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**SUMMARY**

The literature relating to the health and productivity of continuously housed or 'storage fed' dairy cows with particular reference to the adoption of this type of management system was reviewed. Emphasis was placed on means of improving the output of the dairy herd by storage feeding and also crossbreeding and the relationship between the application of these two techniques and the health and welfare of dairy cattle. Although frowned on in some quarters it was considered that provided production figures were used alongside other findings and interpreted with care they could be useful indications of both health and welfare.

Two treatment groups, one containing Jersey cross Friesian cattle (Jx) and the other containing purebred British Friesian's were produced. These two groups were the basis of the study and were continuously housed for five years being allowed to graze naturally only in the last summer of the study.

Shortly after birth calves were weighed and blood samples obtained for immunoglobulin analysis by the Zinc Sulphate Turbidity Test (ZST). During calthood the two breed types showed statistically significant differences for weights ( $P < 0.001$ ) and ZST ( $P < 0.01$ ) results. The Friesian calves had the highest weights but ZST levels were higher for the Jx. Correlations and regressions of weight and ZST were also carried out to determine interparameter relationships. After weaning both groups were housed continuously in a single span cubicle shed and were

storage fed on an identical complete diet until eventually they were allowed to graze in the latter part of their third lactation. Intakes of diet were calculated twice weekly with stock weighing weekly and condition scoring fortnightly. No major health problems were noted during this period and weight gains were more than adequate though again the Friesians significantly outperformed the Jersey cross. After first calving and throughout the experimental period milk samples were obtained weekly at both afternoon and morning milkings from each cow and analysed for butterfat, protein and lactose content.

There was no evidence of continuous housing reducing the level of output of either breed type and fat and protein percentage indicated that nutritionally the system had advantages over a traditional grazing regime . Statistical analysis of weights, yield and milk constituents was undertaken and while yields were found not to be significantly different butterfat and protein percentage were significantly higher in the Jx ( $P < 0.001$ ) and weights lower. Fertility was good throughout the housing period the Jx generally outperforming the Friesians. Indeed the fertility figures and maintenance of fat and protein levels throughout the three lactations indicated advantages in the use of this type of regime.

During an intensive monitoring period over the major part of the third lactation milk samples were also obtained for estimation of somatic cell numbers and chloride concentration at both afternoon

and morning milkings on a monthly basis. In addition at the morning milking a cowside California Mastitis Test (CMT) was performed on each cow by quarter (paddle test) to assess subclinical mastitis incidence. Milk samples were obtained from each quarter giving a positive CMT reaction for bacteriological analysis, somatic cell counts and chloride concentration and when two or more quarters gave a positive CMT result all quarters were sampled. The extent of subclinical mastitis was higher than expected given the background somatic cell (SCC) and total bacterial cell count (TBC) figures for the entire herd the rest of which was allowed to graze in summer. Statistical analysis of these positive CMT milk results was undertaken using an analysis of deviation technique, chloride being found to be significant for group and somatic cell counts for month ( $P < 0.05$ ). Regression and correlation analyses were also carried out and it was found that the most significant parameters were chloride and lactose concentration which were respectively lower and higher in the Jx. However these differences were thought to be a genetic not an indication of mastitis status.

The incidence and prevalence of lameness was low and did not differ significantly from the rest of the herd but the cows were routinely trimmed by the so called 'Dutch Method'.

On the morning of first turnout faeces and blood samples were obtained and thereafter taken monthly for determination of nematode worm burden and pepsinogen status. Worm egg counts were low with only 100 strongyle sp/gram of faeces being found at any

time. Pepsinogen levels were also low although there was a significant ( $P < 0.05$ ) rise with time the highest figure being found in September. However the highest individual figure was only 1238 units and no clinical signs were noted. Group differences were significant ( $P < 0.001$ ) with the Jx constantly having a lower level than the Friesians indicating an improved resistance to abomasal insult.

Neither hypocalcaemia nor hypomagnesaemia presented a problem during the period of housing or after the first turnout. However as with lameness the relatively young age of the cows made it difficult to draw firm conclusions regarding the effect of either continuous housing or breed differences.

Using the results obtained throughout the experimental period heterosis of the Jx was calculated. This was found to be positive for milk yield (except lactation one), protein yield and fat yield. The Jx also had lower calving indices than their Friesian contemporaries.

In summary no definitive disadvantage in terms of health and welfare could be attributed to storage feeding and the major advantages of being able to firmly control input and output and to maximise land use were evident. The major disadvantages highlighted in this study were cost, the requirements of a high quality environment particularly to limit mastitis problems, and that should circumstances demand it, the subsequent introduction of cattle reared in such a manner to grass. The last mentioned

could present considerable management difficulties with fencing and possibly also with parasite infections. Finally although this was not part of the study perhaps the most important disadvantage of storage feeding is public perception. It is felt that in the United Kingdom at least permanent housing is not perceived as desirable despite as shown here, there being little evidence of major welfare problems provided there is a good standard of stockmanship and housing.

The main advantage of crossbreeding was in the quality of milk in terms of fat and protein from the Jx which was significantly better than that given by the Friesian contemporaries. There were few significant differences in terms of health and welfare though the Jx did show an apparent fertility advantage.

**CHAPTER 1**  
**LITERATURE REVIEW**

### 1.1. INTRODUCTION

The literature relating to the health of continuously housed dairy cows will be reviewed. The reasons for this development will be considered with particular emphasis on recent advances within the dairy industry. Means of improving herd output with storage feeding and cross breeding will also be reviewed as will possible consequent diseases . Finally the effects of intensive farming methods on the health of a group of purebred and crossbred dairy cattle will be described and discussed in the light of these various factors.

### 1.2 DAIRY HERD STRUCTURE

In order to understand some of the pressures which have encouraged the scientific study of practical aspects of storage feeding dairy cattle and the use of crossbred cattle, it is necessary to briefly review some of the more important structural changes which have affected the dairy industry in recent times.

From the 1950's there has been a gradual change in the total number of dairy cows and the overall size of the dairy herd. Total cow numbers have dropped by 19% in England and Wales and 13% in Scotland while herd size has increased by 294% and 153% respectively (Table 1.1 and Table 1.2).



Table 1.1 Dairy Cow Population in Great Britain

	1957	1987
	In thousands	
England and Wales	3055.0	2486.3
Scotland	303.6	263.8

(Data from UK and Scottish Dairy Facts and Figures)

Table 1.2 Herd Size in Great Britain

	1957	1987
	cows/herd	
England and Wales	17.5	69
Scotland	36	91

(Data from UK and Scottish Dairy Facts and Figures)

Table 1.3 Milking Systems in Great Britain

	1977	No. of herds 1987	% Change
Cow Sheds	28531	10054	-64.8
Parlours	27981	28456	1.7

(Data from UK Dairy Facts and Figures)

There has also been a marked decline in the number of farmers milking in sheds or byres in the 10 years up to 1987, yet only a small increase in the number of parlour systems being used (Table 1.3). The inference that farmers have either gone out of milk or upgraded to a parlour system is confirmed by the decline in herd numbers (Table 1.4) and also the decrease of 65% in producer numbers from 1965-85.

Table 1.4 Total Dairy Herds in Great Britain

	1977	1987	% Change
No. of herds (x 1000)	57.5	40.3	-30

(Data from UK Dairy Facts and Figures)

This change is also reflected in the overall dairy cow population of the country. Numbers of dairy cows peaked in 1983 (Table 1.5) but the introduction of Milk Quotas in 1984 had a marked effect upon these figures and dairy cow numbers began to decline with a drop of 12% in the 4 years up to 1988. Beef cattle numbers however increased by 2.5% possibly indicating a changeover from dairy to beef by a small number of dairy farmers. Milk Quotas, which were introduced in 1984 to correct the imbalance between output and consumption within the European Community, have themselves resulted in considerable changes, with efficiency of production being more important than physical out-put per se. These changes will be discussed further in relation to quality.

Table 1.5 Cow Numbers in Great Britain

	Dairy Cow Numbers (x 1000)	Beef Cow Numbers
1965	2890	876
1975	3003	1571
1983	3034	1148
1985	2853	1119
1988	2632	1165

(Data from UK Dairy Facts and Figures)

### 1.3 BREED CHANGES

Since the 1950's dairy cow breeds have also undergone a radical change with a decline in Dairy Shorthorn and Ayrshire cows and an increase in Friesian and Holstein (Table 1.6).

Table 1.6 Dairy Cow Population England and Wales

Breed Type	1955	1965 (x 1000)	1985	% Change*
Ayrshire	453.1	415.6	56.1	-87.6
British Friesian	1005.3	1699.4	2188.8	117.7
Holstein	-	-	40.8	40.8
Jersey	64.4	113.8	99.5	54.5
Dairy Shorthorn	626.4	166.7	12.8	-97.9
Others	326.8	251.5	153.0	-53.2
Total Cows	2476.0	2647.0	2551.0	3.0

(Data adapted from UK Dairy Facts and Figures)

\* From 1955-85

Perhaps surprisingly the numbers of Jersey cows in the UK has increased slightly from 1955. The specialised market in Jersey cream products probably accounts for the stability of this breed, which although giving a lower milk yield than all others is comparable in fat yield (Table 1.7).

Table 1.7 Average Milk Yield and Quality 1985

	Milk Yield kg	Butterfat (% by weight)	Protein (% by weight)	Butterfat Yield (kg)	Protein Yield (kg)
British					
Friesian	5729	3.87	3.23	221.71	185.05
Holstein	6406	3.84	3.17	245.99	203.07
Ayrshire	5102	3.96	3.34	202.04	170.41
Dairy					
Shorthorn	5142	3.69	3.27	189.74	168.14
Jersey	3835	5.27	3.80	202.10	145.73

(Data adapted from UK Dairy Facts and Figures)

#### 1.4 MILK YIELDS AND MILK QUALITY

Despite the reduction in the total number of dairy cows in the UK annual milk and butterfat yields have actually increased (Table 1.8).

Table 1.8 Average Milk Yields per annum

	Milk Yield kg x 10 <sup>6</sup>	Butterfat kg x 10 <sup>6</sup>
1965	13512.9	522.95
1985	17804.6	699.72
% increase	32	34

(Data adapted from UK Dairy facts and figures)

However, protein yield of milk has remained relatively static between 1965 until present day. Furthermore, unlike fat yield it has proved much more difficult to manipulate protein output by dietary means.

## 1.5 MANIPULATION OF MILK CONSTITUENTS

### 1.5.1 Milk Protein Content

Although it is difficult, the protein content of milk can be altered by manipulating dietary supply of energy and protein; breed and stage of lactation are also contributing factors.

#### 1.5.1.1 Diet

Various workers have attempted to manipulate the protein concentration of milk by supplementing and substituting feed elements (Table 1.9).

Table 1.9 Manipulation of Protein Content of Milk

Reference	Level of concentrate (kg DM)	Milk Composition (g/kg)	
		Fat	Protein
Castle, Gill & Watson (1981)	6.3	+3.2	+0.2
Mayne & Gordon (1984)	8.4	-1.1	-0.1
Thomas, Aston, Daley & Bass (1986)	10.9	+1.0	-1.3
Sloan, Rowlinson & Armstrong (1986)	10.5	+4.3	-1.0

Restriction of total energy consumption can result in reduced milk protein content. Low concentrate input and restricted access to good quality feeding or feeding poor quality silage can reduce milk protein levels by affecting overall nutrition.

Increasing the protein content of milk is however not easily managed by dietary factors alone as only small improvements are apparent when normal dietary protein is supplemented (Thomas, 1984). In contrast substantial underfeeding of protein will markedly reduce milk protein content (Leaver, 1983). Generally,

in the UK, dietary protein is sufficient to maintain the protein content of the milk.

#### 1.5.1.2 Breeding

Selective breeding can be used to increase protein levels, for example by the introduction of Jersey genes (Ahlborn-Breier, 1989). This aspect will be expanded later.

#### 1.5.1.3 Stage of lactation

Colostrum by its very nature is very high in protein content (Jenness, 1974). This elevated protein level decreases gradually until a minimum between the date of calving and the sixtieth day of lactation, thereafter the protein content increases steadily until the cow dries off (Ng-Kwai-Hang, Hays, Moxley and Monardes, 1981).

#### 1.5.1.4 Age

From the second to third lactation protein content of milk is at a peak, this declines from the third lactation onwards (Ng-Kwai-Hang, et al, 1981).

### 1.5.2 Milk Fat Content

Milk fat content is influenced by stage of lactation and breeding (Crabtree, 1984) but feeding is the most important factor (Sutton, 1984). Thus it can be used as a dietary indicator.

### 1.5.2.1 Diet

Restrictions on fibre intake depress fat percentage in milk, (Table 1.10). Cows should therefore be given access to unrestricted ad libitum forage.

Table 1.10 Effect on milk fat content

Increases	Maintains	Reduces
	<u>Frequent feeding</u>	
	<u>Diet characteristics</u>	
By pass fats	Roughage	Starch
	Fibre/ADF/Cellulose	High intakes
	<u>Diet ingredients</u>	
	Long forage	Sucrose
	Fodder beet	Unsaturated fats
	Fibrous by-products	Ground forage
	Saturated fats	
	Sugar, whey	
	Buffers	

(Adapted from Sutton, J D, 1984).

### 1.5.2.2 Breeding

There are well documented variations between breeds (Table 1.11) and between cows within a breed in milk fat percentage (Leaver, 1983).

Table 1.11 Fat composition of milk

Breed	Fat %
Friesian	3.8
Jersey	5.1

#### 1.5.2.3 Stage of Lactation

Like protein, fat levels are higher in colostrum. These high levels subside until a minimum level is reached between the 6 and 10th weeks of lactation. Thereafter the fat content rises steadily until the cow dries off (Crabtree, 1984).

#### 1.5.3 Milk Lactose Content

Average lactose content of milk is 4.7% which can be changed only marginally by changes in feed intake (Leaver, 1983).

##### 1.5.3.1 Stage of Lactation

Lactose concentration in colostrum is low (2.19%) (Engel and Schlag, 1924) but increases within the first week of lactation, remaining at a steady level (4.7%) until the 20-24th week of lactation when it begins to decline slowly and then more rapidly until the end of lactation (Rook, 1961).

#### 1.6. THE MILK MARKETING BOARDS

The Milk Marketing Boards have recognised that milk quality is not only depressed by inadequate nutrition but also by hygiene and udder infections and have attempted to raise standards by some simple measures of hygiene, for example, by introducing a penalty system for Total Bacterial Counts.



## 1.7 EUROPEAN COMMUNITIES REGULATIONS

In February 1990 the Commission of the European Communities introduced new regulations governing the quality of raw milk (Document 667). Levels for total bacterial counts, somatic cell counts, and the presence of antibiotic residues were set and are shown in Table 1.12 along with the date when they will become effective.

Table 1.12 EC Regulations for raw milk

	From 1.1.93.	From 1.1.95
Plate count 30°C (per ml)	≤ 300,000	≤ 100,000
Somatic cell count (per ml)	≤ 500,000	≤ 400,000
Antibiotics (per ml)		
- penicillin	≤ 0.004 ug	0.004 ug
- other	undetectable	undetectable

## 1.8 HYGIENE OF MILK

### 1.8.1 Resazurin Test

Previous to 1981/82 the hygienic quality of milk was tested by means of a two hour Resazurin test. This technique gave a straight forward pass or fail result and penalties were only incurred after two failed tests. These tests however were found to be unsuitable for assessing the quality of bulk collected supplies. The Resazurin test failed to give the buyer information on the suitability of the milk for processing and passing the test could have encouraged the producer into believing his production methods were satisfactory.

Consequently centralised testing was introduced by all of the milk marketing boards using a new hygienic scheme which involved a Total Bacterial Count Test (TBC) and an assessment of antibiotic contamination.

### 1.8.2 Introduction of Total Bacterial Counts

Each milk marketing board introduced testing of the hygienic quality of milk at different times (Table 1.13).

Table 1.13 Introduction and Review of Total Bacterial Count Tests by month

Introduction and Review of TBC's (month)							
Board	1981	1982	1983	1984	1985	1986	1987
MMB	-	Oct	-	-	-	-	Apr*
Scottish MMB	Apr	-	Apr*	-	-	Apr*	-
Aberdeen & District MMB	-	-	Apr	Apr*	-	-	-
North of Scotland MMB	-	-	-	-	Apr	-	-

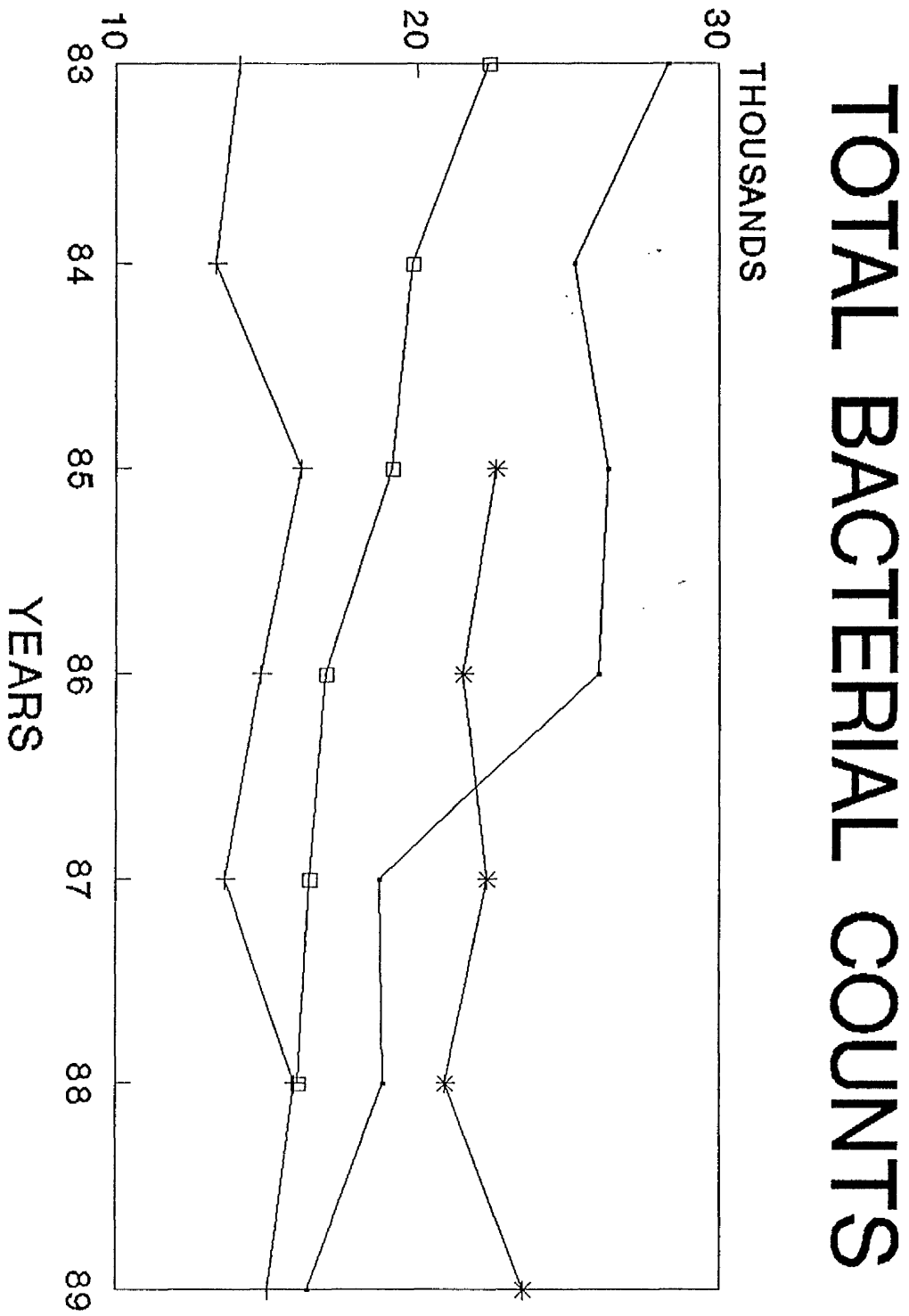
\* Reviewed

Figure 1.1 shows the effect of total bacterial count testing on the average annual TBC recordings for each board.

### 1.8.3 TBC Methodology

In general each milk producer has bulk milk sampled on a weekly basis by the milk marketing boards. The samples are subjected to a Total Bacterial Count test. The test involves mixing a measured quantity of milk with a culture media which is then incubated for 3 days to ensure growth of all viable organisms into colonies. These colonies are then counted using an electronic scanner (Biofoss Colony Counter) and the total number

FIGURE 1.1



of organisms calculated to the nearest thousand per millilitre recorded. These total counts are then used to determine the Hygienic Quality Incentives operated by all of the milk marketing boards.

#### 1.8.4 Hygienic Quality Incentives

The boards do not have a national system for the payment of hygienic quality premiums or for the deduction of levies from unhygienic producers. Each board sets its own criteria for hygienic quality incentives both in terms of premium and penalty (Table 1.14).

Table 1.14 TBC Test Penalty values

Board	TBC Test No. organisms/ml
MMB	100000
Scottish MMB	50000
Aberdeen & District MMB	46000 - 90000
North of Scotland MMB	50000

It must be made clear that generally two or more tests showing these values or above must be made before penalties are incurred. Penalties range from -0.2 to -4.8 pence/litre of milk and can thus greatly decrease the profit margin of a farmer. They are also of value as an indicator of the quality of cleanliness of the cattle and thus by inference as a measure of welfare.

### 1.8.5 Somatic Cell Counts

With the introduction of electronic cell counting techniques (Appendix 5) subclinical mastitis could be monitored. The Milk Marketing Boards began to regularly monitor bulk milk cell counts in 1971 and from 1977 onwards routinely monitored all herds on a monthly basis (Figure 1.2). The Scottish Boards began monthly recording in 1974.

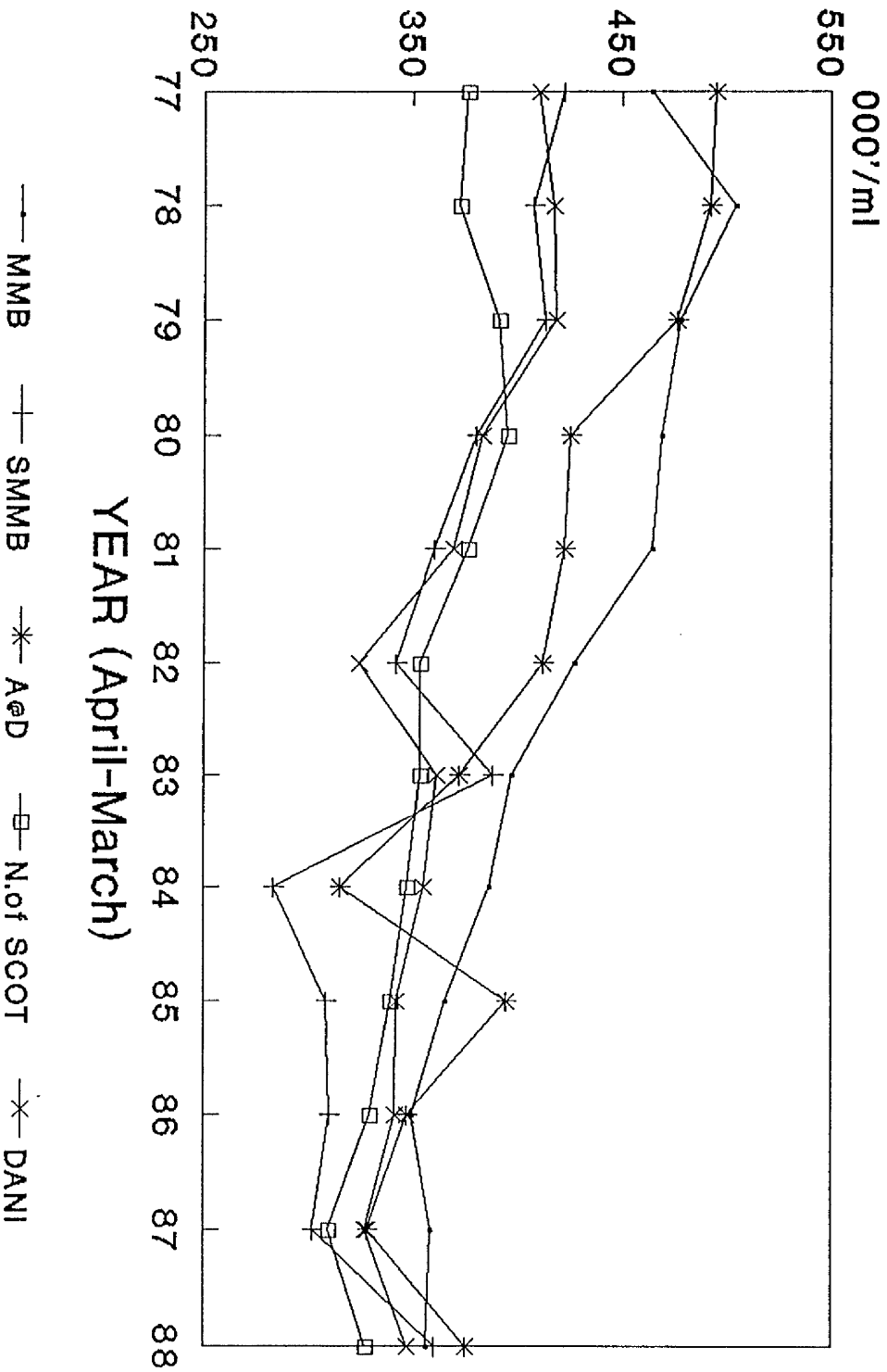
Since the beginning of routine recording, the annual mean cell count in all board areas has fallen practically on a yearly basis until 1987 (Table 1.15). These figures represent a fall of 23%, 28%, 33% and 18% respectively in 10 years of cell count monitoring and by inference an improvement in welfare.

Table 1.15 Somatic Cell Counts

Board	Somatic Cells x 10 <sup>3</sup> /ml.	
	1977	1987
MMB	464	358
Scottish MMB	422	302
Aberdeen & District MMB	492	328
North of Scotland MMB	379	309

Economic factors have had great bearing on the National Cell count. When premium payments were introduced for low Total Bacterial Counts (TBCs) in milk, cell counts also fell. This drop of on average 7% throughout the board areas was probably due to farmers realising that mastitic milk could increase their TBC's. Thus a greater awareness of mastitis was founded (Booth, 1988a and b). A possible reason for the fall around Quota

# CELL COUNTS



introduction was that cows likely to have high cell counts and cows with a poor record of mastitis were preferentially culled as farmers attempted to adjust to their quota restrictions.

