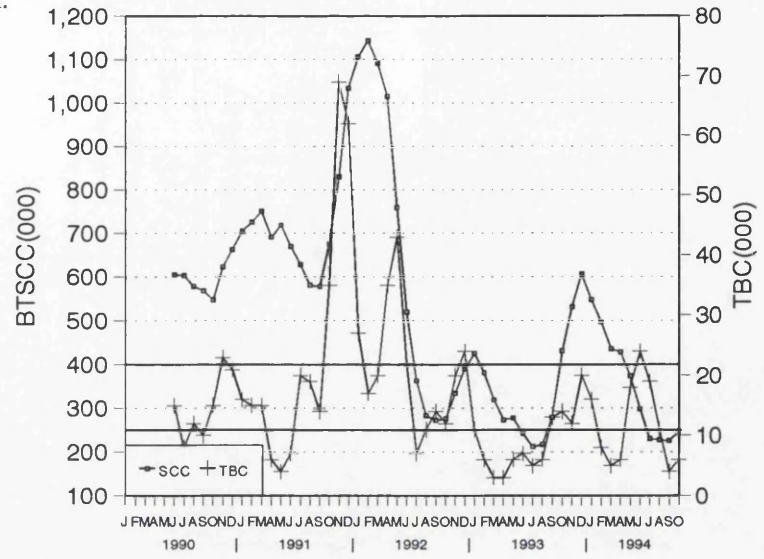
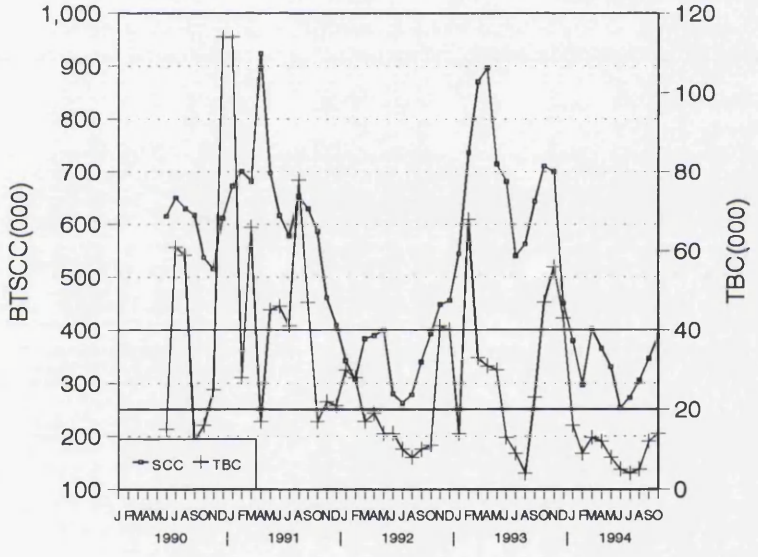


Project Herd 19
Phase-1a



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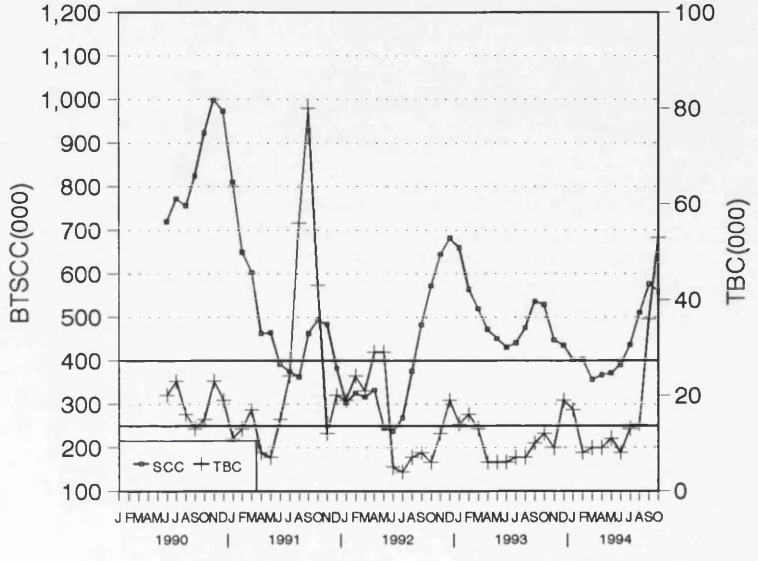
Phase-1a



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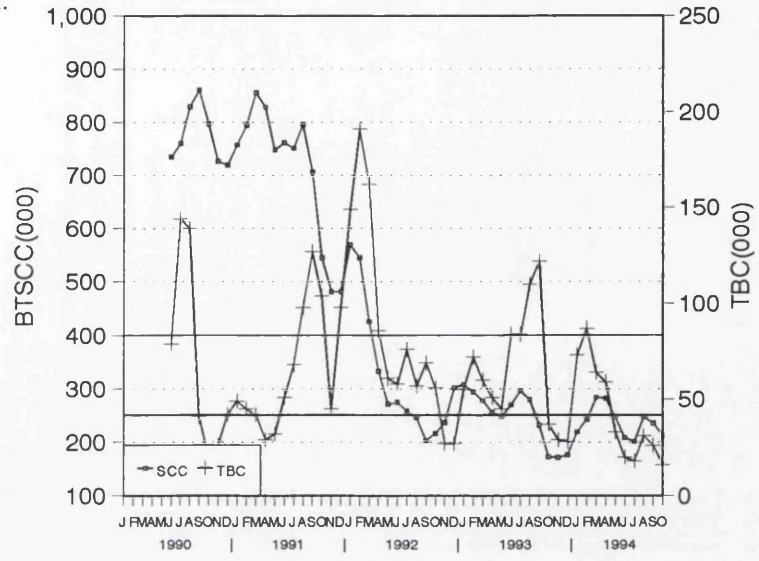
Phase-1a



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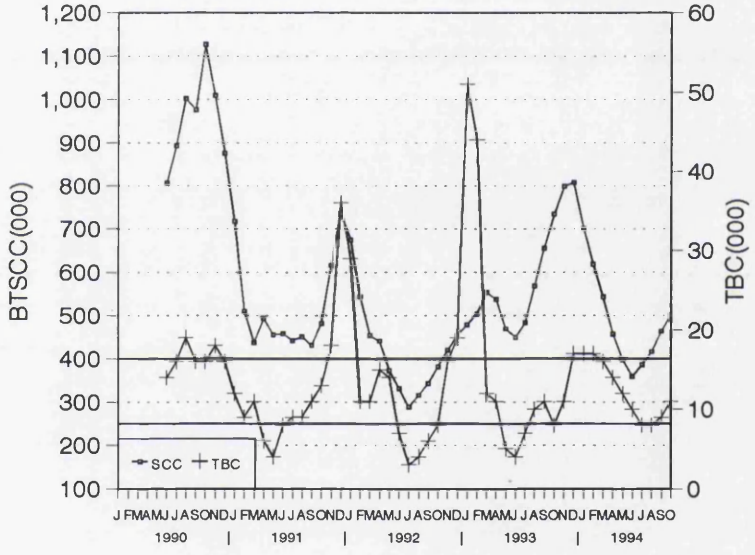
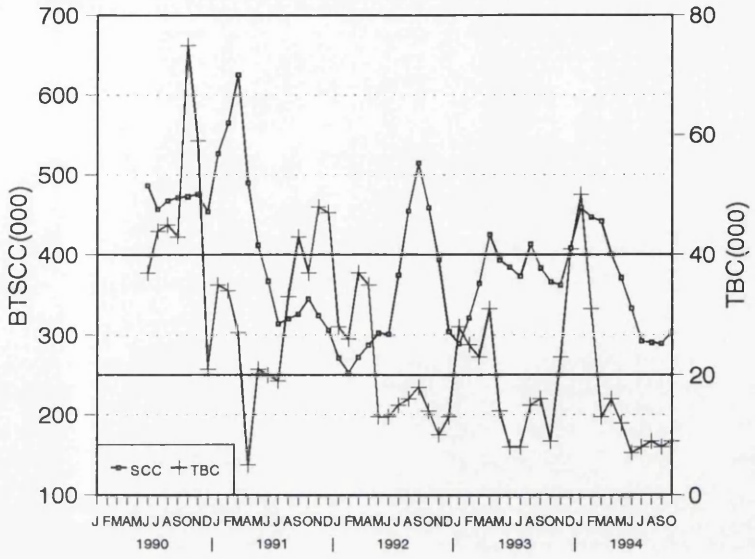
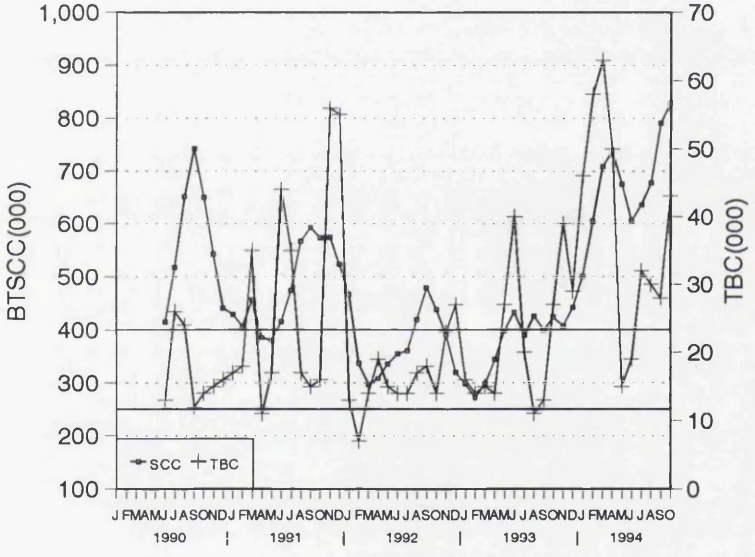
Phase-1a



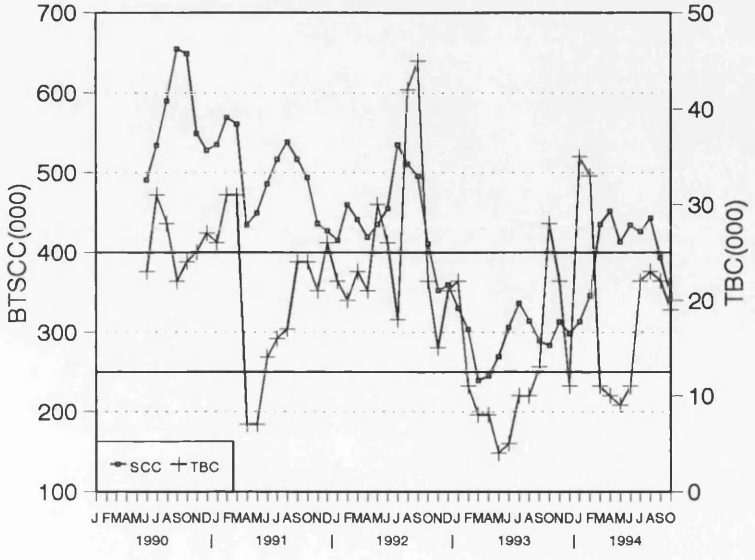
Project Herd 24
Phase-1a



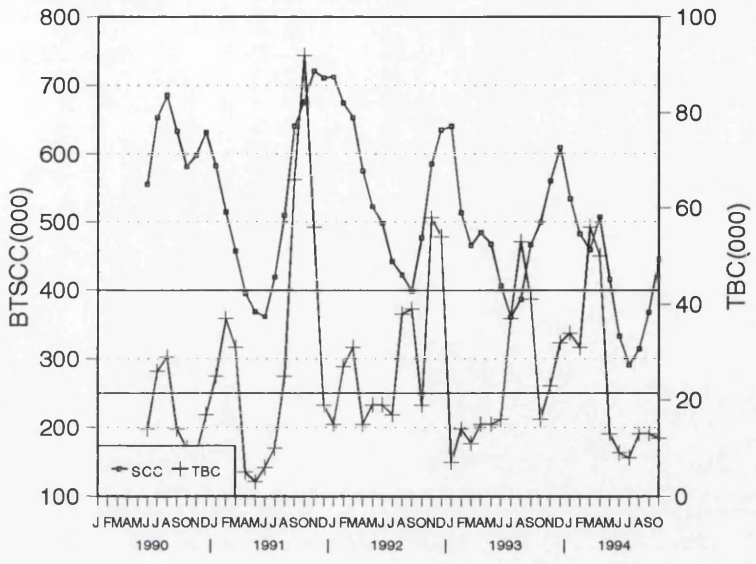
Project Herd 3
Phase-1a & Phase-1c



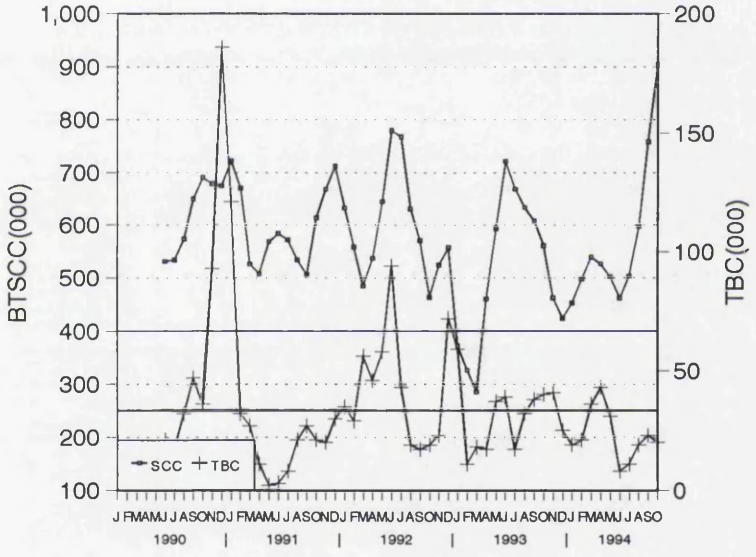
Project Herd 4
Phase-1a & Phase-1c



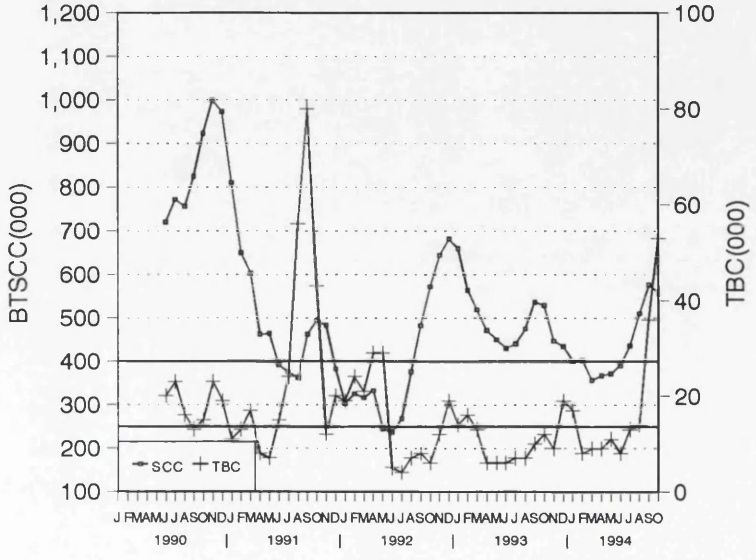
Project Herd 10
Phase-1a & Phase-1c



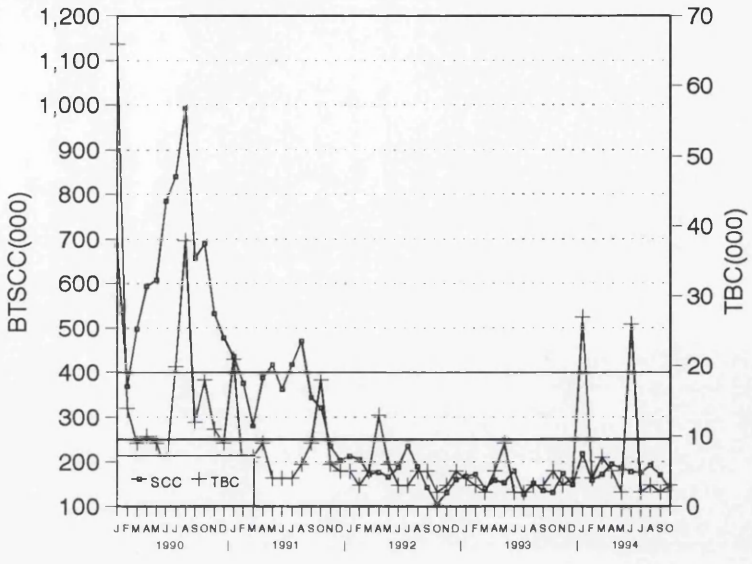
Project Herd 11
Phase-1a & Phase-1c



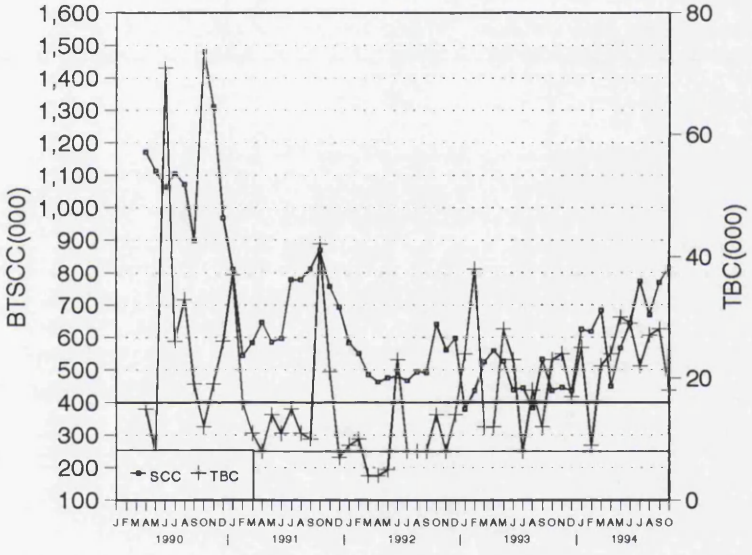
Project Herd 14
Phase-1a & Phase-1c



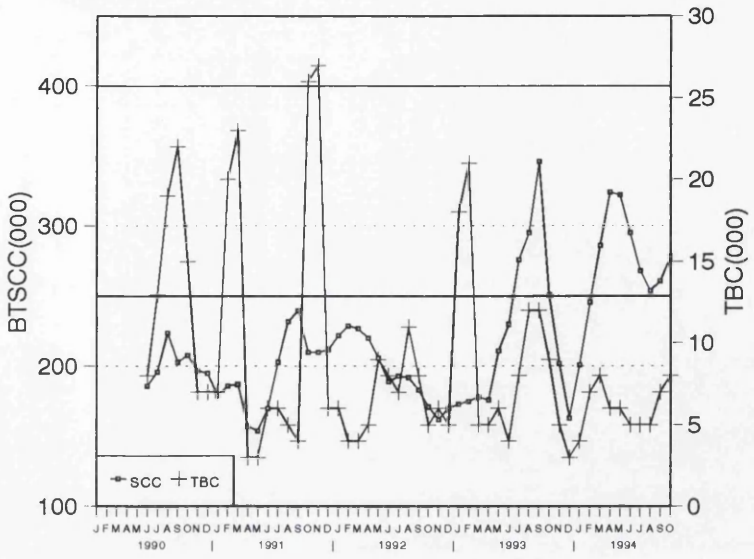
Project Herd 22
Phase-1a & Phase-1c



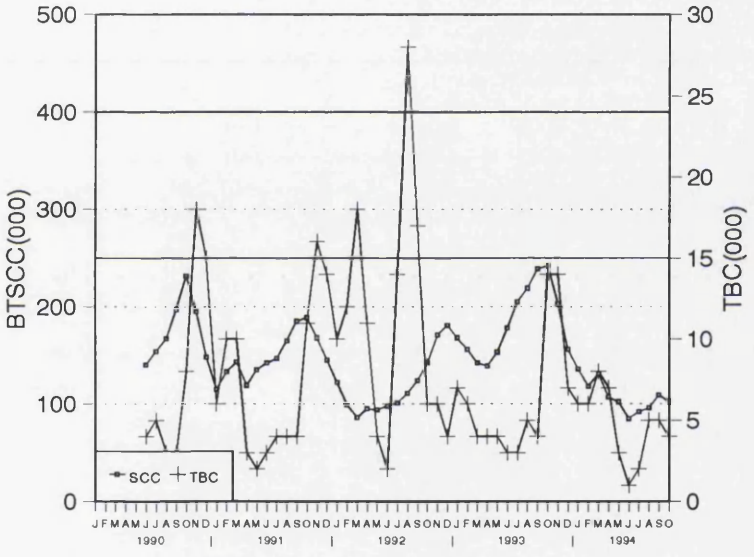
NOSMMB Project Herd
Not statistically selected



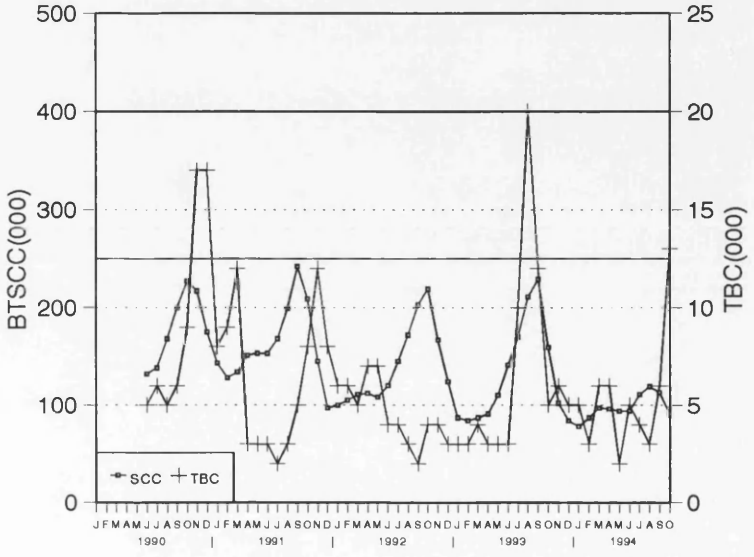
A&DMMB Project Herd
Not statistically selected