#### 1.8.6 Progress

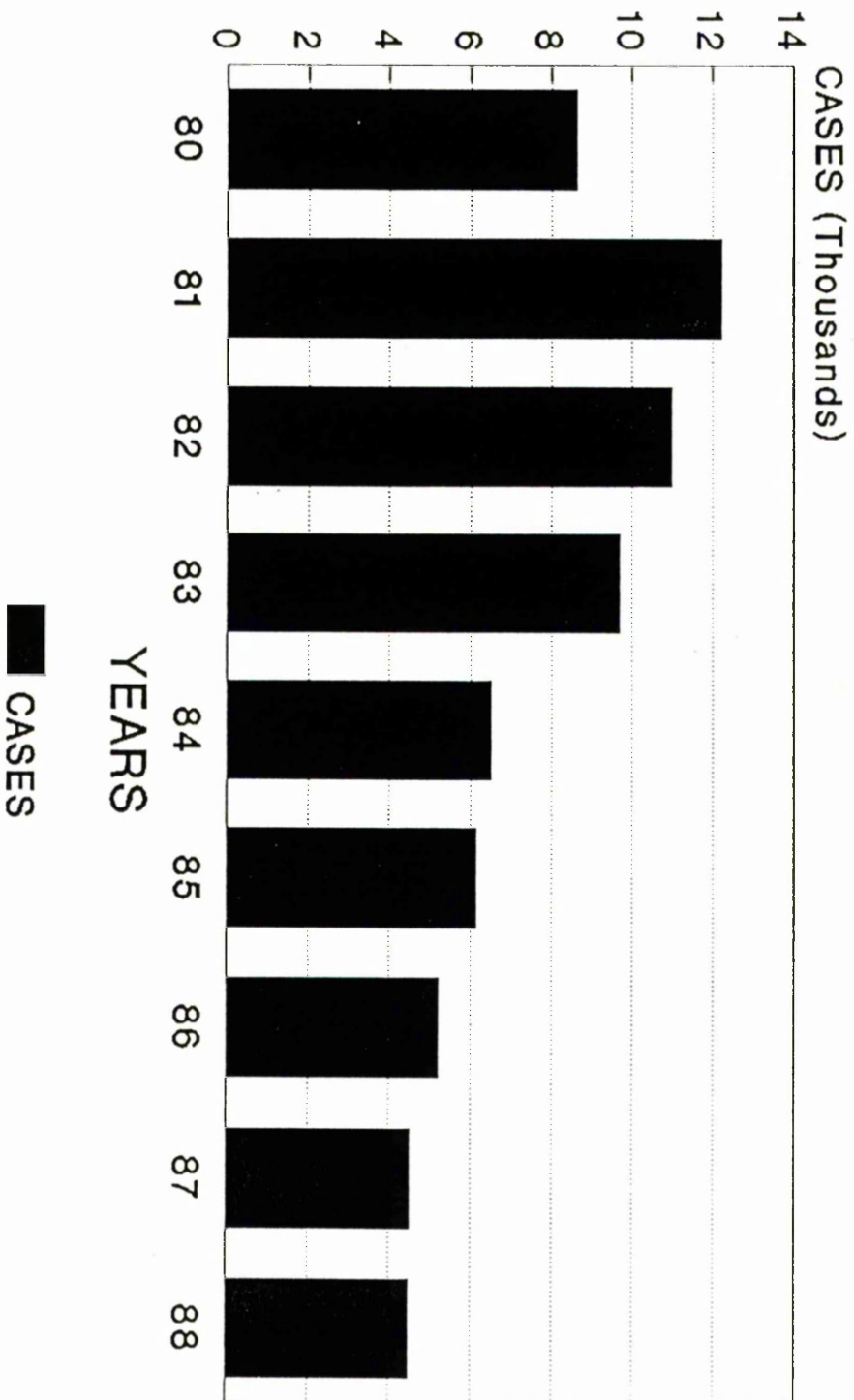
Since the introduction of TBC testing on a national scale Somatic cell counts have gradually fallen and continue to fall although a slight rise in 1988 was evident (Figure 1.2). One major reason for this is that farmers have been made aware that mastitic milk could increase their TBC test results.

Thus the promotion of greater hygiene by the boards in the introduction of premium payments has resulted in a decrease of the incidence of mastitis on farms in both the UK and Scotland as reported by Wilesmith, Francis and Wilson (1986) and the Veterinary Investigation Centre returns (Anon, 1989) (Figures 1.3 and 1.4). Probably the most important factor has been a decrease in cross infection between cows but rapid and effective treatment must also play a part. In this regard it is worth noting that antibiotic failures have also steadily fallen and are now at 2% of all samples though this information is less readily available (Logue, 1989 personal communication).

#### 1.9 MODERN FARMING TECHNIQUES

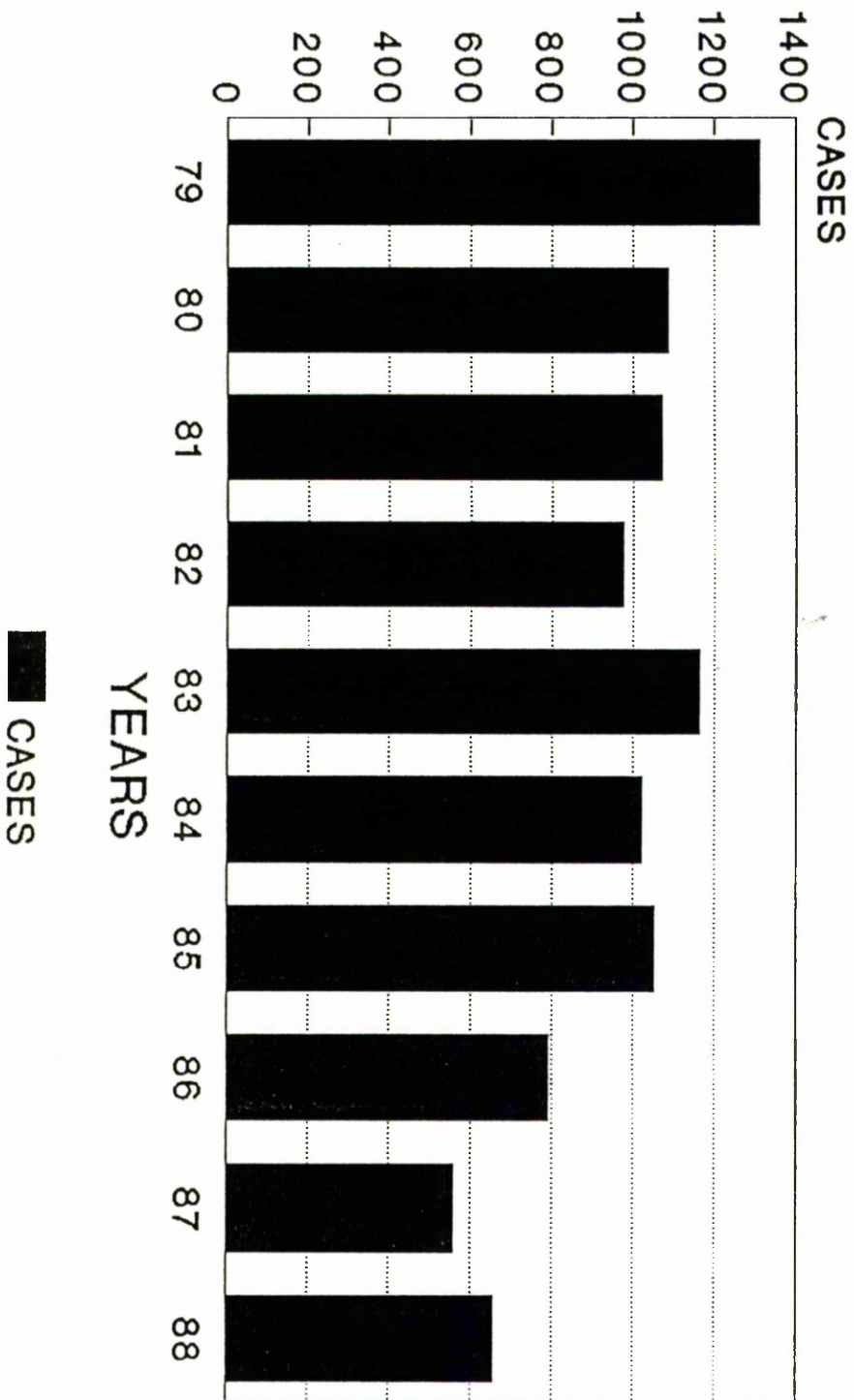
Dairy farming has now become a highly intensive business with large capital outlays, largely associated with housing and parlour systems. A recent estimate for a 'green site system'

# MASTITIS TOTALS (VIDA) U.K.





# MASTITIS TOTALS (VIDA) SCOTLAND



for a dairy with 16 stalls and cubicle housing for 90 cows being £128,101 (Farm Buildings Cost Guide, 1990). This was not inclusive of milking equipment. With such large overheads and inevitable overdrafts farmers have attempted to use modern farming practices to the best advantage. Two of the methods which could possibly contribute to an increased efficiency are storage feeding and crossbreeding.

#### 1.10 STORAGE FEEDING

Storage feeding refers to the system where cattle are housed or partly housed throughout the year. The increasing cost of labour and the decreasing availability of land have dictated considerable changes in dairy herd management and this system of managing dairy cows is now widely practised in some areas of North America and Israel due to economic, climatic and labour factors (Montgomery, Baxter, Owen and Gordon, 1976). While storage feeding has the disadvantage of high cost it has been shown to allow cattle to maintain higher DM intakes and increase grass utilisation per hectare (Phillips and Leaver, 1985) and also leads to higher stocking rates (Roberts and Leaver, 1987) with the use of conserved forage for feeding as opposed to grazing. The system also has the advantage of flexibility allowing the rapid manipulation of calving date to take advantage of changes in milk price without any major changes in management (Roberts and Leaver, 1987). Low priced by-products (for example draff) can also be included in diets reducing overall costs (Roberts and Leaver, 1987). Milk production is well maintained with a broadly similar lactation curve to grazed cattle although

the peak in May and depression in August to October normally associated with grazed dairy cows is not observed (Roberts and Leaver, 1987). Finally it allows the full utilisation of an expensive housing system which otherwise lies idle for 4 to 8 months of the year depending on climate.

By adopting storage feeding external environmental influences are eliminated and this could have an affect on herd health. Two studies have reported that health was not affected by storage feeding (Schingoethe, Parson, Ludens, Tucker and Shave, 1978 and Roberts and Leaver, 1987). However, Roberts and Leaver (1987) stated that regular routine foot trimming and good housing were essential and Anderson, Lamb, Mickelsen, Blake, Olsen and Arave (1977) commented that provision of an exercise yard in the dry period might help to alleviate calving difficulties. It may therefore be summised that lameness and calving difficulties are possible problems in this system.

## 1.11 CROSSBREEDING OF DAIRY CATTLE

### 1.11.1 Introduction

In Britain the majority of dairy cattle are purebred Friesian/Holstein so-called "Black and White". This form of breeding system can give rise to an increased incidence in the inheritance of recessive genes and thereby disease (Stormont, 1958).

### 1.11.2 Principle of crossbreeding

Crossbreeding utilises the fact that breeds of different genetic backgrounds have dissimilar genetic material. Thus progeny of mixed breed pairings will inherit unlike alleles from parents. The first generation cross is termed the  $F_1$ . The  $F_1$  displays an increased heterosis which can be either positive or negative. Positive heterosis is termed crossbred vigour which implies that the  $F_1$  is superior to both of its parents. This type of heterosis is seen in plants (Mendel, 1866) but much more rarely in farm animals where it is usually defined as the  $F_1$  being superior to the mean of the parent values (Figure 1.5).

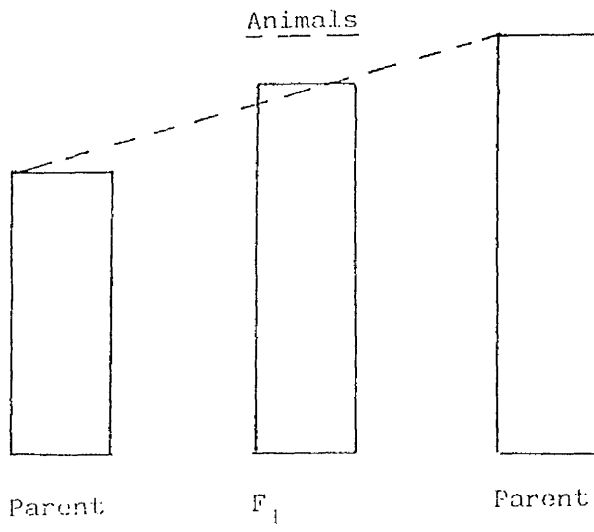
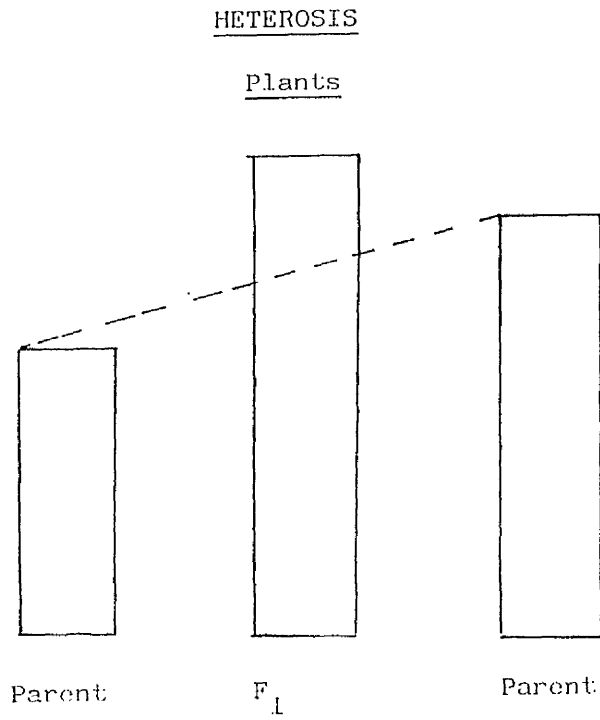
The effects of crossbreeding in dairy cattle, which is usually carried out with the aim of improving only one or two traits, have been extensively reviewed (Pearson and McDowell, 1968 and Turton, 1981).

The main conclusions drawn from these reviews were:

1. Positive heterosis occurs for milk yield but not composition.
2. Positive heterosis occurs for survival and growth rates.
3. Positive heterosis occurs for reproductive traits but these are not consistent.
4. Negative heterosis occurs for survival as calves in early months but positive heterosis to later ages.

Donald (1963) disagreed with Turton on negative heterosis for survival as calves, stating purebred heifer dams produced 14.7%

FIGURE 1.5



dead purebred calves and 6.4% dead crossbred calves. For older dams the values decrease to 2.4 and 3.6% respectively.

### 1.11.3 Economic Factors Relating to Crossbreeding Dairy Cattle

Since the 1980's with the advent of quotas and milk premium payments the 'economic' aspects of crossbreeding have come under closer scrutiny. In addition to the effects upon milk yield and composition these economic factors include survival, growth and feed efficiency and reduction of open days.

#### 1.11.3.1 Milk yield and composition

In a study carried out by Robison, McDaniel and Rincon (1981) positive heterosis was evident in crossbreds for milk yield, milk fat, fat% and fat corrected milk and thus they were less likely to be culled. This positive heterosis was confirmed by Rincon, Schermerhorn, McDowell and McDaniel (1982).

#### 1.11.3.2. Survival

According to Hocking, McAllister, Wolynetz, Batra, Lee, Lin, Roy, Vesely, Wauthy and Winter (1988 a and b) survival is a multicomponent parameter influenced by endogenous biological factors, husbandry/environmental conditions and the economic and physical requirements of the system. The critical areas are parturition of the dam, early calfhood, first conception, first parturition and second conception and also milk yield and milking speed. Crossbred cows had an apparent survival advantage over purebreds and this advantage could be due to hybrid vigour

(Robison et al, 1981). Donald (1963), Vesely, McAllister, Lee Batra, Lin, Roy, Wauthy and Winter (1986) showed that perinatal mortality was significantly lower in crossbred calves in comparison to purebred calves born to purebred dams, but in a review Turton (1981) disagreed stating that apart from the Animal Breeding Research Organisation (ABRO) data on perinatal survival of calves from heifer dams, crossbred calves had poorer survival than purebred calves in the early months of life. As the ABRO experiments were conducted in Great Britain using the normal dairy breeds found in that country it could be contended that these admittedly minority results are more applicable to the present study. Crossbred dairy heifers had a significantly higher chance of completing a lactation and surviving to a second lactation and in addition herd life was reported to be seventeen weeks longer for crossbreds as opposed to purebreds after first parturition (Hocking et al, 1988 a and b). Hocking et al (1988b) considered that these increased survival rates were associated with positive heterosis (hybrid vigour), confirming the earlier findings of Touchberry (1970) and McDowell (1982). Also contributing to survival was the reduction in open day numbers in crossbreds (Rincon et al, 1982) thus crossbreds were more efficient in having an earlier first calving.

#### 1.11.3.3. Growth and Feed Efficiency

Gross feed efficiency has been estimated at 0.610 for Holstein dairy cows by McDowell and McDaniel (1968). However, Neilson, Whittemore, Lewis, Alliston, Roberts, Hodgson-Jones, Mills, Parkinson and Prescott (1983) found that the gross feed

efficiency of high yielding cows was 0.34-0.43. Clearly the methods of estimation have been prone to considerable variation. Nevertheless at a simpler level feed efficiency contributes to the weight of heifers at first parturition. Positive heterosis occurs in crossbreeds for body weight and increases with age through 18 months and declines by 30 months (Robison, Kelly, McDaniel, McDowell, 1980). These authors also state the possibility that cross breeding stimulates the rate of body development but does not influence mature size. This is in general agreement with Lee, Lin, McAllister, Winter, Roy, Vesely, Wauthy, Batra and Atwal (1988) and implies an increased feed efficiency in the young maturing heifers. Hence, genetic effects from heterosis, maternal and additive sources eliminating genetic line effects are collectively believed to be significant (Lee et al, 1988).

#### 1.11.4 Crossbred Populations

In summary the weight of data shows that there are at least some advantages in using crossbred cattle and the practical evidence for this is apparent from the fact that crossbreeds are common dairy cattle in Australia and New Zealand and recently more attention is being given to them in Great Britain with the planned introduction of Brown Swiss semen into a few selected herds (Simm, 1989 personal communication). It is likely that the one perceived major attribute of these cattle is their "stayability" compared to purebreds. The reasons for this are likely to be improved fertility and health though the latter is poorly documented, and yield relative to weight (Donald, Gibson



and Russell, 1977). In Great Britain the pig, sheep and beef industries have long since realised the potential of the crossbred, utilizing a stratified system of breeding which in general suits the country's needs in terms of natural resource and product.

#### 1.11.5 The Jersey Crossbred

The Jersey breed is becoming increasingly used as a genetic resource for crossbreeding. The purebred Jersey has advantages in that it shows early sexual maturity, ease of calving and production of high fat and protein milk (King, 1984). In some cases its low adult size could be considered an advantage and in others a disadvantage. These features are also seen in the crossbred with some additional advantages due to crossbred vigour. Table 1.16 shows the percentage crossbred data adapted from Donald et al (1977) using the parental means of the purebreds as a baseline. In New Zealand a recent study found that positive heterosis at a rate of 3.2-4.8% and 2.1% occurred for milk fat and protein yield respectively. Higher calving rates and fewer calving difficulties were also observed (Ahlborn-Breier, 1989).

The Jersey has been used as either the sire or dam in crossbreeding experiments with a wide range of other breeds including Friesians, Holsteins and Ayrshires. They are also well suited to the production of beef cross calves because of their ability to cope with surprisingly large continental cross calves

(King, 1984) although the calves are not as attractive to contract calf rearers and cattle finishers as Black and White calves. The reason for this is the Jersey crossbred has a significantly larger area of internal pelvic canal per kilogram of body weight than Friesian heifers of the same age (Murray, 1985, personal communication). This produces the ease of calving characteristic with the crossbred.

Table 1.16 Percentage heterosis displayed by Jersey X Friesian (% of parental mean)

Trait	% Heterosis
Weight at 18 months	4.7
Calving Interval	-3.7
Milk yield 305 d 1st lactation	3.6
Milk yield 305 d 2nd lactation	5.7
Total solids 305 d 1st lactation	5.2
Total solids 305 d 2nd lactation	6.5
Milk fat % 1st lactation	1.3
Milk fat % 2nd lactation	0.4
SNF% 1st lactation	1.1
SNF% 2nd lactation	0.8

Adapted from Donald et al (1977).

#### 1.11.6 Disease and Productivity

One of the inevitable considerations in any radical management system is what effect will such a change have on the welfare of the cattle, first and most importantly the influence on their health and productivity, and secondly the related ability of the cattle to express normal behaviour. Without belittling its importance, behavioural aspects will not be discussed here, as no such studies were undertaken in the present work.

#### 1.11.7 Importance of Immunoglobulin Status in Disease Resistance

One of the major factors governing disease resistance in early life is the provision of adequate levels of immunoglobulin received by the ingestion of maternal colostrum. This transfer of maternal antibody in colostrum was first described by Ehrlich (1892) but was not examined in detail until Howe (1921) showed that in the new born animal colostrum globulins were absorbed without change in amounts sufficiently large to alter the composition of blood plasma. Smith and Little (1922) and Smith and Orcutt (1925) recognised the importance of colostrum consumption by calves soon after birth in relation to disease resistance. This passive immunity is dependent upon intestinal absorption of colostrum immunoglobulins during the first twenty four hours of life, thereafter the ability to absorb immunoglobulin declines (Stott, Marx, Menefee, and Nightengale, 1979).

It has been shown that breed differences in colostrum immunoglobulin concentration are wide with the Jersey having the highest total colostrum immunoglobulin (Muller and Ellinger, 1981). This breed difference may have accounted for the significantly higher levels of plasma protein concentration of immunoglobulin in Jersey calves in comparison to Holstein-Friesian calves found by Tennant, Harrold, Reina - Guerra and Laben (1969). However, in their experiment high immunoglobulin levels in the Jersey calves was not a panacea for survival as this group incurred higher losses due to neonatal disease.

#### 1.11.7.1. Breeding for Disease Resistance

With the economic importance of many diseases breeding dairy cattle for disease resistance has been stressed (Mallard, Burnside, Burton and Wilkie, 1983 and Mazengera, Kennedy, Burnside, Wilkie and Burton, 1985) and serum immunoglobulin has been associated with the health status of the cow (Gay, Anderson, Fisher and McEwan, 1965; Lascelles, 1979 and Mallard et al, 1983). Breeding for specific disease resistance has not been considered to be feasible commercially (Gavora and Spencer, 1983, Hohenboken, Muggli, Berggren-Thomas and Norman, 1986) but breeding for general disease resistance may be practicable (Hohenboken et al, 1986). Selection of immune traits in ruminants has been established (Almlid, Steine, Lund and Larsen, 1980 and Windon and Dineen, 1984) and selection for one immune trait can enhance more than one aspect of the immune system (Gavora et al, 1983).

### 1.12 MASTITIS CAUSES AND PREVENTION

#### 1.12.1 Mastitis

##### Introduction

Mastitis is a multifactorial disease with hygiene, milking machines and the environment all playing a part in its aetiology. It is simply an inflammation of the mammary gland and is characterized by pathological damage to the mammary epithelium, followed by subclinical and/or clinical inflammatory reactions. These inflammatory reactions may cause localised and, or generalized pathological changes, (Giesecke, 1975). Milk yield falls and the milk also undergoes physical and chemical change.

1.12.2 The Effect of Mastitis on Milk Yield and Compositional Quality

The effect of mastitis on milk yield and quality has been extensively reviewed (Kitchen 1981, Munro, Grieve, Kitchen, 1984, Harding, 1988). The major accepted conclusions of these reviews are summarised in Table 1.17 and 1.18.

Table 1.17 Effect of mastitis on milk yield and composition

<u>Decrease</u>	<u>Increase</u>	<u>Variable</u>
Milk yield	Chloride	Total Protein (Table 1.18)
Fat		
Lactose		

Adapted from Munro et al (1984).

Table 1.18 Effect on Protein components of milk

<u>Decrease</u>	<u>Increase</u>	<u>Variable</u>
Total Casein	Casein	$\kappa$ Casein
$\alpha$ Casein	$\delta$ Whey Proteins	
$\beta$ Casein	Immunoglobulins	
$\beta$ Lactoglobulin	Serum albumin	
Lactalbumen		

Adapted from Munro et al (1984).

Due to the significant changes in lactose and chloride concentrations in mastitic milk, these parameters have been suggested as a means of diagnosing mastitis (Renner, 1975) . However as mentioned earlier the compositional quality of milk can be altered by other factors, for example, diet and stage of lactation .

### 1.12.3 Clinical Mastitis

This type of mastitis is usually easily recognised by the herdsman and such infections can be promptly treated. In addition to the signs seen in the cow symptoms of clinical mastitis are a drop in milk yield and the presence of clots or exudate in the premilking strippings. However, only a small proportion of infected cows show symptoms at any time (Dodd and Neave, 1970). The remainder showing no symptoms have subclinical mastitis.

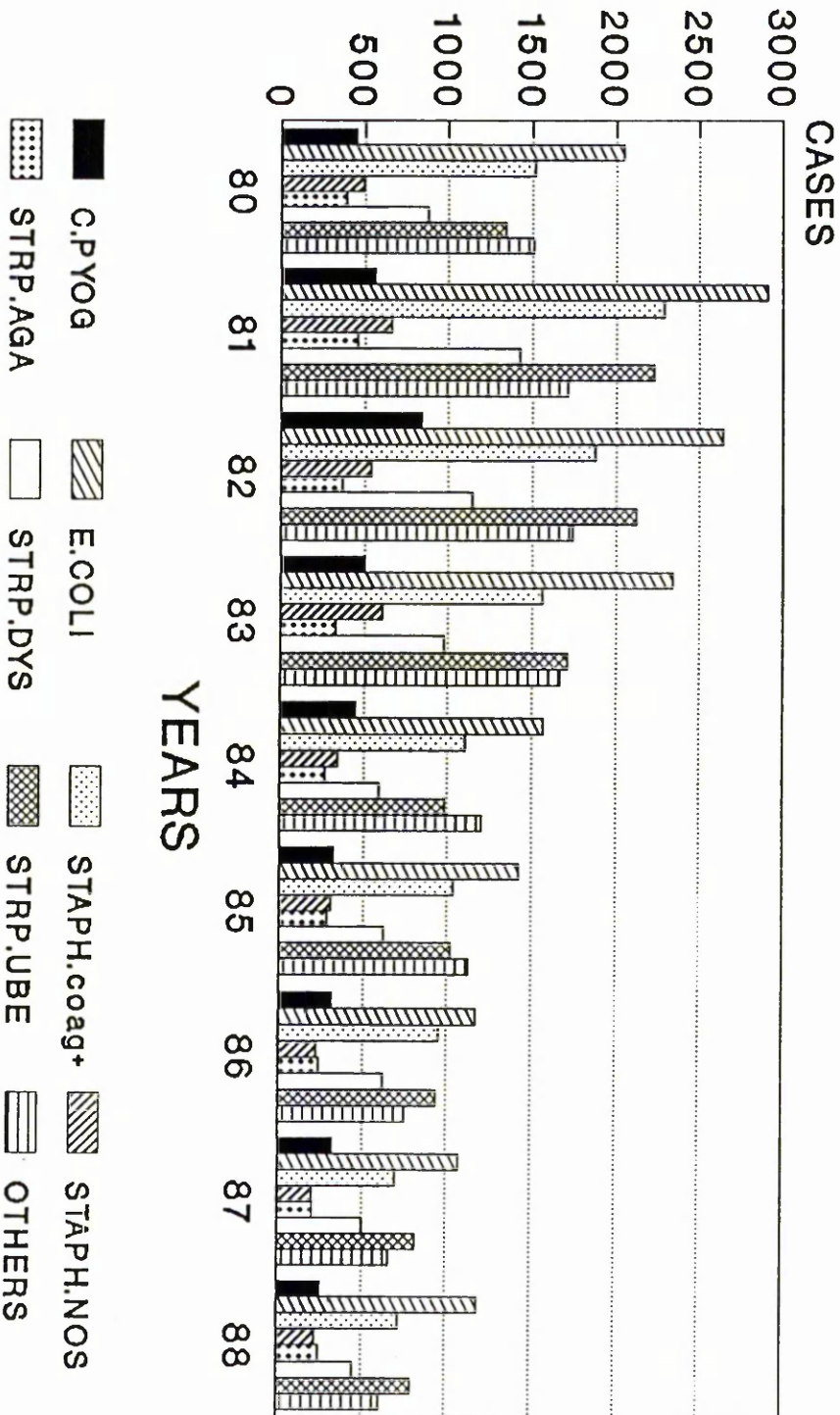
#### 1.12.3.1 Incidence of Clinical Mastitis

Mastitis is an important cause of economic loss to the British dairy farmer. Although the incidence of clinical mastitis has fallen during recent years, the economic impact has risen due to the associated mortality and financial costs of therapy (Wilesmith et al, 1986). During the field trials conducted by the National Institute for Research in Dairying and completed some 15 years ago 87 clinical cases per 100 cows of mastitis were found (Bramley, 1984). In more recent years figures as low as 31.7 cases per 100 cows have been reported Blowey (1986). The

cost of a case of mastitis has been estimated at £40 by Blowey (1986) and £50 by Wilesmith et al (1986). Mastitis is therefore a financial burden to the farmer, with the average 100 cow herd suffering a yearly loss of around £2000 including the cost of the discarded milk.