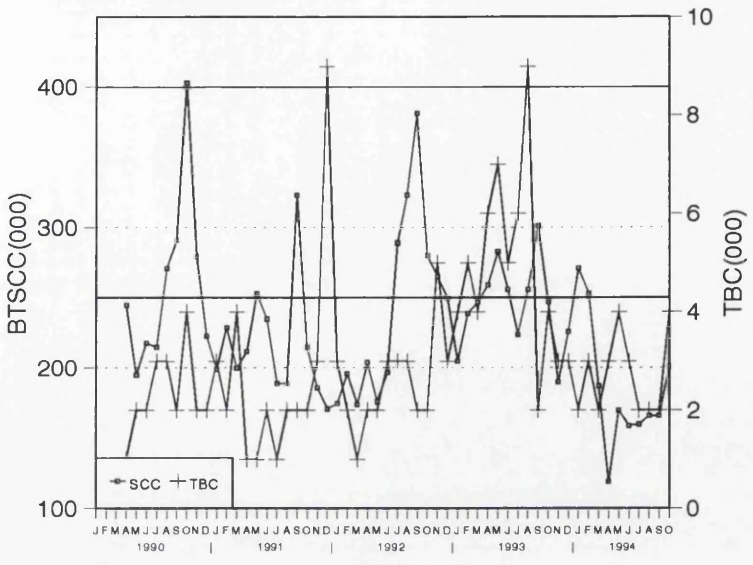
Project Herd 1
Phase-1b



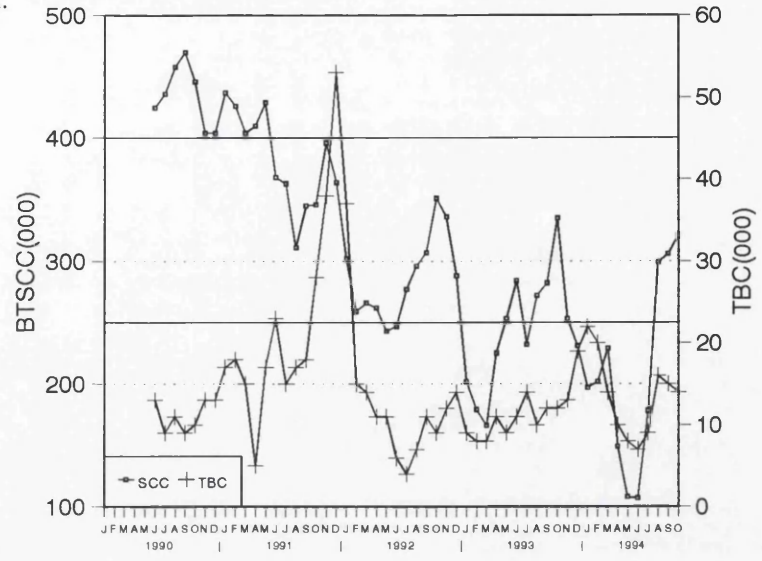
Project Herd 2
Phase-1b



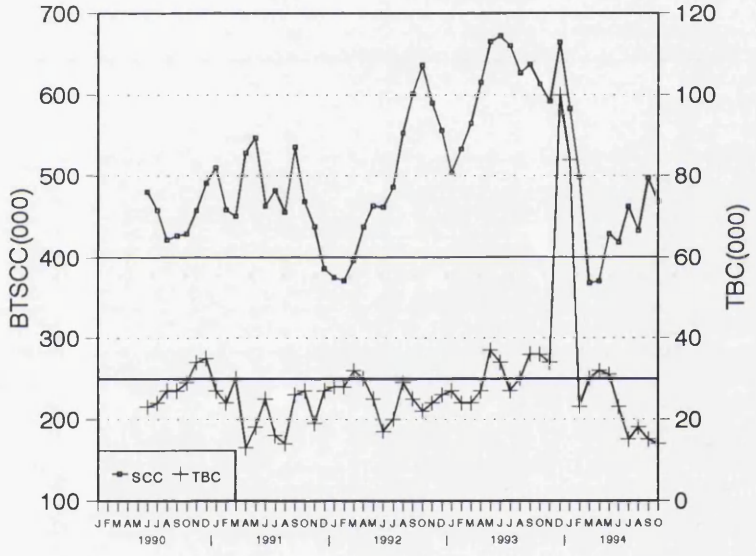
Project Herd 3
Phase-1b



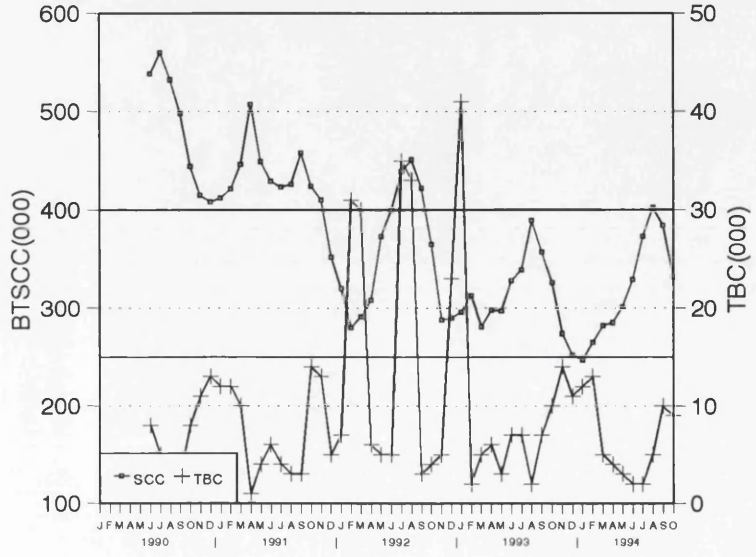
Project Herd 4
Phase-1b (A&DMMB)



Project Herd 1
Phase-2



Project Herd 2
Phase-2



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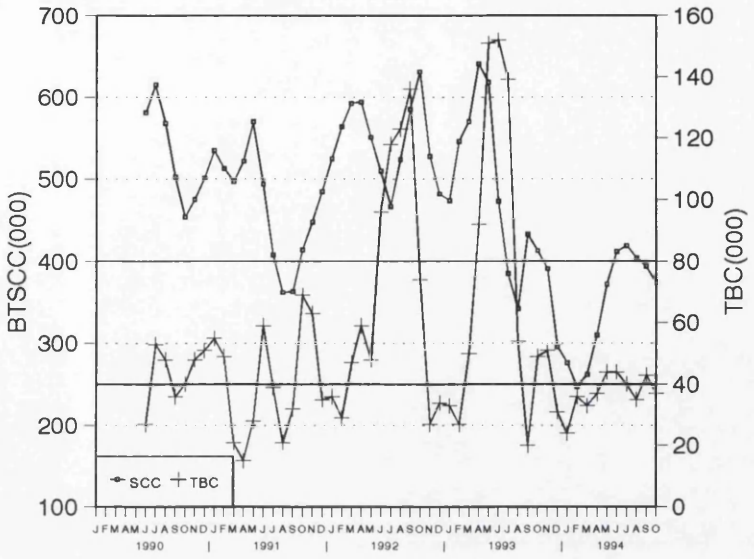
Phase-2



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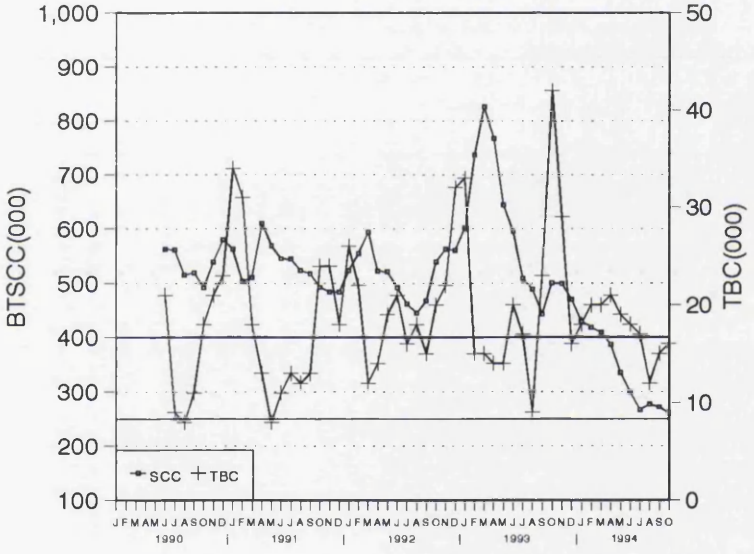
Phase-2



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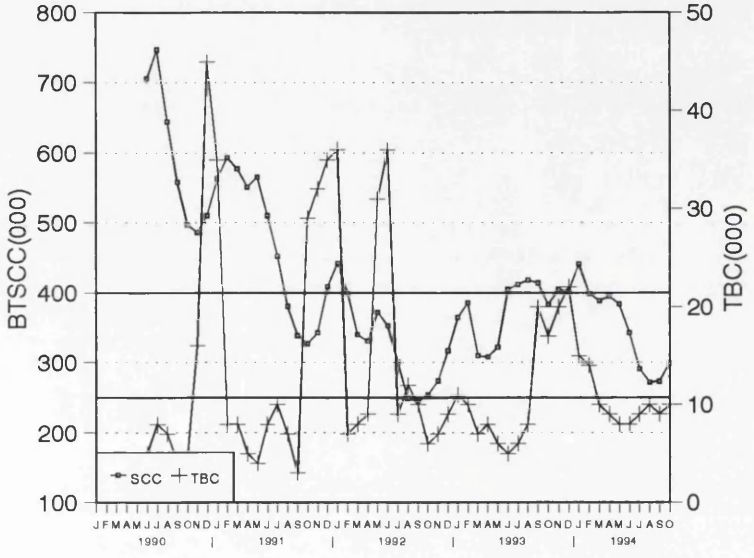
Phase-2



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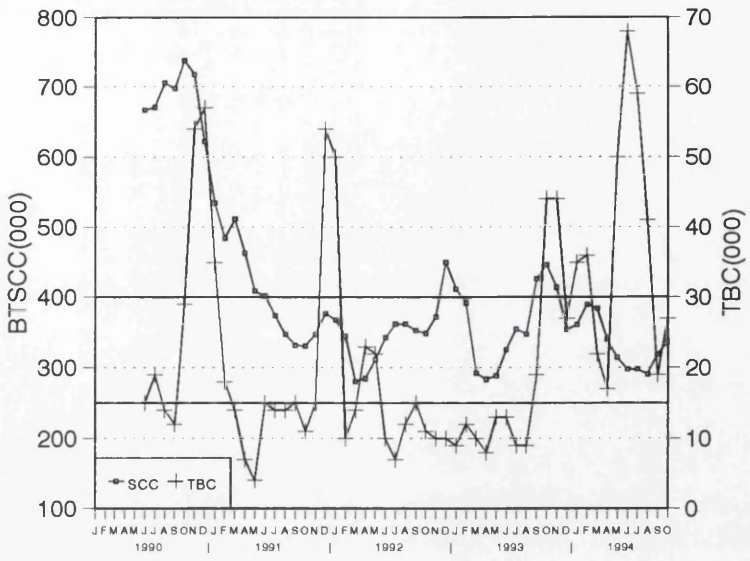
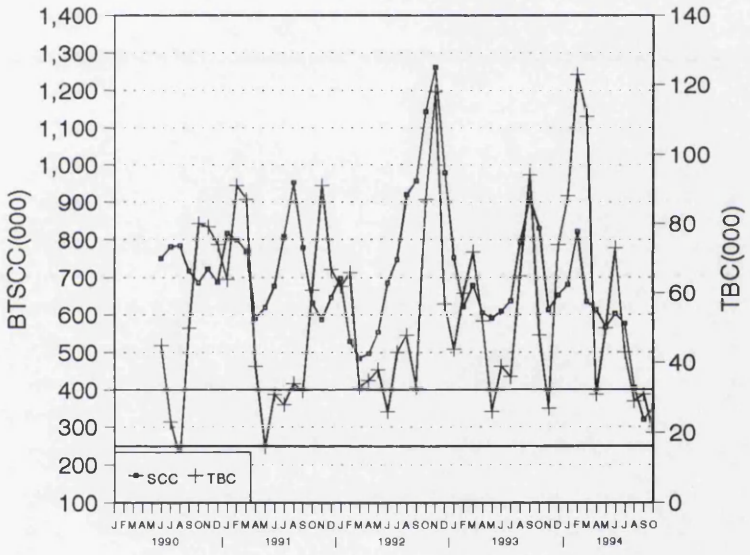
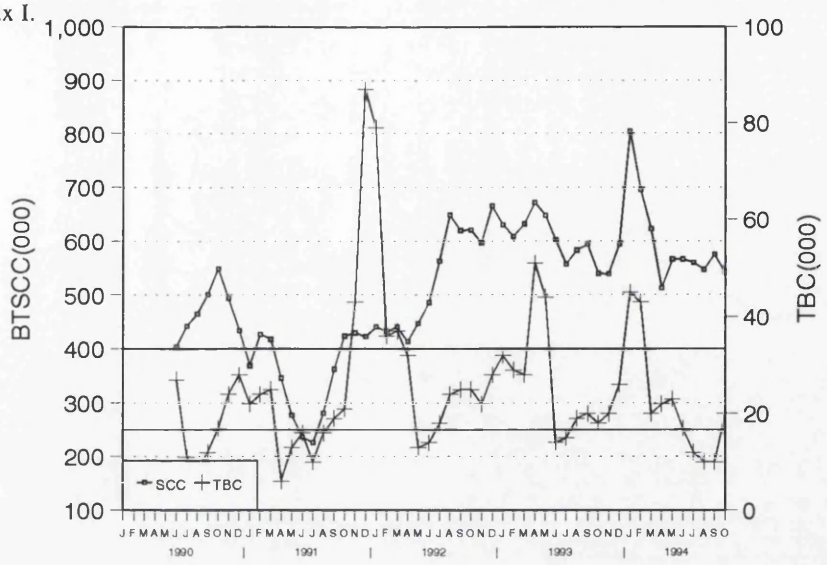
Phase-2



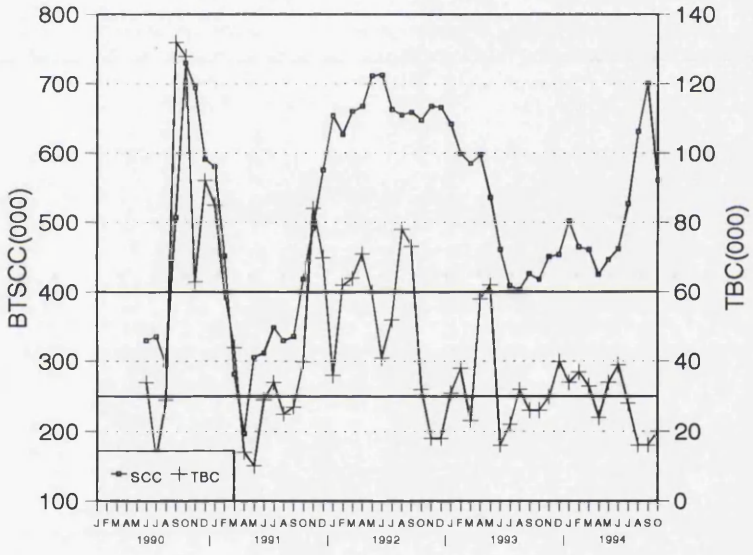
Project Herd 7
Phase-2



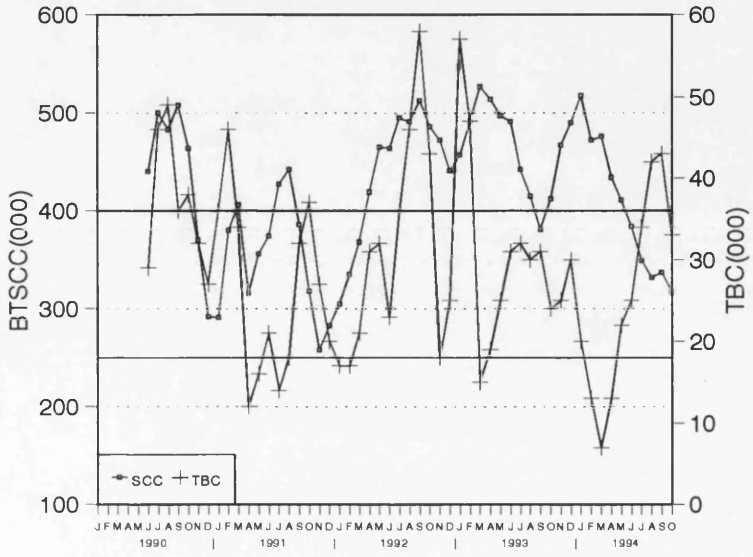
Project Herd 8
Phase-2



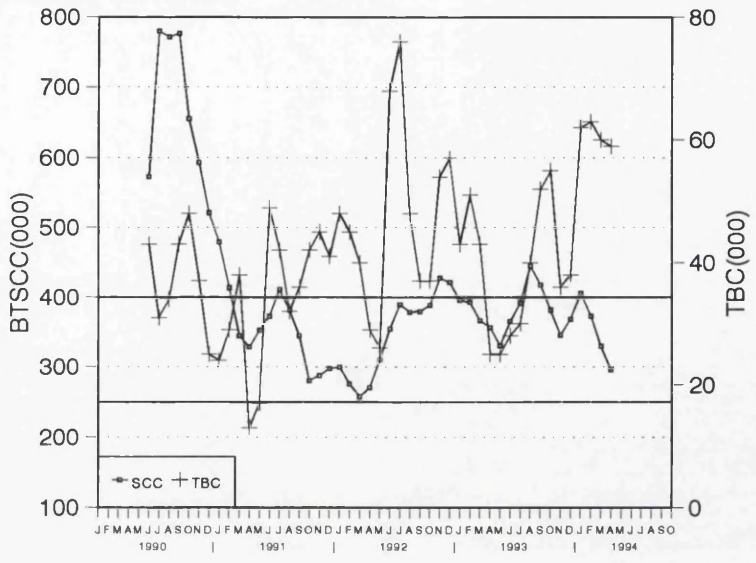
Project Herd 1
Phase-3



Project Herd 2
Phase-3



Project Herd 3
Phase-3



Project Herd 4
Phase-3

APPENDIX II

Mastitis control programme of each project herd after investigation

APPENDIX IIa

Mastitis control profile for 23 Phase-1a project herds

Herd	Size	DCT	PMTD	Cull	MMT	BRH	ACR	B/P[1]	W/D[2]	PapT
1	100	Yes	Yes	15	Yes	Yes	Yes	P	D	Y
2	150	No	No	7	Yes	Yes	Yes	P	D	Y
3	260	Yes	Yes	15	Yes	No	Yes	P	W	Y
4	165	Yes	Yes	12	Yes	Yes	Yes	P	W	Y
5	100	Yes	Yes	6	Yes	Yes	No	P	D	Y
6	80	Yes	Yes	0	No	Yes	n/a	B	D	N
7	60	Yes	No	33	Yes	Yes	No	P	D	Y
8	125	Yes	Yes	0	No	Yes	Yes	P	W	N
9	60	Yes	No	0	Yes	No	n/a	B	D	Y
10	65	Yes	Yes	31	Yes	No	No	P	W	Y
11	85	Yes	No	14	Yes	Yes	No	P	W	N
12	NOSMMB herd									
13	40	Yes	No	13	Yes	Yes	n/a	B	D	Y
14	36	Yes	Yes	8	No	Yes	n/a	B	W	N
15	80	Yes	Yes	10	Yes	Yes	No	P	D	Y
16	70	Yes	No	21	Yes	Yes	n/a	B	W	N
17	120	Yes	No	8	Yes	No	Yes	P	W	Y
18	24	Yes	Yes	25	No	No	n/a	B	W	N
19	100	Yes	Yes	25	Yes	Yes	No	P	W	Y
20	60	Yes	No	8	Yes	Yes	n/a	B	W	N
21	70	Yes	No	17	Yes	Yes	n/a	B	D	Y
22	85	Yes	Yes	7	Yes	No	Yes	P	D	Y
23	35	Yes	No	14	No	Yes	n/a	B	D	Y
24	50	Yes	Yes	10	Yes	Yes	n/a	B	D	Y
25	A&DMMB herd									

[1] Byre (B) or Parlour (P)

[2] Udder preparation: Wet (W) or Dry (D)

APPENDIX IIb

Mastitis control profile for 8 Phase-2 project herds

Herd	Size	DCT	PMTD	Cull	MMT	BRH	ACR	B/P	W/D	PapT
1	65	Yes	Yes	8	Yes	Yes	Yes	P	W	No
2	60	Yes	No	8	Yes	Yes	n/a	B	W	Yes
3	100	Yes	Yes	15	Yes	Yes	Yes	P	W	Yes
4	85	No	Yes	12	Yes	Yes	Yes	P	D	Yes
5	100	Yes	Yes	10	Yes	Yes	N	P	W	Yes
6	70	Yes	Yes	0	Yes	Yes	Yes	P	W	N
7	60	No	No	10	Yes	Yes	n/a	B	W	No
8	60	Yes	No	3	Yes	Yes	n/a	B	D	Yes

APPENDIX IIc

Mastitis control profile for 4 Phase-3 project herds.

Herd	Size	DCT	PMTD	Cull	MMT	BRH	ACR	B/P	W/D	PapT
1	45	Yes	No	4	Yes	Yes	n/a	B	W	No
2	125	Yes	Yes	16	Yes	Yes	Yes	P	D	Yes
3	100	Yes	Yes	5	Yes	Yes	Yes	P	W	No
4	60	Yes	Yes	17	Yes	Yes	Yes	P	W	No

APPENDIX III

Format of confidential May 1993 Scottish Dairy Farm Census

THE MILK MARKETING BOARDS IN SCOTLAND

SCOTTISH DAIRY FARM CENSUS 1993

To be returned by Friday, 21st May, 1993

(Ti. Scottish Dairy Farm Census is conducted under Section 18 of the Scottish Milk Marketing Scheme 1933 and corresponding Sections of the Aberdeen and District, and North of Scotland Milk Marketing Schemes. The terms of the Schemes require Registered Producers to furnish to the Boards, when requested, information relating to the milk produced by them. You are therefore required to answer the undernoted Questions 2 (a) and 7 (a) deemed to be covered by these terms. It is hoped that you will also co-operate with the Boards by completing the answers to the remaining questions. Return of the questionnaire is obligatory under the terms of the Schemes).

Information should relate to the first week in May 1993. The farm should be taken to include all land worked as one unit.

FOR OFFICE USE ONLY

FORM NUMBER 15485

1. FARM SIZE

Please enter the area (in hectares) of grass, crops and rough grazing etc. on the farm in the boxes provided. Enter figures to the nearest whole hectare. (A conversion chart for acres to hectares is enclosed with this questionnaire.)

	Hectares
a) Hectares of grass both for grazing and for mowing (but do not include rough grazing)	
b) Hectares of crops (including fallowland, if any)	
c) Hectares of rough grazing	
d) All other hectares (woodland, roads, buildings, etc.)	
TOTAL HECTARES (check this adds up)	

Is the farm:-

	Tick (✓)
Rented	
Owned	
Part Owned/Part Rented	

2. DAIRY HERD BREEDS

a) Enter in the boxes provided the total number of cows and heifers in milk, and cows in calf but not in milk. Do not include any cows used mainly for suckling calves.

Breed	*Number*
Ayrshire	
Friesian/Ayrshire Cross	
Friesian	
Holstein	
Holstein Cross	
Channel Islands (Jersey/Guernsey)	
Other Breeds and Crosses used for milk	
TOTAL (Check this adds up)	

b) Approximately how many hectares of grass, both for mowing and grazing are used mainly by the above dairy cows and heifers?

	Hectares
Hectares of grass	

3. DAIRY FOLLOWERS

Enter in the boxes provided the total number of dairy followers owned by you. Include all young female dairy stock.

Breed	*Number of Dairy Followers*
Ayrshire	
Friesian/Ayrshire Cross	
Friesian	
Holstein	
Holstein Cross	
Channel Islands (Jersey/Guernsey)	
Other Breeds and Crosses used for milk	
TOTAL (Check this adds up)	

PLEASE CONTINUE ON NEXT PAGE

GJNR,2(1993)152

FOR OFFICE USE ONLY

FORM NUMBER

4. QUOTA CHANGES

Do you have any definite plans to purchase or lease quota within the next twelve months:-

	Tick (✓) One Only
Yes, to purchase extra quota	
Yes, to lease extra quota	
Yes, to both lease and purchase extra quota	
No	

Do you have any definite plans to sell or let quota within the next twelve months?

	Tick (✓)
Yes	
No	

5. BULLS

Enter in the boxes provided the total number of bulls over one year old kept by you on the farm for home use on the dairy herd.

Breed	Number
Ayrshire	
Friesian	
Holstein	
Other pure dairy bulls	
Pure beef bulls (for use on dairy herd)	
Crossbred bulls (for use on dairy herd)	
TOTAL	

6. BREEDING

a) Which of the following breeding methods were used on your dairy cows and dairy heifers in the last 12 months? (Where appropriate tick (✓) more than one box.)

	On Dairy Cows Tick (✓)	On Dairy Heifers (i.e. to calve for first time) Tick (✓)
Natural service		
AI - Technician Service		
- Do-it-yourself		
No breeding		

b) If you used AI on your dairy herd in the last 12 months, did you use beef sires, dairy sires or both?

	Tick (✓) One Only
Beef sires only	
Dairy sires only	
Beef and dairy sires	

c) Do you intend to increase the use of a beef breed bull on your dairy herd in the coming year?

	Tick (✓)
Yes	
No	

d) Enter the approximate total number of live calves which have been born from your dairy cows and heifers in the past 12 months.

	Number
Number of calves	

e) Approximately how many of these calves were bred by AI?

Number

7. MILKING SYSTEMS

a) What type of milking system is used on the farm at present?

Byre	Tick (✓)
With buckets	
With pipeline	

Parlour	Tick (✓)
Herringbone	
Rotary	
Other	

PLEASE CONTINUE ON NEXT PAGE

GSNW, JC1995] 153

FOR OFFICE USE O
FORM NUMBER

7. MILKING SYSTEMS - Continued

b) How many operators are normally working in your byre or parlour at milking time?

	Number
Number	

10 20 40 70 1

8. MASTITIS CONTROL

Please indicate if you are using any of the undernoted mastitis control procedures on a regular basis on all your cows.

a) Teat dipping or udder spraying of cows and heifers in milk.

	Tick (✓)
Yes	
No	

b) Dry cow therapy on all or nearly all cows?

	Tick (✓)
Yes	
No	

c) Which of the following methods, if any, do you use for cleaning cows' udders?

	Tick (✓) One Only
Water and cloth	
Continuous water flow system	
None	

d) Do you have at present a milking machine testing and/or maintenance contract?

	Tick (✓)
Yes	
No	

9. LABOUR

a) How many people in total (including yourself) work regularly on the farm doing farm work or other work connected with the farm business? Show those working full-time and part-time separately?

	Number
Total working full-time	
Total working part-time	
Total regularly working on the farm	

b) How many of the above are members of your family (including yourself) or closely related to you?

	Number
Family labour, full-time	
Family labour, part-time	
Total family labour	

c) How many other people (excluding family) work regularly on the farm?

	Number
Non family labour, full-time	
Non-family labour, part-time	
Total non-family labour	

10. BULK FEED SUPPLIES

Please indicate which, if any, of the following bulk feed systems were used for your milking cows last winter.

Grass Products	Tick (✓)	Other Bulk Feeds	Tick (✓)
Hay		Straw	
Haylage (Tower)		Draff	
Silage, self feed		Kale	
Silage, not self feed		Roots	

11. MILK RECORDING

Are you at present involved in the recording of milk production of individual cows?

	Tick (✓)
Yes- with S.M.R.A.	
- with another organisation or privately	
No	

11. Please indicate here if you would like a copy of the summary report sent to you at the concessionary rate of £2.00 (inc. postage) when available. This offer applies only to registered milk producers.

	Tick (✓)
Please send a copy	
No thanks	

Please check that you have answered the questions on all three pages. THANK YOU.

APPENDIX IV

Project advisory leaflets subsequently supplied to all dairy herds in Scotland

Somatic Cell Counts Are Important

PROTECT YOUR BUSINESS

Comply with this standard.

YOU!

✧✧✧

We can help direct the progress you make.

✧✧✧

For further advice, please contact:

Dr David Logue/John Gunn
Dairy Health Unit
Veterinary Investigation Centre
Auchincruive
AYR KA6 5AE
Tel: (0292) 520318

or Your own Veterinary Surgeon

or Your local SAC Advisory Office

SAC

QUALITY MILK PRODUCTION

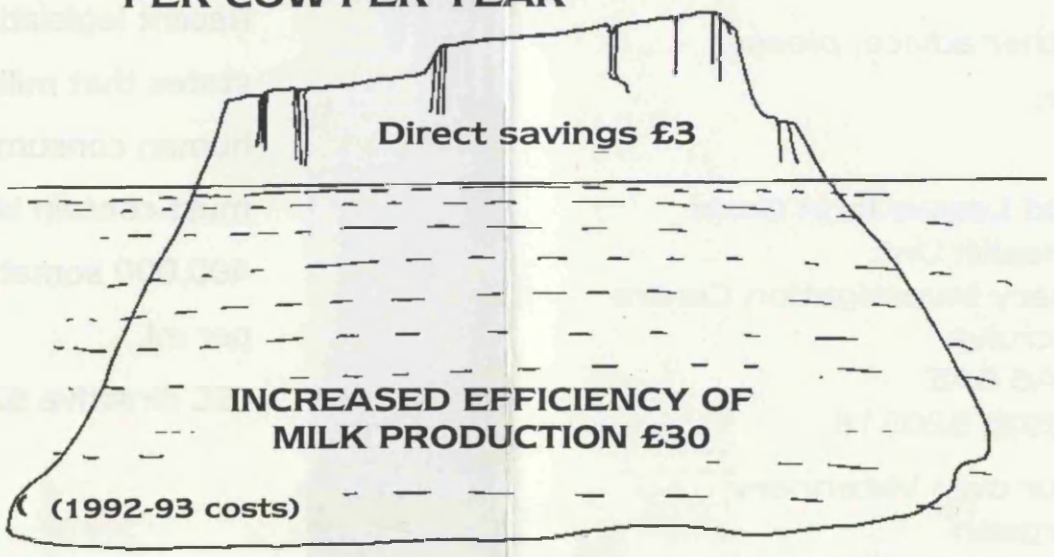
Recent legislation states that milk for human consumption must contain less than 400,000 somatic cells per ml.
(EC directive 92/46)

Gunn, J (1995) ISS

Low Bulk Tank Somatic Cell Count indicates that there is little ***subclinical mastitis*** in the herd and few ***mastitis bacteria*** in the milk.

Bacteria of ***ALL*** types in the bulk milk (from mastitis, environment and poorly cleaned milking machines) are directly measured as the **Total Bacterial Count.**

REDUCING MASTITIS: SAVINGS PER COW PER YEAR



Adequate control of Bulk Tank Somatic Cell Count means an estimated increase in income of £33/cow/year. This comes from both direct and indirect savings.

To encourage low Somatic Cell Counts the buyers of milk have a series of *penalties and premiums.*

Control of your Somatic Cell Count leads to direct savings.

✧✧✧

1994 SMMB Somatic Cell Count Premium/ Penalties

✧✧✧

Cell Count Band (000)	Premium/Penalty pence per litre of milk
less than 250	+0.10
250-400	NIL
401-600	-0.20
more than 600	-0.50

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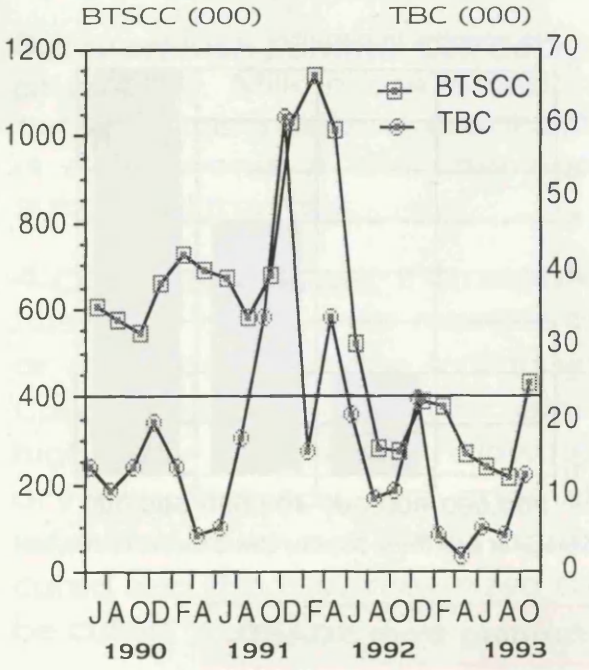
Compare your 3 month geometric average Bulk Tank Somatic Cell Count on your monthly milk statement with the figure on the previous statement. ***Is it moving up or down? What is its relationship with the Total Bacterial Count (a measure of the number of bacteria in the milk).***

Reduction of Bulk Tank Somatic Cell Count in a herd investigated in February 1992.



If the Bulk Tank Somatic Cell count is going up **IDENTIFY:**

1. The ***PROBLEM*** cows by doing Individual Cow Somatic Cell Counts regularly.

2. The ***CAUSE*** by taking milk samples from these cows for bacteriology.

By doing this you can determine the correct course of action.

For further advice, please contact:

Dr David Logue/John Gunn
Dairy Health Unit
Veterinary Investigation Centre
Auchincruive
Ayr KA6 5AE
Tel: (0292) 520318

or Your own Veterinary Surgeon

or Your local SAC Advisory Office

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SOMATIC CELL COUNTS (SCC)

This is a measure of the number of cells in milk. Most of them are white blood cells which are the body's defence against infection.

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The main reason for a high Somatic Cell Count in milk is that the cow has ***AN INFECTION IN AT LEAST ONE QUARTER.***

TREAT CLINICAL CASES

Infection is the Main Cause of High Somatic Cell Count.

Mastitis is caused by bacteria. There are three "types":

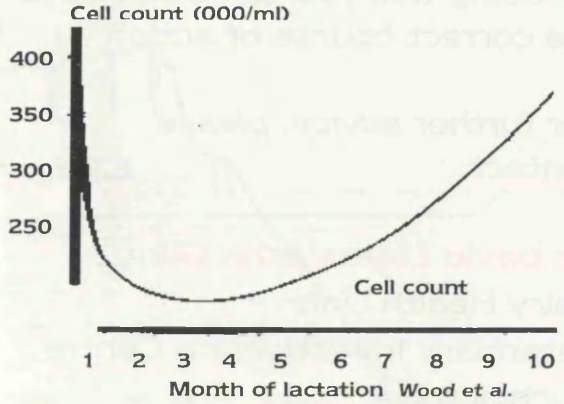
1. Contagious
2. Parlour
3. Environmental

Many of these cases are ***SUB-CLINICAL*** and so are ***NOT OBVIOUS.***

There are some other causes of variation as well as infection. They have a ***MUCH SMALLER EFFECT*** but include:

Stage of lactation in 'normal' cows.

INDIVIDUAL COW SOMATIC CELL COUNT

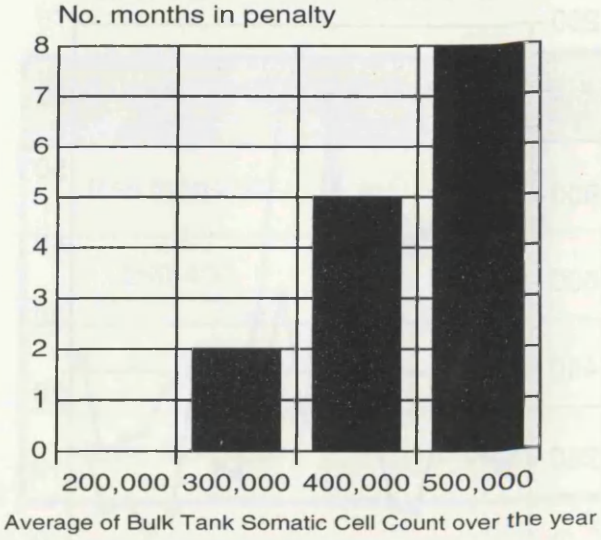


Figures Avoid EC Threshold

The target annual average Bulk Tank Somatic Cell Count needs to be below 250,000/ml to ensure the Bulk Tank Somatic Cell Count of the herd definitely does not exceed the EC threshold of 400,000 cells/ml at some time in the year.

Bulk Tank Somatic Cell Count

Year average against months in Penalty



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*The **minimum** management recommendations are:*

1. All cows should be given ***Dry Cow Therapy*** every year. Cows should be dried off abruptly 2 months before calving and treated irrespective of the amount of milk they are producing.

2. ***Milk high Somatic Cell Count cows last.*** They are carrying infection so milk them last to avoid transferring infection to low cell count cows at milking.

3. ***Dry off high Somatic Cell Count cows early.*** Milk in late lactation naturally has a high cell count but is a lot worse if the cow has subclinical mastitis.

4. ***Cull problem cows.*** If an animal has been treated for mastitis 3 or more times or the Individual Cow Somatic Cell Count remains high early in lactation following dry cow therapy, the original infection may not have been cured and the cow may need to be culled.

from the highest Individual Cow Somatic Cell Count cows into the bulk tank, perhaps by feeding it to calves.

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Veterinary Investigation Centre
Auchincruive
Ayr KA6 5AE
Tel: (0292) 520318

or Your own Veterinary Surgeon

or Your local SAC Advisory Office

USE DRY COW THERAPY

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CONTROLLING YOUR BULK TANK SOMATIC CELL COUNT

EC directive 92/46 on milk hygiene states that the Bulk Tank Somatic Cell Count of milk for human consumption must contain less than 400,000 somatic cells per ml.

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Monitor Your Somatic Cell Count

It is important to monitor the Bulk Tank Somatic Cell Count. ***Is it rising or already too high?***

High cell counts usually mean a number of cows have mastitis (in some cases unknown to you).

Effective prevention and control require that you identify the main mastitis-causing bacteria in your herd:

1. **Identify problem cows** by recording mastitis treatments and Individual Cow Somatic Cell Count.
2. **Identify the main mastitis bacteria:**

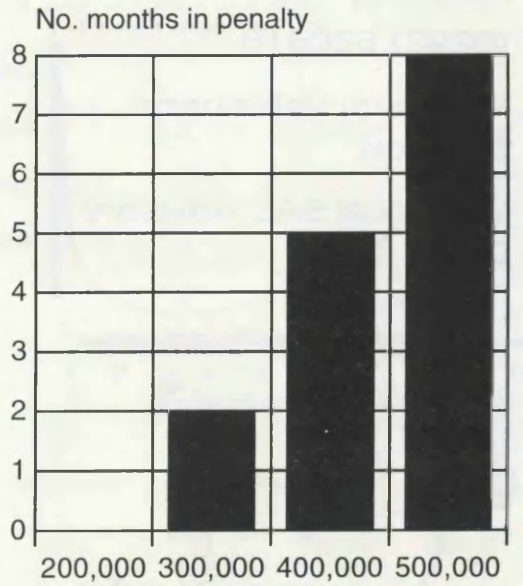
(i) Take milk samples from clinical cases;

(ii) Take milk samples from high Individual Cow Somatic Cell Count "problem" cows.

Aim for Premium

Those herds which do not incur payment penalties achieve an annual average Bulk Tank Somatic Cell Count of under 250,000 cells/ml.

MONTHS IN SCC PENALTY



Average of Bulk Tank Somatic Cell Count over the year

Even these **"Premium"** herds can have a slightly raised Bulk Tank Somatic Cell Count when a lot of cows are calving or nearly dry. However, they have ***VERY FEW INFECTED COWS.***

OBEY THE FIVE POINT PLAN.

1. Teat dip all cows after milking.

2. Treat all cows with antibiotic at drying off.

3. Identify and treat clinical cases promptly.

4. Cull chronic cases.

5. Have the milking machine tested at least annually.

PLUS

CLEAN YOUR ENVIRONMENT

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Post-milking teat dipping is an important way of preventing infection with ***Staph. aureus*** and ***Strep. dysgalactiae.***

All cows should receive Dry Cow Therapy. If this does not cure the infection you should consider culling the cow.

Treatment does ***NOT ALWAYS REDUCE*** the Somatic Cell Count of the cow to a "normal" level i.e. once a quarter has been infected, the Somatic Cell Count may remain high.

USE DRY COW THERAPY

from becoming infected.

Cows with 3 or more clinical cases in 1 lactation or consistently high Somatic Cell Count may be carrying ***Staph. aureus.*** They should be milked last to avoid contaminating the milking machine and dairyman's hands and spreading bacteria to uninfected cows with low Somatic Cell Counts.

For further advice, please contact:

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Veterinary Investigation Centre
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Tel: (0292) 520318

or Your own Veterinary Surgeon

or Your local SAC Advisory Office

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MASTITIS BACTERIA: 1 "Parlour" organisms

Mastitis is the main cause of high Somatic Cell Counts. Bacteria are the main cause of this infection. Two bacteria are able to survive and spread in the PARLOUR:

Staph. aureus;

Strep. dysgalactiae.

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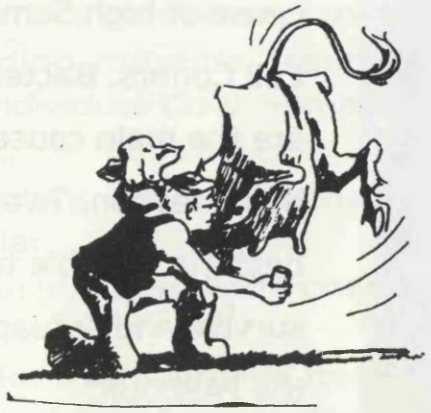
Staph. aureus can survive on the skin of both the teat and dairyman's hands, as well as on udder cloths.

Strep. dysgalactiae can also survive on teat skin, especially where there are lesions such as chaps and warts.

DIP TEATS



The Somatic Cell Count is a measure of the number of white blood cells in the milk. As part of the body's defence mechanism these cells move into the milk to kill infection by absorbing the bacteria. Unfortunately ***Staph. aureus*** bacteria can survive and multiply **within** the somatic cells away from the effect of anti-biotic. Later they break out beginning the cycle of Somatic Cell Count increase and the spread of infection all over again.

CULL CHRONIC COWS

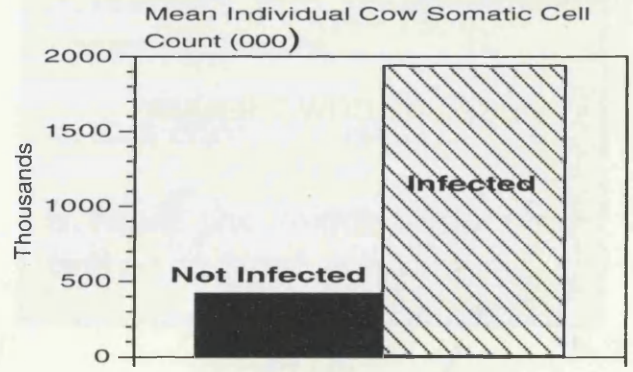
dysgalactiae causing high Somatic Cell Count in your herd?

To find out you should:

1. Identify problem cows;
2. Identify bacteria.

Send individual cow milk samples into the laboratory for bacteriology. Then you will know the ***CAUSE*** of your problem and be able to take ***APPROPRIATE*** control measures.

STAPH. AUREUS
Effect on Somatic Cell Count*



** in problem herds*

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Since ***Strep. agalactiae*** only survives within or upon the cow, this is the place to kill it! In the udder it is sensitive to antibiotics, either as milking cow tubes or dry cow therapy. On teats it is easily killed by reputable post-milking teat dips.

To identify high Somatic Cell Count cows you should arrange for regular monthly Individual Cow Somatic Cell Counts.

1. Help Prevent Spread

The herd should be divided by Individual Cow Somatic Cell Count (high/low) and the high cell count infected cows milked last.

Use a post-milking teat dip and Dry Cow Therapy.

2. Identify Bacteria

Send individual cow milk samples into the laboratory for bacteriology. Then you will know the ***CAUSE*** of your problem and be able to take ***APPROPRIATE*** control measures.

Small groups of the problem infected cows can rejoin the rest of the herd following antibiotic treatment and reduction of their cell count.

4. Continue to Monitor

Treatment does **NOT CURE ALL** cows. Continue to monitor Individual Cow Somatic Cell Counts and maintain all preventive measures.