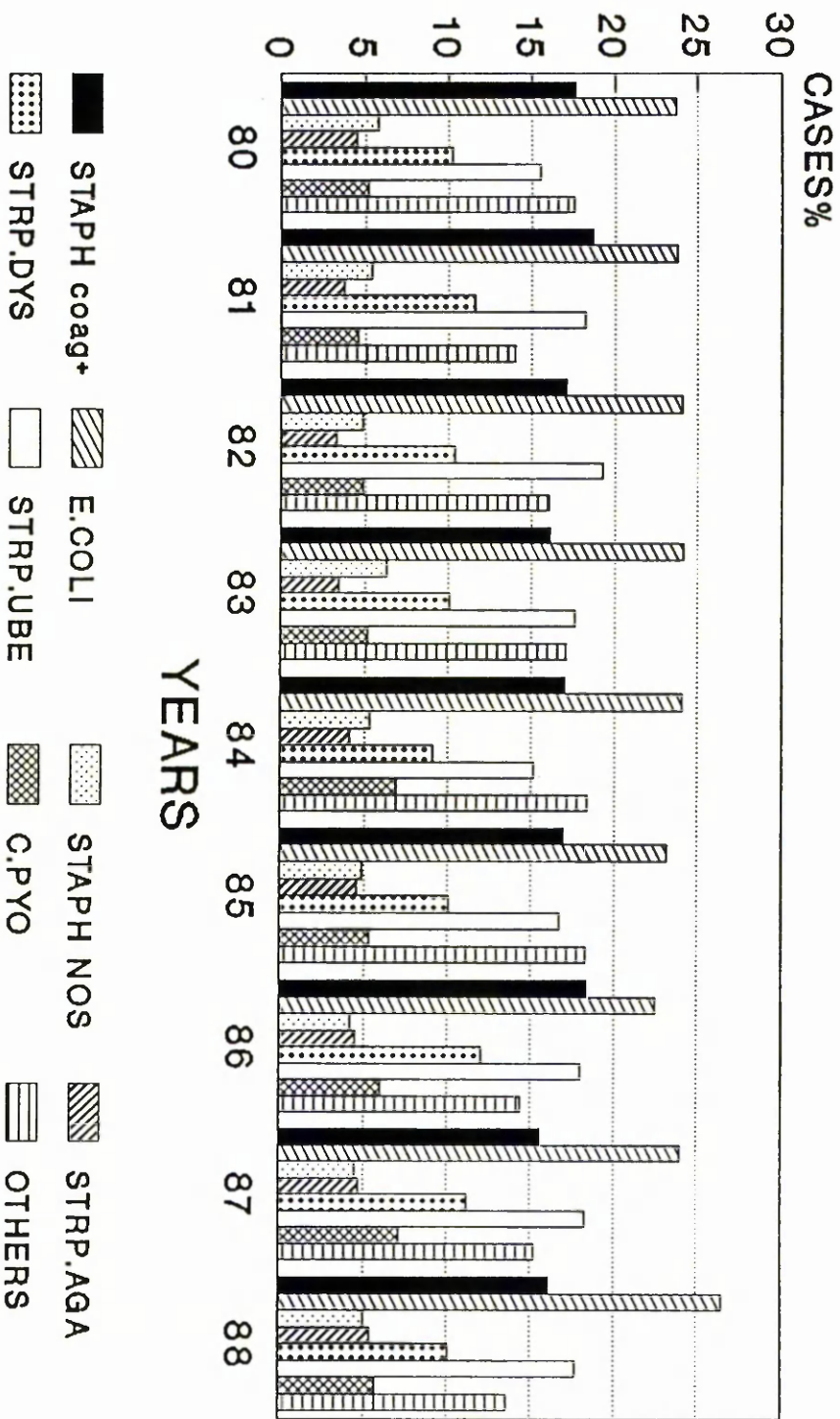
In the years since the earliest epidemiological studies mentioned above, a gradual change in the causal organisms of mastitis has been observed. Staphylococcus aureus has been supplanted by Escherichia coli as the most frequently isolated organism associated with mastitis (Table 1.19). These changes have been influenced by management changes and mastitis control measures (Wilesmith et al, 1986) and can be seen in Figures 1.6, 1.7, 1.8. 1.9 which give a breakdown of mastitis pathogen isolation and percentage of cases for UK and Scotland (Anon, 1989).

# MASTITIS U.K. (VIDA)

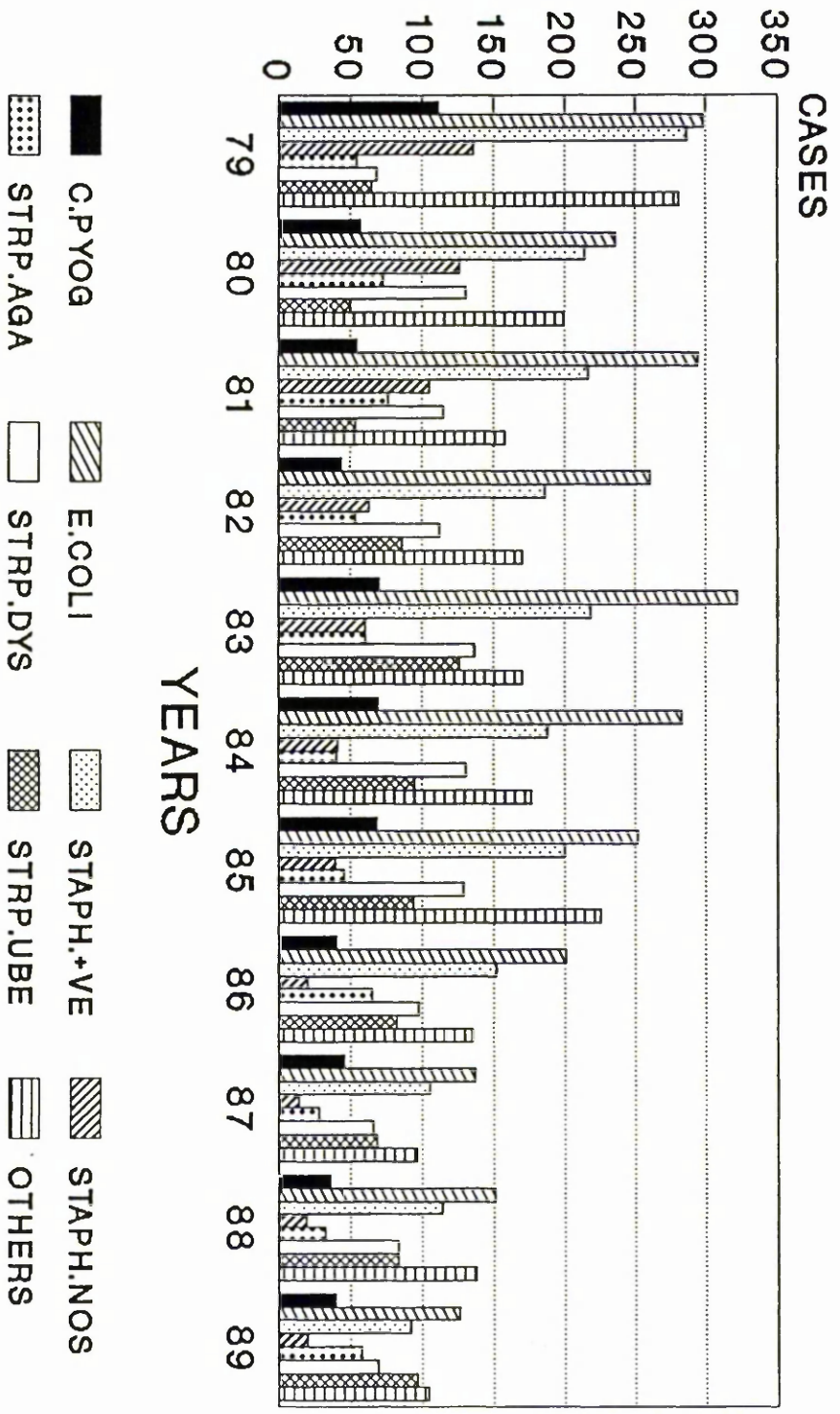




# MASTITIS U.K. PERCENTAGE BREAKDOWN



# MASTITIS SCOTLAND (VIDA)



# MASTITIS SCOTLAND PERCENTAGE BREAKDOWN

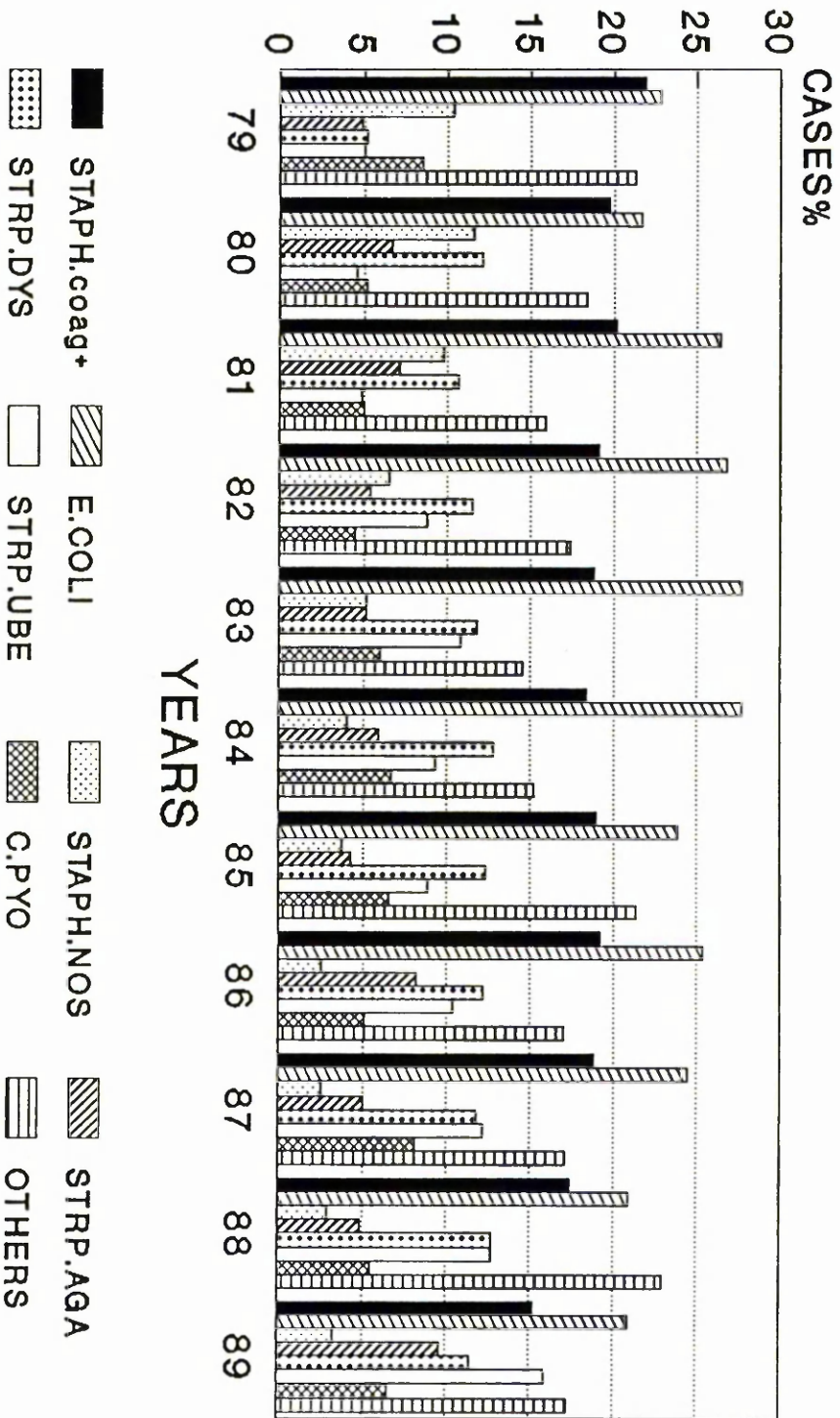


Table 1.19 Incidence of clinical mastitis by major pathogen

Year	% cases/year					
	A <sup>1</sup>	B <sup>2</sup>	C <sup>3</sup>	D <sup>4</sup>	E <sup>5</sup>	F <sup>6</sup>
Pathogen						
<u>Staph. aureus</u>	57.2	37.5	15.7	14.8	5.9	9.6
<u>Esch. coli</u>	5.8*	5.5*	19.4	18.3	9.9	16.2
<u>Strep. agalactiae</u>	5.3	3.1	1.8	1.4	-	8.0
<u>Strep. uberis</u>	5.9	17.7	13.4	16.9	26.2	0.2
<u>Strep. dysgalactiae</u>	13.1	20.1	8.6	8.9	5.4	4.8

\*coliforms

Year of research	Author
A - 1962/63	Dodd and Neave (1970)
B - 1967/68	Wilson and Kingwell (1975)
C - 1980	Wilsemith <u>et al</u> (1986)
D - 1982	Robinson, Jackson and Marr (1983)
E - 1983	Robinson, Jackson and Marr (1983)
F - 1985/86	Schukken, Grommers, Van de Geer, Brand (1989)

1-5 - UK  
6 - Netherlands

#### 1.12.4 Subclinical Mastitis

Subclinical mastitis is by definition not recognised clinically and is primarily diagnosed by somatic cell count (Appendix 5). Counts of greater than or equal to  $0.5 \times 10^6$  cells/ml with no clinical symptoms are generally taken as being indicative of sub-clinical mastitis. The Cowside California Mastitis Test (CMT) or Paddle test (Appendix 2) is also a cheap and useful tool in the diagnosis of this type of mastitic infection. However, it is not highly accurate.

#### 1.12.4.1 Incidence of Subclinical Mastitis

During the Mastitis Field Experiment 3 research project (Kingwill, Neave, Dodd, Griffen and Westgarth, 1970) 55% of cows presented with subclinical mastitis. Nine years later this had been reduced to a 32% incidence (Wilson and Richards, 1980). There have been no recent surveys of this carefully controlled type but as bulk somatic cell counts are an indication of subclinical mastitis incidence it can be presumed that incidence continues to fall (Figure 1.2) with a drop of 26% (mean for GB Boards) in the ten years from 1977. Booth (1988a) suggested that the current level may be less than 25%.

The National Mastitis Awareness Campaign has therefore succeeded in making farmers more cognisant with cell counts and TBC's and is slowly reducing the incidence and prevalence of clinical and subclinical mastitis.

#### 1.12.5 National Mastitis Awareness Campaign

In 1971 the Milk Marketing Board began to monitor Somatic Cell Counts from the bulk tank on a regular basis and in 1972 the National Mastitis Awareness Campaign was introduced (Booth, 1988b).

The Scheme was initially a six part programme aimed at controlling mastitis.

1. Good stock management. Nothing can replace the well trained careful stockman.

2. Prevention of infection by sound hygienic routines during milking for example teat dipping with a suitable teat dip after each milking.
3. Prompt treatment of existing clinical infections on every cow at drying off.
4. Cull incurable cows, for example, cows with chronic or recurring mastitis and also those with badly damaged udders.
5. Monitor progress with monthly bulk milk cell counts and react to findings.
6. Efficient milking, maintain and test milking machines regularly.

The control measures have now been restricted to a "5 part plan" encompassing 1, 2, 3, 4 and 6 of the above. These measures properly implemented can control most mastitic infections although the environmental organisms such as Esch. coli and Strep. uberis have proved more intractable than the Cow-side infections such as Strep. agalactiae and Staph. aureus (Booth, 1988b). In view of present advice it is surprising that immediate treatment of clinical cases was not one of the major components of this very first programme.

#### 1.12.5.1. Progress

From the introduction of the campaign in 1972 until the latest survey carried out in 1983 the number of herds practising "teat disinfection" has risen from 17% to 68% with 90% now using dry cow therapy and 66% of herds carrying out milking machine testing

(Marshall, 1984). Application of the practices have thus doubled and presumably continue to increase (Booth, 1988c).

1.12.6. Environmental Effects on Mastitis.

Inevitably cows are subjected to a large number of environmental influences. These include housing, bedding and hygiene at milking and also seasonal factors. These can be simply subdivided into the external and internal environment which can be further subdivided (Tables 1.20.and 1.21).

Table 1.20 External Environment

<u>Regional</u>	<u>Local</u>
climate	topography
geography	shelter
tradition	pasture

Table 1.21 Internal Environment

<u>General herd environment</u>	<u>Specific herd environment</u>	<u>Milking environment</u>
housing	stall design and function	methods
indoor climate	bedding	equipment
feeds used	feeding regime	hygiene
management and stockmanship	manure/slurry disposal	walkways/ collecting pens

#### 1.12.6.1 The External Environment

This will not be discussed in detail as storage feeding eliminates the majority of external environmental influences (Roberts and Leaver, 1987).

##### (a) Climate, Geography and Tradition

Dairy cattle are susceptible to extremes of climate and outbreaks of mastitis have been ascribed to chilling of the udder (Ewbank 1968; Sainsbury and Sainsbury 1978 and Bakken 1981) and possibly by the lowering of disease resistance.

Geography determines population densities and so can have some interesting effects upon mastitis. Small isolated communities have small herds and milk production is thus generally less intensive than in areas such as the UK where market forces have encouraged larger herds. In the latter case machine milking and other less labour intensive practices are required. Furthermore, because of more sophisticated marketing there is a greater imposition of hygienic standards. All of these factors have implications on mastitis (Anon, 1987)

##### (b) Topography, Shelter and Pasture

These aspects, the so-called 'local environmental factors', have not been widely researched to determine their influence on the incidence of mastitis. 'Summer mastitis' is the major exception



to this and its association with Hydrotea irritans has been well documented (Thomas, Over, Vecht and Nansen, 1987).

Although little scientific evidence exists, practical experience has shown that local weather conditions, muddy footpaths and barbed wire can be contributory factors in the development of udder disease and hence predisposing to mastitis (Anon,1987).

#### 1.12.6.2 The Internal Environment

The systems for housing and milking dairy cows differ markedly worldwide according to climate, topography and tradition. However, there are essentially three forms:

1. The cow house or byre
2. The milking parlour (with or without loose housing)
3. The milking bail (which may be fixed or portable)

Generally there is a difference in how each system contributes to mastitis, although it has been recognised that housing of any type increases mastitis incidence (Bramley, 1982), the effects being most marked for such pathogens as coliforms and Strep. uberis (Bramley, 1989). Wilesmith et al (1986) stated that 65% of mastitis cases occur between October and March. Studies of interactions between the types of housing and udder disease usually revealed that the highest rates of mastitis were found in housed herds especially when housing in byres ( Ekesbo, 1966 and Bakken, 1981). Continuous housing throughout the year contributes to higher frequencies of mastitis (Anon 1987).

The high incidence of mastitis in byres has been attributed to poor mobility and an inability to adjust to the herds social hierarchy. The resultant stress is believed to influence the effectiveness of udder resistance factors. Essentially there are two areas of importance:

- a. milking hygiene
- b. housing hygiene

(a) Milking Hygiene

It has been shown that proper hygienic controls at milking reduced the spread of pathogenic bacteria and cross infection between cows of the major mastitic pathogens Staph. aureus and Strep. agalactiae has been reduced by application of these controls (Wilson and Kingwill, 1975 and Bramley, 1981). Strep. dysgalactiae cross infection was also controlled by these measures ( Wilson and Kingwill, 1975 and Bramley, 1981) but not entirely as this organism has also been demonstrated as an essential player in the "summer mastitis" syndrome (Stuart, Buntain and Landridge, 1951; Tarry, Wilson and Stuart, 1978 and Hillerton, Bramley and Broom, 1983). While teat disinfection post milking had no effect on the transfer of bacteria during or between milkings it was nevertheless found to decrease the rates of new infection by reducing the teats exposure to bacteria (Grindal, 1988a).

It has been suggested that irradiation of these organisms can be achieved by segregation, treatment and culling of infected animals and proper hygienic controls and certainly this has proved possible for Strep. agalactiae (Bramley, 1981). Storage feeding allows for well controlled grouping of cattle (Roberts and Leaver, 1987) and can thus aid any segregation and treatment policies.

The role of milking machines in mastitis have been discussed by Thiel (1975) O'Shea (1983) and Grindal (1988a and b).

(b) Housing hygiene

Housing generally is correlated with an increase in teat lesions (Keller, 1977; Karlsson and Gustafsson, 1978 and Bramley, 1989) and also increased exposure to certain bacteria (Francis, 1989 and Bramley, 1989) thus housing generally leads to a higher incidence of mastitis. Environmental bacteria and their sources are detailed in Table 1.22.

Table 1.22 Common Environmental Pathogens and their Sources

Organism	Source
<u>Escherichia coli</u>	Faeces, contaminated bedding, widespread environmental contaminant
<u>Streptococcus uberis</u>	Faeces, cow skin, tonsil, vulva, rumen, belly, lips and bedding.
<u>Klebsiella sp</u>	Faeces, bedding, ubiquitous in nature.
<u>Pseudomonas aeruginosa</u>	Soil, water, faeces, ubiquitous in nature.
<u>Bacillus sp</u>	Spores widespread, faeces.
<u>Streptococcus dysgalactiae</u>	Flies ( <u>Hydrotea irritans</u> ), faeces, throat, vulva.
<u>Corynebacterium pyogenes</u>	Flies ( <u>Hydrotea irritans</u> ), faeces, mouth, vaginal discharge.

Much research has been undertaken on the contributory factors of housing on mastitis. These have been extensively documented (Anon 1987 and British Mastitis Conference, 1989). The size of cubicle and style of partition are very important contributory factors in mastitis and the interactions between stall dimension, type of bedding and dung removal have all been extensively researched (Ekesbo 1966; Bakken 1982 and Dodd, Higgs and Bramley, 1984). In general the higher the level of comfort the less mastitis (Klastrup, 1978; Wilson and Richards 1980 and Bakken, 1982).

Studies on the ability of environmental bacteria to multiply in bedding have also been carried out (Jasper, 1980 and Francis, 1989). Francis (1989) concluded that the greatest multiplication

of organisms in bedding occurred in the first 12 hours, the extent varying according to material (Table 1.23).

Table 1.23 Multiplication rates in bedding

Organism	Type of bedding		
	Old Manure	Straw	Hardwood Chips
Klebsiella sp	x 2500	x 1800	x 100
Coliforms	x 1000	x 180	x 25

All of the organisms also multiplied in paper and pine sawdust. The multiplication rate of Strep. uberis was much smaller than that of other organisms but occurred in all bedding materials.

The act of lying down creates an incubator effect under the cows udder with temperatures rising to approximately 37°C. Bacteria thus have ideal conditions to multiply and the increased contact time between teats and bedding give bacteria increased penetration time. Furthermore Francis and Sumner (1979) reported that cows lie down for up to 14 hours per day and higher yielding cows tend to lie down longer. The ambient temperature also effects the amount of lying time. When temperatures fall from November to January cows lie down for greater periods. During the summer, storage fed cows may have an increased incidence of 'coliform' mastitis due to the increased growth rate of coliform bacteria in bedding during the warmer period (Russell, 1983). Roberts and Leaver (1987) stated that storage feeding had no detrimental effect on cow health but found a higher than average

incidence of mastitis in the first year of their experiment in comparison to the cows in the herd which were not in the experiment.

Housing cows without litter has also been associated with a high incidence of mastitis from 6.8 to 17.3% in loose housing and 8.6 to 6.8% in the cow house (Ekesbo, 1966). This higher incidence is possibly due to the increase of teat tramps from 1.4 to 10.1% in loose houses with and without bedding respectively and from 3.9 to 10.1% in the byre/cow house with and without bedding.

### 1.13 MINERAL IMBALANCES

#### 1.13.1 Introduction

The minerals calcium, magnesium and phosphorus play an important part in metabolic and nutritional disease in cattle. Any disturbance of this mineral balance can lead to disease, the most common conditions being milk fever and grass tetany. Both are acute conditions and can be fatal unless treated. The first is primarily the result of hypocalcaemia and the latter hypomagnesaemia. Normal blood concentrations of these minerals are shown in Table 1.24.

Table 1.24 Normal Blood concentrations of calcium, magnesium and inorganic phosphorus

Normal range concentrations in blood plasma\*

	Metric Units	S I Units
Calcium	8.5-11.5 mg/100ml	2.12-2.87 mmol/l
Magnesium	1.8- 3.2 mg/100ml	0.74-1.32 mmol/l
Inorganic phosphorus	3.5- 6.0 mg/100ml	1.13-1.94 mmol/l

\*Supplied by Scottish Veterinary Investigation Service, SAC.

### 1.13.2 Milk Fever

Milk fever is a complex disease but it is essentially a failure of calcium haemostasis and is most commonly seen around parturition and in early lactation.

In the normal cow some 8 g of calcium is maintained in the body fluids by homeostasis at approximately 10 mg/100 ml in blood by a constant interchange with bone. Hence there is calcium input to blood from feed and bones whilst at the same time an equal amount is removed in faeces (endogenous calcium) or is again incorporated into new bone formation. In other words bone degradation by osteoclast activity acts as the homeostatic control. This is influenced by a variety of factors but most particularly thyrocalcitonin and parathyroid hormone (Boda and Cole 1954; Payne 1967). Probably the most important role of parathyroid hormone is its role in the stimulation of hydroxylase production in the kidney. This enzyme is the catalyst for the second step in the conversion of vitamin D to its active hormonal form, 1,25 dihydroxyvitamin D<sub>3</sub> which assists in the absorption of calcium from the gut and its mobilisation from bone (Kelly,

1988). The in utero calf has an increasing demand for calcium until at full term 0.2 g of calcium per hour is needed that is almost 5 g per day. This requirement is replaced at parturition by a fivefold increase in demand for lactation (1 g calcium per hour) thus 12.5% of the total body calcium is removed per hour in the lactating cow. Most if not all cows fail to meet this increased calcium requirement being unable to mobilise bone degradation sufficiently. As a result a transient hypocalcaemia occurs at this time (Hove, 1986). This can progress to the clinical condition of 'milk fever' in some cows.

#### 1.13.2.1 Incidence of Milk Fever

The national incidence of milk fever in the UK dairy herd has increased from an estimated 3-4 per cent in 1960 (Leech, Davies, MacRae and Withers 1960) to around 9 per cent in 1975 (Mullen, 1975). VIDA returns (1985-1989) reflect this variability (Figures 1.10 and 1.11) and also demonstrate differences between regions and season, the overall figure proposed by Kelly (1988) of 7% thus seems to be a reasonable estimate.