For further advice, please contact:

Dr David Logue/John Gunn
Dairy Health Unit
Veterinary Investigation Centre
Auchincruive
Ayr KA6 5AE
Tel: (0292) 520318

or Your own Veterinary Surgeon

or Your local SAC Advisory Office

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MASTITIS BACTERIA: 2 "Contagious" organisms

Mastitis is the main cause of high Somatic Cell Counts. Bacteria are the main cause of this infection. One bacteria, *Strep. agaiactiae* is a particular problem. Having once entered a herd it can easily spread to infect most of the cows.

Gunn/Logue (1995) 1.5M

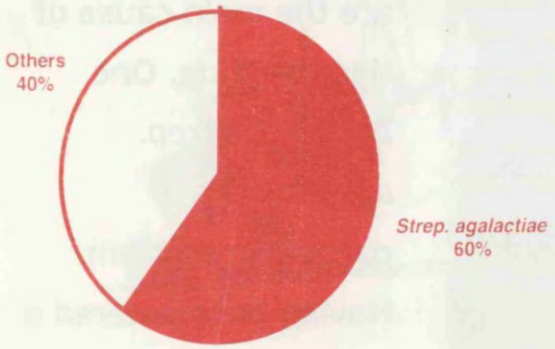
survives *WITHIN* the udder.

It is highly infectious, spreading to clean cows at milking time on clusters contaminated by high Somatic Cell Count infected cows, dirty cloths and milkers hands.

Strep. agalactiae is currently the most common cause of high Somatic Cell Counts in Scottish dairy herds.

HIGH SOMATIC CELL COUNT HERDS

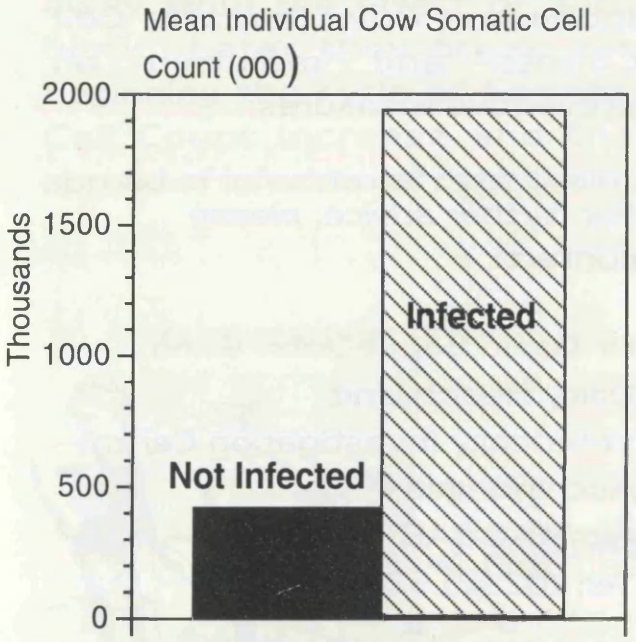
Mastitis Bacteria



increase in the Individual Cow Somatic Cell Count and thus of the Bulk Tank Count.

STREP. AGALACTIAE

Effect on Somatic Cell Count*



** in problem herds*

Is *Strep. agalactiae* causing high Somatic Cell Count in your herd? The *only* way to find out is to send individual cow milk samples from cows with mastitis and/or high Somatic Cell Counts into the laboratory for bacteriology. Only then will you know the *CAUSE* of your problem and thus the *APPROPRIATE* control measures to take.

TREAT CLINICAL CASES

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DIRTY ENVIRONMENT = MORE ENVIRONMENTAL MASTITIS!

DIP TEATS

Keep the environment clean and dry by scraping passages and cubicles frequently and renewing bedding daily.

Allow cows to stand for at least 30 minutes after milking in a **CLEAN** collecting yard while the teats are closing tight and the teat dip is drying.

For further advice, please contact:

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Dairy Health Unit
Veterinary Investigation Centre
Auchincruive
Ayr KA6 5AE
Tel: (0292) 520318

or Your own Veterinary Surgeon

or Your local SAC Advisory Office

SAC

MASTITIS BACTERIA: 3 "Environmental" organisms

Mastitis is the main cause of high Somatic Cell Counts. Bacteria are the main cause of this infection. Two causes of mastitis, bacteria called *E. coli* and *Strep. uberis*, are found widely in the environment so mastitis caused by them is often called "environmental mastitis".

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Herds which have controlled other causes of mastitis using post-milking teat dipping and Dry Cow Therapy can still have problems with environmental mastitis.

The Dry Period

The rate of ***INFECTION*** with environmental mastitis bacteria is three to four-fold greater ***DURING THE DRY PERIOD*** than during lactation. Although infection becomes established during the dry period, it will generally not become obvious until after calving.

USE DRY COW THERAPY

Are *E. coli* and *Strep. uberis* Causing High Somatic Cell Count in Your Herd?

The only way to find out is to send milk samples from cows with mastitis into the laboratory ***BEFORE*** treatment. Only then will you know the ***CAUSE*** of your problem and thus the ***APPROPRIATE*** control measures to take. This information will also help decide the best way of treating the next mastitis case.

Treat Clinical Cases Quickly and Give a Full Course

If *E. coli* and *Strep. uberis* are a problem in ***YOUR*** herd it is important to treat immediately to cure the infected cows and so avoid transfer of infection to clean cows.

TREAT CLINICAL CASES

Milking Machine Service

Have your milking machine checked at least twice a year to ensure it is not helping to cause environmental mastitis. Correct any problems ***IMMEDIATELY.***

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APPENDIX V

Publications based on material contained in this thesis

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RESULTS OF OUR APPROACH TO MASTITIS CONTROL IN SCOTLAND

David Logue*, John Gunn* & David Fenlon#, * Dairy Health Unit, SAC Veterinary Services, Auchincruive; # Department of Bacteriology, SAC, Aberdeen

Introduction

Before addressing the given title it must be said that it implies that there is some authority or overall control over how mastitis is tackled in Scotland and that somehow this is vested in the particular group that is being represented here. Since this is certainly not the case it is best to set the record straight right from the start. However we are delighted to have the chance to share with you our results, conceptions and, perhaps misconceptions about this fascinating and frustrating disease. In this paper we will be presenting data, particularly that pertaining to subclinical mastitis, which were collated with the help of the three Milk Marketing Boards in Scotland and other colleagues. This project was coordinated by John Gunn and his part has been indispensible. The other collaborators are listed in Table 1. However there are a considerable number of others who have contributed in some way to the information presented here.

Table 1. Collaborators in the study of subclinical mastitis in Scotland.

D. Taylor, Glasgow University, Vet. School	D. Todd SMMB
J. McIssac A&DMMB	D. Platt, Glasgow University, Dept. Bacteriology
M. MacLeod NoSMMB	

Having been to all of the British Mastitis Conferences since they started in 1988 it would also be unwise to give the impression that we have been hiding some magic cure distilled from something or other in Scotland and have not let on to the rest of the UK. Our approach to mastitis is therefore quite predictable and follows some well trodden paths. Because of our interest we will tend to discuss the general rather than the particular. We appreciate that some would prefer the individual farm problem-solving presentation. In our experience they generally concentrate on one aspect and the overall message which we hope those farmers here will take away fails to be fully stated.

Our approach and thus this paper can be summarised under three headings:

1. Define the problem

2. Identify the main methods of control and prevention

3. Convince the farmer

1. Define the problem

In the past Scotland has always had larger herd sizes than most other parts of the UK but in recent years this difference has become less with quite a number of regions in England matching the Scottish average herd size of 91 cows. Despite the impact of quotas slowing the trend there continues to be a steady increase in the number of cows in the herd. One of the reasons for this last change has been the decline in the cowshed and in the number of herds. In the last decade in Scotland there has been a reduction in the proportion of producers using a byre milking system from 46% to 31%, a fall of nearly one third. The small rise in the number of parlours (see Table 2) belies the overall fall of around a fifth in the number of herds. One consequence of this is that the number of cows being milked, and by inference cared for, by one person has risen from 60 in the early 1980s to 70 now (Table 3). Within Scotland and indeed the UK the area around Aberdeen (A&DMMB) perhaps shows the future with an average herd size of 121 cows.

Table 2. Proportion of different milking systems in Scotland.

SYSTEM	1981	1990	Av herd size in 1990
Byre	46%	31%	55 cows
Parlour	54%	69%	106 cows

[adapted from The Structure of Scottish Milk Production 1990]

Table 3. Milking system and its effect upon number of cows per milker.

SYSTEM	No. cows/milker		Av herd size in 1990
	1981	1990	
Byre	37	39	55
Parlour	79	85	106
Overall	59	69	91

[adapted from The Structure of Scottish Milk Production 1990]

The first problem we must come to terms with when discussing inputs for mastitis diagnosis, control and prevention is the need to understand the management systems and in particular the economic and labour pressures in these larger herds. It is the demands of time which are paramount and especially those related to managing cows to reduce mastitis. Often the latter measures are seen as of less immediate priority than other farm needs such as silage making.

The second part of defining the problem is to attempt to monitor the extent of mastitis. All are well aware that there are essentially two types of mastitis:

i) *clinical*

ii) *sub clinical*

The distinction between the two is somewhat arbitrary and can be misleading

i) Clinical mastitis

The most accurate appreciation of clinical mastitis can be achieved by examining farm records, the details of mastitis treatments and most importantly the bacteriological identification of the cause. Unfortunately, in our experience, sufficient information on the last parameter to give an authoritative estimate of the predominant organism acting on the farm is rare. However even without bacteriology some insight into the problem can be achieved providing that the farm records properly and preferably puts this information in an easily accessible database such as DAISY. For example, on one farm there were a substantial number of cases of mastitis occurring around 100 days and not, as is often the case, in early lactation (Figure 1). Indeed this breakdown substantially changed the perception of both farmer and vet. In another instance there appeared to be a relationship with lameness. Unfortunately in neither example were there sufficient laboratory results available to relate these findings with a particular pathogen though both were thought to be of 'environmental origin'.

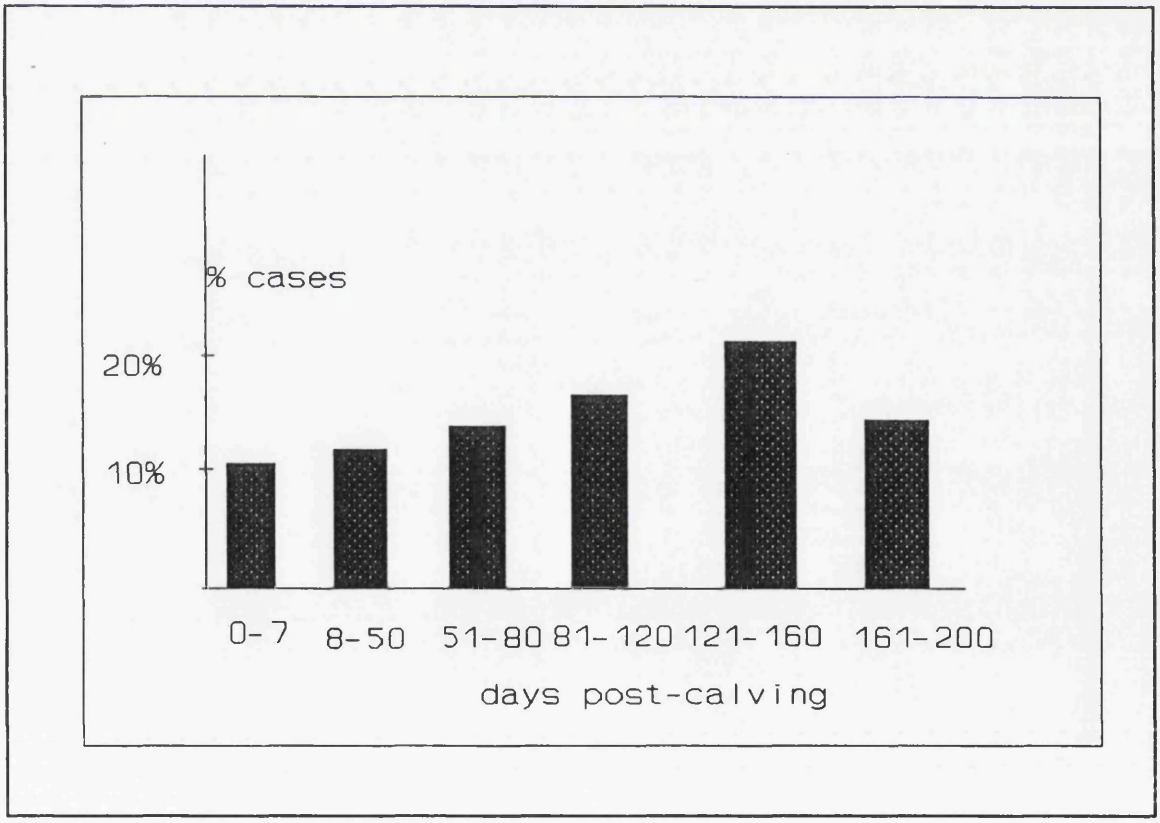

Figure 1. Incidence of clinical mastitis by days post-calving.

This reluctance to examine sufficient milks is a combination of three factors:

a) cost

b) inconvenience

c) hope.

Since in our project investigating herds with high Somatic Cell Counts (see later) we offered farmers free bacteriological examination of clinical mastitis samples and received hardly any, we are forced to the conclusion that, despite what might be thought, inconvenience and hope have as large an influence as cost! The hope is that the results from one sample will be representative of the whole. In many cases it is further compounded by the hope that they will be valid from one year to the next. Unfortunately neither need be the case. Table 4 shows the apparent change in proportion of isolates from the main herd at the SAC Auchincruive Crichton Royal Farm in two successive years (1987-89). Thus we would recommend that any farm interested in mastitis control should aim to sample about 20% to 25% of their clinically affected quarters for bacteriological examination and take the sample PROPERLY. The fresher the sample the more accurate the bacteriology so it is worth trying to ensure that it is kept cool and processed as soon as possible.

Table 4. Clinical mastitis and sampling variation between years.

Mastitis	Year 1	Year 2
Number of incidents	63	86
Number of samples	23	30
E. coli	17	11
Staph. aureus	3	6
Strep. dysgalactiae	1	11
Strep. uberis	0	1
Others	2	4

The problem of obtaining good information about the organisms acting on any one farm has forced us to another method of defining the problem. This involves taking a more general view and using the pooled results of similar laboratory tests on mastitic milk samples submitted to the 8 veterinary investigation centres in Scotland by farmers and their veterinarians. We rely totally on diagnostic field samples to give us this background and under these circumstances the data can present problems of interpretation; but some comparisons are more acceptable than others. For example comparison of the proportions of diagnoses of the different mastitis causing organisms throughout the Great Britain has remained remarkably steady over the last decade. However there does appear to be a consistent difference between these overall figures and those for Scotland alone which show firstly that there would appear to be more 'cowside' or 'contagious' organisms diagnosed in Scotland (*Streptococcus agalactiae, Staphylococcus aureus* and to a lesser extent *Streptococcus dysgalactiae*) and secondly that one particular environmental organism (*Streptococcus uberis* or SPUB in Figure 2) appears to be less common. Obviously one

could blame sampling bias for this difference but cynically it seems unlikely that Scottish farmers are any more interested in mastitis than their counterparts elsewhere.

So this raises a number of questions:

i) why is there this difference?

ii) why, if there is more cowside mastitis in Scotland, is this not readily detectable in differences in the Somatic Cell Counts in Scotland compared to the rest of the UK. (See Figure 3).

iii) why are the proportions of these major organisms in the UK so consistent when we are told that the incidence of mastitis, whether clinical or sub clinical, is declining (2) and bulk tank somatic cell counts appear to confirm this.

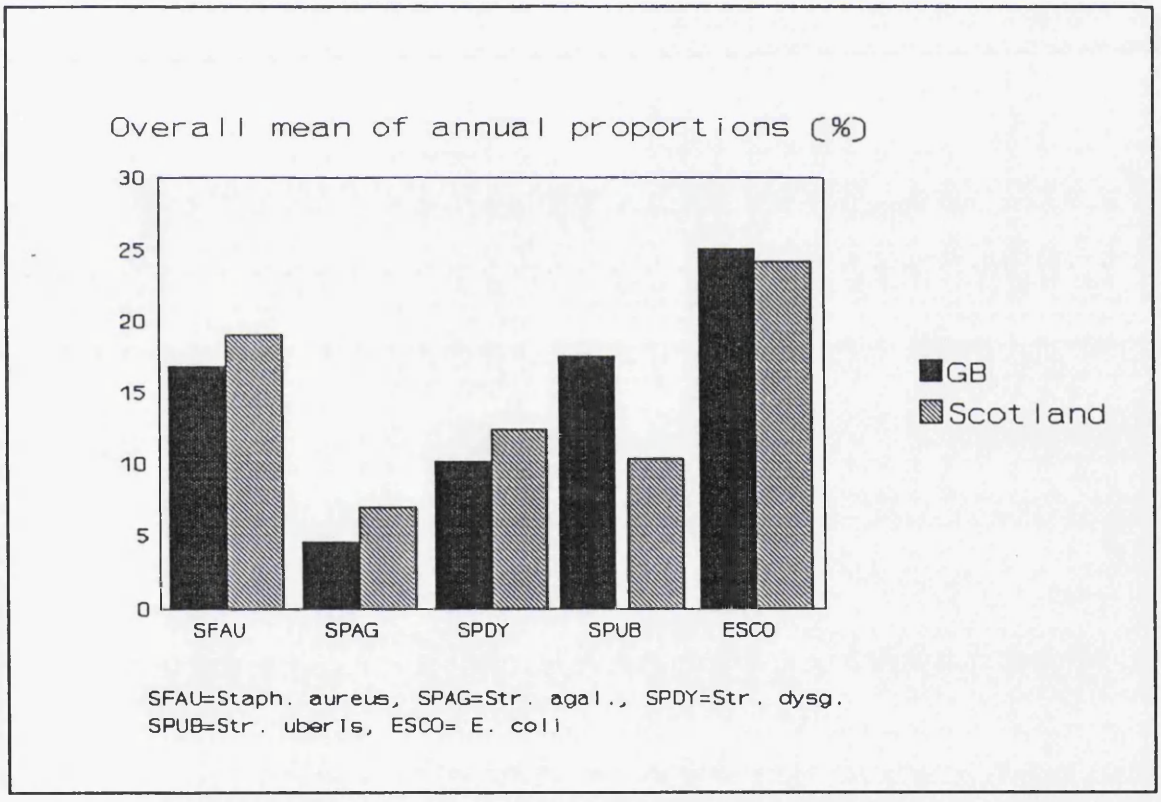

Figure 2. Mean proportion of bacterial isolations for major pathogens in milk samples in Britain and Scotland, 1980-1992.

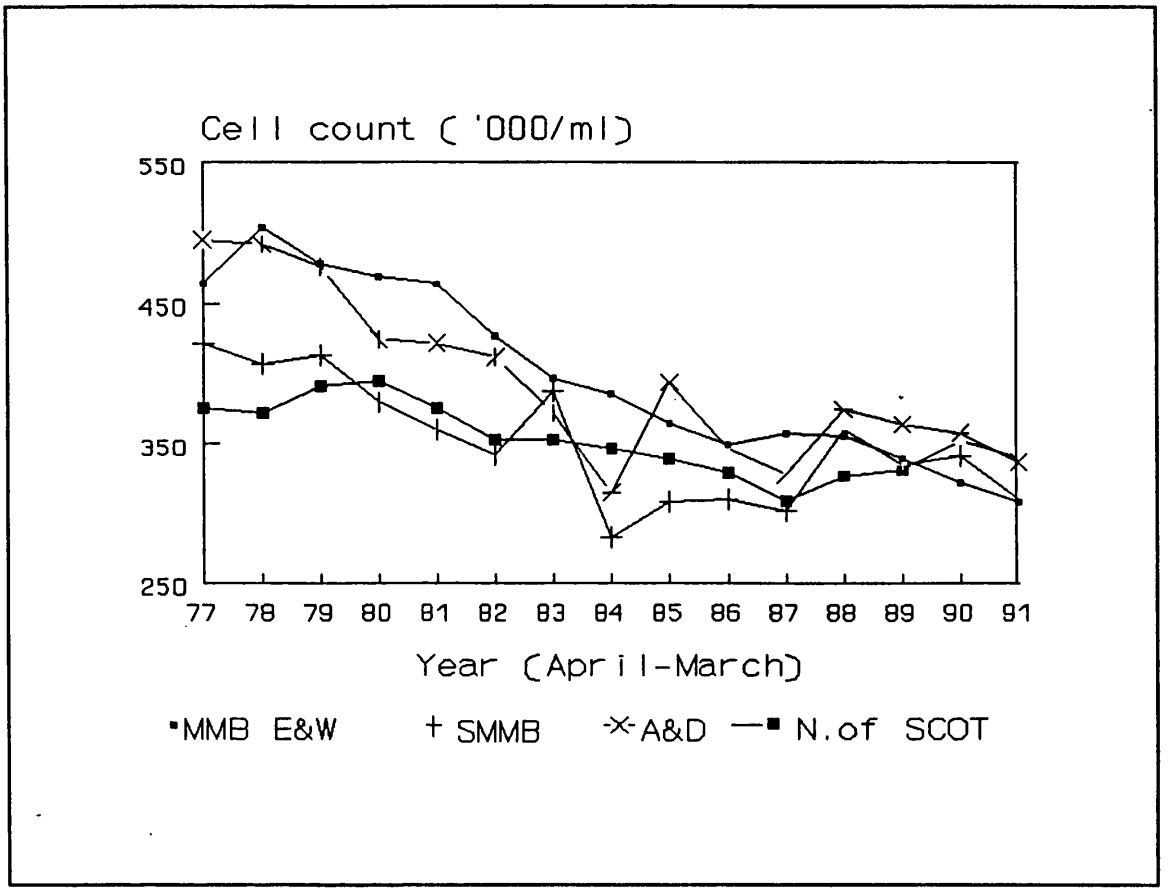

Figure 3. Changes in mean somatic cell count.

ii) Sub clinical mastitis

These are some of the questions which have been plaguing us for some time and indeed still do! The more so when at the very first of these conferences in 1988 Beck & Dodd (1) reminded the audience that the main cost of mastitis to the farmer was the level of hidden (subclinical) mastitis. However it was not until the industry realised somewhat belatedly the full implications of the first EC Directive on milk quality in 1986 that there was sufficient interest to examine this problem from a broader perspective than just trouble-shooting on an individual farm.

In attempting to define subclinical mastitis there are some advantages over the clinical disease in that a visit and bacteriological examination of milk samples can identify cases and give fairly accurate prevalence figures. Examination of data from old herd investigations by SAC Aberdeen has shown that although there were difficulties at the beginning and end of lactation there was a good correlation between the presence of infection (virtually irrespective of the organism) in the quarter and a high somatic cell count and this relationship could still be drawn for the udder as a whole (Figure 4). In other words high individual cow somatic cell counts mean an infected cow and the higher the mean cell count over a period the more likely the cow was to be infected (Figure 5). Thus it is possible to sample cows regularly

to determine their individual somatic cell count (by comparison to bacteriology this is quick and cheap) and rank them in a 'pecking order' of likelihood to be subclinically infected. Samples taken from among this group are more likely to yield a significant pathogen and so indicate which are the major subclinical pathogens acting in the herd. This information is vital in determining the best immediate control strategy.

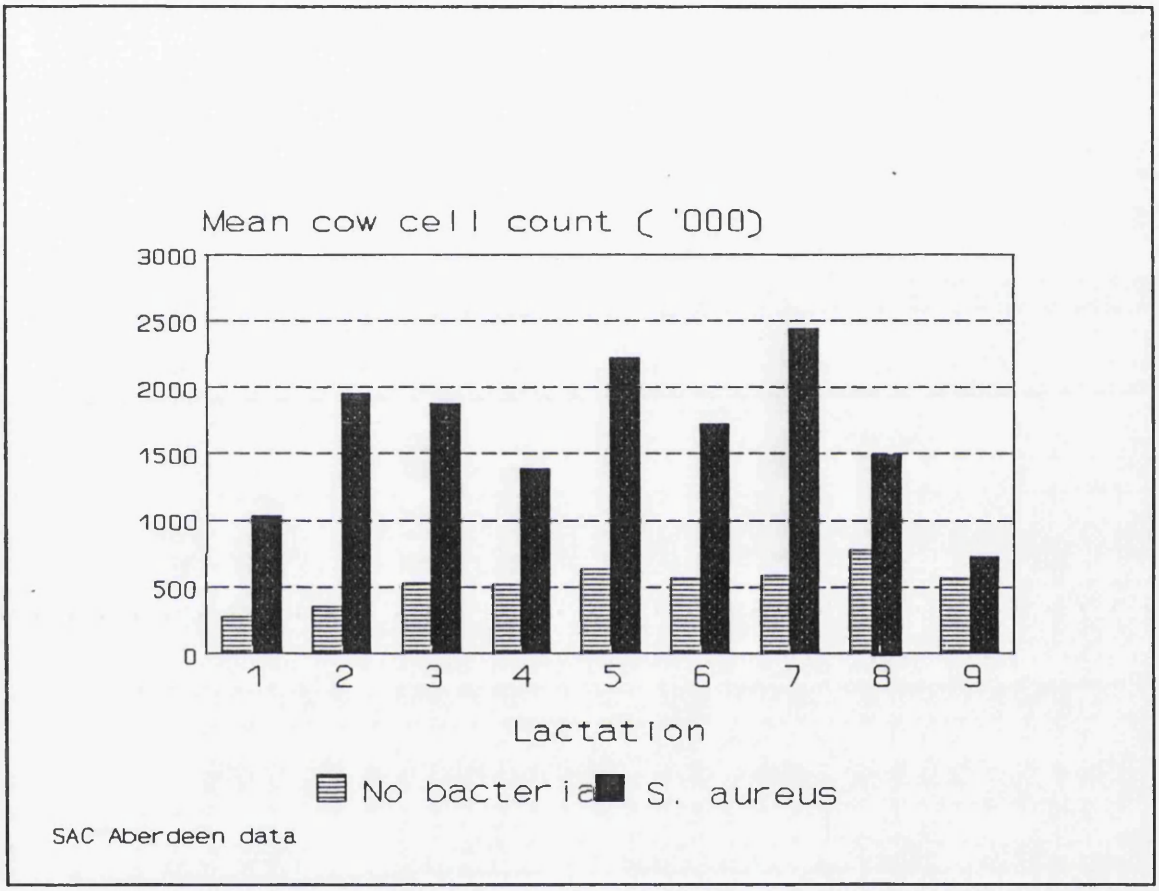

Figure 4. Effect of presence of a major pathogen (*S.aureus*) upon individual cow somatic cell count by parity.

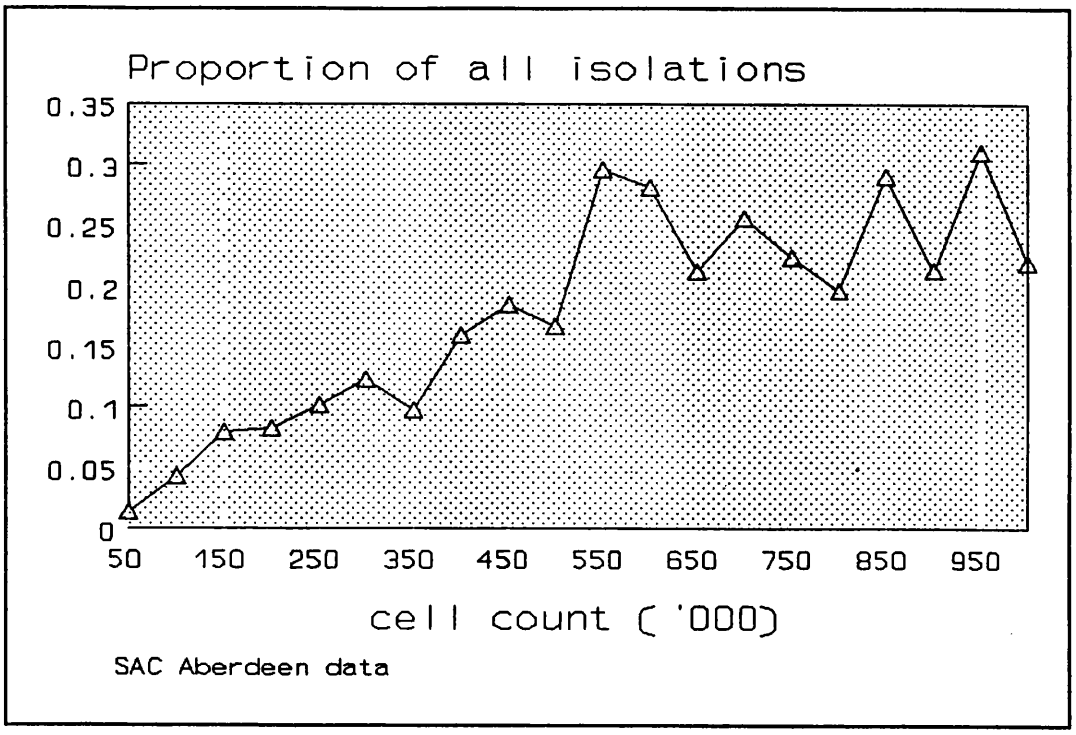

Figure 5. Likelihood of obtaining a bacterial pathogen with increasing individual cow cell count.

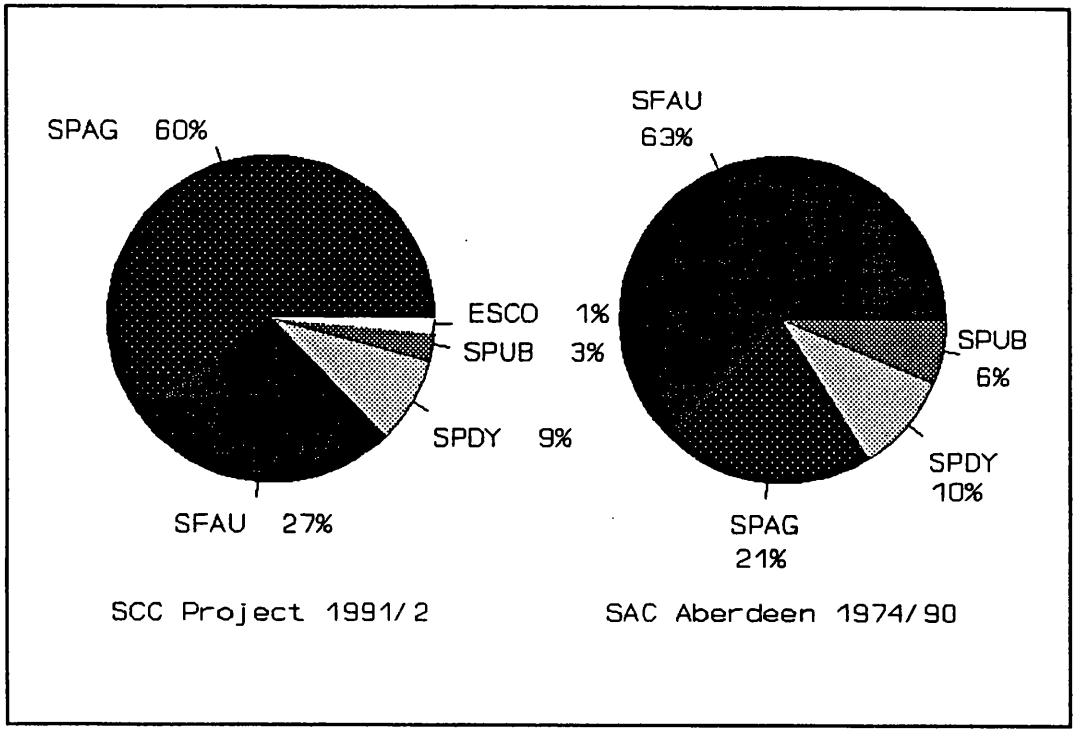

Figure 6. Frequency of isolation of different bacterial pathogens from samples taken in the Cell Count Project and by SAC, Aberdeen. SPAG - *Streptococcus agalactiae*, SPDY - *S.dysgalactiae*, SPUG - *S.uberis*, SFAU - *Staphylococcus aureus*, ESCO - *Escherichia coli*.

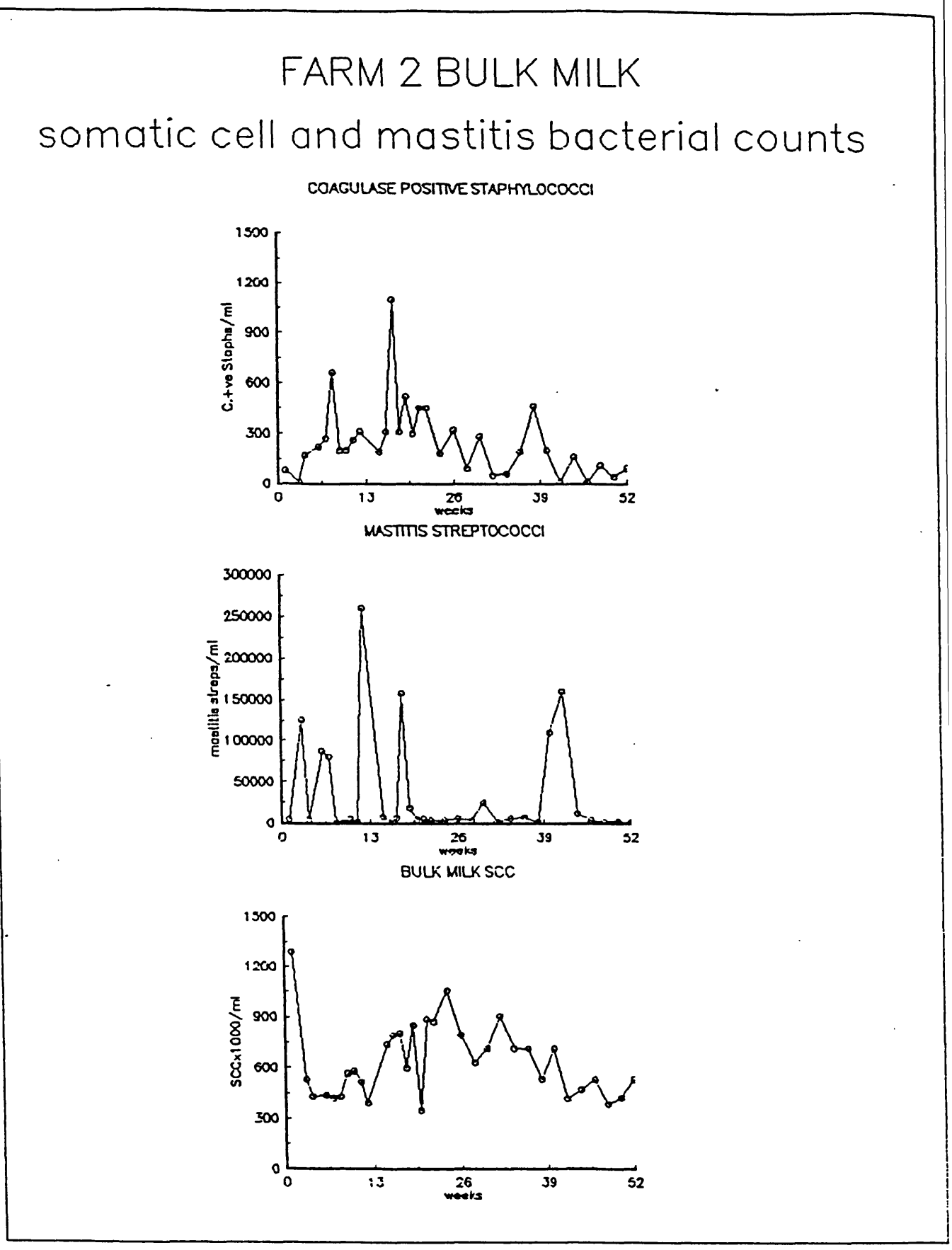

Figure 7. Variation in weekly bulk milk cell count, recovery of staphylcocci and streptococci on one farm.

Study of the Aberdeen data showed that *Staphylococcus aureus* was the most common isolate (Figure 6) however these herds were investigated because they were experiencing a 'mastitis problem' they were not selected specifically because they had a high bulk tank somatic cell count (BTSCC). The equivalent results taken from a representative group of high cell count herds identified and examined over the last two years shows that the pecking order of these major organisms has changed with *Streptococcus agalactiae* now the predominant isolate (Figure 6). It was present in 84% of these high cell count herds and accounted for 60% of all the significant isolates.

Surprisingly we could find no clear correlation between monthly herd BTSCC and Total Bacteria Count using the full database of the SMMMB. Nevertheless it was possible to use a milk sample taken from the bulk tank for culture as an 'advance' warning that mastitis-causing streptococci and particularly *Streptococcus agalactiae* were endemic. However the erratic nature of its recovery from bulk tank milk means that a negative result cannot be conclusive (Figure 7). The relatively lower numbers of *Staphylococcus aureus* shed by the infected udder mean that this organism is not reliably identified by this technique.

2. Identify the main methods of control and prevention

Since the Veterinary Investigation Centre (VIC) data indicate that almost half of all clinical cases and virtually all the sub-clinical cases in Scotland are caused by the three major so-called 'cowside' 'contagious' or 'parlour' organisms and there are now financial incentives and penalties for low and high bulk tank somatic cell counts it would seem prudent to make controlling these the highest priority. Furthermore in this endeavour we are fortunate because comprehensive measures for controlling these forms of mastitis have been developed from research which began over half a century ago - The FIVE POINT PLAN. The major problem is in persuading the farmer to apply them without using some short-cuts or ignoring them when it does not suit. We have to try to educate the farmer that success depends upon using ALL of these strategies in combination, not just what suits, and instil a COMMITMENT to the cause. Finally we must communicate a plan of action to those farms having problems; a plan which is simple, is tailored to the individual farm, states the priorities clearly and gives sensible targets. All are much easier to talk about than to do!

Unfortunately these five strategies seem to be less successful for the one third of cases of clinical mastitis in Scotland caused by the 'environmental organisms'. These have very much caught the farmers imagination, in some cases to such a degree that all thoughts of the others are ignored or certainly pushed to the bottom of the priorities list. One reason for this is the intractable nature of this latter group. Since a whole conference was devoted to environmental mastitis in 1989 and we have nothing new to add we propose to concentrate on the former save to make what might appear to some to be the facetious comment that one important method of control is to clean up the environment and look hard at your calving facilities! If you like a sixth point to add to the 5 point plan is REDUCE ENVIRONMENTAL CONTAMINATION!

Education

The very fact that so many farms have bulk tank somatic cell counts (BTSCC) in excess of 400,000 is evidence that we have not been as successful as we would have liked. But is it all the fault of the advisors? It is interesting to note that those farms in the SMMB who milk record have a lower BTSCC than the rest (Figure 8). In fact milk recorded herds are 3 times

less likely to incur a cell count penalty. We believe that much of this greater advantage stems from the greater attention to detail which these herds have learned either from hard experience or from listening to good advice (or both). The discipline of recording is something else which is very hard to impart yet it is what separates the best from the rest. Finally there is the need for tighter reproduction management which brings the veterinarian onto the farm more regularly with his attendant advice and discussion. All these are 'education factors' which should not be ignored. However the best factor is getting the farmer off his farm and on to 'good' farms to see how other farmers manage to do it better. For some reason that always makes a bigger impact than all the pontificating that this paper represents.

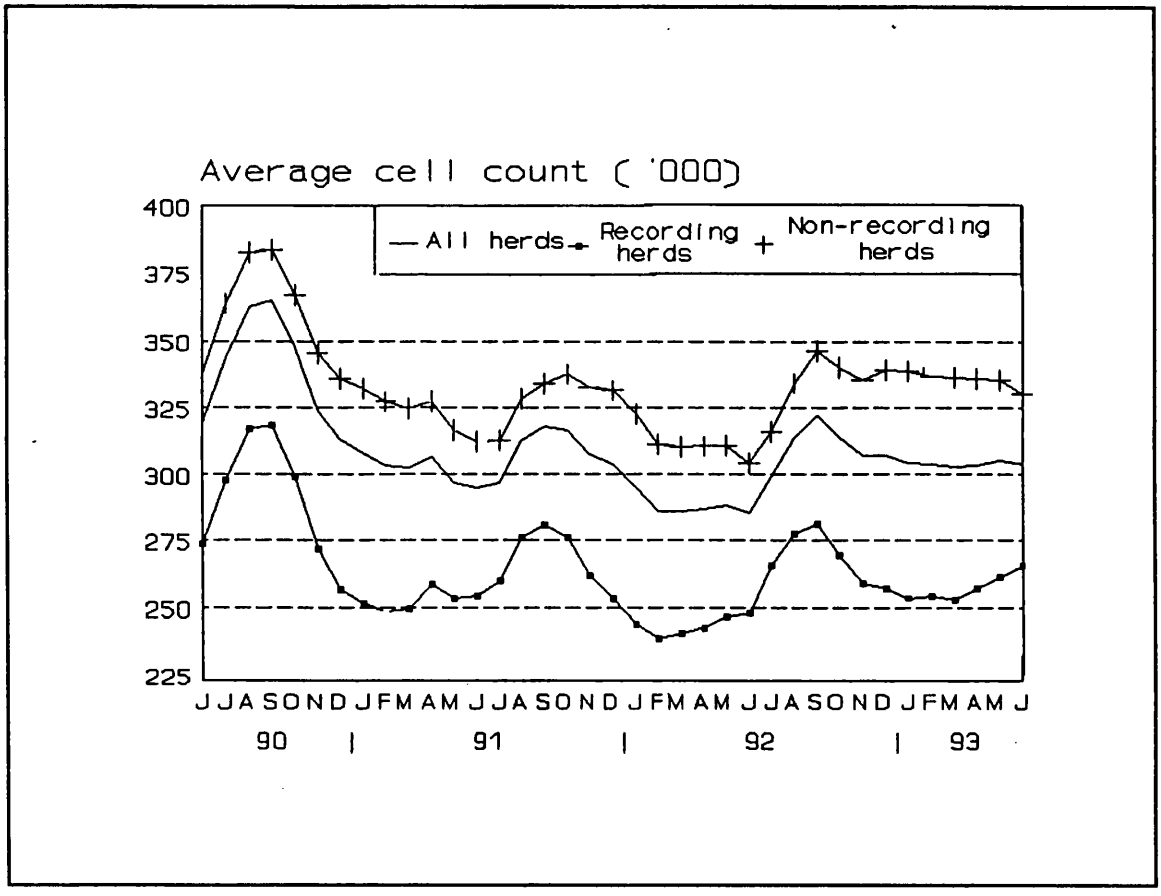

Figure 8. Bulk tank cell counts from the SMMB: Effect of milk recording.

When asked in a postal survey, the number of farmers who admitted to not following the two vitally important aspects of the 5 point plan in the control of *Streptococcus agalactiae*, post milking teat dipping and dry cow therapy, was in our opinion staggeringly high (Table 5)

Table 5. Proportion of farms failing to fully apply good mastitis control measures.

Area	Do not teat dip/spray	Do not use DCT
SMMB	44%	19%
A&DMMB	23%	8%
NoSMMB	22%	26%
Scotland total	42%	18%

[Adapted from 'The structure of Scottish milk production, 1990]

Worse, this survey did not ask whether the farms teat dipped ALL year round. Our experience is that many do not teat dip or spray in the summer. A further illustration of this failure and its importance was seen in a small survey by SAC Aberdeen and the A&DMMB comparing farms with a BTSCC greater than 400,000 (i.e. in penalty) and similar sized farms which have either had some 'borderline results' in the last year or have maintained a low value throughout (Table 6).

Table 6. Relationship between BTSCC and some management parameters.

Type of herd	High	Borderline	Low
Mean BTSCC (x10)	633	374	191
Mean annual yield	5322	6393	6278
Buy in replacements	80%	20%	10%
Post milk teat dip/spray	30%	100%	100%
Possess ACR	50%	50%	100%
Yearly machine test	20%	70%	90%
% herd >5th lactn	10%	22%	21%

The high BTSCC farms obviously do not control their management inputs as well as they should and particularly they do not disinfect teats after milking.