#### 1.13.2.2 Predisposition to milk fever

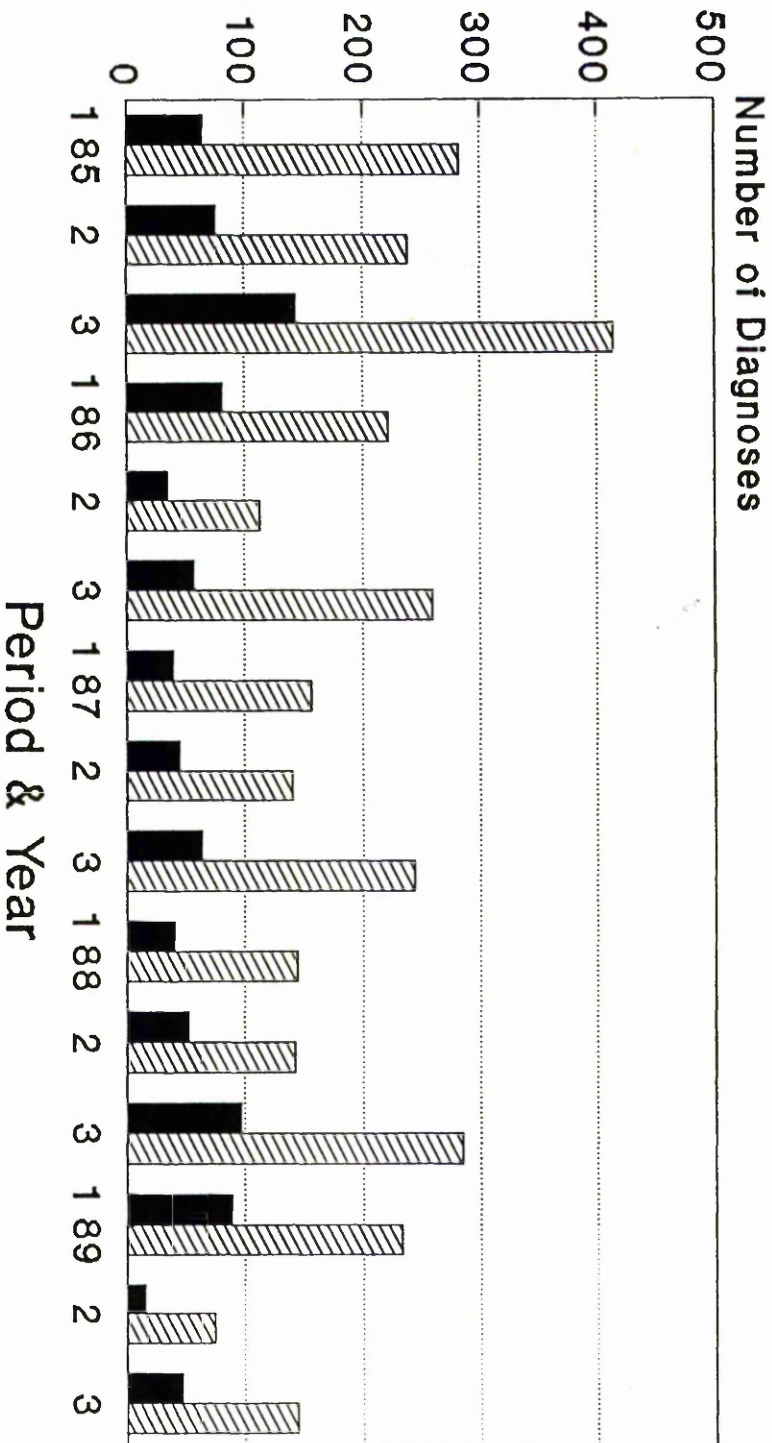
Many factors predispose the dairy cow to milk fever.

##### a) Breed

Predisposition to milk fever is heritable (Dyrendahl, Henricson and Jonsson 1972; Jonsson 1978 and Harris 1981). The Jersey is the most susceptible of the major European dairy breeds (Hibbs Krauss, Monroe and Sutton, 1946; Harris, 1981; Littledike, Young



# HYPOCALCAEMIA In G.B. & Scotland



1-Mar, Ap, May 2-Ju, Jul, Au 3-Sep, Oc, Nov

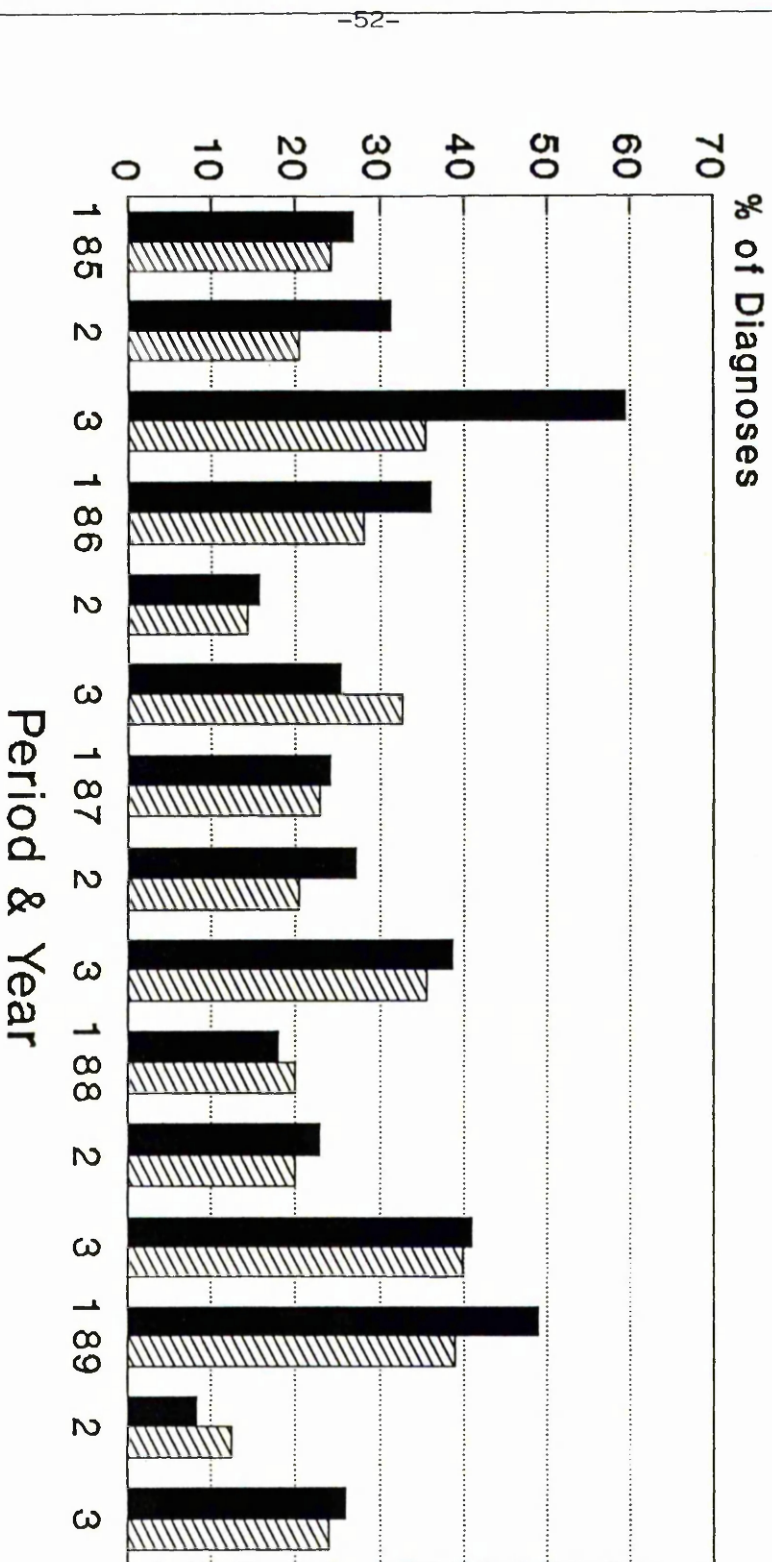
■ Scotland    ▨ Great Britain

FIGURE 1.11

# HYPOCALCAEMIA

In G.B. & Scotland

% OF TOTAL SUBMISSIONS



■ Scotland    ▨ Great Britain

1-Mar, Ap, May 2-Jul, Jul, Au 3-Sep, Oc, Nov

and Beitz, 1981, and Kelly, 1988).

b) Age and Milk Yield

High yielding cows have an increased susceptibility to parturient hypocalcaemia (Jonsson, 1960; Rogers, Grainger and Earle, 1979 and Curtis, Erb, Sniffen and Smith, 1984) but by no means do all high yielders succumb.

First calf heifers rarely exhibit parturient paresis, but there is increased susceptibility to milk fever with age (Little and Wright, 1926; Payne, 1970; Jonsson 1978; Kelly, 1988 and MacPherson, Moisey, MacLeod and Roberts, 1988).

This increase is possibly due to:

- i. Increasing yield with age
- ii. The reduction in osteoclast activity with age leading to lowered ability to respond to emergency demand (Payne, 1967, and Mayer, Ramborg and Kronfeld, 1969).

(c) Diets

Prepartal diets high in calcium cause calcium homeostasis to be governed by absorption from the intestinal tract. Calcium mobilisation from bone at this time is low as the high dietary intake of calcium stimulates secretion of thyrocalcitonin which reduces this mobilisation. The sudden increase of demand for calcium at the commencement of lactation results in a precipitate fall in plasma calcium and emergency measures to maintain calcium levels by diverting calcium from bone fail due

to the relatively high concentration of thyrocalcitonin which inhibits mobilisation of calcium from the bone (Yarrington, Capen and Black 1977). Impaired gastrointestinal function also occurs at this time. Conversely diets low in calcium cause mobilisation of calcium from bone mediated by stimulation of the parathyroid gland producing parathormone which in simple terms acts as an antagonist to calcitonin and therefore emergency calcium is more easily obtained from bone at parturition (Boda and Cole, 1954; Boda, 1956; Jonsson, 1978 and Goff, Horst, Beitz and Littledike, 1988).

A diet high in phosphate in the late stage dry period has been implicated in milk fever whilst low dietary phosphate in the presence of high calcium assists in prevention (Curtis et al, 1984). Thus one must concur with Kelly (1988) that opinion differs as to the merit of feeding high phosphate diets to dry cows. The importance of the Ca:P ratio in pre-partal diets was stressed by Boda and Cole (1954). However, more recently Jonsson (1978) found that Ca:P ratios were not important and Kelly (1988) also considered this to be the case when Vitamin D status was kept high. Overfeeding of dairy cows prior to calving also predisposes to milk fever (Jonsson and Pehrsan, 1983 and Pehrsan, Jonsson and Anderson 1986).

There is little literature relating dietary protein to incidence of parturient hypocalcaemia but Curtis, et al (1984) found that high protein diets decreased the incidence of milk fever.

d) Disruption of alimentary activity

Periods of alimentary stasis commonly occur at parturition and can lead to interruptions in the uptake of calcium from the alimentary tract. This interruption can lead to clinical hypocalcaemia. Older cows frequently suffer from inappetance close to parturition and thus dietary calcium uptake is cut off (Moodie and Robertson 1961 and 1962, and Payne 1967). There is a possibility that oestrogen may induce alimentary stasis (MacFarlane 1967). If alimentary stasis occurs a lag period follows which prevents calcium uptake from the gut and parturient hypocalcaemia results if sufficient calcium is not available from bones (Littledike et al, 1981).

1.13.2.3. Hypomagnesaemia and Hypophosphataemia in relation to Milk Fever

An imbalance of either or both magnesium and phosphate can also predispose to hypocalcaemia (Littledike, et al 1981). Calcium mobilisation is reduced in hypomagnesaemic steers, dry cows and lactating cows and plasma calcium concentrations consequently decline even when the degree of hypomagnesaemia is only slight (0.76 mmol/litre). Contreras, Manston and Sansom, (1982) and Van de Braak, Van't Klooster, Malestein (1987) suggested that insufficient magnesium during the dry period, even in the absence of hypomagnesaemia for much of that time, can decrease the cows ability to mobilise calcium at parturition and thus cause milk fever.

Hypophosphataemia has also been implicated as a predisposing factor in milk fever (Marr, Moodie and Robertson, 1955 and Littledike, Whipps and Schroeder, 1969).

#### 1.13.3. Grass Tetany.

Cattle and sheep receiving inadequate magnesium in their diets are susceptible to grass tetany. The disease has been categorised into three forms depending on clinical signs (Rogers, 1979). Figures 1.12 and 1.13 show the incidence of reported cases of hypomagnesaemia and also demonstrate regional and seasonal differences.

- (a) Tetanic Syndrome type - characterised by nervousness, muscle twitching, ataxia, convulsions, recumbency with spasms (opisthotonos).
- (b) Paretic type - listlessness, staggering, paresis, recumbency and coma without spasms.
- (c) Subclinical types - depression of appetite and milk yield, slight nervousness, anaemia and udder oedema.

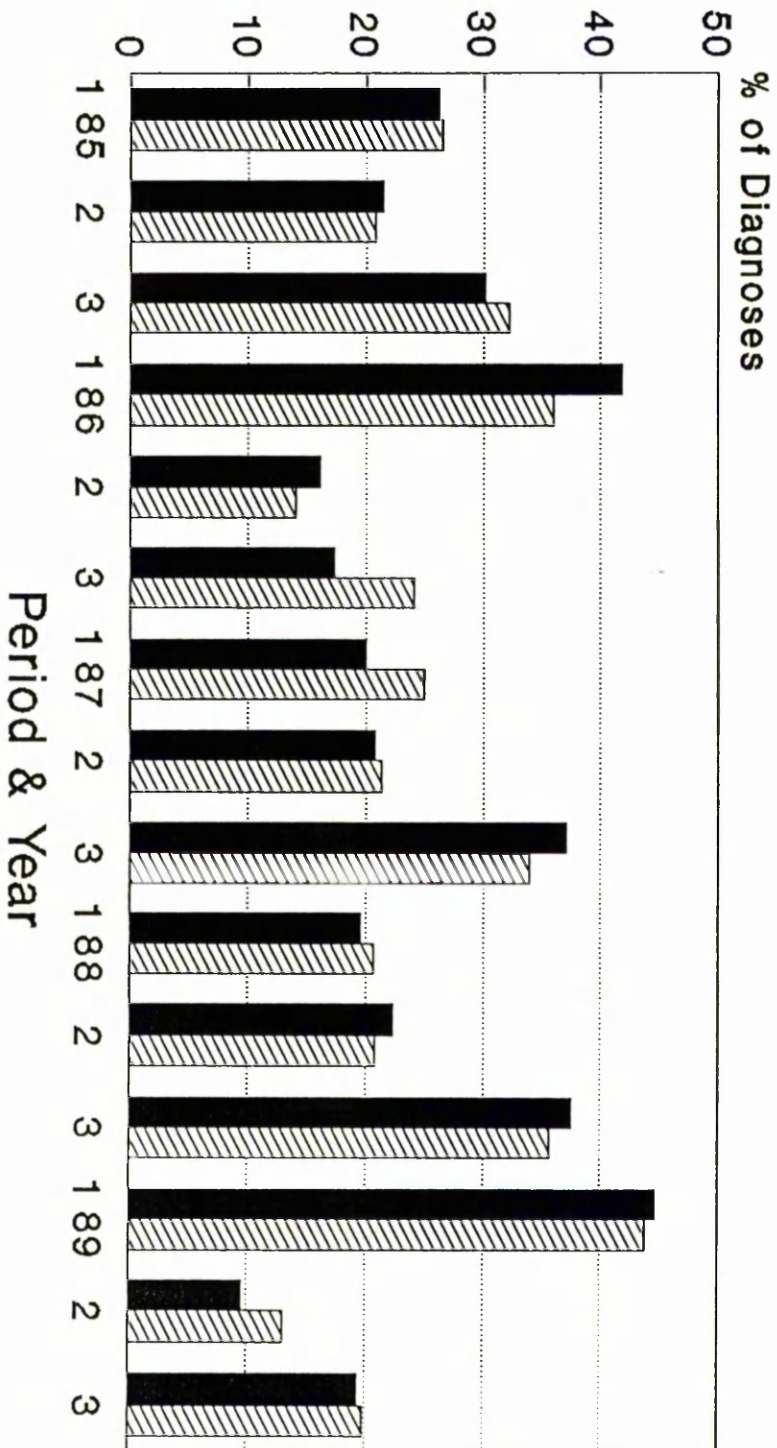
Similarly the management of the cows has allowed a further subdivision of the condition (Littledike, et al 1981).

- i. Spring type: affecting cows a few days after turnout.
- ii. Winter type: affecting cows fed winter rations in confinement.
- iii. Out-winter type: affecting cows in late winter that have been maintained throughout the winter on sparse pasture plus supplementary hay.

# HYPOMAGNESAEMIA

In G.B. & Scotland

**% OF TOTAL SUBMISSIONS**

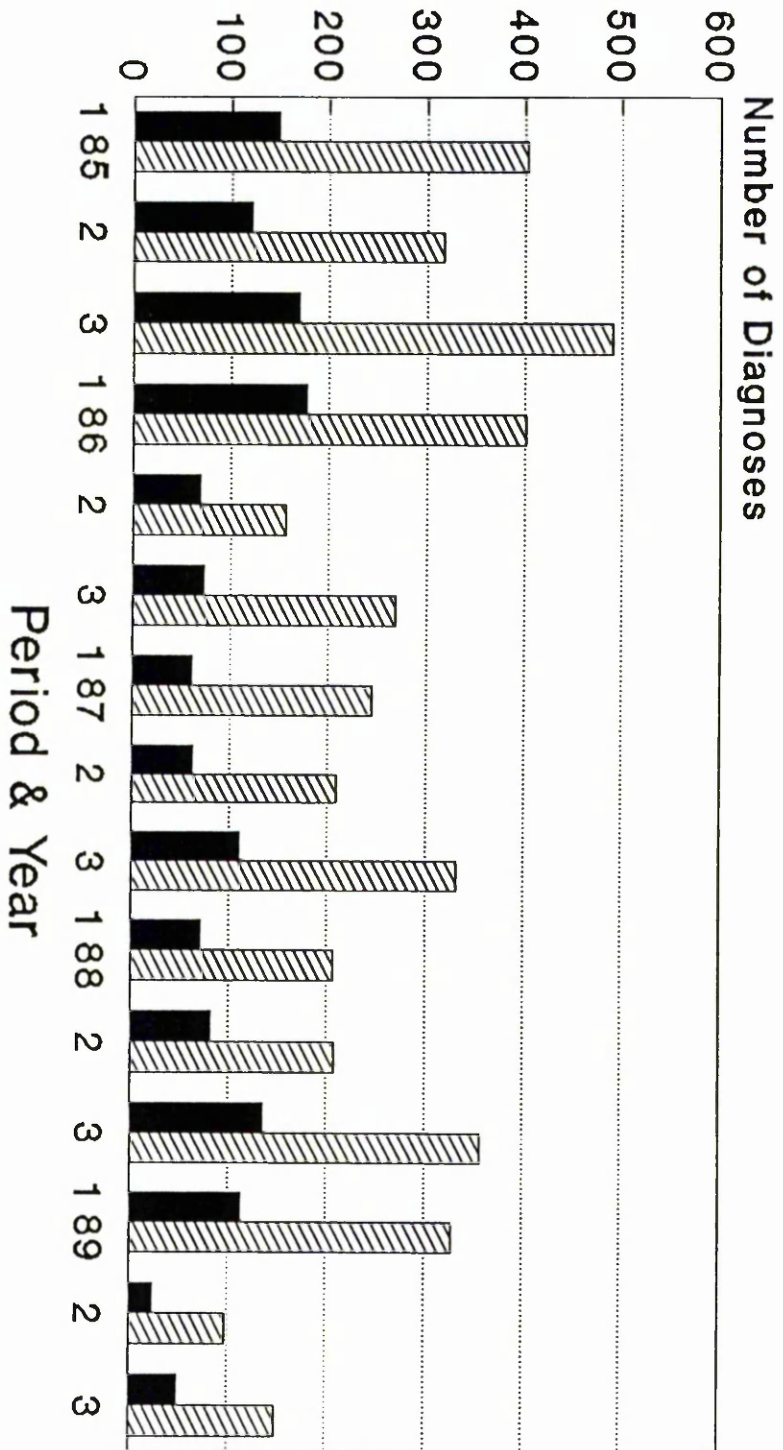


■ Scotland    ▨ Great Britain

1=Mar,Ap,May 2=Ju,Jul,Au 3=Sep,Oc,Nov



# HYPOMAGNESAEMIA In G.B. & Scotland



■ Scotland    ▨ Great Britain

1=Mar, Ap, May 2=Ju, Jul, Au 3=Sep, Oc, Nov



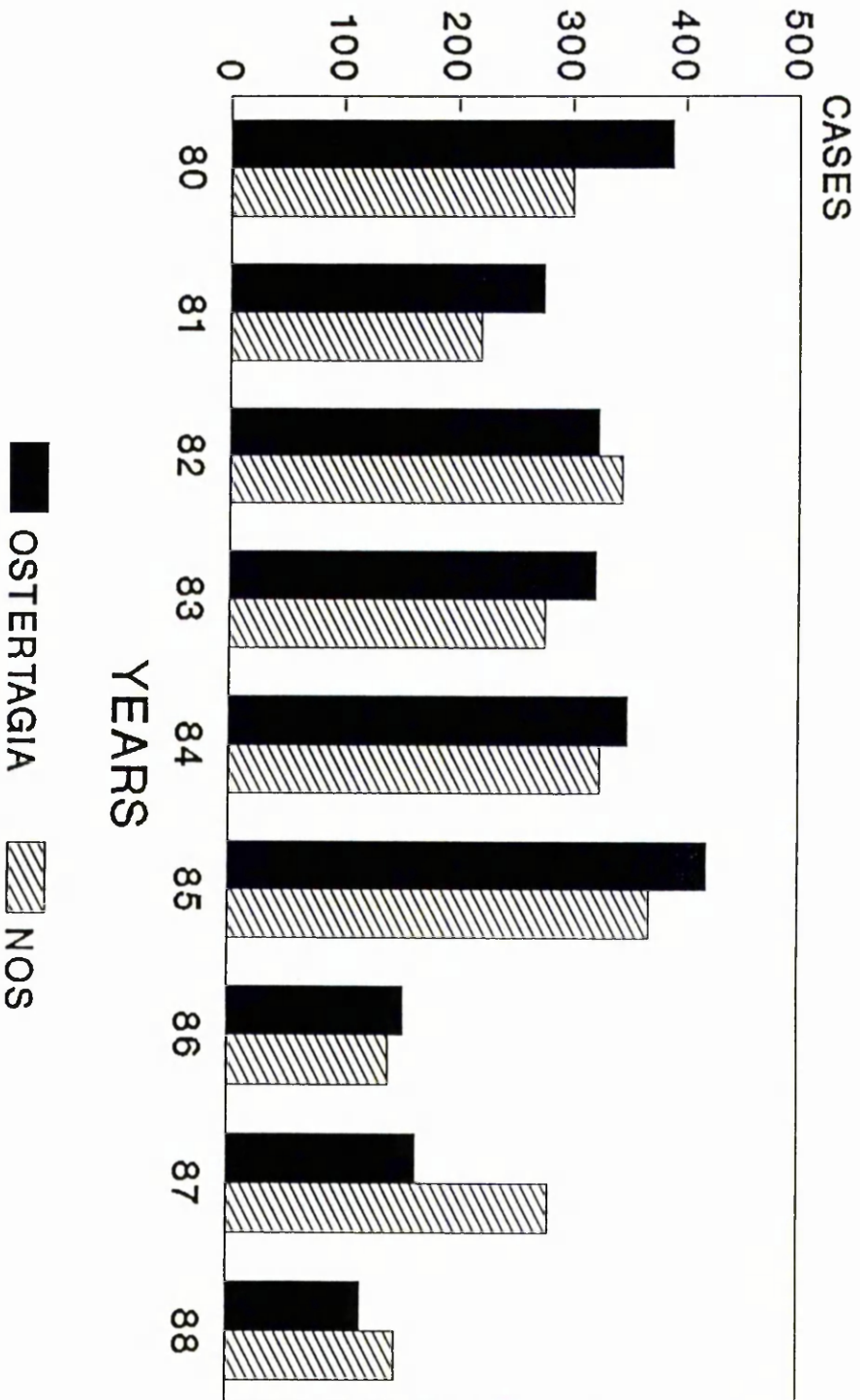
Grass tetany is thus of major importance as it can rapidly be fatal creating economic loss to the farmer. Payne (1967) considered it to be a sequel of increased productivity from grassland and suggested that there were three main factors. Firstly there was increased fertiliser usage particularly potash which aggravated hypomagnesaemia and that often this was then compounded by increased grazing pressure and finally minimal concentrate feeding. These factors were also demonstrated as important in Scotland by Butler (1963).

#### 1.14 NEMATODES

##### 1.14.1 Introduction

In Britain, many different roundworm parasites of cattle have been identified but few are regarded as economically important. Ostertagia ostertagi and Dictyocaulus viviparus are considered to be the most important pathogens in south west Scotland, the former affecting the abomasum, the latter the lungs (Armour, 1970 and Oakley, 1982). The incidence of clinical Parasitic Gastroenteritis (PGE) in the UK is very difficult to estimate but it is certainly high and as can be seen from VIDA returns (1985-1988, Figure 1.14) can vary quite considerably between years. Bairden and Armour (1981) quoted an incidence of infection of 83% in abomasa examined, therefore, clinical infection rates are high.

# PARASITIC GASTROENTERITIS (VIDA)



#### 1.14.2 Ostertagia ostertagi

Bovine ostertagiasis has been extensively investigated and all authors agree that Ostertagia ostertagi is the most pathogenic and economically important of the gut roundworms in the UK (Armour, Bairden, Duncan, Jennings and Parkins, 1979, Bairden and Armour, 1981 and Chalmers, 1983).

The presence of large numbers of O. ostertagi species in the abomasum results in extensive pathological and biochemical changes and severe clinical signs, in particular a profuse watery diarrhoea.

##### 1.14.2.1 Pathogenesis and epidemiology

It is now known that these changes are maximal when the larvae reach maturity and begin to emerge from the gastric glands. Two types of disease are caused by these nematodes.

1. Type I - accumulating infective third stage larvae ( $L_3$ ) from hatchings in late Spring leads to the emergence of adult forms 18 to 21 days after ingestion of  $L_3$  with abomasal damage (Murray, Jennings and Armour, 1970). This usually occurs from July until October (Armour, 1970).

Although primarily a disease of young dairy cattle, ostertagiasis can affect older animals particularly if there is no previous exposure to the disease. Armour (1970) stated that there was no age immunity. Furthermore acquired immunity would seem to be

slow to develop, field trials showing that a significant level of immunity was not present in calves until the end of their first grazing season. This immunity falls during inwintering but after turnout the following spring it is rapidly re-established on ingestion of overwintered  $L_3$  and the disease is usually of a transient nature with the established worms being quickly expelled (Armour, 1970). Strong acquired immunity developed in cattle after the second and third grazing year and adult stock in endemic areas are thought to be highly immune to reinfection (Armour 1974).

2. Type II - this type of ostertagiasis is believed to be due to the larvae undergoing a diapause similar to that of insects. This inhibition at the early fourth stage ( $EL_4$ ) is believed to be due to the falling temperatures of late autumn.

Adult forms emerge 14 to 17 days after the inhibited  $EL_4$  stages resume their development (Armour and Bruce, 1974). The  $EL_4$  stages begin their development in successive waves thus causing repeated damage to the abomasal mucosa resulting in a more severe form of the disease in comparison to Type I. Type II ostertagiasis occurs from March to May (Armour, 1974).

The resulting damage to the abomasal wall by both types I and II ostertagiasis results in impaired digestion due to the elevation of the abomasal pH to 5 which results in impaired peptic activity and diarrhoea. Plasma/serum pepsinogen levels rise at this stage

and this can be used as a diagnostic tool. Adults can develop and lay eggs within 3 to 4 weeks (Armour, Bairden, Oakley and Rowlands, 1988) and can be detected in the faeces by direct microscopy or more usually numbers are estimated by flotation (Gordon and Whitlock, 1939 and Armour, 1974).

## 1.15 PARASITIC BRONCHITIS

### 1.15.1 Introduction

Dictyocaulus viviparus the causal agent of parasitic bronchitis, also known as husk or hoose, is the only roundworm responsible for respiratory disease in Britain.

#### 1.15.1.1 Pathogenesis and Epidemiology

Adult female lungworms lay embryonated eggs in the respiratory tract of the host, these may hatch but development beyond the second larval stage cannot take place at body temperature (Daubney, 1920 and Taylor and Michel, 1952).