Commitment

The most important part of any control programme, particularly for those farms attempting to reduce their BTSCC, is COMMITMENT. We can identify the major organisms, advise treatment; milking of high risk cows last, early drying off and dry-cow therapy, checking the milking machine, and in some cases culling and so on. This is the easy part, it is the man at the sharp end who must really want to do it not just talk about it! Application of these measures to a greater or lesser extent in a small number of statistically representative farms which were given a specific advisory input (essentially one visit and regular visual updates of BTSCC and individual cow cell count [ICSCC]) has shown that progress can be made and that it was greater than in an 'unhelped' control group (Figure 9). It can be seen that at least initially the latter were also making progress so all the articles in the farming press and other advisory inputs have had a positive effect. However these mean values hide our failures.

Figure 10 shows that within the SCC Project some farms have made excellent progress and unfortunately some have not. We are still investigating the reasons for this difference but it is clear that some are more COMMITTED than others!

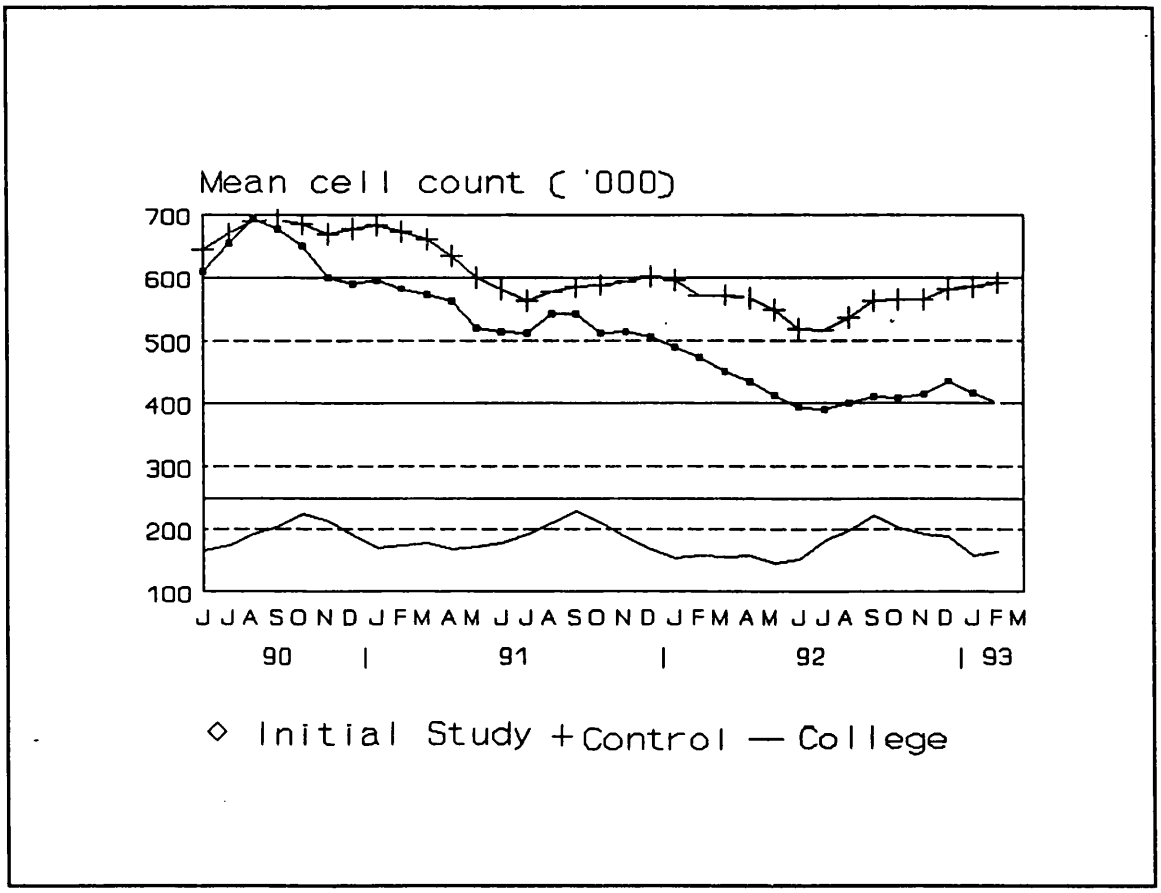

Figure 9. Progress in the Cell Count project showing changes in the bulk tank cell count for herds in the initial study and the control group (and also the SAC herds).

The main difference in the strategy used in this project over that propounded in the '5 point plan' has been the availability and application of ICSCC as means of identifying those cows which contribute most to the general level of infection in the herd and then attempting to reduce this by treating and/or culling as many as possible within the restrictions of quota and the organism(s) identified. Whereever possible these cows should also be milked last. It must also be pointed out that the higher the herd average is over 250,000 the more likely it is to incur frequent penalty (>400,000) (see Figure 11) so our target is not to get herds under 400,000 but under 200,000! It must be emphasised that treatment during lactation, in our experience, will not necessarily result in a dramatic reduction of ICSCC what we are trying to do is REDUCE THE LEVEL OF INFECTION in the herd. This will only be TEMPORARY unless the herd applies all the other strategies we have already mentioned.

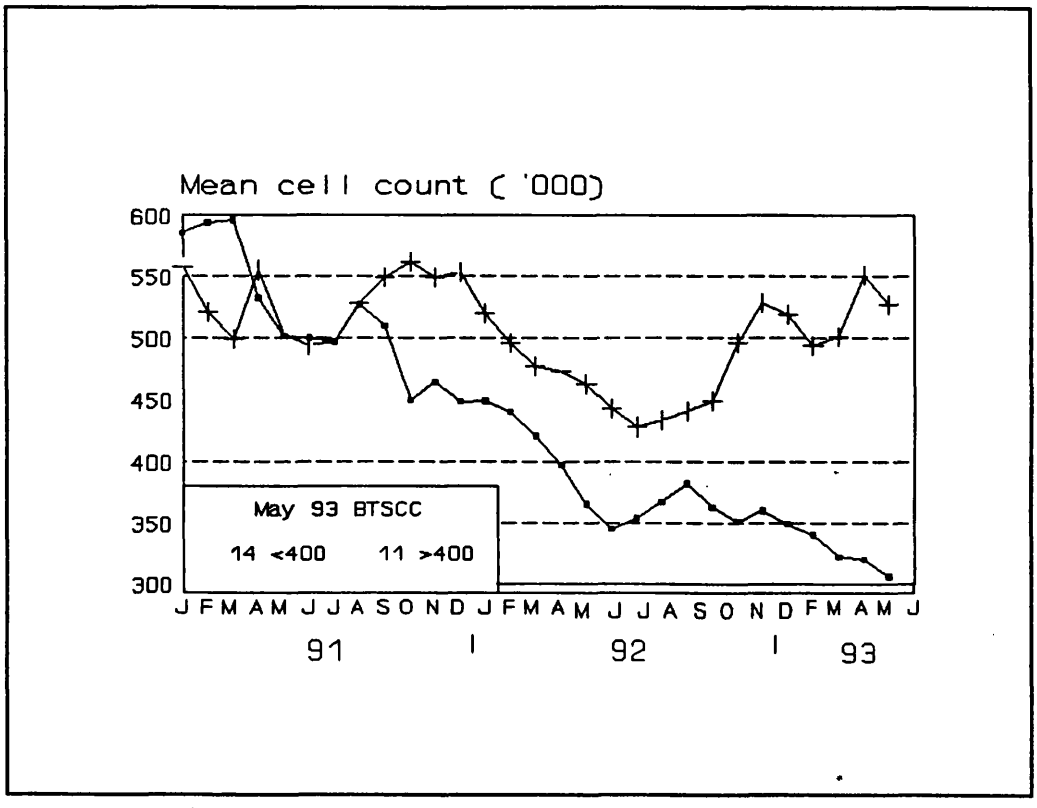

Figure 10. Cell Count Project herds, comparison of the bulk tank cell count between 14 'responding' herds and 11 'non-responding' herds.

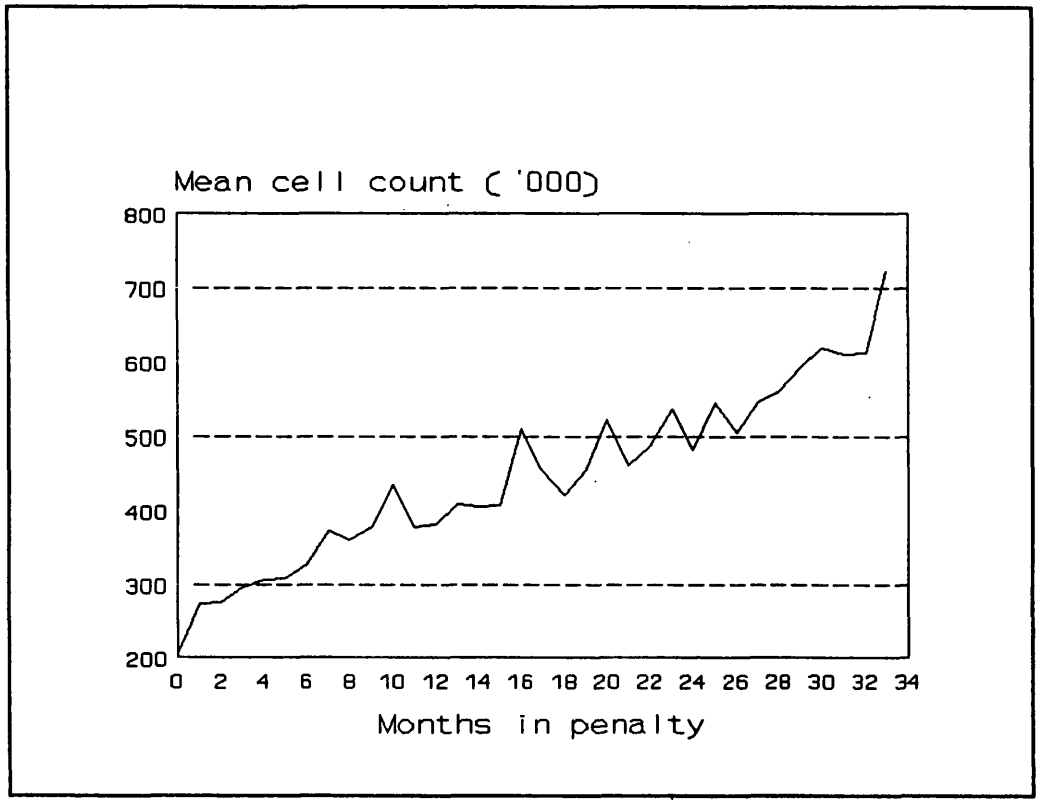

Figure 11. Mean bulk tank cell count versus months in penalty bands for SMMB herds.

Further strategies

During this study a number of the participants expressed an interest in pre-milking teat dipping and thanks to the collaboration of a commercial company we were able to allow them the use of an experimental product over the period November to January last winter. We stress that this was not a controlled trial but we took the rather pragmatic view that anything that concentrated the farm on the need for good hygiene was worthwhile. Examination of the BTSCC and TBC profiles of these few herds with the winter before and comparing them with similar 'non users' illustrated that this was probably the case though these few results are hardly a compelling argument for their widespread recommendation (Tables 7 & 8). Nevertheless it merits further more controlled study.

Table 7. Effect of premilking teat dipping on bulk tank TBC.

Comparison with same period in previous year	Better	Same	Worse
User farm	4(44%)	4(44%)	1(11%)
Non-user	4(27%)	4(27%)	7(47%)

Table 8. Effect of premilking teat dipping on BTSCC.

Comparison with same period in previous year	Better	Same	Worse
User farm	6(67%)	0	3(33%)
Non-user	8(53%)	3(20%)	4(27%)

In a similar vein we have examined whether there was any merit in giving a second treatment of dry-cow therapy, 3 weeks after drying off, to cows with a higher than average ICSCC. In this case the cows used were our own and we were able to pair the 40 cows and impose some experimental discipline but we were not able to prevent animals from being culled! The extra treatment had no significant effect upon infections caused by *Staphylococcus aureus*, nor did it significantly lower the ICSCC in the subsequent lactation, though there was a trend towards a lower figure. However there was a significant reduction in the number of 'missing' i.e. culled quarters at the end of the post treatment lactation ($p < 0.05$). Thus the general trend was that this extra treatment gave a slight advantage and we would like to repeat this with larger numbers. Smith and colleagues (3) also reported a small advantage when they gave two long-acting cloxacillin treatments with 2 weeks between finding 10.6% of staphylococcal infections persisting to calving compared to 20.3% in those treated singly. The problems in eliminating this organism are considerable. Examination of isolations at drying off and calving indicated a cure rate of just over 40% substantially lower than some reports (3,4) but still well within other estimates which range from as low as 20% (5).

These very low cure rates make it clear that we need to understand more about the relationship between this organism and our cows for it is a rare farm indeed which does not have some cows infected by it. We have just started examining isolations of *Staphylococcus aureus* from our own herds and from other herds and subjecting them to bacterial DNA fingerprinting. The results are very preliminary but we have found both within and between herd differences and similarities, for example apparently the same organism has been identified in herds as far apart as Ayr and Perth. We believe that this type of information will be needed to help us understand why this organism is so persistent and also to augment the present research into the production of a vaccine, which perhaps is not so far away now as it was ten years ago (6).

In short there is no new breakthrough, no magic injection, just confirmation that the only way to control mastitis is by hard work, application of simple principles and attention to ensure that these are correctly applied. To recap these are:

1. REDUCE THE LEVEL OF INFECTION (OR CONTAMINATION)

2. LIMIT THE POSSIBILITIES OF TRANSFER OF INFECTION

3. LIMIT THE EFFECT OF PREDISPOSING FACTORS

3. Convince the farmer

As can be seen some farmers have been sufficiently convinced to enter into a control programme with real commitment and have consequently been very successful. The problem is convincing the rest who are in cell count payment penalty bands that it is worth their while to do the same. We have made a calculation of the cost of BTSCC penalty last winter using information from the two groups of herds i.e. those given project advisory input and those without. The advantage in terms of cell count penalty alone between these two groups was of the order of £20/herd/month over last winter. Note that this ignores the higher numbers of antibiotic failures, higher TBC figures and lower milk fat and protein which we estimate could cost a further £10/month. These comparisons are not theoretical they were based on actual milk sales data and mean a loss of around £3/cow/year at present quality prices. No big deal some might say- and that is one reason why some farms are not actively reducing their sub clinical mastitis and why penalties for high BTSCC figures will continue to rise! However these same farms should note that Beck & Dodd (1) estimated halving the incidence of mastitis i.e. both clinical and sub clinical would result in a benefit of £33/cow/year. Taking all these figures together then improved control of mastitis should mean some £35 to £40 improved gross margin per cow. Of course to achieve this involves extra labour, teat-dip, dry cow tubes, and ICSCC counts but, on the plus side, there would be less treatment long-term.

These sort of figures are being continually being thrown at farmers to convince them of the need to think more deeply about a number of aspects of their management of their dairy cows and if they are all added up the figure would make one wonder if it is possible to make a profit at all! However even if those associated with the loss of efficiency are somewhat optimistic and the losses are only half of this estimate or even a third a loss of margin of £10/cow is not something to be ignored in these recessionary days.

Remember these calculations were done with last years milk quality payments. All the signs are that whoever is buying the milk this winter is going to make the financial returns for high quality milk even more attractive and of course equivalently less attractive for the lower quality and that this trend is going to continue as long as the buyers can choose their source of milk. Under commercial conditions, it takes something like two years to get the level of infection down sufficiently to be sure that there is good control of the BTSCC. Can those in penalty bands, or indeed near the borderline, afford to wait? We hope that those farmers in the audience have been convinced, and also that veterinarians and other advisors have been stimulated to try and instil a greater awareness, of the need for, and reasons behind, the present strategy of mastitis control.

Acknowledgements

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Please read the instructions overleaf then complete the abstract box below.

Commission/Session: Animal Management and Health

Title: A Study of High Somatic Cell Count Herds in Scotland : Preliminary Results

Names & Addresses of Authors: J Gunn, D N Logue & D P Arnot, The Scottish Agricultural College, Auchincruive, Ayr KA6 5HW

Abstract

Monthly Somatic Cell Counts (SCC) and Total Bacterial Counts (TBC) from June 1990 of all SMMB producers (2300) were used to examine trends and to select 23 representative producers who consistently had a Bulk Tank SCC>400,000 cells/ml. Individual cow SCC data was used to selected individual cows for quarter sampling on the basis of a 3-month Geometric Mean SCC>283,000 cells/ml (Linear Score 5 and above). Eight hundred and two (36%) significant isolations were made from a total of 2392 quarter samples of which 474 (20%) were *Streptococcus agalactiae*, 229 (9%) were *Staphylococcus aureus*, 63 (3%) *Streptococcus dysgalactiae*, 24 (1%) were *Streptococcus uberis* and 10 (0.4%) were *Escherichia coli*. Multiple isolates were recovered from 59 (2%) of the quarter samples. Thus *S. agalactiae* was found on 19 (83%) of the herds accounted for 57% of all significant single isolations, *S. aureus* for 29%, *S. dysgalactiae* for 10%, *S. uberis* for 4% and *E. coli* for 1%. Since June 1990 the average SCC for the 23 herds has decreased by 26% from 585,000 cells/ml in June 1990 to 433,000 cells/ml in Dec 1993. Adequate control measures consistently reduce the SCC, particularly in those herds with high SCC due to *S. agalactiae*.

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Commission/Session: Animal Management and Health

Title: Subclinical Mastitis in 'Problem Herds' in Scotland

Names & Addresses of Authors: D N Logue, J Gunn, & D P Arnot, The Scottish Agricultural College, Auchincruive, Ayr KA6 5HW and D Fenlon, The Scottish Agricultural College, 581 King Street, Aberdeen AB9 1UD

Abstract

A retrospective examination of data from some 58 herds was undertaken recently. These herds all in N.E. Scotland were subjected to an examination for subclinical mastitis because of either high Somatic Cell Counts (SCC), high Total Bacterial Counts (TBC) or complaints of a high incidence of clinical mastitis. Milk from every cow in the herd was examined either using a composite sample from all 4 quarters (4475 samples), or individual quarter samples (4000). The SCC of each sample was also determined and other epidemiological data noted. A pathogenic organism associated with mastitis was isolated from all herds. In particular *Streptococcus agalactiae* was found in 62% of herds. The proportion of positive samples was highest in the composite group (39%) than quarter (14%), but the decision to take composite samples was not random. In any case the relative order of prevalence of isolates was the same, *Staphylococcus aureus* was the most common accounting for over half of all significant isolates followed by *S. agalactiae* and *Streptococcus dysgalactiae*. The presence of these organisms was associated with a significant rise in SCC and also in the age of the cow. It is evident that standard preventive measures were not fully implemented on these farms.

Gunn, J (1994) 1+4

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Commission/Session: Animal Management and Health

Title: Genetic Fingerprinting of *S. aureus* from Mastitic Milk Samples

Names & Addresses of Authors: D J Platt, D Candish, Royal Infirmary, Glasgow G4 0SF and H E Pyett, I W Smith, L Sommerville, J Gunn & D N Logue, The Scottish Agricultural College, Auchincruive, Ayr KA6 5HW

Abstract

Staphylococcal mastitis is a major problem in that it is common, often subclinical and difficult to eradicate. A better understanding of the organism and its epidemiology will be required before optimally effective control measures can be implemented.

The application of DNA based methods of strain discrimination provides not only valuable epidemiological data but also the means to rationally select strains for further investigation of pathogenic mechanisms.

Strains of *S. aureus* were isolated from herds with high bulk tank somatic cell counts (problem herds) in different parts of Scotland. DNA fingerprints were generated using Hha 1 and Sau 3A digestion of *S. aureus* genomic DNA. Toxin and haemolysin production were also determined. In most individual herds there was more than one strain of *S. aureus* but also evidence of multiple cows infected with the same strain. Different strains were associated with different geographical regions. Examples of 'in herd' and 'herd to herd' analysis of strain variation on the basis of DNA fingerprints will be presented to illustrate the epidemiology together with correlation of toxin, haemolysin and somatic cell count data.

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Please read the instructions overleaf then complete the abstract box below.

Commission/Session: Animal Management and Health

Title: The Effect of Mastitis Control Procedures on Bulk Tank Somatic Cell Counts in Scottish Herds

Names & Addresses of Authors: J Gunn & David Logue, The Scottish Agricultural College, Auchincruive, Ayr KA6 5HW, D Fenlon, The Scottish Agricultural College, 581 King Street, Aberdeen AB9 1UD, D Young, SMMB Paisley PA3 1TJ and A Sword, Scottish Agric. Statistics Service, Univ of Edinburgh, Kings Blds, Mayfield Rd., Edinburgh EH9 3JZ.

Abstract

There is considerable scepticism amongst producers about the value of the major elements of mastitis control. This is especially the case for those producers in penalty wishing advice about how they might improve their SCC figures. Recently we were able to combine the raw Scottish Milk Marketing Board (SMMB) census data for Dairy Facts and Figures 1993 relating to mastitis control for all 2187 SMMB producers with the actual performance of the herds. In addition further data was acquired from the Aberdeen and District MMB comparing high SCC herds, borderline herds and low SCC herds.

The objective of the study was to compare and combine this information from a large number of herds in Scotland (total 2216) with the more limited data obtained by the two smaller but more in depth studies of subclinical mastitis in Scotland. In order to reinforce the importance and value of the major elements of mastitis control enshrined in the five point plan and thus give producers further evidence that infection with *Streptococcus agalactiae* was an important factor in many off those herds with a high SCC

In summary SMMB herds which post-milk test dipped had an average bulk tank SCC of 235,000 compared to 298,000 for those which did not. Similarly those which used dry cow therapy had an average SCC of 260,000 compared to 339,000 for those which did not. Other factors correlated with a lowered SCC figure from these studies were the importance of udder preparation before milking, in particular the use of use of individual paper towel, regular milking machine testing, breeding all replacements on the farm, and the general standard of management of the herd including culling policy.

Gunn J (1995) 180

DEFINITIONS OF QUALITY AND FACTORS AFFECTING IT: MILK HYGIENE

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ABSTRACT

The implementation of EC 92/46 will produce some difficulties for UK milk producers in particular the minority (approximately 10%) who have a bulk tank somatic cell count consistently over the accepted threshold of 400,000. Field studies in Scotland have shown that the main cause of high bulk tank somatic cell counts in herds in Scotland was due to subclinical mastitis and that the most common cause of this was a bacteria *Streptococcus agalactiae* which responds well to the major elements of mastitis control. The other common pathogen associated with high bulk tank SCC figures *Staphylococcus aureus* was more difficult to control but nevertheless the application of herd specific advice, even in this case, resulted in significant progress being made. Reduction of a bulk tank somatic cell count from above the EC standard of 400,000 to substantially below this should result in an increase in annual gross margin of over £30 per cow.

INTRODUCTION

Due to the furore created by the deregulation of milk marketing the implications of the recent EC Milk Hygiene Directive (92/46) are only now beginning to be grasped by the milk industry. This is despite the fact that these regulations were first presented in 1985 (85/397). The regulations cover the production and placing on the market of raw milk, heat treated milk and milk based products from cows, sheep, goats and buffaloes. This paper will only discuss the requirements as they affect the production of milk from the cow. Furthermore it should be realised that at the time of writing there are still discussions about the way in which these regulations are to be interpreted and enshrined in UK legislation.

There are two important areas in this Directive. The first is the specification of the general conditions for hygiene and the animal health standards of the holding. This is usually ignored in any discussion of this impending legislation yet it could cause considerable problems. The second is the standards for the raw milk itself and this has been the subject of much recent debate.

GENERAL HYGIENE AND ANIMAL HEALTH

We are fortunate in the UK to have freedom from Tuberculosis and Brucellosis. In fact this does not mean that these two diseases are unknown and there are particular problems in finally eradicating the former in some areas of the UK (Anon, 1994). Furthermore, since the removal of restrictions upon the movement of animals throughout the EC, it is more likely that any individual farm which buys-in stock could be infected with either of these diseases (or others!). The

regulations have a further 'catch-all' phrase namely; that the cattle will not show any 'symptoms' of infectious diseases communicable to man through milk. At present we must assume that the authorities will interpret this as has been done in the past. However we are only too aware that this might not be the case in future and recently there have been a number of studies in Scotland examining the interaction of *Escherichia coli* O157 (Synge et al., 1993), *Listeria monocytogenes* (Low *et al.*, 1993), *Staphylococcus aureus* (Platt *et al.*, 1994) and *Salmonella typhimurium,* in particular phage types 204c and 104. (Platt and Smith 1991, Hunter J pers. com. 1994), and *Leptospira hardjo* (Logue 1992) with the domestic ruminant especially the cow.

The regulations also require that the cow should have a general state of health unimpaired by any 'visible disorder' and which 'are not suffering from any infection of the genital tract with discharge, enteritis with diarrhoea and fever or inflammation of the udder'. Again it is the interpretation which could present a problem. Lameness, mastitis and vulval discharge are very common conditions of the dairy cow. In very round figures each affects approximately one fifth of the dairy cows in the UK annually though obviously not all of these cases will be severe (Esslemont and Peeler 1993). While the incidence of each of these conditions is not independent of the others the correlation's are not high. Therefore potentially around one half of the dairy cows in the UK could be removed from milk production at some time during their lactation. Finally it is a requirement that all animals are 'clean and well kept' and that the holding has the 'capability of isolating infected animals'. It can be seen that these aspects of the regulations have some far reaching implications and will more directly affect the majority of our producers since approximately 80% regularly comply with the raw milk standards (Booth 1994). However it is the raw milk standards which have caused the greatest misunderstanding and controversy.

RAW MILK STANDARDS

The raw milk standards require that the milk contains neither any added water, nor any residues above defined maximum levels. Leaving aside the reduction in maximum residue levels for penicillin, which have been effectively halved, the main thrust of the standards has been in attempting to improve the 'hygienic' standard of the milk being collected from producers. The two simple parameters used are the 'plate count at 30°C (per ml)' or 'total bacteria count' (TBC) and the 'somatic cell count' (SCC). The standards are shown in Table 1.

Table 1. Raw milk standards.

Criteria	"Drinking milk" + lesser manipulations	Until 1998 for "major manipulations" e.g. cheese
Cell Count (per ml) (SCC)	400,000[1]	500,000
Plate Count (per ml) @30^{0}C (TBC)	100,000[2]	300,000

[1] 3 month geometric mean [2] 2 month geometric mean

In order to encourage producers to meet these standards all the UK boards and now buyers of milk have systems of premiums and penalties. In fact the EC standard for TBC has little or no impact on herds in the UK with less than one producer in a thousand failing to meet this standard and that usually only temporarily. Furthermore progress has been inexorable with the average TBC in the UK falling from almost 25,000 to under 15,000 over the last decade (Booth 1994). However the SCC threshold does have a major impact upon the UK producer with milk from over a fifth of producers failing to meet the standard in March 1991 when all the MMBs in the UK introduced penalties and premiums for this parameter. This is illustrated by the Scottish figures in Table 2 which mirror those of the UK as a whole (Booth 1992).

Table 2. Bulk tank SCC distribution of Scottish dairy herds: March 1991

BTSCC band*	SMMB	A&DMMB	NOSMMB
Less than 250,000	1294 (47%)	61 (40%)	53 (51%)
250-400,000	835 (31%)	54 (36%)	25 (24%)
401-600,000	335 (13%)	26 (17%)	17 (%16)
More than 600,000	195 (7%)	11 (7%)	9 (9%)

* based on a 3 month geometric mean

We were fortunate that the three Milk Marketing Boards in Scotland, the Scottish (SMMB), the Aberdeen and District (A&DMMB) and the North of Scotland (NOSMMB) recognised the need for 'hands on' experience of this problem and commissioned some research into the situation in Scotland. It is this data which will be primarily used to illustrate the major importance of subclinical mastitis particularly in relation to elevated bulk tank SCC figures but also to some elevated TBC figures as well.

THE TOTAL BACTERIAL COUNT

This parameter is a very direct measure of the extent of contamination of the milk counting the number of contaminating bacteria in the milk expressed as numbers per ml. The importance of this contamination in relation to the keeping qualities of the milk was recognised as long ago as 1952 when the reazurin test for the hygienic quality of bulk milk as it left the farm was introduced in the UK. This was replaced by the TBC in 1982 and at present almost 80% of producers in the UK produce milk with a TBC below 20,000-well below the EC standard (Booth 1994). There are three major sources of the bacteria in the bulk tank milk:

1. Dirt on the teat etc. gaining entry at milking
2. Inadequate washing and cleaning of the milking machine between milking.
3. Milk from a cow with mastitis excreting a large number of pathogenic organisms.

All of these sources can be further multiplied if the storage of the milk is poor. However within the UK there are fairly stringent requirements for the cooling capacity of the bulk tank and so milk will not be collected by the tanker if it is not below 4°C (or very close to this).

A survey by Jeffrey and Wilson (1987) in the A&DMMB found a large proportion of mastitis pathogens in the TBC of the bulk milk tank samples which had figures greater than 45,000. They estimated that mastitis was the main cause of the elevation in 43.8 % of the cases of high TBC . In the majority of these cases the major pathogenic bacteria were identified as being mastitis causing streptococci species (Jeffrey & Wilson 1987). This figure was remarkably close to the ADAS estimate of 45% of high TBC problems being due to mastitis (Marshall 1991). More recently the relationship of the TBC of the bulk tank in herds with a high somatic cell count has been studied in the same population of 150 herds (A&DMMB) (Fenlon *et al.*, 1994). This work has shown that the level of mastitis streptococci was much greater in herds with a high SCC (>400,00) than in either borderline herds (mean SCC figure 374,000) or the low (mean SCC figure 191,000). This study has also shown that there was a high correlation (r^2 = 0.827) between the streptococcal count and the SCC. In exactly half of these cases the pathogen *Streptococcus agalactiae* was identified. There was less of a correlation between the other major pathogen *Staphylococcus aureus* and SCC (r^2=0.686). This was not altogether surprising given that, in high SCC herds primarily affected by either one of these bacteria, the maximum number of *S. aureus* identified in the bulk tank milk was some 10 times less than for *S. agalactiae.* In other words in those herds with a high SCC and a high fluctuating TBC figure (20,000 to 100,000) are quite likely to be infected with *S. agalactiae* and this may be identified by examining the bulk tank milk (Fenlon *et al.*, 1994). In fact as a result of investigations into affected herds it can be shown that one cow can excrete sufficient *S. agalactiae* to raise the TBC over 50,000 (Fenlon pers. com. 1991). Thus examination of bulk tank milk can only be seen as a prelude to a more intensive examination of individual cows in the herd.

In summary both the TBC and SCC are frequently elevated by subclinical mastitis.

However it is the latter parameter which is more directly affected by this disease.

THE SOMATIC CELL COUNT

It has been recognised for almost a century that normal milk contains a variety of cells (Prescott and Breed 1910). Early studies involved identifying and counting these cells in stained smears of milk examined under the light microscope. Thus Blackburn *et al.*, (1955) correctly identified the presence of considerably more than normal numbers of polymorphs in the milk of cow with mastitis. However they had real difficulty defining the origin of many of the other cells. For example macrophages were commonly identified as epithelial cells for quite some time (Jensen and Eberhart 1974). It is now accepted that the major cell types in normal milk in order of magnitude are macrophages, polymorphonuclear neutrophils, T & B lymphocytes and epithelial cells (Fitzpatrick 1992). The proportion of these cells, in particular the polymorphs and to a lesser extent the macrophages, varies with the physiological status of the cow. Thus in early and late lactation and in infection these two are excreted in larger than normal numbers and contribute to the well described changes in SCC with stage of lactation (Wood & Booth 1983, Fitzpatrick 1992).

Clearly SCC counts by smear examination would not be practical method of monitoring a large number of herds. It was not until electronic counting became widely available in the 1970s that the use of SCC figures as advisory tools and a method of assessing milk quality began to be seriously considered. Standard methods were recommended to the International Dairy Federation and are still being revised and refined (Heeschen and Ubben 1994). At present there are two major electronic techniques for somatic cell counting, the Coulter Counter and the Fossomatic. It is important to realise that both utilise different principles in counting. The Coulter count is based on particle size and has to be calibrated to count fixed somatic cells. The Fossomatic is an automated microscope and counts flourescently stained cells in a small drop of test material on a rotating disc. The latter system facilitates automation very readily and is the most widely used technique for large numbers of samples. Although there are standard methods and there has been a regular quality control of milk laboratories in the UK through the MMB of England and Wales the situation following deregulation is not yet clear. There are therefore potential problems ahead if farmers start to question figures and ask different laboratories to examine duplicate samples (Heeschen and Ubben 1994). The cell count can be performed on milk from three sources, the bulk tank, the cow i.e. a composite of all four quarters and the quarter itself.

Quarter somatic cell count: The quarter cell count (QSCC) is the most accurate way of monitoring changes in the udder as the sample will be unaffected by the mixing of milk from the other quarters. A quarter cell count threshold of 500,000 has been suggested as being indicative of subclinical mastitis particularly if it was accompanied by the bacterial isolation of a known pathogen (Griffin Morant and Dodd 1987). Study of the results from a number of herd

investigations conducted between 1974 and 1990 by SAC Aberdeen has allowed the analysis of some 5860 quarter SCC figures.

Table 3. Proportion of significant bacteria isolated with increasing quarter SCC (QSCC)

Isolate	SCC<283,000	SCC 283-566,000	SCC>566,000
Number examined	3309	922	1576
No significant isolate	97%	90%	61%
Significant isolate	3%	10%	39%

This study has shown unequivocally that the higher the SCC the more likely one is to isolate a mastitis pathogen but that one can isolate potentially pathogenic bacteria from low SCC quarters (Table 3).

Table 4. Effect of age and presence of *Staphylococcus aureus* upon mean quarter SCC (QSCC)

		Mean QSCC*	
	Lactation 1	Lactation 3	Lactation 7
S. aureus isolated	661,000	3,040,000	3,178,000
No significant isolate	147,000	193,000	265,000

* Based on log transformed data

In addition it can be clearly seen from table 4 that at the level of the quarter the presence of a pathogenic bacteria is a much more important influence than age. Furthermore it is our belief that the rise seen with age in the samples with no significant isolate is due to failure to detect a pathogenic organism or a 'non-specific' reaction of the cow to a previous infection (or both). In other words a normal QSCC should be considerably less than 400,000.

Individual cow somatic cell count: Unfortunately the collection of quarter milk is not very practical. However in Scotland since 1989 individual cow somatic cell counts (ICSCC) could be done on the same milk sample taken for fat and protein estimation. Despite the problem of the mixing of the milk from all 4 quarters evidence from North America indicated that the ICSCC presented a tremendous opportunity to identify cows affected by subclinical mastitis. Ali and Shook (1980) reported the need to use log transformed figures in analysis of such data and this has developed into a 'linear score' based on a $\log_2$ scale and been adopted by the National Co-operative Dairy Herd Improvement Program. (Jones 1986). In fact the threshold used in table 3 are based on this transformation with the range of 283,000 to 565,999 being Linear score 5. Our own observations based on further SAC Aberdeen data involving 5416 composite milk samples (i.e. from all milking quarters of the cow), over the same period as before, confirm

that with care the ICSCC can indeed be an extremely useful tool. In the first instance, as with the quarter SCC data, there was ample evidence that infection with a 'pathogenic' bacteria was directly related to an increased SCC (see Table 5).

Table 5. Effect of *Staphylococcus aureus* and month of lactation upon individual cow SCC (ICSCC)

	Mean ICSCC* for that month of lactation			
	1	2	5	10
S.aureus isolated	468,000	555,000	750,000	1,180,000
No significant isolate	172,000	119,000	190,000	373,000

* Based on log transformed data

Furthermore, as with the quarter SCC, the presence of such a bacteria far outweighed other influencing factors such as the stage of lactation and age (Logue *et al.*, 1993). Table 5 also shows that in infected cows there is a strong tendency for the SCC to rise more quickly as drying off approaches. The Aberdeen database also provided an opportunity to examine the effect of restricting bacteriological investigation to cows with ICSCC above a given threshold. Table 6 shows the proportion of the major pathogens grouped by whether the cell count was less than linear score 5, linear score 5 or greater than linear score 5. The limits of Linear Score 5 band of 283-566,000 were chosen for investigation because this was a recognised scale for the interpretation of SCC data, fell within our spectrum of 'normal' as defined by the quarter SCC study, was of the same order as the 250,000 for quarter SCC advised by Griffen *et al.*, (1987), and provided a margin round the EC limit of 400,000. Furthermore only 10% of samples with an ICSCC below 283,000 yielded significant isolates compared to 47% from those with greater than that. A significant pathogen was isolated in 58% composite samples with an ICSCC in excess of 566,000 i.e. LS6. This analysis showed that sampling high ICSCC cows was likely to yield a greater percentage of 'significant' isolates ($p < 0.001$) but that low ICSCC cows could yield a significant isolate.

Table 6. Proportions of significant mastitis pathogens isolated by SAC Aberdeen.

	Samples (%) by category			
	Individual Cow SCC (ICSCC) expressed in thousands			
	All	ICSCC<283	ICSCC 283-566	ICSCC >566
No sig isolate	69.89	88.33	68.1	39.23
Sig isolate	26.34	10.33	26.94	53.51
S.aureus	15.46	7.12	17.03	28.87
S.agalactiae	8.36	1.94	8.08	19.55
S.dysgalactiae	1.38	0.53	1.08	3.03
S.uberis	0.94	0.67	0.75	1.51
E.coli	0.20	0.007	0	0.55

Although the bacteriological investigation of each herd was extensive, further epidemiological data was not available. At the herd level, BTSCC data would have allowed correlation with the prevalence of infection and thus a direct comparison with the findings of Pearson *et al.*, (1972) and Pearson & Greer (1974). However, when the median ICSCC results from 5212 cows in 55 Aberdeen herds were used as an estimation of the herd BTSCC the cow infection prevalence for the BTSCC range less than 500,000 was 26%, 500,000 to 1,000,000 was 33% and greater than 1,000,000 was 65%, these results were in excellent agreement with Pearson & Greer (1974).

In summary this study showed that it would be possible to screen cows by ICSCC and by only sampling those with an elevated SCC increase the efficiency of bacteriology and yet still achieve a reasonable estimate of the relative proportions of the major bacteria causing subclinical mastitis on any particular farm.

Bulk tank somatic cell count: In 1975 Booth warned that there was an increasing acceptance in Europe that the bulk tank somatic cell count was a measure of the quality of the milk. This has now become a fact. Unfortunately a single BTSCC is not a reliable measure of herd infection though it can be improved by averaging a number of counts (Wilson & Richards, 1980, David and Jackson 1984). The national cell count in the UK has shown two periods of marked fall, in 1975-1976 and in 1983, both attributable to increased culling of cows (Logue *et al.*, 1993). At present approximately two-thirds of UK herds fall within the range 200-599,000 cells/ml (Booth, 1992).

Analysis of the bulk tank somatic cell databases of the three milk marketing boards in Scotland by Gunn (1991 unpublished) showed that, like the rest of the UK, herds with a high BTSCC were a sizeable problem. Counts as high as 1,430,000 cells/ml have been recorded in Scotland last year (Gunn 1994 unpublished). Study using the "Contribution Index" developed by Schukken *et al.*, (1992) showed that persistently high SCC herds in Scotland were found to make

a significant contribution to the overall SCC and thus, in contrast to the findings of Schukken *et al.*, (1992) in Ontario, could not be ignored (Gunn 1994 unpublished).

High somatic cell count herds: Analysis of the entire SMMB dataset for BTSCC over the past 4 years has showed an almost linear relationship between the annual mean BTSCC and the number of months continuously in penalty. This analysis also showed that an annual mean BTSCC performance of less than 250,000 was required to completely avoid being in excess of the standard at some time in the year. This was especially the case where a tight calving pattern emphasised the physiological SCC rise caused by a greater preponderance of late and early lactation cows (Logue *et al.*, 1993). Using this data (Gunn 1994 unpublished) selected an initial 27 herds with a consistently high BTSCC. Two subsequently stopped producing milk during the study and have been ignored. The remainder were the subject of 33 herd investigations. These farms were visited and assessed for mastitis control. Bacteriological examination of quarter milk samples from all cows with a 3 month geometric mean in excess of 283,000 was undertaken. These findings permitted herd specific advice about mastitis control. The effectiveness in control was then monitored using the monthly BTSCC and TBC data and reported to each individual producer at periodic intervals (approximately quarterly) in a graphic form.

Examination of the distribution of the bacteria isolated showed that, as with the Aberdeen data, *S. agalactiae* and *S. aureus* were the most prevalent mastitis pathogens in high BTSCC herds. *S. agalactiae* was found in 83% of the original herds. It also accounted for 57% of the significant mastitis pathogens isolated from cows with lactation mean ICSCC in excess of 283,000 as compared to 29% *S. aureus*. This was the converse of the SAC Aberdeen data and illustrates that these high somatic cell count herds are a discrete entity (Logue *et al.*, 1993). Indeed 22 years ago in N. Ireland Pearson *et al.*, (1972) described isolating *S. agalactiae* from 48% of high BTSCC (annual mean > 1,000,000) herds and none in the low herds (< 300,000). A re-examination of those herds which failed to make substantial progress (and were still over 400,000) after approximately 1 year showed a change in distribution with *S. aureus* becoming the most prevalent mastitis pathogen (Logue *et al.*, 1993). However *S. agalactiae* was still far too frequent an isolate given that most had treated all *S. agalactiae* carrier cows during lactation and claimed to have treated all cows with dry cow therapy. In other words despite the reduction in the level of infection of *S. agalactiae* the deficiencies identified in the mastitis control programme even at the second visit, particularly attention to detail in milking routine, resulted in a failure to contain spread from those animals which were not cured This meant a continuation of the *S. agalactiae* infection and a relative increase in *S. aureus* which is less responsive to antibiotic.

In a second study the BTSCC profile of herds which moved below 400,000 when premiums were introduced but then exceeded this figure after January 1992 allowed a small investigation of what factors, if any, caused this short-term improvement. As in the first group *S. agalactiae* was the most prevalent mastitis pathogen found in an in-depth investigation of 4 of these herds. The

proportion of isolates were not significantly different from the original or 'initial' group and there was no evidence that either their mastitis control practices or herd management were different. It would appear that initially these herds merely culled 'problem cows' and succeeded in temporarily lowering their BTSCC but in failing to alter their inadequate mastitis control measures they also failed to maintain their position.

The study of these various high BTSCC herd investigations and comparison with 4 herds with a low BTSCC (<250,000) failed to find any unique factor influencing the BTSCC apart from the need to reduce the level of infection and to apply the recommended mastitis control programme as completely as necessary. These studies showed that a strategy emphasising the most important points based on the herd ICSCC profile and major pathogens will reduce the BTSCC more quickly than merely general advice to apply the 'Five Point Plan'. There was a highly significant reduction ($p < 0.0001$) in the group mean BTSCC of the initial herds which fell from 605,000 in 1990/91 to 414,00 in 1993 with over half being no longer over 400,000. This average was considerably lower than their contemporaries, who had a final figure of just under 600,000 (Gunn 1994 unpublished). All these herds (i.e. studied and contemporaries) were also the recipients of considerable general advice through articles in the farming press, meetings by SAC and the three MMBs in Scotland and on-farm advice through a number of agencies including their veterinarian. We are not denigrating such advice indeed we have unequivocal proof based on a survey of just over 2,000 farms that it is of great value. This survey showed that, in order of importance, dry cow therapy, the presence of a good recording system, post milking teat dipping and regular testing of the milking machine were all associated with a lower BTSCC (Gunn *et al.*, 1994). The need for such specific advice in high BTSCC herds to enable them to make rapid progress is further highlighted by the particular problems which *S. aureus* presents. This organism is very difficult to effectively treat either during lactation or in the dry period thus control, while maintaining quota, can be difficult (Logue *et al.*, 1993). The finding of different *S. aureus* genotypes both within and between farms, using DNA fingerprinting techniques, has indicated that there may be an underlying difference in *S. aureus* pathogenicity but these studies are very preliminary (Platt *et al.*, 1994).