First stage larvae ( $L_1$ ), passed in faeces contaminate the area beyond the faecal pat and develop through the second ( $L_2$ ) and third ( $L_3$ ) stage to the infective stage. The  $L_3$  stage has the ability to overwinter on pasture (Jarrett, McIntyre and Urquhart, 1954; Jarrett, McIntyre, Urquhart and Bell, 1955; Allan and Baxter, 1957 and Downey, 1973) and thus can infect cattle at turnout.

The infective larvae migrate on pasture for example in water and

on cattle feet to contaminate most areas. Pilobolus sporangia has been shown to cause larval dispersion due to fungal spore explosion (Robinson, 1962). Jorgensen (1982) suggested that suppression of P. sporangia was associated with reduced larval counts on pasture.

Infective larvae are ingested by cattle and the disease cycle begins. Primary infection can be divided into four stages (Urquhart, Jarrett and McIntyre, 1973).

1. Penetration Phase.
2. Prepatent Phase.
3. Patent Phase.
4. Post Patent Phase.

Biochemically the disease is a compensated respiratory acidosis (Fisher and McIntyre, 1960). The partial pressure of oxygen of arterial blood may be 30% (normal 97-98%) in infected animals and this probably accounts for the poor exercise tolerance of affected animals (Oakley, 1982).

Adults can develop and start laying within 3-4 weeks of ingestion (Armour, 1974 and Armour et al, 1988). The eggs hatch in faeces and the presence of larvae in faeces can be detected by the Baermann Technique (Appendix 8).

CHAPTER 2  
MATERIALS AND METHODS

## 2.1. INTRODUCTION

The Materials and Methods described below were all used in this study. A short description of the farm and farming methods has also been included to give the reader a broader understanding of the background to the research. Analytical Techniques are detailed in Appendices 1-8.

## 2.2 THE FARM

The Crichton Royal Farm is part of the Scottish Agricultural College and lies in the South West of Dumfries on the banks of the River Nith (Map Ref NX983 736 1). The farm is 253 ha in size and lies between 0-75 metres above sea level. Soil ranges from sandy loam in the east to silty clay in the west and the average rainfall is 1041 mm per annum. It is self sufficient in silage production and cereals (120 ha) and fodder beet (5 ha) are also grown and utilized as feed stuff. At the time of this study the farm was basically two separate self sufficient dairy units, the Acrehead unit milking 140 cows and being used for systems analysis and the main Crichton steading milking 200 cows carrying out strategic research and development.

### 2.2.1 Grazing and Wintering

Turnout usually occurs in mid April and by May there is no housing, the cows are continuously grazed at around 7.4 cows/ha in the late spring and early summer. After first and second cut silages have been made the number of cows per hectare are reduced to 3.5. There are of course experimental exceptions. Inwintering generally begins around mid October.



### 2.3 THE COWS

Although the general farm policy has been to use purebred Friesian/Holstein cows a group of Jersey cross Friesians (Jx) were bred in 1983. These animals and a group of purebred "control" Friesians were the basis for this and other studies of crossbred cattle.

Purebred Friesians were selected as dams for the breeding of both control and Jx and were artificially inseminated (AI) to calve in the Autumn of 1983. The dams were inseminated on alternate days with either Jersey or Friesian semen. Three Jersey and three Friesian (part Holstein) bulls were used in rotation to cover all cows. Details of the genetic qualities of the bulls are shown in Tables 2.1 and 2.2.

Table 2.1 Bulls - Improved Contemporary Comparisons (ICC)

#### Jersey Bulls

FT - Fyn Tved (Danish)  
NC - No Capo (Danish)  
ECS - Ellerdine Cindy Snowball (British)

	Weighting	Milk Yield	Fat (kg)	Protein (kg)	Fat (%)	Protein (%)
FT	42.7	393	40.4	16.3	0.54	0.07
NC	4.8	-1.01	-0.5	-3.3	-0.17	-0.03
ECS	452.2	249	18.5	8.5	0.05	0.07

Table 2.2 Friesian Bulls

B Pros - Bartonhoo Prosperity (British)  
BP - Bartonhoo Plutonium (British)  
DP - Dalesend Performer (British)

	Weighting	Milk Yield	Fat (kg)	Protein (kg)	Fat (%)	Protein (%)
BPros	614.1	494	17.3	12.2	-0.01	-0.09
BP	606.2	793	25.5	17.5	-0.07	-0.17
DP	3681.0	254	14.4	6.6	0.11	-0.05

There were 117 first inseminations, 53 to a Jersey bull and 64 to a Friesian bull. Initially all returns were put to the Friesian bull but subsequently a Hereford bull was used as a natural service sweeper.

## 2.4 THE CALVES

### 2.4.1 Allocation of Calves to Experiment

No selective allocation of Jx heifers or Friesian heifers was made.

### 2.4.2 Rearing of Youngstock

Calves were left with their dam for 24 hours after birth and in addition to natural suckling each calf was given 2 litres of 'bulked' colostrum by stomach tube and was weighed and ear tagged within 9 hours of birth. At the end of this 24 hour period calves were removed from their dam and placed in individual pens and fed cows milk twice daily for 4 to 7 days. During this period peripheral blood was removed for the estimation of colostral immunoglobulin uptake by the Zinc Sulphate Turbidity Test (ZST) (Appendix 1).

Subsequently the calves were rehoused individually and fed 3 litres of warm water with 450 g of commercial high-fat milk powder once per day until weaning 28 days later. Calf concentrates, water and hay were also available at all times. Following weaning at 28 days the calves remained in their individual pens to enable feed intakes to be measured.

When the calves reached 8 weeks of age they were grouped in pens of 8 and at 11 weeks removed to the rearing unit - a large single span cubicle shed. Jx and Friesian heifers were kept in separate pens and their group feed intakes were monitored on a weekly basis. Either silage or straw was fed and both groups were weighed fortnightly to monitor daily live weight gain (DLWG). Generally in the rearing unit silage was fed ad libitum with a homemix of 80:20 barley/soya at 2.5 kg/head/day fed twice daily but due to the very poor summer in 1983, the heifers received an ad libitum straw, barley and granstock mixture as their basic feedstuff ( ME 10.2 MJ/kg DM ).

In October 1984 a complete diet silage mix containing barley, soya, minerals and fish meal of ME 11.5 MJ/kg/DM was offered ad libitum to both groups.

## 2.5 FIRST INSEMINATION

All the heifers irrespective of origin were reared to calve as "2 year olds" in the Autumn of 1985. Therefore at approximately 15 months of age all heifers were synchronised by 2 injections (ten days apart) of prostaglandin analogue (Lutalyse-Upjohn Limited) and subsequently inseminated by Hereford semen. Pregnancy was diagnosed by a manual examination of the uterus by a Veterinary Surgeon at 8 to 16 weeks gestation. Cattle were fed silage alone prior to calving.

## 2.6 MANAGEMENT AND FEEDING POST CALVING FIRST LACTATION

After calving both groups of heifers were reduced to 16 per group. This was done on a last to calf basis and since only 16 Jx calved this could not be selective for that group. The data for the excluded Friesians was not analysed.

Both groups were fed a complete diet ad libitum containing silage, barley, sugar beet pulp, soya bean meal and minerals throughout their lactation (Table 2.3). A breakdown of the mineral supplement is shown in Table 2.4. Intakes were measured on a daily basis with refusal weight being taken twice weekly. The amount of fodder offered was adjusted on a daily basis to give a refusal of between 5 and 10% DM, generally around 5%. ME of the diet was also adjusted in stages throughout the lactation.

Table 2.3 Diet formulation per 1000 kg of silage (fresh weight)

	Barley	Soya Bean Meal (44%)	Sugar Beet Pulp (Molassed)	Minerals	ME MJ/kg DM of complete diet
Lactation 1					
Weeks 1-33	260	80	70	10	12.2
Weeks 34-42	115	30	-	5	11.5

Table 2.4 Mineral Content

Ash	2.6%	
Phosphorous	2.5%	
Magnesium	5.0%	
Calcium	7.5%	
Sodium	8.5%	
Vitamin A		320,000 I.U/kg
Vitamin D <sub>3</sub>		80,000 I.U/kg
Vitamin E (Tocopherol acetate)		400 I.U/kg
Iron	3000 mg/kg	
Cobalt	30 mg/kg	
Manganese	5000 mg/kg	
Copper	400 mg/kg	
Zinc	400 mg/kg	
Iodine	250 mg/kg	
Selenium	18 mg/kg	

## 2.7 SUBSEQUENT INSEMINATIONS

### 2.7.1. Bulls Used for Insemination

All cows were bred to a Limousin bull for second calving and a Charolais for third. For fourth bulling Belgian Blue semen was used on the Jx and Friesian on the Friesian contemporaries. In the last season pregnancy was diagnosed using real-time ultrasound.

## 2.8 FEEDING DURING SECOND AND THIRD LACTATIONS

Throughout the second lactation a complete diet was fed ad libitum, proportions again being altered in stages during the lactation (Table 2.5). Mineral breakdown was shown in Table 2.4. Amounts fed were adjusted to give a 5-10% refusal. During lactation three silage and fodder beet were fed ad libitum, intakes were however not measured. At the end of the third lactation the cows were turned out to grass for the first time. Housed dry cows were fed silage alone.

Table 2.5 Diet formulation per 1000 kg of silage (fresh weight)

	Barley (44%)	Soya Bean Meal (44%)	Sugar Beet Pulp (Molassed)	Fish Meal	Minerals	ME MJ/kg DM of complete diet
Lactation 2						
Weeks 1 -33	135	5	95	7.5	5	11.7
Weeks 24-44	37.5	10	25	-	2.5	10.9

## 2.9 GRAZING

On the morning of 13 April 1988 the cows were first turned out to grass alongside an ongoing anthelmintic trial onto "clean pasture". The grass was a perennial ryegrass sward sown in 1975. Stocking rates were 7.4 cows/ha, in the early season, reduced to 3.6 cows/ha after 1st and 2nd cut silages had been made. During the grazing period concentrates (12.8 MJ/kg DM) were fed at a rate of 2 kg/cow/day fed in 1 kg lots at each milking. Grazing continued until 16 October 1988 when the cows were brought into the cubicle housing. Plate 1 shows a Jx at grass.



Plate 1      Jx at grass

## 2.10 HEALTH RECORDS

Three concurrent records of health and management were kept for the stock:

1. **The farm diary** into which each health incident was entered on a daily basis by the stockmen in charge; for example clinical mastitis, lameness, calving/calving difficulty and culls. In addition any on farm treatment was also recorded.
2. **The Veterinary Treatment Book** which was completed by the Veterinary Surgeon on each visit. This records the diagnosis and treatment.
3. **Veterinary Investigation Centre Records** - Samples taken by either the Veterinary Surgeon or stockman to aid diagnosis were generally submitted to the Veterinary Investigation Centre (VIC) where a full record of all samples and the results of the diagnostic tests was retained on file.

## 2.11 INTENSIVE MONITORING OF CONTINUOUSLY HOUSED CATTLE

### 2.11.1 Clinical Mastitis

Cases of clinical mastitis were detected by the dairyman based on the presence of clots, blood or exudate in the pre-milking strippings. All cases were recorded in the farm diary along with treatment and in addition wherever possible a sterile sample of milk was obtained for bacteriological analysis (Appendix 3). In less obvious cases a somatic cell count (Appendix 5) and chloride quantification (Appendix 4) were also performed.



### 2.11.2 Subclinical Mastitis

Beginning on the morning of 14 November 1987 and continuing to 15 September 1988, each cow in the group was tested monthly in two ways. Firstly a Cowside California Mastitis Test (CMT), paddle test was performed on each quarter (Appendix 2) at morning milking to assess subclinical mastitis incidence and secondly a somatic cell count (Appendix 5) and chloride concentration (Appendix 4) was undertaken on a whole milk samples obtained at morning and afternoon milking.

A sterile sample of milk was obtained from any quarter giving a positive CMT reaction for bacteriological analysis (Appendix 3) and also for somatic cell count and chloride measurement . In some cases where more than one quarter was badly affected the remaining quarters were also sampled. In addition milk quality was also assessed on the whole milk samples.

### 2.11.3 Milk Quality

Milk was obtained by sampling each collecting jar after the cow was milked. The milk was decanted into a universal container (25 ml) containing one Lactab Mark III (30mg potassium dichromate plus 20 mg salt, Thomson Capper Limited) as a preservative. Samples were obtained from each cow at both the morning and afternoon milking. Fat, protein and lactose content of the whole milk samples were obtained using an infra-red autoanalyser by the method of Biggs (1979).

#### 2.11.4 Gastrointestinal and Respiratory Parasites

On the morning of turnout, 13 April 1989, and thereafter monthly faeces and blood samples were obtained from each animal. The faecal samples were examined for the presence of nematode eggs and Dictyocaulus larvae (Appendices 7 and 8) and the blood samples were used in the estimation of serum pepsinogen (Appendix 6).

#### 2.11.5 Lameness

Lame cows were also recorded in either the stockmens or veterinarians diaries depending on treatment involved. The cattle were examined routinely once yearly and those with overgrown feet routinely pared by commercial foot trimmers using the so-called "Dutch Method".

#### 2.11.6 Reproduction

All the major reproductive events: pregnancy diagnosis, calving, service, and diagnosis and treatments for infertility were recorded. If an abortion occurred then, in addition to statutory obligations, if at all possible foetuses, placentae and blood samples from the dam were presented to the local VIC for investigation.

#### 2.11.7 Milk Fever and Grass Staggers

Cows were observed after calving to ensure hypocalcaemia did not occur. If milk fever was suspected 500 ml of calcium borogluconate 40% was administered intravenously. Such a case

was then observed for a further 24 hours and further treatment administered if necessary. If hypomagnesaemia was even suspected it was treated immediately. Also prophylactic calcined magnesite 60 grams per head was fed along with silage at night during the early post-turnout period.

## 2.12 COW WEIGHTS AND CONDITION SCORES

Cows were weighed weekly and condition scored fortnightly by the method described by the National Institute of Research in Dairying (NIRD).

## 2.13 STATISTICAL ANALYSIS

### 2.13.1 Calf and Cow Data

Data for liveweight, liveweight change, ZST, calf:cow ratio by weight, 305 day yields, gestation length, number of open days and gross efficiencies were analysed for significant difference between treatments using one-way analysis of variance. Correlation between calf ZST and weight were also carried out. Analysis by Chi-Square test was carried out for mastitic quarters and number of services to determine the critical value of  $\chi^2$  for group differences.

### 2.13.2. Intensive Monitoring Data

#### 2.13.2.1 Monthly bulk milk samples

Lactose, fat, protein, somatic cell count and chloride content of monthly bulk milk samples were analysed for significant difference between groups using one-way analysis of variance. Correlations and regressions of somatic cell counts, chlorides and lactoses were also carried out to determine relationships between these parameters.

#### 2.13.3. CMT Postitive Milk Samples

CMT positive somatic cell counts and chlorides could not be analysed by simple one-way analysis of variance due to the unbalanced structure of the data obtained. Therefore these two variates were analysed by regression of variate on factor and subsequent analysis of variance on deviation from the regression line, using a linear model. This analysis of deviation due to each factor (group, month, quarter and all interactions) using analysis of variance was by a process of elimination; starting with the factor which displayed the greatest residual variation and ending with the factor which displayed the least (Tables 3.39 and 3.40). This analysis was carried out using the modelling capacity of the Genstat IV statistical package.

CHAPTER 3

RESULTS

## RESULTS

For all tables in the results section, the following star coding is used to express significant difference (sig diff) between means:

\*\*\* - Significant difference at  $P < 0.001$

\*\* - Significant difference at  $P < 0.01$

\* - Significant difference at  $P < 0.05$

N.S. denotes no significant difference

Jx refers to Jersey X Friesian's and Fr to the Friesian contemporaries

### 3.1 YOUNGSTOCK DATA

#### 3.1.1 Calf Weights and Zinc Sulphate Turbidity Tests

The calf weight and Zinc Sulphate Turbidity Test (ZST) results are shown in Table 3.1. Only data regarding female animals was recorded as all males were sold within two weeks. Friesian calves were significantly heavier than the Jx calves but had significantly lower ZST figures.

Table 3.1 Calf weights and Zinc Sulphate Turbidity Tests

	Jx	Fr	SED	Sig Diff
No. of observations	16	16		
Weight (mean kg)	35.13	45.25	1.801	***
ZST (mean ZST units)	27.4	15.9	3.18	**
Correlation ZST and Wt				
Individual Groups	0.153	-0.37		
Both Groups		-0.46		

### 3.1.2 Liveweight and Liveweight Gains

Group Liveweight (LW) and Liveweight Gains (LWG) between the birth of calves and their calving (at approximately 2 years old) are presented in Tables 3.2 and 3.3 respectively. It can be seen that the Friesian calves out performed the Jx calves at every stage.

Table 3.2 Liveweight Means (kg)

	Jx	Fr	SED	Sig Diff
Number of observations	16	16		
Birthweight	36.00	44.20	1.346	***
Weaning (4 weeks)	46.38	57.00	1.553	***
Eight weeks	68.31	81.53	1.842	***
Bulling (15 months)	258.40	314.30	13.040	***
Calving (24 months)	393.80	464.90	14.620	***

Table 3.3 Mean Liveweight gains

	Jx	Fr	SED	Sig Diff
Number of observations	16	16		
Birth - weaning (4 weeks)	0.3705	0.4571	0.194	***
Weaning - 8 weeks	0.783	0.876	0.0297	**
8 weeks - bulling (15 mth)	0.522	0.643	0.316	**
Bulling (15 mth) - Calving (24 mth)	0.537	0.597	0.496	NS
Birth -calving (24 mth)	0.532	0.626	0.0212	***

### 3.1.3 Forage and Concentrate Intakes

Forage and concentrate intakes were monitored by group from birth to calving (Table 3.4), the Friesian calves having higher intakes at every stage.

Table 3.4 Forage and concentrate intakes (kg DM/head/day)

	Jx	Fr
Number	16	16
Milk powder to 4 weeks	0.28	0.37
Concentrates to 8 weeks	1.16	1.28
Complete diet 20.1.84 - bulling	4.05	4.80
Bulling - calving	5.85	6.51

#### 3.1.4 Bulling

All heifers were synchronised by two prostaglandin (Prosolvin-Intervet) injections with an interval of ten days between and at approximately 15 months of age were artificially inseminated (AI) by Hereford semen. Repeats were also served by AI and pregnancy diagnosed by a veterinarian at approximately 20 weeks. Of the 34 animals, 33 were found to be in calf with only one Jx being found to be empty. No statistical analysis by Chi-square was attempted. Subsequently one Friesian was also found to be empty.

#### 3.2. FIRST PARTURITION

All heifer calvings were observed and all calving difficulties were noted as part of another study (Murray, 1985 personal communication). Of the Jx heifers five required minimal assistance to ensure safe calving. In the Friesian contemporary group two required mechanical assistance and four minimal assistance. Two Jx calves were stillborn, no Friesians were stillborn. However, there was no statistical significance (Chi-square) found in the number of assisted calvings ( $x^2=0.351$ ). Calves were weighed shortly after calving and dams were weighed within 4 days of calving, group differences being calculated



(Table 3.5). Ratios of calf weight to dam weight were also calculated and are also presented in Table 3.5. Although the ratio of calf to cow weight was substantially less for the Jx this was not significantly different from that of Friesian.

Table 3.5 Calf dam weights and ratios

	Jx	Fr	SED	Sig
Number of observations	14	16		
Calf weight (kg)	34.0	36.3	2.68	NS
Cow weight (kg)	378.8	448.1	13.87	***
Ratio calf wt:Cow wt	11.32	13.08	1.309	NS

### 3.2.1 Length of first gestation

Both groups were inseminated by Hereford semen and the gestation length of each heifer was calculated and group differences were analysed (Table 3.6). Again the mean figure was lower for the Jx and there was no significant difference.

Table 3.6 Gestation length (days)

	Jx	Fr	SED	Sig
Number observations	16	16		
Gestation length (days)	287.6	295.8	8.14	NS

Dams then entered the main dairy herd where they were maintained as two separate groups, each group being fed the same complete diet throughout the lactation.

### 3.3. FIRST LACTATION

#### 3.3.1 Feeding Regime

Diets were altered at stages throughout the lactation to maintain adequate energy for milk production. Diet 1 was fed for the first 33 weeks of the lactation and Diet 2 from weeks 34-42. Table 3.7 summarises Table 2.3 shown earlier.

Table 3.7 Metabolisable Energy of Complete Diets 1 and 2 in Lactation One

	ME MJ/kg DM of complete diet
Diet 1	12.2
Diet 2	11.5

#### 3.3.2 Group Intakes

The complete diet was fed to give a ten per cent refusal and group intakes were calculated (Table 3.8). As noted previously the Friesian contemporaries had a higher intake than the Jx.

Table 3.8 Group intakes (kg DM/day)

	Jx	Fr
Number in group	16	16
Intake kg DM/day	16.08	17.49

### 3.3.3. Milk Production

Milk production and composition was monitored by the Scottish Milk Marketing Board (SMMB) monthly. 305 day yields as calculated by the SMMB are presented in Table 3.9, with the only significant difference being the higher butterfat and protein percentage of the Jx.

Table 3.9 305d yields

	Jx	Fr	SED	Sig Diff
Number of observations	16	16		
Yield (litres)	4044	4567	274.6	NS
Fat (%)	4.909	4.220	0.1316	***
Protein (%)	3.528	3.291	0.0554	***
Fat yield (kg)	198.1	191.5	11.64	NS
Protein yield (kg)	142.5	149.7	8.66	NS

As a routine, weekly milk yield and composition was monitored on the farm. These figures were used to calculate Gross Efficiency.

### 3.3.4 Gross Efficiency

Gross efficiency was calculated using the following equation.

Gross efficiency (GE) = Energy Out/Energy In.

Energy out = (fat x 0.0623) +(Solids Not Fat (SNF) x 0.331)-0.381

Energy in = Complete Diet Intake x ME (MJ/kg DM)

The lactation was split into 3 periods for the purpose of these calculations as in Table 3.10. Significant differences were only found between periods.

Table 3.10 Gross Efficiency

	Jx	Fr
Number of observations	16	16
Period 1 (Weeks 1-14)	0.542	0.518
Period 2 (Weeks 15-28)	0.363	0.332
Period 3 (Weeks 29-42)	0.330	0.362
	SED	Sig Diff
Group	0.1696	NS
Period	0.0208	***
Group*Period	0.0294	NS

Figure 3.1 shows the Gross Efficiency and mean liveweight curve throughout the lactation.

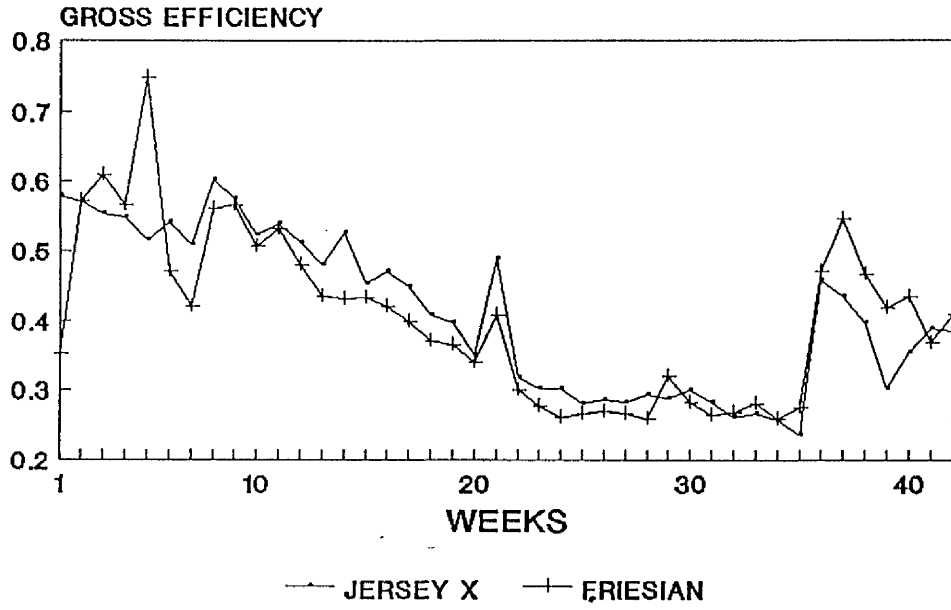
### 3.3.5 Weights

The two groups of cattle were weighed at weekly intervals. Between group differences in weight continued to be significantly different (Table 3.11). This information was split into the same three periods as Gross Efficiency.

Table 3.11 Weights

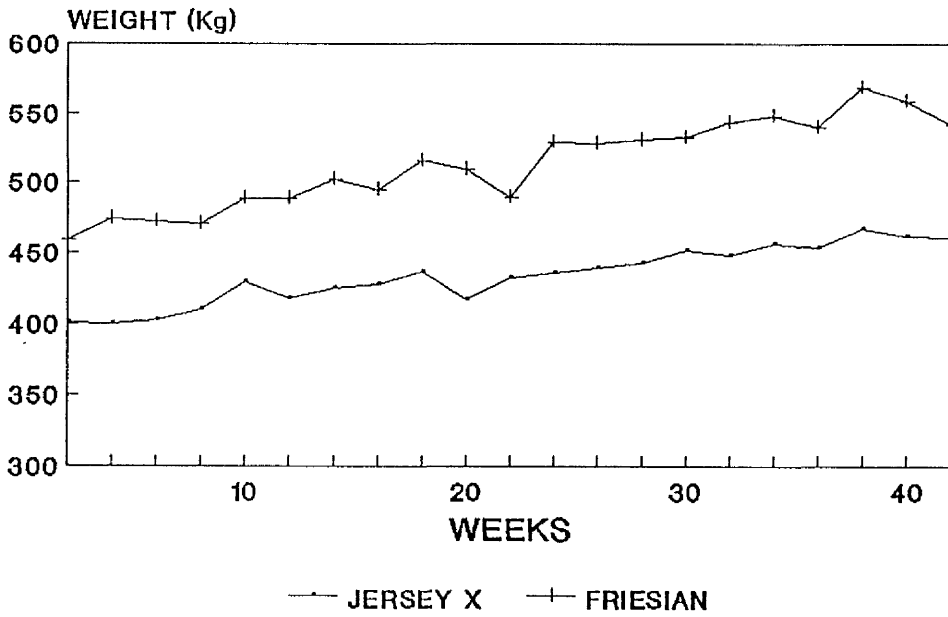
	Jx	Fr
	Weight (kg)	
Number of observations	16	16
Period 1 (Weeks 1-14)	412.1	479.6
Period 2 (Weeks 15-28)	433.1	514.0
Period 3 (Weeks 29-42)	457.1	547.9
	SED	Sig Diff
Group	3.78	***
Period	4.63	***
Group*Period	6.54	NS

# GROSS EFFICIENCY



LACTATION 1

# MEAN WEIGHTS



LACTATION 1

Mean group change was also calculated (Table 3.12). No significant differences were noted.

Table 3.12 Liveweight change

	Jx	Fr
	Weight change (kg/day)	
Number of observations	16	16
Period 1 (Weeks 1-14)	0.24	0.44
Period 2 (Weeks 15-28)	0.19	0.29
Period 3 (Weeks 29-42)	0.19	0.11
	SED	Sig Diff
Group	0.28	NS
Period	0.34	NS
Group*Period	0.48	NS

### 3.3.6 Bulling

Bulling for second calving was initiated on the 25th of November 1985 and halted on the 19th of February 1986. Each cow was artificially inseminated by Limousin semen. The number of cows holding to each service is shown in Table 3.13.

Table 3.13 Number holding to service

	Jx	Fr
Number of observations	16	16
One service	9	11
Two services	6	3
Three services	1	1
Four services	-	1
Total No. Services	24	24

No statistical analysis was attempted.

### 3.3.6.1 Open days

The number of days between calving and bulling (open days) were also calculated (Table 3.14).

Table 3.14 Open Days

	Jx	Fr	SED	Sig Diff
No. observations	16	16		
Open Days	92.3	81.7	8.58	NS

### 3.3.7. Health Problems

Throughout the lactation the cattle were observed for any health problems. One Friesian had a retained placenta. One Jx suffered from metritis as did two Friesian heifers.

Two cases of clinical mastitis were observed one in a Jx and the other in a Friesian. The Jx was diagnosed as having "blackspot" in the right hind teat, this teat suffered from recurring mastitis throughout the lactation. Diagnoses of the causal organism in the Friesian heifer was not carried out. Lameness was not a problem with only one case being observed in a Friesian heifer.

### 3.4 SECOND PARTURITION

At calving the cattle were observed for calving difficulties and were assisted when required. The Jx group required no assistance, but one calf was born dead. In the Friesian group 4 animals were minimally assisted and no dead calves were found in this group. Statistical analysis by chi-square was not attempted.

Almost all calves were weighed after birth, with dams being weighed approximately 4 days post calving. Group differences in calf weight, cow weight and calf to post calving dam weight ratios were calculated (Table 3.15). As before only the weights of the dams were found to be significantly different.

Table 3.15 Calf, dam weights and ratios

	Jx	Fr	SED	Sig Diff
Number of observations	14	14		
Calf weight (kg)	46.4	47.9	2.24	NS
Cow weight (kg)	457.7	510.2	17.87	**
Ratio calf wt:cow wt	9.94	10.78	0.595	NS

#### 3.4.1. Length of Second Gestation

The length of gestation was also calculated and group differences analysed (Table 3.16). As in the first gestation the Jx had a shorter gestation but not significantly so.



Table 3.16 Gestation length (days)

	Jx	Fr	SED	Sig Diff
Number of observations	14	14		
Gestation length (days)	286.9	291.6	2.71	NS

### 3.5 SECOND LACTATION

#### 3.5.1. Feeding Regime

Diets in lactation 2 were altered as in lactation 1 to maintain adequate energy but had a lower concentrate input. Table 3.17 summarises this with Diet 3 was fed for the first 33 weeks and Diet 4 for weeks 34-44 .

Table 3.17 Metabolisable Energy of Diets 3 and 4 in Lactation Two

	ME MJ/kg DM of Complete Diet
Diet 3	11.7
Diet 4	10.9

#### 3.5.2. Group Intakes

The complete diet was fed to give a 10% DM refusal and group intakes were calculated. Again Friesian intakes were higher (Table 3.18) although the difference was less than in the first lactation.

Table 3.18 Group Intakes

	Jx	Fr
Number in group	16	16
Intake (kg DM/day)	15.23	15.80

### 3.5.3 Milk Production

Milk production was routinely monitored on the same basis as the first location and calculated 305 d milk yields are presented in Table 3.19. As before the percentage fat and protein were significantly different in favour of the Jx but the Friesian cattle gave significantly higher yields.

Table 3.19 305 d yields

	Jx	Fr	SED	Sig Diff
Number of observations	16	16		
Yield (litres)	4873	5603	296.1	*
Fat (%)	5.026	4.217	0.1316	***
Protein (%)	3.719	3.339	0.0747	***
Fat yield (kg)	244.2	234.9	12.98	NS
Protein yield (kg)	181.0	186.2	9.97	NS

Weekly milk yields and composition were again obtained routinely and were used to calculate Gross Efficiency.

### 3.5.4 Gross Efficiency

Gross efficiency was calculated as in Lactation 1. Again the lactation was split into 3 periods (Table 3.20).

Table 3.20 Gross Efficiency

	Jx	Fr
Number of observations	14	14
Period 1 (Weeks 1 -13)	0.726	0.679
Period 2 (Weeks 14-26)	0.456	0.473
Period 3 (Weeks 27-39)	0.494	0.532
	SED	Sig Diff
Group	0.0201	NS
Period	0.0246	***
Group*Period	0.0347	NS

Figure 3.2 shows the Gross Efficiency and mean liveweight curve for the lactation.

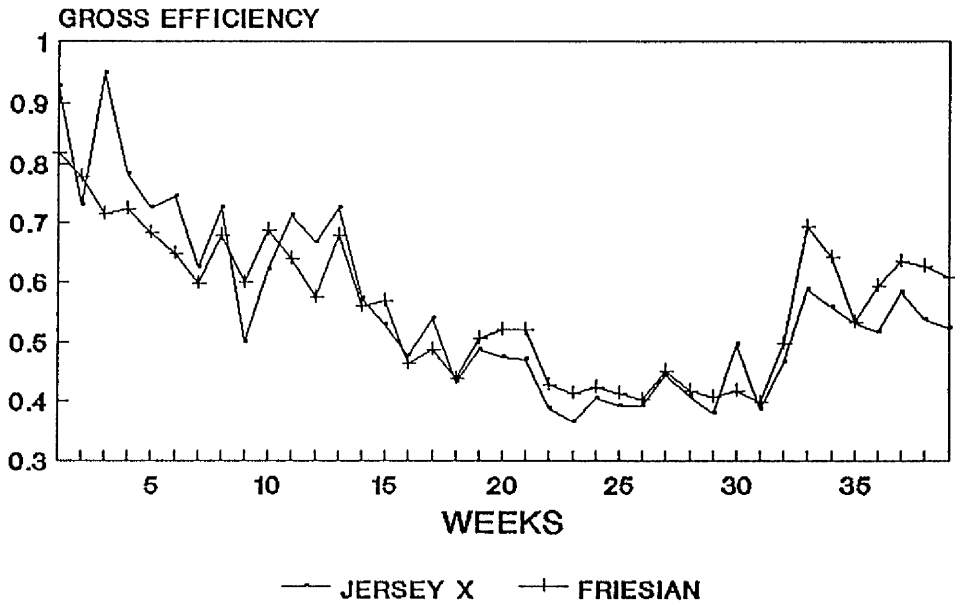
### 3.5.5 Weights

As before the cattle were weighed weekly. Group mean live weights were calculated (Table 3.21) as before these are divided into 3 periods. In each period the Friesian cattle weighed more than the Jx.

Table 3.21 Mean liveweights

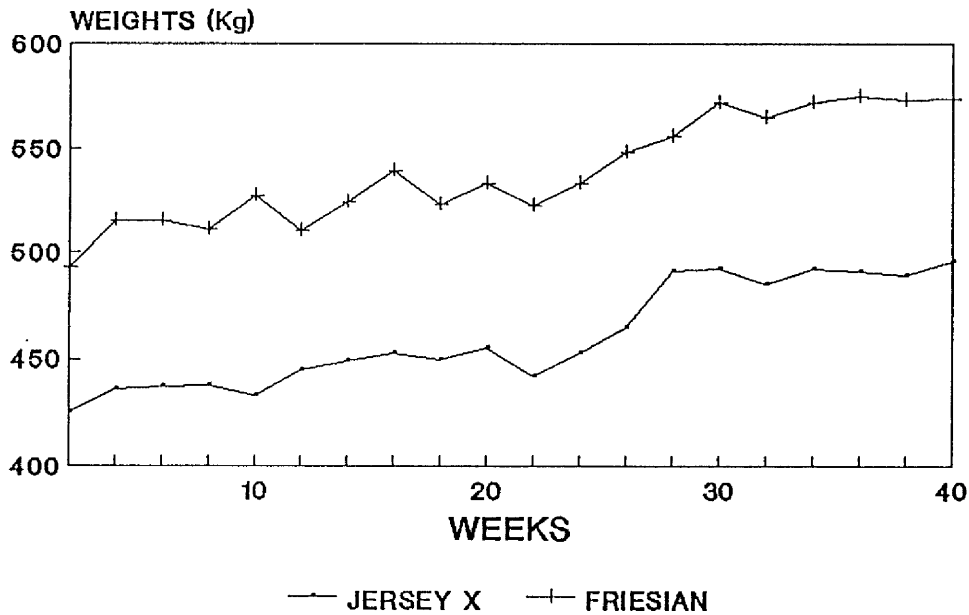
	Jx	Fr
	Weight (kg)	
Number of observations	14	14
Period 1 (Weeks 1-13)	437.6	513.6
Period 2 (Weeks 14-26)	453.0	533.0
Period 3 (Weeks 27-39)	490.9	569.6
	SED	Sig Diff
Group	2.55	***
Period	3.17	***
Group*Period	6.54	NS

# GROSS EFFICIENCY



LACTATION 2

# MEAN WEIGHTS



LACTATION 2

Mean liveweight change over the 3 periods was also calculated (Table 3.22).

Table 3.22 Liveweight change

	Jx	Fr
	Weight change (kg/day)	
Number of observations	14	14
Period 1 (Weeks 1-13)	0.24	0.31
Period 2 (Weeks 14-26)	0.23	0.33
Period 3 (Weeks 27-39)	0.34	0.28
	SED	Sig Diff
Group	0.26	NS
Period	0.34	NS
Group*Period	0.47	NS

### 3.5.6 Bulling

Bulling for third calving commenced 25 November, 1986 and ended on the 18th February 1987. Each cow was artificially inseminated with Charolais semen. The number of services per cow are shown in Table 3.23.

Table 3.23 Number of services

	Jx	Fr
Number of observations	14	14
One service	10	10
Two services	4	4

No analysis by chi-square was attempted.

### 3.5.6.1 Open Days

The number of days open was calculated from date of calving to date of bulling (Table 3.24).

	Jx	Fr	SED	Sig Diff
Number of observations	14	14		
Days open	71.1	80.5	9.4	NS

### 3.5.7. Health Problems

Health throughout the lactation was monitored. One cow from each group had a retained placenta. Five cows all Jx, had mastitis, but no investigation of the causal organism was undertaken. Each animal was treated with Leo Yellow (Trademark - Leo Laboratories). Lameness became more of a problem with three Jx and three Friesians being treated. Two Jx and one Friesian were culled. One Friesian died but the cause of death was not established.

## 3.6 THIRD PARTURITION

The third calving of both groups was relatively trouble free, with only one Jx calf being born dead and one Friesian requiring slight assistance. Again calves were weighed shortly after birth and dams within approximately 4 days. Differences between group calf weights, cow weights and calf : cow weight ratios were calculated (Table 3.25).

Table 3.25 Calf, dam weights and ratios

	Jx	Fr	SED	Sig Diff
Number of observations	12	12		
Calf weight (kg)	45.9	51.8	1.754	**
Cow weight (kg)	482.0	572.0	22.800	**
Ratio calf wt:cow wt	10.54	11.11	0.571	NS

### 3.6.1. Length of Third Gestation

Gestation length in days was calculated and group differences analysed (Table 3.26).

Table 3.26 Gestation length (days)

	Jx	Fr	SED	Sig Diff
Number of observations	13	13		
Gestation length (days)	278.9	285.4	6.59	NS

## 3.7 THIRD LACTATION

### 3.7.1. Feeding Regime

During this lactation the feed intakes of the cattle were not recorded. The cattle were however fed silage (ME 10.9 MJ/kg DM) ad libitum supplemented by fodder beet (ME 13.1 MJ/kg DM). Parlour concentrates were also offered at 1 kg/head/milking.

### 3.7.2. Milk Production

Monthly milk recording was carried out by the Scottish Milk Records Association. Group differences in 305 d yields were determined as before and are shown in Table 3.27. On this occasion, as in Lactation 1, only butterfat and protein percentages were significantly different.

Table 3.27 305 d yields

	Jx	Fr	SED	Sig Diff
Number of observations	12	12		
Yield (litres)	5261	5841	331.3	NS
Fat (%)	5.043	4.413	0.1243	***
Protein (%)	3.777	3.429	0.0610	***
Fat yield (kg)	264.8	258.0	16.38	NS
Protein yield (kg)	198.2	200.3	11.83	NS

### 3.7.3 Weights

Cattle were weighed on a weekly basis to monitor liveweight change (Tables 3.28, 3.29, respectively). No divisional periods were made and no significant difference in liveweight change was noted.

Table 3.28 Mean liveweight

	Jx Weight (kg)	Fr Weight (kg)	SED	Sig Diff
Number of observations	14	14		
Weight (kg)	497.1	602.2	5.92	***

Table 3.29 Liveweight change

	Jx	Fr	SED	Sig Diff
Number of observations	14	14		
Weight (kg) change/week	1.48	1.15	1.358	NS



### 3.7.4 Bulling

Bulling for fourth calving began on 24 November 1987 and ended 12 February 1988. Belgian Blue semen was used on the Jx cows and Friesian semen on the Friesian contemporaries. The number of services are shown in Table 3.30. Statistical analysis by chi-square was carried out but there was no significant difference between groups.

Table 3.30 Number of Services

	Jx	Fr
Number of observations	11	11
One service	5	9
Two services	4	2
Three services	2	-
Total services	19	13

#### 3.7.4.1. Open Days

A significant difference between the two groups for the number of open days from calving to service date was noted (Table 3.31).

Table 3.31 Open Days

	Jx	Fr	SED	Sig Diff
Number of observations	14	14		
Days open	51.9	73.6	9.72	*

### 3.7.5 Health Problems

Health throughout the lactation was again monitored. One abortion in a Jx was recorded but no specific diagnosis was

reached as to the cause.

The incidence of mastitis increased with 14 cases of clinical mastitis being found in the 28 cows. Nine in the Jx group and 5 in the Friesian group. One of the Jx group became a chronic mastitic cow and lost a quarter. Analysis by quarter (chi-square) showed no significant difference between groups  $\chi^2 = 1.306$  (Table 3.32). Lameness was not as great a problem in lactation three with three Jx suffering and one Friesian, these were all treated.

Table 3.32 Example of Chisquare analysis

	Jx	Fr	
Number of mastitic quarters	9	5	
Number of non-mastitic quarters	47	51	
Chi-square (Expected counts) are printed below observed counts			
	9 (7.0)	5 (7.0)	Total 14
	47 (49.0)	51 (49.0)	98
Total	56	56	112

$$\text{Chi-square} = 0.571 + 0.571 + 0.082 + 0.082 = 1.306$$

degrees of freedom = 1

Critical value of  $\chi^2 = 3.84$  (NS)

In addition to the routine health monitoring carried out by the herdsman at this time, intensive monitoring of mastitis status and nematode infection was initiated.

### 3.8 INTENSIVE MONITORING

#### 3.8.1 Mastitis

The number of recorded clinical mastitis cases have already been discussed for each of the three years. However, in year three a more detailed investigation of mastitis was undertaken involving culture of most subclinical cases and the regular monthly monitoring of subclinical status by the use of a cowside California Mastitis Test (CMT) and the examination of the identified positive quarters by bacteriological culture, somatic cell count and chloride estimation.

When used in this chapter Tank Somatic Cell Count refers to the milk for sale in the tank that is from all milking cows. Individual somatic cell count refers to the milk from an individual cow all quarters, quarter somatic cell count refers to the milk from a single quarter.

#### 3.8.2 Individual Somatic Cell Counts, Chlorides and Lactose Concentrations

On the day of Cowside testing individual milk samples were obtained at morning (am) and afternoon (pm) milkings from all animals and analysed for somatic cells, chloride content and lactose content, yields were also obtained to enable calculation of weighted means to be carried out. Monthly group mean individual somatic cell counts, chlorides and lactose concentrations are presented in Tables 3.33, 3.34 and 3.35, respectively.

Table 3.33 Individual somatic cell counts

	JX			FR		
	am	pm	Weighted Mean	am	pm	Weighted Mean
November	0.235	0.306	0.258	0.129	0.250	0.173
December	0.304	0.393	0.330	0.433	0.547	0.482
January	0.308	0.503	0.382	0.393	0.335	0.367
February	0.861	0.330	0.666	0.793	0.539	0.740
March	0.331	0.325	0.325	0.353	0.345	0.346
April	0.214	0.235	0.221	0.546	0.365	0.505
May	0.156	0.452	0.282	0.249	0.241	0.247
June	0.234	0.314	0.264	0.130	0.247	0.179
July	0.292	0.471	0.361	0.470	0.518	0.490
			am	pm		Weighted Mean
Significant Difference	Group		NS	NS		NS
	Month		NS	NS		NS
	Group*Month		NS	NS		NS
SED			0.24	0.1386		0.1884

Table 3.34 Individual Chlorides

	JX			FR		
	am	pm	Weighted Mean	am	pm	Weighted Mean
November	24.06	20.37	22.73	23.89	22.56	23.42
December	23.56	22.31	23.21	23.53	26.13	24.58
January	22.47	21.47	22.10	26.13	25.00	25.69
February	22.29	20.59	21.73	26.27	24.27	25.61
March	25.18	21.59	23.97	25.47	24.93	25.30
April	24.06	22.82	23.62	27.11	26.66	26.84
May	26.24	24.18	25.36	30.82	30.27	30.63
June	26.65	27.82	27.02	32.10	35.39	33.41
July	27.64	28.27	27.88	37.09	32.32	35.37
				am	pm	Weighted Mean
Significant Difference	Group			***	***	***
	Month			***	***	***
	Group*Month			*	NS	NS
SED				1.27	1.66	1.224

Table 3.35 Individual Lactose

	JX		Lactose			Weighted Mean
	am	pm	Weighted Mean (%)	FR		
				am	pm	
November	4.913	4.986	4.936	4.777	4.679	4.715
December	4.816	4.821	4.819	4.663	4.544	4.595
January	4.912	5.018	4.981	4.789	4.807	4.797
February	4.786	4.789	4.787	4.690	4.699	4.695
March	4.512	4.471	4.787	4.441	4.449	4.436
April	5.151	5.133	5.143	4.812	4.792	4.815
May	4.737	4.679	4.705	4.523	4.497	4.509
June	4.719	4.809	4.672	4.636	4.669	4.489
July	4.208	4.662	4.239	4.135	4.200	4.282
Significant Difference	Group			am	pm	Weighted Mean
	Month			***	***	***
	Group*Month			***	***	***
SED				NS	NS	NS
				0.828	0.078	0.0931

In addition correlations and regressions between each parameter were carried out to determine interparameter relationships (Tables 3.36 and 3.37).

Table 3.36 Correlations

	Cell Count			Chloride		
	am	pm	Weighted Mean	am	pm	Weighted Mean
Cell count am	-	-	-	-	-	-
Cell count pm	-	-	-	-	-	-
Cell Count						
Weighted Mean	-	-	-	-	-	-
Chloride am	0.07	-	-	-	-	-
Chloride pm	-	0.150	-	-	-	-
Chloride						
Weighted Mean	-	-	-0.100	-	-	-
Lactose am	0.173	-	-	-	0.440	-
Lactose pm	-	-	-0.220	-	-0.450	-
Weighted Mean	-	-	-0.220	-	-	-0.53

Table 3.37 Regressions: chloride, lactose and somatic cells.

Y variate	Regression Equation	RSD Variation about Y	% variation accounted for	n	Significance
am					
Chloride	$0.45 \times \text{cell count} + 25.69$	21.79	0.1	258	NS
Lactose	$-0.08 \times \text{cell count} + 4.74$	0.10	2.6	252	**
Lactose	$-0.03 \times \text{chloride} + 5.48$	0.08	18.6	252	***
pm					
Chloride	$2.20 \times \text{cell count} + 23.95$	33.64	1.8	251	*
Lactose	$-0.17 \times \text{cell count} + 4.70$	0.81	4.6	247	***
Lactose	$-0.02 \times \text{chloride} + 5.29$	0.07	20.4	247	***
Weighted Means					
Chloride	$0.86 \times \text{cell count} + 25.11$	21.41	0.6	251	NS
Lactose	$-0.11 \times \text{cell count} + 4.76$	0.08	4.5	247	***
Lactose	$-0.03 \times \text{cell count} + 5.56$	0.06	27.8	247	***

Note It is clear that there are significant regressions of x on y on the data, but the % of variation accounted for was at the greatest 27.8. Therefore x is only one factor in the variation of y and many other factors must be influencing the y data. This statement is relevant to all regressions presented.

Examination of the correlation between each monthly meaned individual somatic cell count with the overall mean for that animal (based on 9 monthly counts) is shown in Table 3.38.

Table 3.38 Correlation of Monthly Means

Month	Correlation with Mean of Months 1 to 9
1	0.718
2	0.629
3	0.849
4	0.283
5	0.287
6	0.262
7	0.760
8	0.852
9	0.310

As can be seen correlations varied from a maximum of 0.852 to a minimum of 0.262. Table 3.39 shows that the cumulative correlation increased with the number of readings apart from months one and two.

Table 3.39 Cumulative Correlation of Individual Somatic Cell Counts

Mean of Months	Correlation with Mean of Months 1-9
1	0.718
1+2	0.688
1+2+3	0.835
1+2+3+4	0.843
1+2+3+4+5	0.849
1+2+3+4+5+6	0.939
1+2+3+4+5+6+7	0.969
1+2+3+4+5+6+7+8	0.997
1+2+3+4+5+6+7+8+9	1.000

### 3.8.3 CMT Examination

CMT Examination of all quarters was carried out at the am milking (Appendix 2). Due to the highly subjective nature of this test it was decided not to grade results but to sample all quarters which gave a positive result from trace upwards. In addition animals with more than 2 positive quarters had all quarters sampled. Table 3.40 shows the number of quarter samples taken per month and the number of cows these samples relate to.

Table 3.40 Number of quarter samples and cows by month

	No. of samples	No of cows
November	5	4
December	7	5
January	10	4
February	10	5
March	15	8
April	24	11
May	13	5
June	15	10
July	13	8
Total	112	60

### 3.8.4. CMT Positive Milks

All positive CMT samples were analysed for somatic cells and chloride content. Due to the unbalanced number of results obtained statistical analysis was carried out using an analysis of deviance technique (Table 3.41 and Table 3.42).

Somatic cell counts, chlorides and the lactose content of all positive (CMT) milks were also analysed for correlation and by regression to establish interparameter relationships (Table 3.43



Table 3.41 Somatic Cell Count: CMT positive milk samples  
 Y variate: Somatic Cell Count

Terms	Residual V2=DF	SS	Change V1=DF	SS	Mean Change	Variance Ratio	Sig Diff
Initial Model Constant	109	3606.87	*	*			
Modification to Model							
Group	108	3582.60	1	24.26	24.26	0.80	NS
Month	100	3029.53	8	553.08	69.13	2.28	*
Quarter	97	3011.26	3	18.27	6.09	0.20	NS
Group*Month	90	2957.37	7	53.89	7.70	0.25	NS
Group*Quarter	87	2914.51	3	42.86	14.29	0.47	NS
Month*Quarter	65	2714.19	22	173.32	7.88	0.26	NS
Group*Month*Quarter	54	2630.88	11	110.31	10.03	0.33	NS

Table 3.42 Chloride Concentration: CMT positive milk sample  
 Y variate: Chloride concentration

Terms	Residual		Change		Mean Change	Variance Ratio	Sig Diff
	V2=DF	SS	V1=DF	SS			
Initial Model Constant	102	12581.8	*	*			
Modifications to Model							
Group	101	10244.6	1	2337.2	2337.2	23.04	***
Month	94	9744.5	7	500.1	71.4	0.70	NS
Quarter	91	9727.5	3	17.0	5.7	0.06	NS
Group*Month	85	9484.7	6	242.8	40.5	0.40	NS
Group*Quarter	82	9460.1	3	24.6	8.2	0.08	NS
Month*Quarter	62	7855.2	20	1604.9	80.2	0.79	NS
Group*Month*Quarter	51	7535.0	11	320.2	29.1	0.29	NS

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and Table 3.44). As can be seen correlation co-efficients were not high.

Table 3.43 Correlation of somatic cell counts, chloride content and lactose % (CMT positive milks)

	Cell Count	Chloride
Cell count	-	-
Chloride	0.48	-
Lactose	0.27	0.28

Table 3.44 Regression: Somatic Cell Counts, Chloride Content and Lactose % (CMT positive milks)

Y variate	Regression Equation	RSD Variation about Y	% variation accounted for	n	Significance
Chloride	$0.90 \times \text{cell count} + 33.99$	95.85	22.3	102	***
Lactose	$-0.03 \times \text{cell count} + 4.60$	0.15	20.7	102	***
Lactose	$-0.02 \times \text{chloride} + 5.25$	0.14	28.3	102	***

### 3.8.5 Bacteriology of CMT Postive Milks

Bacteriological examination of all positive CMT milks was carried out (Appendix 3) in order to identify causal organisms. Isolates per month are shown in Table 3.45.

Table 3.45 Bacterial Isolates (CMT positive milks)

Month	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Jul	Total
Organism										
SFUA	1	-	1	5	8	9	6	7	3	40
SPDY	1	3	4	6	4	6	-	3	5	32
SPUB	-	-	1	-	-	-	-	-	-	1
COPY	1	-	3	-	-	-	-	-	-	4
SFEP	-	4	-	1	-	-	-	2	-	7
SPFA	-	4	-	-	1	-	-	1	2	8
ESCO	-	1	2	-	-	-	-	1	-	4
BALI	1	-	-	-	-	-	-	-	-	1
BRAN	-	-	-	-	-	1	-	-	-	1
COBO	-	-	-	-	-	-	-	-	2	2
Total	4	12	11	12	13	16	6	14	12	100

Key SFAU	-	<u>Staphylococcus aureus</u>
SPDY	-	<u>Streptococcus dysgalactiae</u>
SPUB	-	<u>Streptococcus uberis</u>
COPY	-	<u>Corynebacterium pyogenes*</u>
SFEP	-	<u>Staphylococcus epidermidis</u>
SPFA	-	<u>Streptococcus faecalis</u>
ESCO	-	<u>Escherichia coli</u>
BRAN	-	<u>Branhamella species</u>
COBO	-	<u>Corynebacterium bovis</u>
BALI	-	<u>Bacillus licheniformis</u>

\*Corynebacterium pyogenes is now Actinomyces pyogenes

Bacteria isolated were subjected to antibiotic sensitivity testing (Appendix 3). The majority of isolates were sensitive to broad spectrum antibiotics. However all Staph. aureus isolates (which accounted for 40% of the total) were beta lactamase producers and thus resistant to penicillin.

### 3.8.6 Somatic Cells as an Indication of Mastitis

In addition to the statistical analyses obtained the proportion of CMT positive milks in relation to number of somatic cells was calculated (Table 3.46). The presence of 500,000 somatic cells per millitre of milk is indicative of mastitis.

Table 3.46 Positive milks as related to Somatic Cells

Somatic cells	Number of milk samples*	Proportion of total
≤200,000	24	21.4%
≤500,000	39	34.8%
≥500,000	49	43.8%
Total	112	

\*NB not all were necessarily CMT positive

#### 3.8.6.1 Relationship Between Mastitis and Number of Somatic Cells Present In Milk

The number of CMT positive cows (Table 3.40) was directly associated to the number of bacterial isolates obtained (Table 3.45) with 60 positive cows giving 112 milk samples and 100 isolates. Thus 89% of the samples yielded an organism and 76% if Staph epidermidis and Strep faecalis are excluded. Somatic cell counts of >500,000/ml of milk are generally regarded as being indicative of subclinical mastitis, in this experiment however >200,000 somatic cells/ml appeared to be surprisingly useful, in terms of yielding a significant isolate..

### 3.8.7 Milk Yield Throughout Intensive Monitoring Period

Milk yield at turnout to inwintering was monitored as a routine. Group differences and percentage drop in yield over this period are shown (Table 3.47).

Table 3.47

	Jx	Fr
No observations	14	14
Milk yield at turnout (litres/day)	18.9	21.1
Milk yield at inwintering (litres/day)	10.7	10.6
Percentage change	-43.4	-49.8

This was not analysed for significant differences between groups as the cows were in late lactation and it was expected that yields would fall during this period, with subsequent drying off beginning in September.