Economic loss: Unfortunately at present the various penalty and premium schemes built around these hygienic standards are not indicating to producers the relative importance of the BTSCC. For example in most structures exceeding the EC threshold for TBC will result in 10 times the penalty cost per litre of milk than the BTSCC. It was calculated by Logue *et al.*, (1993) that the difference in milk quality payments between the assisted herds and their contemporaries amounted to plus £3/cow (in the herd) per year. However that does not take into account other gains most particularly the increased efficiency of milk production as a result of less mastitis. Based on figures calculated by Beck and Dodd (1989) it was estimated these herds were probably gaining by the order of £33/cow (Logue *et al.*, 1993). Subsequent independent case studies in

Scotland calculated that by reducing the BTSCC of the herd from 600,000 to just under 400,000 the herd should result in an increase in gross margin of £34 /cow (Treacey 1994). These figures are of the same order of magnitude as the range of £29 to £84/cow quoted by a number of authors (Pearson *et al.*, 1972, Lucey *et al.*, 1986, and Esslemont and Peeler 1993).

Thus assuming an average herd in Scotland the cost per annum at present is of the order of several thousand pounds and this is likely to become greater. Investment now in the proper control of subclinical mastitis will not only protect the long term viability of the business but should pay for itself handsomely as the buyers of milk strive to acquire the highest quality product that they can.

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A STUDY OF MASTITIS BACTERIA AND HERD MANAGEMENT PRACTICES TO IDENTIFY THEIR RELATIONSHIP TO HIGH SOMATIC CELL COUNTS IN BULK TANK MILK

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SUMMARY

Thirty dairy herds, selected to cover a wide range of bulk tank somatic cell count (BTSCC) values, were used to study the relationship between the levels of the principal species of mastitis-causing bacteria, herd management practices and the BTSCC. A good correlation was found between the number of mastitis streptococci (*Streptococcus agalactiae, S. dysgalactiae* and *S. uberis*) found in bulk tank milk and the BTSCC. *Staphylococcus aureus* was less significantly correlated to BTSCC, but was of increasing importance in borderline BTSCC herds, where lower excretion levels into milk were unlikely to trigger hygiene penalties and so alert producers to the presence of a significant mastitis problem. High BTSCC herds had significantly lower yields and were less likely to use a post-milking teat dip or to have a regular programme of milking machine maintenance or automatic cluster removal. These herds also tended to buy in replacements rather than breed their own. Overall the management of high BTSCC herds showed less commitment to implementing mastitis control procedures than herds with a consistently low BTSCC.

KEYWORDS: Mastitis; somatic cell counts; *Staphylococcus aureus; Streptococcus* spp; herd management.

INTRODUCTION

In a series of Directives, 85/397/EEC and 92/46/EEC, the European Community has adopted the somatic cell count (SCC) as one of the basic measurements of milk hygienic quality for intracommunity trade (United Kingdom Dairy Facts and Figures, 1993). In order to encourage producers to meet the EC standard of < 400 000 cells ml^{-1} in milk for human consumption, all the United Kingdom milk

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marketing boards (MMB) have incorporated bulk tank somatic cell counts (BTSCC) as a component of their milk quality payments. Premium and penalty payments are calculated on the basis of the 3-month geometric mean of weekly BTSCC measurements. Together with the pre-existing hygienic payments based on a standard for total bacterial count (TBC), dairy herds are now facing increased financial pressure to produce milk of high hygienic quality.

These two measures of milk hygienic quality are frequently adversely affected by the same factors. In particular a common cause of high TBC is the presence of large numbers of mastitis bacteria excreted into the milk by subclinically infected cows, which also produce large numbers of somatic cells. A survey by Jeffrey and Wilson (1987) in the Aberdeen and District Milk Marketing Board (ADMMB) region found that a preponderance of mastitis bacteria caused an elevation of TBC in 43.8% of bulk milk samples which consequently incurred hygiene penalty. The majority of these mastitis bacteria were identified as streptococcal species (Jeffrey & Wilson, 1987). It was also recognized that the percentage of infected cows increased with BTSCC elevation (Jones *et al.*, 1984). In Scotland, *Streptococcus agalactiae* was found to be the most common subclinical mastitis infection causing high BTSCC (Logue *et al.*, 1993).

The objectives of the present study were firstly to confirm that many producers with high BTSCC also had difficulty in consistently meeting the TBC standard due to contamination of milk by mastitis bacteria, and secondly to determine the main bacterial 'contaminant' in milk in herds with BTSCC figures around the EC standard of 400 000 ml^{-1} which rarely incur hygiene penalties. Quantitative bacteriology using selective media enumerated the significant mastitis pathogens within the bacterial population of bulk tank milk samples. These data were combined with BTSCC and herd management information in an analysis of factors associated with elevated BTSCC.

MATERIALS AND METHODS

The 12-month study began in May 1990 and covered the period when producers were first informed that a payment penalty scheme based on SCC would be introduced by the ADMMB. Thirty producers were selected by the Board on the basis of their arithmetic mean BTSCC for the previous 12 months (June 1989–May 1990) to cover a range of cell counts, designated 'low' (<250 000 cells ml^{-1}), 'borderline' (250–450 000 cells ml^{-1}) and 'high' (>450 000 cells ml^{-1}). At weekly intervals, later reduced to fortnightly, the latest routine bulk milk samples collected by the tanker driver at every collection from the 30 study herds were taken from overnight refrigeration at 4°C at the Board's laboratory. The BTSCC figures used in this study were those determined in the appropriate week by ADMMB for the production of a rolling geometric mean.

The bacteria identified and enumerated for the purpose of this study (*Staphylococcus aureus, Streptococcus agalactiae, S. dysgalactiae* and *S. uberis*) were recognized to be significant mastitis pathogens (Bramley & Dodd, 1984; Jeffrey & Wilson, 1987). Mastitis streptococci were counted on a specific streptococcal agar, using a pour plate technique on 10-fold dilutions in maximum recovery diluent

(MRD) (lab m Ltd) described by Wilson and Jeffrey (1987). Presumptive identification of mastitis streptococci was based on colony morphology and haemolytic pattern on blood agar; biochemical characteristics were established by sugar fermentation and catalase test. Antigenic-typing using 'Streptex' reagents (Wellcome Diagnostics Ltd) was used to confirm the identification. Staphylococci were isolated and enumerated by spreading 0.1 ml of milk and a 10-fold dilution of the milk in MRD on Kranep agar (Oxoid Ltd) and identified by blood-agar colony morphology and confirmed as coagulase positive *S. aureus* using a 'Staphaurex' test kit (Wellcome Diagnostics Ltd)

At the end of the study the producers whose bulk milk had been monitored were contacted by the Milk Board Regional Officer and herd management information was collected by an interview questionnaire completed on 29 of the 30 farms. The herd management information included number and age of lactating cows, culling and replacement policy, and calving pattern. The elements of a mastitis control programme used on the farm such as post-milking teat dipping, dry-cow therapy and automatic cluster removal (ACR) were also recorded.

RESULTS

The mean counts of mastitis streptococci and *S. aureus* from the samples collected during the course of the investigation were calculated. Statistical analysis of the data showed a significant relationship ($P < 0.001$) between the level of mastitis-causing bacteria in the bulk milk and the BTSCC. The mean level of mastitis streptococci was much greater (geometric mean 1469 cfu ml^{-1}) in high BTSCC herds than in either the borderline (geometric mean 557 cfu ml^{-1}) or low BTSCC group (geometric mean 114 cfu ml^{-1}) (Table I).

Figure 1 illustrates the linear regression analysis of $\log_{10}$ BTSCC on $\log_{10}$ mean streptococci. The herd categories remained as discrete groups along the regression line;

$$\log \text{BTSCC} = 4.614 + 0.3498 \log \text{strep}$$

Table I
Relationship between bulk tank somatic cell count (BTSCC) category and mean level of main mastitis bacteria

Herd category (BTSCC range 1000 ml)	*No. of herds*	*BTSCC (×1000 ml^{-1})*	*Log_{10}(BTSCC) (geometric mean ×1000 ml^{-1})*	*Log_{10} mean mastitis streptococci ml^{-1} (geometric mean ml^{-1})*	*Log_{10} mean Staphylococcus aureus ml^{-1} (geometric mean ml^{-1})*
High (467–969)	11	648	5.800 (631)	3.167 (1469)	2.458 (287)
Borderline (274–443)	8	370	5.558 (361)	2.746 (557)	2.321 (209)
Low (136–247)	11	188	5.269 (186)	2.055 (114)	1.602 (40)
SED (27 df)			0.0408	0.1845	0.1560

*Standard error of difference between two means.

indicating mastitis streptococcal count was highly correlated with BTSCC (r^2= 0.827, $P < 0.001$) over the whole range of cell counts.

Regression analysis of the *S. aureus* data showed that this mastitis pathogen count was not as highly correlated with the BTSCC (r^2=0.686) (Figure 2). The regression equation

$$\log \text{BTSCC} = 4.880 + 0.3134 \log \text{staph}$$

showed a highly significant relationship ($P<0.001$), though not as good as that for the mastitis streptococci. The high and borderline BTSCC results are less distinctly separated on the *S. aureus* scatter plot than on that for the streptococci. The addition of *S. aureus* to the mastitis streptococci in the regression analysis explains slightly more of the percentage variance (71.2) compared to the streptococci alone (68.1).

Incorporating the *S. aureus* and mastitis streptococci separately into the same regression equation,

$$\log \text{BTSCC} = 4.530 + 0.2709 \log \text{strep } 0.1391 \log \text{staph}$$

revealed that mastitis streptococci have a significantly greater effect on the cell count than staphylococci, confirming the results in Table I. This highlights that the problem in high BTSCC herds is associated with mastitis streptococci. The presence of *S. aureus* in bulk milk was distributed more evenly between high and borderline herds.

Figures 3 and 4 present the quantitative bacteriology data from two different high BTSCC herds to illustrate the difference in the level and pattern of excretion of *S. aureus* and mastitis streptococci, respectively, in bulk milk samples. The level of mastitis streptococci reached in excess of 250 000 cfu ml^{-1} in bulk tank milk,

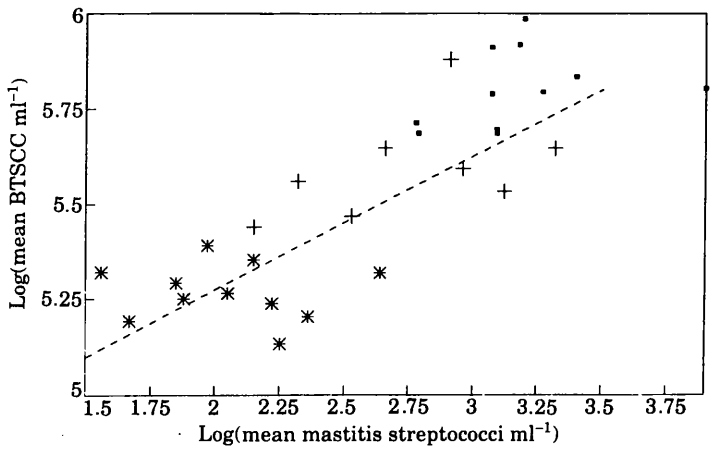

Fig. 1. Regression analysis showing relationship between $\log_{10}$ mean bulk tank somatic cell count and $\log_{10}$ mean bulk tank mastitis streptococcal count.

whereas the maximum *S. aureus* level was 17 000 cfu ml^{-1}. The predominant mastitis pathogen was identified in 31 of 330 bulk milk samples from high BTSCC herds where the total mastitis bacteria exceeded 10 000 cfu ml^{-1}. *S. agalactiae* was the predominant pathogen in 13 such samples; 12 were mainly *S. uberis*, and *S. dysgalactiae* was typed in a further six samples.

Analysis of the herd management data from the questionnaire, summarized in Table II, indicated that there were major differences in management practices, particularly between high BTSCC herds and the rest. High BTSCC herds tended to be younger, with a lower proportion of cows older than fifth lactation. These herds also bought-in replacements rather than bred their own heifers. High

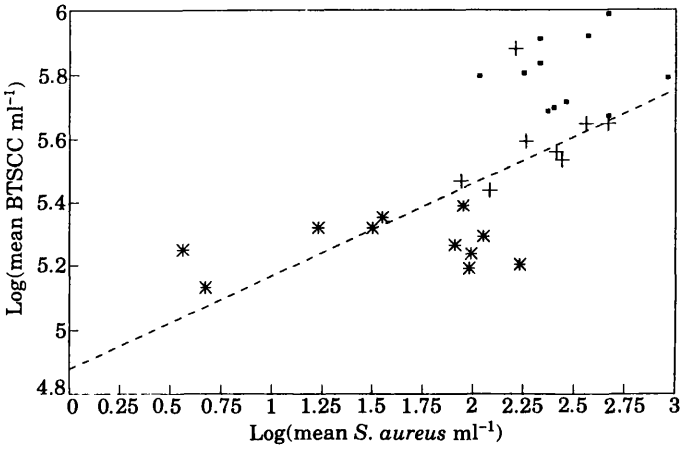

Fig. 2. Regression analysis showing relationship between $\log_{10}$ mean bulk tank somatic cell count and $\log_{10}$ mean bulk tank *Staphylococcus aureus* count. (■), High; (+), borderline; (*), low.

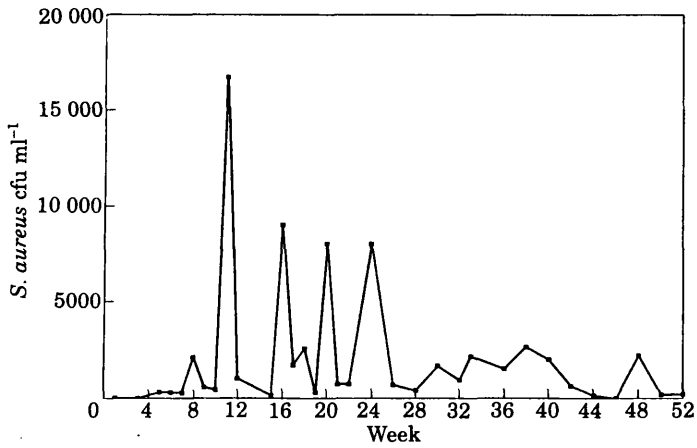

Fig. 3. The numbers of *Staphylococcus aureus* in bulk tank milk over the period of study from the herd with the highest mean level of *S. aureus* in the bulk milk.

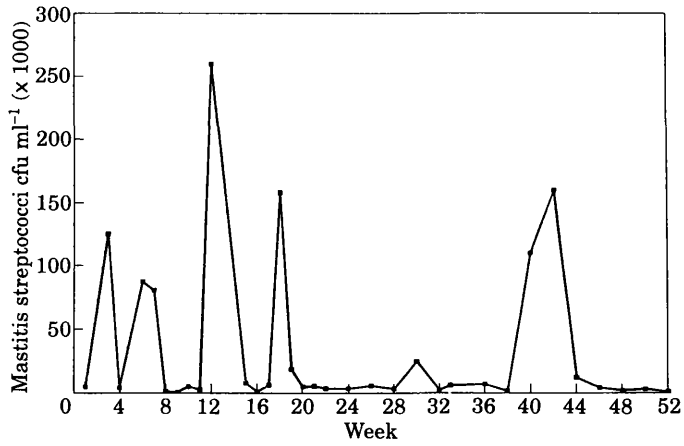

Fig. 4. The numbers of mastitis streptococci in bulk tank milk over the period of study from a herd with the highest mean level of *Streptococcus agalactiae* in the bulk milk.

Table II
Relationship between bulk tank somatic cell count (BTSCC) and some management parameters

Type of herd	*High*	*Borderline*	*Low*
Mean BTSCC (×1000)	648	370	188
Mean annual yield	5391	6657	6207
Size of herd (mean)	60–250 (106)	32–140 (90)	80–231 (133)
Buy in replacements	9/11	2/7	2/11
Post-milk teat dip/spray	5/11	6/7	11/11
Possess ACR*	5/11	3/7	9/11
Yearly machine test	5/11	5/7	9/11
Percent herd >fifth lactation (average/herd)	11%	21%	23%
Mean culling rate	17.8%	20.9%	16.3%
Main reason for culling	Mastitis related	Infertility/low yield	Infertility/old age

*ACR, automatic cluster removal.

BTSCC herds were less likely to use a post-milking teat dip or to have a regular programme of milking machine maintenance. High and borderline herds were less likely to have automatic cluster removal. Although the culling rates were similar for all herds, mastitis-related causes were most frequently cited as the reason for culling in high BTSCC herds compared to borderline and low BTSCC herds, where old age and infertility were claimed as the principal causes.

DISCUSSION

Jeffrey & Wilson (1987) reported that 43% of TBC hygiene failures in the ADMMB region were due to the predominant presence of mastitis bacteria, 90%

of which were mastitis *Streptococci* spp. (*S. agalactiae, S. dysgalactiae* or *S. uberis*). A single cow with clinical mastitis may excrete mastitis streptococci at >10 000 000 cfu ml^{-1} (Cousins & Bramley, 1981), which can potentially increase the TBC of the bulk milk in a 100 cow herd by 100 000 cfu ml^{-1}. In this study, high levels of mastitis streptococci were found in high BTSCC herds, 31 bulk samples were identified in which mastitis streptococci were present in numbers > 10 000 cfu ml^{-1}. Since persistent subclinical infection is a common consequence of infection by mastitis streptococci, infected cow(s) may remain undetected. A high TBC may provide the only evidence of such subclinical infection with mastitis streptococci, particularly when associated with a high BTSCC.

S. aureus numbers were considerably lower than those of mastitis streptococci, confirming the findings of Cousins and Bramley (1981) that *S. aureus* were excreted from an infected udder in lower numbers than streptococci. Jeffrey and Wilson (1987) found *S. aureus* to be the predominant bacterium in only 3.6% of TBC failures. This is in contrast to the prevalence of the organism in individual cow milk samples from whole herd bacteriological surveys. In a non-quantitative analysis of results of herd tests in the ADMMB region between 1974–1990 undertaken on herds with a clinical mastitis problem, *S. aureus* was the most common isolate, accounting for 65% of all significant mastitis bacteria and was present in 16% of all samples tested (Logue *et al.*, 1992). Nevertheless in the present study, a significant level of *S. aureus* infection was detected in bulk milk samples from high and borderline BTSCC herds, and can be a significant cause of elevated BTSCCs in borderline herds, where the TBC of the bulk milk remains consistently below the penalty levels of the Milk Boards.

Hutton *et al.* (1989) observed that managers of low SCC herds were more likely to attend meetings, pay more attention to details and have a greater awareness of mastitis control practices. In Scotland, Logue *et al.* (1993) noted that farms in the Scottish Milk Marketing Board area which recorded milk yields had lower BTSCC figures than those that did not and were three times less likely to incur an SCC penalty. They suggested that awareness and commitment were therefore very important in mastitis control. The results of our questionnaire also suggest that these factors contribute to the low BTSCC in some herds.

In a review of the effect of the milking machine and mastitis (IDF, 1987) overmilking appeared not to be a significant cause of new teat infection. However, in a study of high (460 000 cells ml^{-1}) and low (175 000 cells ml^{-1}) BTSCC herds, Hutton *et al.* (1989) reported automatic cluster removal (ACR) was more frequently found in the low SCC group. The findings of the current study support this observation and the use of ACR has been shown significantly to reduce BTSCC (Logan, 1993). This may be because with ACR the vacuum is shut off before the cluster is removed reducing irregular vacuum fluctuations within the machine and reducing the risk of backflow of milk on the teat with the consequent risk of penetration of the teat duct (Kingwill *et al.*, 1977). Also, the presence of ACR was associated with better maintained milking equipment. This is significant since penetration of the teat by mastitis bacteria is more likely to occur in a poorly functioning milking machine in which there is excessive vacuum fluctuation or an incorrect pulsation rate. The high BTSCC herd milking machines had poorer testing records than those used for other herd groups. The excessively

high vacuum level in a poorly functioning milking machine may result in teat damage. Teat lesions are susceptible to colonization by as few as 100 cfu *S. aureus* (Bramley *et al.*, 1979) and this bacterium (and also *S. dysgalactiae*) can readily penetrate the teat canal to establish an udder infection. It is well documented that the open teat orifice makes the udder more susceptible to infection by pathogens after milking than before milking (Kingwill *et al.*, 1977) and that post-milking teat dipping reduces infection. In the high BTSCC herds, the absence of post-milking teat dipping allowed continued cross-infection.

A further significant difference between the high BTSCC herds and those with lower cell counts was a lower number of cows above fifth lactation. Age is known to result in higher individual cow somatic cell counts (ICSCC), however in uninfected quarters there would appear to be little or no age effect (Gunn *et al.*, in preparation). Thus by inference the rise is due to increased prevalence of subclinical mastitis. This coupled with the fact that more low BTSCC herds breed their own replacements and are less likely to introduce infection from another herd suggests that there is an interrelationship between a number of different factors. However the effects of such culling on mastitis will be limited if mastitis hygiene control measures are not put in place (Natzke & Everett, 1975). The lower overall yield was particularly noticeable in the high BTSCC group and may have been due to a combination of clinical and subclinical mastitis coupled with the lower numbers of high yielding order animals.

There are limits to the information which can be gained from the examination of bulk milk for mastitis organisms. 'Environmental' mastitis organisms such as *Escherichia coli* originating from the udder cannot be differentiated from those arising from faecal contamination. However, the organisms in the present study were shown by Veterinary Investigation Centre data (VIDA, MAFF) to be responsible for almost half the clinical cases and virtually all the subclinical cases of mastitis in Scotland. It is possible to identify the predominant bacterial species causing the TBC failure and provide an advance warning of a streptococcal mastitis problem, particularly *S. agalactiae.* It is also possible to determine if there is an underlying problem. Gonzalez *et al.* (1986) found that bulk milk levels of *S. agalactiae* in excess of 4000 cfu ml^{-1} gave a moderately high correlation with at least 7% of the herd shedding the bacterium; their study was less conclusive for *S. aureus.* This study has shown that a mean level of streptococcal mastitis bacteria in the milk of 1000 cfu ml^{-1} (log_{10} 3.0) suggest a definite streptococcal mastitis problem. This will often cause sporadic high TBC in the bulk milk and potential hygiene failures. When a herd has a BTSCC which remains obstinately around 400 000 ml^{-1}, with few if any TBC failures, the presence of *S. aureus* in the bulk milk at levels of 100 cfu ml^{-1} (log_{10} 2.0) is indicative of a problem in the herd.

In summary high BTSCC herds generally failed to implement standard mastitis control procedures fully. This was well illustrated by the herds with consistent low BTSCC which practised such a policy.

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