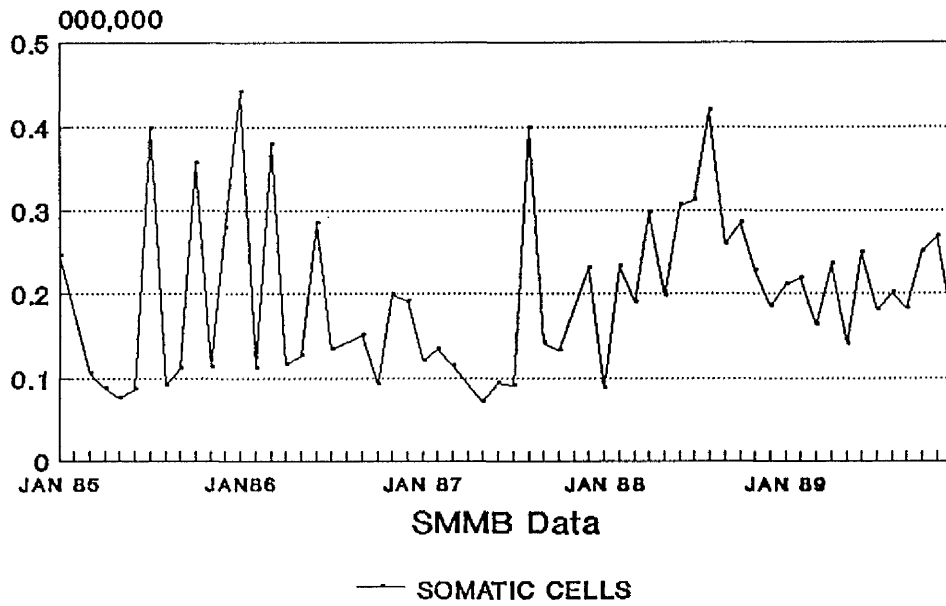
### 3.9 TANK SOMATIC CELL COUNTS AND TOTAL BACTERIAL COUNTS

Milk samples from the tank were routinely examined by the SMMB for somatic cells and bacteria. The trends for both parameters are shown in Figure 3.3 from 1985 onwards. Statistical analysis of the results was undertaken and there were no significant differences between years, months and year\*month interactions.

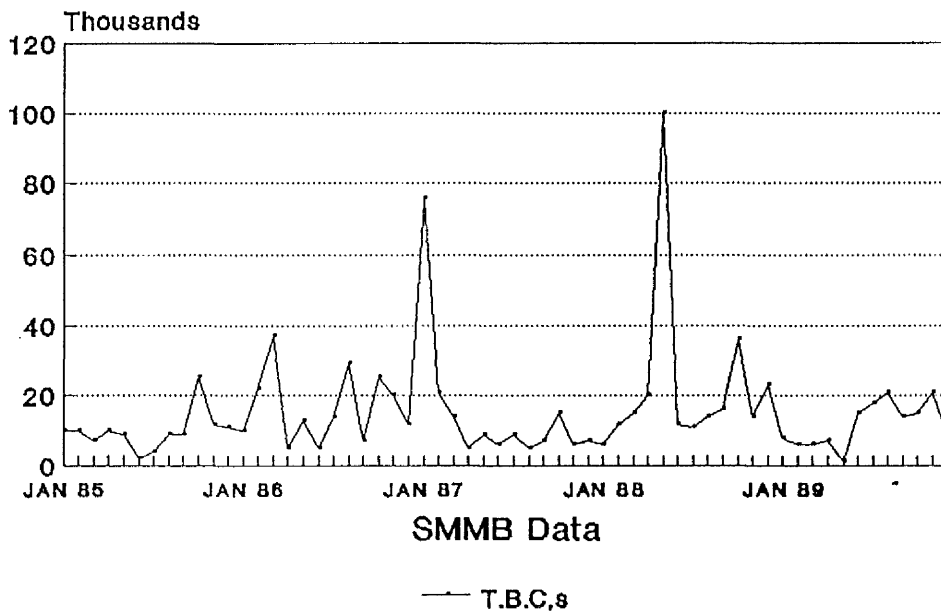
Correlation and Regression analyses were also undertaken to determine relationships between parameters. The correlation factor was very low (0.048) and as expected the regression was not significant (Table 3.48).



# CRICHTON ROYAL FARM



# CRICHTON ROYAL FARM



Yearly means for each factor are shown in Table 3.49.

Table 3.49      Yearly Means  
                 Somatic Cell Counts and Total Bacterial Counts

	Somatic Cell Count x 10 <sup>3</sup>	TBC x 10 <sup>3</sup>
1985	178	9.8
1986	203	15.5
1987	154	16.9
1988	250	23.3
1989	205	11.8

### 3.10 Effect of Turnout

On observation at turnout it was obvious that the animals were wary of their new found freedom, with the majority standing 'heads down' for approximately 30 minutes. Fresh grass was also an unknown feed quantity, and initially grazing was not the first priority. After the initial shock the cattle became more inquisitive and began to explore their surroundings. Electric fences caused some consternation with some cows actually licking the wire! Barbed wire fencing also left its mark with many cuts appearing in the first few days. During the first morning of turnout most animals settled and by mid day were behaving much more normally although they were still constantly wary of their surroundings. By the next day most had relaxed in their new environment and were "happy" to be outside. One Friesian, however, never came to terms with the outdoors and was culled as she endangered the safety of the herdsman. Apart from this case, within a week it was as if these animals had been outside all

their lives.

As was expected milk yield fell throughout the grazing period with drops of approximately 47% from April until September. Another group of Autumn/Winter calving cows on the same farm (which had not been continuously grazed previously) had a drop in milk yield of 67% from turnout to housing showing that there was no inherent differences associated with grazing continuously housed cattle . Breed differences between the Jx and Friesian were not significant.

### 3.11 NEMATODE INFECTION STATUS

#### 3.11.1 Presence of Nematode and Dictyocaulus species

Worm eggs were not detected in faecal samples until 5 months after turnout. In this sampling period four Friesians and one Jx were found to have a worm egg burden of 100 strongyle species/gram of faeces. Dictyocaulus larvae were not found at any time.

#### 3.11.2 Pepsinogen Evaluation

Serum pepsinogen levels were calculated and analysed by group and month for statistical significance (Table 3.50). These increased as the season progressed and showed a significantly lower set of values for the Jx.

Table 3.50 Serum Pepsinogen

	Jx	Fr
No. of observations	14	14
April	558	630
May	617	771
June	584	695
July	740	847
August (missing values)		
September	760	895

Significant Difference	
Group	***
Month	*
Group*Month	NS
SED	73.6

### 3.11.3 Hypocalcaemia and Hypomagnesaemia

No clinical cases of hypocalcaemia or hypomagnesaemia were seen throughout the experimental period. However, in lactation five, outwith this study period, two Jx succumbed to milk fever.

### 3.12 HETEROSIS

In this experiment there were no purebred Jersey cows available. Therefore heterosis was estimated for milk yield and constituents by calculating the percentage difference between the national data for the Jersey and British Friesian and using this figure to estimate the purebred Jersey yield from the observed Friesian yield.

For example:

Milk yield (litres) National Data			Experiment	
Jersey	British Friesian	% difference	Jersey	Friesian
3835	5729	33.1	unknown	4873

$$\begin{aligned}
 \therefore \text{Jersey} &= 4873 \times 33.1 \\
 &\quad \underline{\quad\quad\quad} \\
 &\quad\quad\quad 100 \\
 &= 3260
 \end{aligned}$$

Percentage heterosis could thus be calculated (Table 3.51).

Table 3.51 Percentage Heterosis from National Yields and milk constituents

National figures used as conversion factor of contemporary Friesian data to estimate Jersey data.

	Jersey	Friesian	% difference		
Yield (litres)	3835	5729	33.1		
Fat yield (kg)	202.1	221.7	8.8		
Protein yield (kg)	145.7	185.1	21.3		
	Jersey*	Jersey X	Friesian	% Heterosis	
		observed	expected	observed	
Lactation One					
Yield (litres)	3260	4044	4067	4873	-0.6
Fat yield (kg)	174.6	198.1	183.3	191.5	8.1
Protein yield (kg)	117.8	142.5	133.8	149.7	6.3
Lactation Two					
Yield (litres)	3748	4873	4676	5603	4.2
Fat yield (kg)	214.2	244.2	224.8	234.9	8.6
Protein yield (kg)	146.5	181.0	166.4	186.2	8.7
Lactation Three					
Yield (litres)	3908	5261	4875	5841	7.9
Fat yield (kg)	235.3	264.8	246.9	258.0	7.2
Protein yield (kg)	157.6	198.2	179.0	200.3	10.7

\*On basis of National data corrected for contemporary Friesians.

Expected Jx yield =  $\frac{\text{Jersey} + \text{Friesian}}{2}$

2

that is the mean of the parental data.

In addition the percentage deviation from the baseline Friesian data obtained was calculated for weights, gestation length and days open. This is not a calculation of heterosis but merely an observation of breed difference as heterosis could not be calculated in these parameters due to the absence of the purebred Jersey (Table 3.52).

Table 3.52 Percentage Difference Between Breeds

	Jersey X	Friesian	% Difference
Calf weight (kg)	35.1	45.3	-22.5
Weight (kg) at first calving	393.8	464.9	-15.3
Weight (kg) at second calving	457.7	510.2	-10.3
Weight (kg) at third calving	482.0	572.0	-15.7
Gestation Length			
First	287.6	295.8	-2.8
Second	286.9	291.6	-1.6
Third	278.9	285.4	-2.3
Open Days			
First calving to second bulling	92.3	81.7	11.5
Second calving to third bulling	71.1	80.5	-11.7
Third calving to fourth bulling	51.9	73.6	-29.5

1.0

**CHAPTER 4**  
**DISCUSSION**

#### 4.1 INTRODUCTION

The results obtained in this study will be discussed both in relation to continuous housing and to crossbreeding and pay particular reference to the affects on the general health and related welfare of the cattle.

It is worth stressing that this assessment has largely involved the exhibition of clinical disease and production parameters. Although some considered observation regarding stress and contentment were made these were not sufficiently regular to allow a significant assessment of the relative disadvantages of continuous housing in this regard. Common sense and the observation of cattle at turnout would indicate that they would prefer at least some period grazing naturally but as has already become apparent there are some production advantages in using continuous housing. With increased public concern about these issues it is important that reliable information about such practices is presented dispassionately.

#### 4.2 EFFECT OF MANAGEMENT AND BREEDING PROGRAMME ON HERD HEALTH

##### 4.2.1. Youngstock Health

The health of animals reared from birth to first calving was not adversely effected by continuous housing. This was of no surprise as it is virtually normal practice in south west Scotland for autumn born calves to remain inside until after silage or hay making and it is now becoming increasingly common for calves to be kept inside all summer and not be turned out



until their second year in order to completely avoid roundworm and lungworm infections during this most susceptible period.

In this study mean ZST levels were significantly higher ( $P < 0.001$ ) (Table 3.1) for the Jx calves although these were the lightest of the two groups (Table 3.2). Relations between ZST and calf weight showed a correlation factor of  $-0.46$  that is as 46% of the ZST levels increased calf weight decreased. This is in direct conflict with the findings of Fisher and Logue (1989) who found a trend at the Crichton Royal Farm for the heaviest calves to have the highest ZST levels. They also stated that contrary to the literature there was no survival advantage at the Crichton Royal Farm for a calf having a high ZST and considered that the low mortality rates were due to the high standard of calf care practised. However as there was no reason to believe that the Jx calves received preferential treatment another factor must account for this variation. It is suggested that the higher ZST levels are due to positive heterosis and this agrees with King (1984).

At its simplest this effect could simply be due to the Jx being quicker to stand and suckle, but there is no data to confirm this. Overall the health of both Jx and Friesian calves was excellent during this growing period. No outbreaks of respiratory disease were recorded confirming the findings of Webster (1986) who stated that animals kept in the same group were less likely to contract infections of the respiratory tract

between 6 and 14 weeks of age. Note that this does not necessarily mean that animals were not challenged by respiratory viruses or bacteria. Unfortunately this was not examined, but evidence from a monitoring exercise in 1989 conducted on similar groups of young calves showed serological evidence of challenge by Respiratory Syncytial Virus (RSV) (Macleod, 1989, personal communication). Further, it is presumed since some pneumonias were reported in other calves which were not part of this study that this virus (at least) was probably active in the earlier period. There was no clinical evidence of either gastrointestinal or respiratory nematodes during this period though of course this was not unexpected.

#### 4.2.2. Weights, Forage Intakes and Gross Efficiencies

Mean daily liveweight gains ranged from 0.37 to 0.88 kg per day and the general health of the animals was excellent throughout this period. These figures given in full in Table 3.3 were comparable to those of a group of autumn calvers (DLWG 0.66 kg) grazed during summer and storage fed during winter (Fisher, 1989). There was therefore no evidence of a production advantage to continuous housing. Mean liveweights between the Jx and Friesian groups from birth to first calving were significantly different ( $P < 0.001$  Table 3.2) with the Jx consistently having the lower body weight. This agreed with the earlier data of King (1984). This difference continued throughout the experimental period (Tables 3.11, 3.21, 3.28). This is not surprising since, at least in the first two lactations the Friesians had a higher

dry matter intake and a similar gross efficiency (Tables 3.10 and 3.20). There was no evidence of any interaction with disease.

Closer examination of the figures for efficiency revealed that although not significant here (Table 3.10 and 3.20) it may indeed be the case that the Jx has a slightly higher efficiency particularly in early lactation. Furthermore in the third lactation where efficiency was not calculated the Jx outperformed the Friesian in combined fat and protein yield (Table 2.27). The significant difference ( $P < 0.001$ ) of efficiency found between lactation periods was presumably due to differences in rate of weight gain during the lactation. Differences between lactations was the result of yield and weight differences. Note both groups continued to put on weight throughout the period of recording. In fact these considerations must be viewed with caution since there is a wide variation associated with the formulae for calculating gross efficiency and there is a large variation in the quoted figures for high yielding cows (McDowell and McDaniel, 1968 and Neilson et al, 1983). Increased feed efficiency is, however, considered to be a trait of the young maturing crossbred (Lee et al, 1988), but this increased efficiency, although stimulating body development is not considered to influence mature size (Robison et al, 1980).

#### 4.2.3 Effect on Fertility throughout Experimental Period

The overall proportion of heifers in calf after the first

breeding period was 94% with all but two heifers calving. This figure was higher than the figure of 85% quoted by Leaver (1979) but was in close agreement with the 91% (excluding 2% late embryonic losses) reported in similar groups of cattle at the Crichton Royal Farm in 1990 (Logue, 1990 personal communication). At second, third and fourth matings 63%, 71% and 64% respectively held to first insemination. These figures were significantly higher than the rates of 52% and 49% found by Warren (1979a) in a survey undertaken in the North and East of Scotland.

Overall the percentage Black and Whites holding to first service was high (73%) in comparison to 58% found by Watson, Jones and Saunders (1987) and the 58% found by Warren (1979a) over the same time period.

Various factors could be associated with this high conception rate in storage fed cattle.

1. Maintenance of condition score at 2 plus at calving providing the desirable condition score of 2 at service (Warren, 1979b).
2. Maintenance of high quality protein intake (King, 1971).
3. A more controlled weight loss in early lactation (King, 1971).
4. Availability of trace elements (Hidiriogluo, 1979).
5. Availability of vitamin complexes in particular beta carotene (Lotthammer, 1979 and Hurley and Doane, 1989).
6. Low stress (Webster, 1986).

Although the trace element and vitamin levels of the complete diet were not measured it is believed that the complete diet including grass silage offered to the cattle contained adequate supplies of these nutrients. No clinical evidence of deficiencies were observed but no blood samples were taken to confirm this. However the conception rate to first service was, comparable to the 63% found in selenium treated heifers on this farm (Kelly and McPherson, 1986) and higher than that found by these authors in the non-treated heifers (42%). For this reason it is believed that selenium deficiencies had not occurred.

One of the reasons given by Marsh (1983) for beginning a storage feeding regime is when a poor conception to first service is experienced at turnout and or at subsequent rehousing. This study showed a consistently high conception rate in the cows with only one first calved cow being found empty 35 days after the insemination programme had concluded and one abortion in the third lactation. Maintenance of such high pregnancy rates could have been due to a number of factors and while it is impossible to give any clear opinion as to their relative importance, it is tempting to suggest that they were largely the result of the low stress associated with the very stable relatively small (16 cows) groups. However, to test such ideas fully one would require three experimental groups of cattle , one continuously housed, one run at grass and the third constantly intermixed with other cattle. Blood sampling for mineral analysis would also be pre-requisite to determine mineral status of all animals concerned as opposed

to the presumption here that these were adequate.

#### 4.2.4 Parturition

The majority of heifers required no assistance at calving with only 11 (33%) needing minimal assistance and 2 (6%) requiring a mechanical aid. This latter figure is consistent with the findings of Russell (1983) in grazed first calving heifers. Of the 33% given minimal assistance the possibility of over cautiousness on the part of the herdsman must be considered. Normally on this farm assistance is only given to animals as a last resort, however, it was considered that the small size of the Jx heifers caused the herdsman to be overprotective at parturition. From the findings of Murray (1985) it is believed that these crossbreds would have been able to deliver without aid.

One advantage of continuous housing is greater control of heifer condition and weight at calving. Traditionally autumn calved heifers are in fact late summer calvers, calving at grass and in good grazing years they can calve down at a higher condition score than is desirable. It was interesting to note that the highest calf:cow weight ratio was seen at the second parturition. This was probably a sire effect but nevertheless was an interesting observation which merits further study, since it may be that an "easy calving sire" should be used on second calvers as well as first.

At second and third parturition the Jx required no assistance with calving although one calf was born dead on each occasion. This ease of calving was consistent with the finding of Murray (1985) and according to King (1984) it is generally agreed that the ease of calving which is a feature of the purebred Jersey is also the case for the crossbred. At the second calving four Friesian contemporaries required minimal assistance but at the third only one required assistance.

The survivability of the calves from these crossbred cattle is clearly of importance and in this study the calves of the Jx cows had a higher perinatal mortality (9%) than the calves from the purebred dams (0%). This agreed with the findings of Turton (1981) but disagreed with Donald (1963) and Vesely et al (1986). However, it must be remembered that the calves of the Jx were three breed crosses and the calves of the Friesian, except in parturition four, were two breed crosses and this possibly had some bearing on their relative survival.

A calving index of 365 days is desired by many farmers (Leaver, 1979) yet many farms fail to achieve this despite rigorous culling. Calving indices of 395 days were usual in 1979 and there is little evidence that this has changed markedly. This index is considered to result in a 8% reduction in calf crop (Ellis and Esslemont, 1979). Further reductions from this are thus highly desirable. In this experiment the calving index from first calving to second calving was 376 days , this was to be

expected due to the tendency to calve first calf heifers rather earlier than the bulk of the herd and to allow them a slightly longer recovery period or "make-up" before rebreeding.

In this instance there was a noticeable 11 days difference between the two breeds the Jx having the higher number of days open. This factor could be accounted for by a greater need for the Jx to recover (or "make up" ) since they were put into calf at a much lower weight (258 kg) in comparison to the Friesian (314 kg). However, this hypothesis contradicts King (1984) who considered early maturity to be one of the important features of this breed.

During the second period (second calving to third calving) the calving index was considerably reduced (358 days). At this point the Jx index was 16 days shorter, due to a reduction in days open (71.1 compared to 80.5) and gestation length (278.9 compared to 285.4 days).

Unfortunately, fourth calving occurred outwith the time span of this study and since some were swept up with a beef bull calculating accurate gestation lengths was not possible. However, based on the mean gestation length of 295 days then the calving indices for the Jx was 347 days and the Friesian 369 days. This difference in number of days open was statistically significant ( $P < 0.05$ ) and the resultant difference in calving index represents a theoretical increase in calf crop of 6% for the Jx group. This



shortening of calving index could be a product of the relationship between hybrid vigour and improved fertility parameters (Pearson and McDowell, 1968). Turton (1981) and Rincon et al (1982) also found a reduction in open days in crossbreds. However, this short calving index would also have a penalty in terms of overall milk yield, a factor which will be mentioned later. Nevertheless, with an estimate of some £60 per cow due to a 24 day difference in number of days open (Esslemont, 1990) the cost of loss in milk production is not significant.

Throughout the study period no clinical cases of hypocalcaemia were noted and similarly no cases of hypomagnesaemia occurred. This was despite the fact that the purebred Jersey is the most susceptible of the major dairy breeds to hypocalcaemia (Harris, 1981; Littledike et al, 1981 and Kelly, 1988). However two cases in the Jx cows were seen in the year following this study period so by inference the 50:50 Jx is also probably more susceptible. This predisposition to hypocalcaemia could however have been offset by the relatively young age of these cattle (Payne, 1970; Jonsson, 1978 and Kelly, 1988).

The lack of hypomagnesaemia was to be expected as the major factors predisposing to this condition are the use of fertilisers and increased grazing pressure and minimal concentrate feeding (Butler et al, 1963 and Payne, 1967). Clearly the continuously housed cattle were thus not subjected to this type of regime and when they were first turned out their behaviour and the feeding

of 2kg of concentrate and of prophylactic calcined magnesite with the 'buffer' silage at night during the early post turnout period were sufficient to prevent any clinical exhibition of this condition.

#### 4.3 MILK YIELD AND MILK CONSTITUENTS

As might be expected Friesian milk yield increased with age reaching 5841 litres (305 day yield) in the third lactation. This was a slightly higher figure than the national yield for the British Friesian of 5729 litres. Protein and fat yields also increased each year. In fact protein % and yield are considered to increase with age peaking at third lactation (Ng-Kwai-Hang et al, 1981). Fat also increased throughout this period, but this was probably a consequence of feeding rather than an age effect (Sutton, 1984). As in previous work, storage feeding maintained milk yield and quality (Marsh, 1983, Roberts and Leaver, 1987) the peaks and troughs seen in grazed animals due to forage availability and weather obviously being of little consequence in this type of management system. Thus, there was no evidence of a production disadvantage due to storage feeding.

Although the Jx were a consistently lower yielding breed than the Friesians the difference was only significant ( $P > 0.05$ ) in lactation two. It must, however, be remembered that the calving index of the Jx was lower than that of the Friesian contemporary and so the figure of 347 days for the Jx would slightly penalise milk yield. No statistical significance for fat and protein yield was found between the two groups although fat and protein

percentages were significantly different ( $P < 0.001$ ) throughout all three lactations. Therefore, although the Jx was consistently lower in total milk yield, the higher percentages of fat and protein obtained resulted in a similar yield of the two components in milk. Hence, farmers who are being paid a premium for fat would be well advised to consider the Jersey crossbred as an option for improving profit margin.

#### 4.4 CLINICAL MASTITIS

Incidence of clinical mastitis increased with age with only two cases (6%) in lactation one but thirteen (46%) in lactation three. This final figure was markedly higher than the 31.7% reported by Blowey (1986) and coincided with a high tank TBC and SCC (Figure 3.3). It is considered that storage feeding was a contributory factor since it is well documented that mastitis incidence increases with housing (Ekesbo, 1966; Bakken, 1982 and Bramley, 1989) and in particular with continuous housing (Anon, 1987). It is suggested by these authors that Esch. coli and Strep. uberis are the organisms most likely to be associated with clinical mastitis in housed cattle. In a recent review by Jones (1990) much consideration was given to the role of Esch. coli in clinical mastitis. Although not all clinical cases of mastitis were subjected to a full bacteriological examination it is believed that the majority of clinical mastitis cases in this study were not due to Esch. Coli. A study of isolates from mastitic milk from the whole Crichton Royal Farm herd during this same period confirmed Strep. dysgalactiae as the most common

pathogen (9%) followed by Esch. coli (6%) and Staph. aureus (3%). Furthermore, Staph. aureus and Strep. dysgalactiae were the predominant isolates in the period of intense monitoring indicating a fairly close relationship between clinical and subclinical mastitis in this herd. It should be noted that during this period a considerable proportion of these isolates were from subclinical cases and these could have been due to secondary infection following a primary attack by another organism.

#### 4.5 TANK SOMATIC CELL COUNTS AND TOTAL BACTERIAL COUNTS

No statistical significance between years, months and year\*month interactions were found between these parameters and correlations were low (4.8%). From Table 3.45 it is obvious that the yearly mean for these parameters were well below the National average and the yet to be introduced EC criteria (Document 667).

Booth (1988a and b) suggested that an interparameter relationship existed between the total bacterial counts (TBC) and somatic cell counts (SCC) of the bulk tank milk, stating that when total bacterial counts were introduced not only did number of bacteria in milk fall but so did the number of somatic cells. It is suggested that the initial fall was because farmers perceived a direct relationship between the addition of mastitic milk to their bulk tank and high total bacterial counts and therefore greater care was practised in the identification and treatment of clinical mastitis. Subsequently and because of this improved

care, fewer cows had subclinical mastitis and consequently there was a gradual reduction in somatic cell counts. The introduction of the new EC criteria should thus bring about a further rise in milk quality and a concomitant increase in cow care hygiene practices.

#### 4.6 LAMENESS

In total throughout the three years of the experiment only eleven cases of lameness were observed, this was an incidence of 12.5% in the three years, which was slightly lower than the 15-17% incidence currently expected (Collick, Ward and Dobson, 1989, Esslemont and Wassell, 1990) and was directly comparable to similar age groups conventionally managed at Crichton Royal Farm.

With the estimated cost of a case of lameness being £238 (Esslemont, 1990) it is in the farmers interest to keep incidence of lameness to the lowest possible level. Rowlands, Russell and Williams (1983) reported an increase in lameness with housing, this however, does not appear to be the case in this study though it must be admitted that the study took place over the early life-time of these cattle a time of low incidence. In addition the low rate of lameness was aided by the high standard of hoof care maintained with routine foot trimming by the 'Dutch method' undertaken yearly. Roberts and Leaver (1987) recommended a high standard of foot care in storage fed herds to prevent serious outbreaks of lameness. No breed differences were found in the incidence of lameness with 6 Jx and 5 Friesians being affected. However a subsequent investigation has shown that the Jx cattle

do appear to have harder hooves (Logue and Bradley, 1990).

#### 4.7 INTENSIVE MONITORING

##### 4.7.1 Subclinical mastitis

During this period of intensive monitoring individual somatic cell counts for the two groups were not significantly different although both chloride and lactose concentrations were ( $P < 0.001$ ). The latter also showed significant differences between months and also for group\*month interactions, with the Jx generally having lower chloride levels and higher lactose levels than their Friesian contemporaries.

As somatic cells (Booth, 1988 b) and chloride and lactose concentrations (Renner, 1975) are considered to be the most useful tools in the diagnosis of mastitis, correlation and regression analyses was also carried out. The literature indicates that as somatic cell numbers rise, the chloride concentration of milk should also rise but lactose concentration should fall. In fact low correlations (maximum 22%) were found between somatic cell counts and the other two parameters, but a correlation of -53% (maximum) was found between lactose and chloride concentrations with a significant regression ( $P < 0.001$ ) for these factors (Table 3.36 and 3.37). However, this regression between lactose and chloride concentrations was possibly confounded by the "breed" difference, with the Jx having a higher natural lactose and lower chloride content of milk.

The recent interest in the use of one individual somatic cell count as a means of studying the epidemiology of mastitis in general and the control of bulk tank somatic cell counts in particular (Heuston and Heider, 1985 and Heuston, Heider, Harvey and Smith, 1987) makes the results of Tables 3.38 and 3.39 worthy of note. Although the actual numbers used here were small and in these calculations the actual figures rather than a log transformation were used they nevertheless agree well with the data analyses of Wood and Booth (1983) in which the correlation of month to overall mean varied from 0.45 to 0.60. In general terms there is now considerable information indicating a quite good relationship between lactational individual somatic cell counts and overall milk yield with estimates of a reduction in yield of over 3kg/day for cows in excess of 500,000 cells/ml of milk (Jones, Pearson, Clabaugh and Weald, 1984 and Reneau, 1986). The main point to be taken from this study is that reliance upon one or two samples alone particularly in older cows (these were all third lactation animals) means that in a fair proportion of cattle the diagnosis of a high cell count could be a considerable overestimate (or under -estimate) of the lactational mean. Further-more study of the individual somatic cell count of cows which had a known high somatic cell count in at least one quarter revealed further diagnostic difficulties in that some animals had surprisingly low individual somatic cell counts despite having at least one quarter with a cell count in excess of 500,000. Thus for epidemiological work, and in particular if one is using this individual somatic cell count as a basis for culling, the more information about the cow that is

available the better.

Turning to quarter sampling, the number of cows giving a positive CMT result increased until a peak was reached in April of 11 CMT positive cows per 28 cows; an incidence of 39% somewhat higher than the current level of 25% suggested (Booth, 1988a). However, these cattle were all in their third lactation with relatively high mean somatic cell counts  $343 \times 10^3$  and  $392 \times 10^3$  for the Jx and Friesians respectively and so this might be expected. From the peak in April CMT positive cows decreased to 8 per 28 cows (29%) possibly due to a reduced challenge after turnout (Bramley 1982) though other effects related to well-being and milking machine overhaul cannot be ruled out. Furthermore background clinical infection rate in the entire herd was 44 cases per 100 cows per annum and similar to the incidence rate of the continually housed cattle thus it is difficult to claim that this rate of infection was a direct result of the continuous housing of these cattle.

Some 62% of the mastitis cases occurred between November and March, a figure similar to the 65% occurrence found by Wilesmith et al (1986). Staph. aureus and Strep. dysgalactiae were the most common bacteria associated with subclinical mastitis in this experiment accounting for 36% and 29% of the isolates. Strep. uberis however only accounted for 0.9% of the isolates despite its apparent importance in other herds (Bramley, 1989).



Statistical analysis of chloride content and somatic cell counts in positive milk samples was complex due to the uneven number of samples. The quarter somatic cell counts like individual somatic cell counts varied significantly by month ( $P < 0.05$ ) as did chloride concentration by group ( $P < 0.001$  Table 3.39 and 3.40) and illustrate the difficulty of using just one individual somatic cell count as a means of identifying cows with subclinical mastitis. It is again suggested that the significantly different chloride results were a factor of breed milk composition.

In contrast to the low correlations between individual cow chloride and somatic cell count levels, these parameters were highly correlated in the positive quarter samples (48%) with a statistically significant regression ( $P < 0.001$ ). Lactose was not as closely correlated (28% maximum). Thus as somatic cell counts increased chloride content also increased and chloride content could be regarded as a means of detecting mastitis (Renner, 1975). However, these results confirm that cell counts of individual quarters along with microbiological examination is the most accurate method for diagnosis of subclinical mastitis in general agreement with Pearson, Wright, Greer (1970) and Newbould (1974). However, the proportional findings between somatic cell counts and incidence of mastitis in this experiment indicated that a full study of quarters with a SCC of over 200,000 somatic cells/ml in a quarter milk sample as opposed to the traditionally accepted threshold of 500,000 cells/ml would be well worthwhile.

There is also evidence that in considerations of the practical application of individual (that is 4 quarter) somatic cell counts that this will also apply as only 55% of cattle with a quarter sample of greater than 500,000 had an individual somatic cell count of more than 500,000 but a further 45% had an individual cell count less than 400,000. With the increasing use of individual somatic cell counts by the Scottish Milk Records Association the implications on how this data is used in mastitis investigations is clear.

#### 4.7.2 Other Diseases

No Dictyocaulus larvae were found in any faeces samples which were taken until the September after turnout, and only minimal infection by strongyle species was evident. Pepsinogen levels showed a significant ( $P < 0.001$ ) breed difference there was also a significant ( $P < 0.01$ ) monthly variation.

While it is known that  $L_3$  numbers on permanent grazing pastures at Crichton Royal Farm are low (Kelly, 1987 personal communication) the cattle were probably not primed before turnout as strongyle  $L_3$  presence in silage is not known and is presumed to be zero. Further the presence of an age immunity to O. ostertagia is emphatically denied by Armour (1974). Strongyloides species have been found to cause infection in housed sheep in SW Scotland and it is possible that challenge by this nematode, probably by skin infection, could give some immunity to other nematode species (Mitchell, 1990, personal

communication). Nevertheless these findings are of considerable interest since firstly a continual rise in pepsinogen levels throughout the grazing period occurred and secondly those of the Jx cattle remained lower. It is tempting to speculate that this was a result of positive heterosis causing a breed resistance but since these pepsinogen levels did not ever exceed the accepted clinical figure it is possible that this might be due to a simple physiological difference.

#### 4.8 HETEROSIS

Maintenance of the two different groups of cattle in a highly controlled environment was an ideal background for measuring heterosis of the Jersey crossbred in comparison to their Friesian contemporaries. The Jx exhibited varying degrees of heterosis for different parameters throughout the lactation (Table 3.49). Low adult size is one of the features regarded as important in the Jersey (King, 1984) and the crossbreeds exhibited this feature. Milk yield showed a positive heterosis, and while this agreed with the findings of Pearson and McDowell (1968) and Turton (1981) the extent of heterosis was considerably less than that reported by these workers. However, although milk yields were lower in the Jx than in the Friesian cattle, milk combined fat and protein yield was actually a little higher than the value of the Friesian contemporaries. This agreed with the findings of a positive heterosis by Robison et al (1981) and Ahlborn-Breier (1989). When percentage heterosis was calculated using the mean on the national data for Jersey and Friesian

purebreds as the baseline (Table 3.49) it was found that although milk yield in the first lactation displayed a slightly negative heterosis, it exhibited a positive heterosis in the second and third lactations. This apparent increase with age concurred with the findings of the majority of authors (Pearson and McDowell,1968; Turton,1981 and Rincon et al,1982).

In this study, again using the national purebred data for calculation, positive heterosis was also found for fat and protein yield. This was in contrast to Pearson and McDowell (1968) and Turton (1981) who concluded that positive heterosis was not seen for milk constituents. However, Robison et al(1981) found that fat yield and percentage were parameters which could be affected by so-called hybrid vigour.

Calving index was also reduced for the crossbred apart from the first year, though this parameter was a little confounded by the shorter gestation length of the Jx. Nevertheless, the reduction would aid the longevity of the cow in the herd and so reduce culling rates (Hocking et al, 1988a). As already mentioned the increased number of open days between first calving and second bulling could have been due to the relatively low weight of the crossbred at calving. In other words. while the Friesian heifers were ready for bulling at 15 months of age with a mean weight of 314 kg, the Jx heifers with a mean weight of 258 kg were not. If this is the case it would be a considerable disadvantage for the Jx since economics are forcing more and more farmers to calve heifers as 2 year olds.

**CHAPTER 5**  
**CONCLUSIONS**

### 5.1 STORAGE FEEDING

The main limitation of a grazing system for cattle is that in practice it is difficult to maximise dry matter intake and maintain sward quality at critical times of the grazing year. If partial or a complete storage feeding systems are utilised then dry matter intakes can be increased along with milk output per se (Phillips and Leaver, 1985). In addition with the decreasing availability of land, utilisation of grass to best advantage is required. Storage feeding allows for higher stocking rates with higher utilisation of grass per hectare due to cutting the crop at strategic times. The major disadvantage of the system is the increased cost with many farmers requiring additional silage and slurry storage. Purchase of additional machinery would also be a financial burden (Roberts and Leaver, 1987). However, many farmers would have some farm buildings and machinery readily available.

Marsh (1983) found that on one commercial farm labour requirements by storage feeding were reduced although there was a need for more specialist labour. At the Crichton Royal Farm gross margins of between £1596 and £1822/ha for storage feeding compared favourably to £1347/ha in a conventionally managed herd grazed from April to October (Roberts and Leaver, 1987). The change from a conventional system to storage feeding system must therefore be considered carefully on an individual farm basis.

The system also allows for greater management control and the increased flexibility obtained could for example allow a rapid manipulation of calving date to take advantage of high milk prices in July and August. The simplified grass management can also be a bonus to farmers with widely dispersed grassland which takes time and effort to utilise by grazing. Housing in a controlled environment also prevents stress and the poor conception rates often found immediately after turnout and inwintering can be avoided by this type of regime.

## 5.2 EFFECT ON HERD HEALTH

In this study no significant health problems were encountered as a result of continuously housing cattle for four years from birth. However, in the light of the experience gained here the one disease which could cause problems is mastitis, in both clinical and subclinical forms. The incidence of clinical mastitis was low in the first year of the study but rose to 46% in lactation three. This was higher than the annual national mastitis incidence of 32% reported by Blowey (1986) in conventionally managed herds. The level of subclinical mastitis in the last year was also high, with a maximum of 39% of cows having either a high cell count or a significant mastitis causing organism isolated in one month. Clearly there are a large number of factors influencing incidence of mastitis but it is believed that, in part at least it can be attributed to storage feeding.

It is worth noting that taking both clinical and subclinical cases one of the most commonly found bacteria was Staph. aureus,

an organism notoriously difficult to eradicate. This organism could possibly have been associated with between cow transfer and by inference this means that a very high standard of hygiene at milking time is vitally important in such herds. This is reinforced by the fact that both the somatic cell counts and total bacterial count figures from the Crichton Royal Farm in general were well below the national average.

An increase in lameness incidence as described by Rowlands, Russell and Williams (1983) did not occur and the incidence of lameness was lower than that reported by Collick et al (1989). Hence, if buildings, feeding management and foot care are good storage feeding should not present any serious difficulties in this regard. Similarly an increase in calving difficulties were not observed with only 14.7% needing assistance throughout the three calving periods. This was without the use of an exercise paddock as advocated by Anderson et al (1977).

Thus, in general it can be said that storage feeding had no detrimental effect on herd health but there is a need for good housing, regular foot trimming and a high standard of milking hygiene and other environmental considerations in particular the hygiene and comfort of cow cubicles. For example during this study every cow had access to a cubicle and space to feed along with all the others at the feed-face.



While clearly under some climatic conditions storage feeding could actually be desirable, the public perception in the UK is that cattle should be permitted to graze naturally for at least part of the year. Indeed this is already being legislated for in some Scandinavian countries. However, the use of such models may be of great value as experimental models for the study of the effect of housing environment upon cow behaviour and some diseases.

### 5.3 CROSSBREEDING

In this study the crossbreds showed positive heterosis for milk yield and composition and also for the reproductive traits. It is considered that the most significant of these findings was the shorter calving index in the Jx compared to that of the Friesian. This was estimated as representing an increase in calf crop of 6%. However, it must be pointed out that this difference improved with age. Hence, to utilise this type of positive heterosis the cows must be kept as long as possible and by inference replacements of Jx must be kept to a minimum. The normal 25% replacement rate of the herd practiced in Great Britain would thus not realise the full potential of the advantage.

Maintenance of a 50:50 crossbred herd would cause replacement problems since a number of purebreds would have to be maintained to breed replacements. Line crossing and criss-cross breeding could be utilised to obviate the need for either keeping some

purebred animals or buying in replacements, but a 50:50 cross would not then be attained. However, buying in of the first generation cross would be the most suitable option for the majority of commercial dairy farmers. The pig industry has applied this technology with great success and there seems to be no technical reason why it could not be employed for cattle. However, as with crossbred sows if animals are bought in they must come from a known and reputable source and be quarantined for a period to prevent the dissemination of any disease present in the new stock.

Finally with ovum transfer slowly becoming a more practical and economic on-farm technique it is possible that in the not to distant future crossbred cows could be implanted with crossbred eggs.

## APPENDIX 1

### ESTIMATION OF GAMMAGLOBULINS BY ZINC SULPHATE TURBIDITY TEST

Principle New born calves absorb immunoglobulins from the colostrum of their dams and these immunoglobulins assist significantly in the survival of calves during their first few weeks of life (McEwan, Fisher, Selman, 1968). A turbidimetric method for the detection and estimation of blood gammaglobulin levels was first described for human serum by Kunkel in 1947 (McEwan et al, 1968).

As the immunoglobulins are similar in structure and reaction behaviour to gammaglobulins it has proved possible to apply the zinc sulphate turbidity (ZST) test as an indirect method of measuring calf immune status (McEwan, Fisher, Selman and Penhale, 1970).

#### Reagents:

1. Solution of Zinc Sulphate (BDH Limited)

2.08 g (7.233 m mol)  $Zn_4.7 H_2O$ . Made up with deionised  $H_2O$ .

Store in a bottle fitted with a  $CO_2$  trap.

2. Working Solution

1:10 dilution of stock solution ie 0.72 mmol  $ZnSO_4.7 H_2O$ .

This solution should be freshly prepared to prevent errors due to  $CO_2$  uptake by the reagent and should be used at  $20^{\circ}C$  as the test is temperature dependent.

Standard

3 ml 47.08 mmol/l solution Barium Chloride ( $\text{BaCl}_2 \cdot 2\text{H}_2\text{O}$ )  
added to 100 mmol/l  $\text{H}_2\text{SO}_4$ .

Turbidity produced by the  $\text{BaSO}_4$  formed is read using an EEL  
colorimeter.

Method

- i. 0.1 ml of serum from each sample is added to 2 colorimeter  
tubes (a) and (b).
- ii. Add 6 ml deionised  $\text{H}_2\text{O}$  to tube (a) (blank).  
6 ml working strength zinc sulphate to tube (b).
- iii. Mix by gentle inversion, leave for 1 hour for turbidity to  
develop.
- iv. Remix and read in EEL colorimeter against a 623 filter  
using the blank of each pair to zero the colorimeter.
- v. Results expressed as ZST units and are obtained by  
multiplying the EEL reading by 10.

Results > 20 ZST units - normal  
(that is sufficient immunoglobulin)

< 20 ZST units - inadequate  
immunoglobulin levels

## APPENDIX 2

### CALIFORNIA MASTITIS TEST (CMT)

Schalm and Noorlander (1957)

#### Principle

This test is based on an anionic surface active reagent (detergent) releasing DNA from cells present in milk and forming a coagulum with the DNA. In mastitic milk there is a large increase in inflammatory cells (largely polymorphnuclear leukocytes) and thus the coagulum becomes apparent to the naked eye (Carroll & Schalm, 1962).

#### Test Reagent

10 cc Teepol  
100 cc Distilled Water  
\*Bromocresol Purple to colour slightly

\*The Bromocresol Purple is added to aid the recognition of pH changes in milk and is not an essential part of the reagent.

The test was used on farm as a cowside test but it can also be used in the laboratory.

#### Method 1 (On farm)

Approximately 3 mls of foremilk from each quarter is stripped into the shallow cups of a paddle (each paddle is divided into 4 cups) and mixed with an equal volume of CMT reagent.

The milk and CMT reagent are mixed by a gentle circular rotation of the paddle. The amount of coagulation present is determined by eye and is graded from trace to 3+. This test is subjective,

however in the hands of the skilled operator the grades are directly proportional to the number of somatic cells present in the milk, see Table.

Relationship between CMT and Somatic Cells Present in Milk

CMT Reaction	No. Somatic Cell present
Trace	≤ 500000
1+	≤ 1000000
2+	≤ 2000000
3+	≤ 4000000

### APPENDIX 3

#### BACTERIOLOGY OF MILK SAMPLES

##### Principle

Bacteriology of positive CMT milks from cattle diagnosed as suffering from either clinical or subclinical mastitis was carried out to identify the causal organisms and to obtain antibiotic sensitivity patterns.

##### Materials and Methods

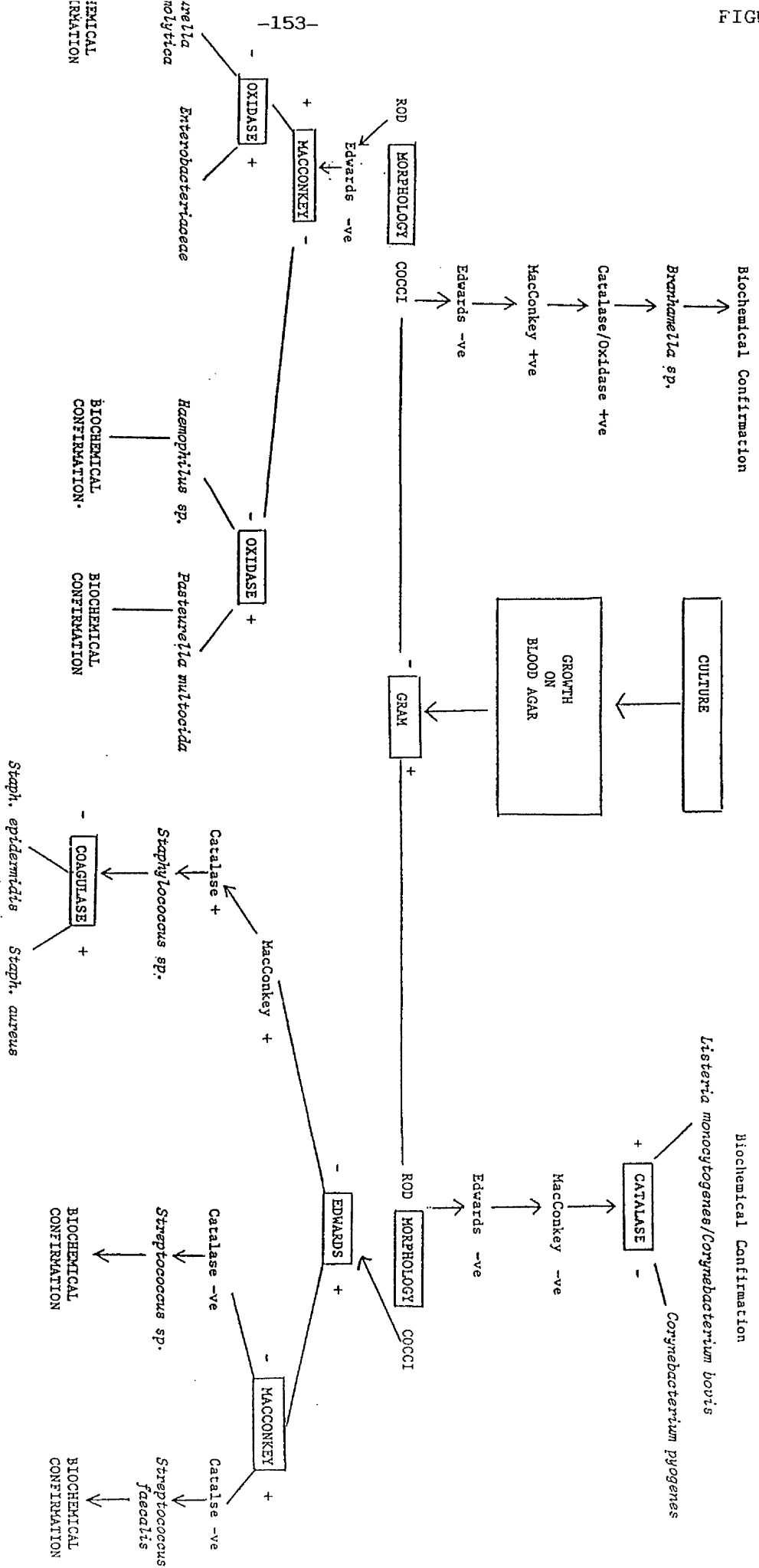
###### Identification of bacteria

Within 3 hours of sampling the milks were examined by Gram smear and bacteriological culture was instituted using the methods described by Central Veterinary Laboratory's Bacteriological Laboratory Guide 1988 as a reference. Culture was carried out on 3 synthetic media reconstituted to manufacturers instructions, namely.

1. Blood agar - Oxoid blood agar base No. 2 (CM271) with 5% defibrinated sheep red blood cells.
2. Edwards - Edwards agar base (Oxoid, CM27) with 5% defibrinated sheep red blood cells.
3. MacConkey agar - (Lab M, Lab 2)

Incubation of the inoculated plates was carried out for 18 hours aerobically at 37°C. After incubation the plates were the examined for growth. Bacterial colonies were identified by a combination of biochemical analyses (See Figure 1). In the case of Staph. aureus a slide agglutination test was used to confirm

Identification of Common Mastitic Bacteria on growth characteristics and gram stain





the presence of the enzyme coagulase (EDTA 0803, Difco Laboratories). If growth was not present at 18 hours incubation the plates were incubated for a further 18 hours and discarded if they remained negative.

In addition milk was also inoculated into a Food and Drug Administration (FDA) plate.

FDA plate - nutrient agar base (Oxoid CM3)) to which is added a fully antibiotic sensitive Oxford Staphylococcus, Staphylococcus aureus, (National Collection of Type Cultures (NCTC) 6571).

A central core was cut aseptically from the agar and 3-4 mls of milk pipetted into this well. On incubation the Oxford Staphylococcus (NCTC 6571) present colonised the agar and could be readily observed. If antibiotic or any other inhibitory substance was present in the milk diffusion of this occurred into the agar around the well and prevented growth of the Oxford Staphylococcus. Milks were thus reported as being positive/negative for inhibitory substances.

#### Antibiotic Sensitivity Testing

Antibiotic sensitivity testing was carried out using the standard Veterinary Investigation Service (VIS) method of Jackson (1981) for Enterobacteriaceae and other appropriate organisms. In brief: Select a colony and suspend in 0.5 ml peptone water (Oxoid CM9) add a further 2.5 ml of peptone water and spread

evenly over the surface of an agar plate (Oxoid: Sensitest CM 409 or ISO-Sensitest CM 471). The aim is to produce "colonies nudging each other". Lay chosen antimicrobial discs on the surface using a disc dispenser which will dispense eight discs in a circle allowing a ninth to be placed in the centre. Incubate overnight at 37°C. Control organisms should also be tested to check on each batch of media. Sensitive and resistant organisms are available as control organisms from the Bacteriology Department, Central Veterinary Laboratory, Weybridge. At the very least these 'control' organisms should be used to examine each batch of media.

#### Interpretation of Results

Generally growth within 2 mm of the edge of the disc denotes resistance. For cephalosporins resistance was shown by growth within 4.5 mm of the edge of the disc. Nitrocefin discs used to identify beta-lactamase production (Francis and Carroll, 1986).

The antibiotics used in this study are shown in Table 1.

Table 1      Antibiotics Tested

Antibiotic	Gram +ve/-ve non-enterobacteriaceae	Gram-ve enterobacteriaceae
Cloxacillin	✓	-
Neomycin	✓	✓
Erythromycin	✓	-
Tetracycline	✓	✓
Novobiocin	✓	-
Nitrofurantion	✓	✓
Penicillin G	✓	✓
Streptomycin	✓	✓
Synulox	✓	✓
Ampicillin	✓	✓
Nafcillin	✓	✓
Cefuroxime	✓	✓
Polymixin	✓	✓
Gentamycin	✓	✓
Choramphenicol	-	✓
Sulphonamide	-	✓
Trimethoprim	-	✓

#### APPENDIX 4

##### ESTIMATION OF MILK CHLORIDE CONTENT

###### Principle

Mastitis causes a rise in the chloride content of milk. Measurement of chloride concentration can thus be used as an aid in the diagnosis of mastitis (Renner 1975).

Chloride measurement was carried out using a Corning 920 chloride meter.

Milk is added to an acid buffer and placed around two silver electrodes which generate a constant emission of silver ions. These combine with the chloride ions present in the milk to form a precipitate of silver chloride. When the chloride supply in the sample is exhausted, that is all that has been precipitated, free silver ions appear in the solution and thus the conductivity changes. This is detected by the sensing electrodes and the readout is terminated.

###### Materials and Methods

1. Corning 920 Chloride Meter
2. 20 mls milk
3. Acid Buffer

Accuracy

Reproducibility

+ 1 m Eq/l at 135 m EqCl/l with a 100 ml sample

+ 2 m Eq/l at 135 m EqCl/l with a 20 ml sample

Range 10 - 350 MEqCl/l.

Linearity

Better than 1 m EqU/l Resolution 1 m Eqcl/l

Long Term Stability

Better than 1 m EqCl/l

1 m EqCl/l = 1 m mol

## APPENDIX 5

### MILK CELL COUNTS USING COULTER COUNTER MODEL TAI I

#### Principle

When cells are suspended as the only particles in their size range in an electrolyte they can be counted individually by a Coulter Counter. This allows the reliable determination of the total somatic cell count of milk (Pearson, Wright, Greer, 1970; Newbould, 1974 and 1978; Heeschen, 1975, Ginn, Packard and Thompson 1979). The method used is as specified in the 1975 International Dairy Federation Seminar

The Coulter Counter can also be modified and calibrated with reference latex particles to count cells of differing sizes. Thus differential counts of the cells in milk is also possible. (Sheldrake, Hoave, Woodhouse, McGregor, 1977; Dohoo, Meek, Martin, 1984 and Wright, Taylor, 1986).

This is a useful facility as mastitic milk contains varying numbers of polymorphnuclear leucocytes and macrophages which have been mobilised to fight infection. It is a particularly useful tool in the diagnosis of subclinical mastitis (Wright, Taylor, 1986).

#### Materials and Methods

##### a. Pretreatment

Fix milk as quickly as possible with Somafix (Coulter Electronics) 50 g/l formaldehyde at a ratio of 3 drops 0.2 ml to

10 mls milk. Mix gently. Heat to  $55^{\circ}\text{C} \pm 1^{\circ}\text{C}$  and maintain for 25 minutes (NB this allows the milk contained in a plastic universal to attain  $55^{\circ}\text{C}$  (for 10 mins). After dilution and heating it is only the cell nucleus which remains.

When cooled to room temperature the samples are suitable for cell counting or may be stored at  $20^{\circ}\text{C}$  for 3 days/ $4^{\circ}\text{C}$  for 5 days. Prolonged storage is however not recommended as some alteration in cell size may occur.

b. Cell Counting Method

- i. Allow sample to attain Room Temperature
- ii. Mix gently and dilute 1:100 with Somaton (Coulter Electronics)
- iii. Mix by inversion and heat to  $80 \pm 1^{\circ}\text{C}$  for 11 mins
- iv. Cool to room temperature
- v. Set Coulter Counter and eliminate interference eg vibrations
- vi. Mix samples by inversion (twice) prior to decanting into accuvettes
- vii. Monitor counts closely to observe fluctuations in counting speed or pattern which may indicate orifice blockage
- viii. Repeat dilutions and counts on any sample exhibiting atypical counts in relation to CMT/Chloride/Bacteriological isolate.

Interpretation of Results

0.5 ml of a 1/100 suspension is counted and population numbers displayed in channel 7 of the TALL. This is either the particles counted at 5.04 mm leucocyte size and above (cumulative population) selected or the particles counted between 5.04 and 6.35 mm (differential population) selected.

Cell sizes are shown in Table 1 using a using a Somacount Coulter Control (Coulter Electronics) Sample and with the cumulative population of cells for this sample. Channel 7 shows a cumulative population count of 2.014 particles per 0.5 ml of suspension. Therefore:

$$\begin{aligned} \text{at a 1/100 dilution true count} &= 4028 \times 100/\text{ml of milk} \\ &= 402800/\text{ml or } 0.4 \times 10^6/\text{ml} - \text{Somatic Cells} \end{aligned}$$

Table 1 Somacount Coulter Control Assay

Channel	Cumulative Pop	Cell Size mm equivalent spherical diameter
1		1.26
2	4881	1.53
3	4581	2.00
4	3217	2.52
5	2518	3.17
6	2207	4.00
7	2014	5.04
8	478	6.35
9	191	8.00
10	135	10.08
11	89	12.70
12	64	16.00
13	32	20.10
14	18	25.40
15	4	32.00
16	1	40.30

The printout for cumulative, differential populations and % volume differences for a low cell count (normal) milk and a high cell count (mastitic) milk are shown in Table 2 and 3



respectively. Also the graphs obtained from these milks are shown in figures 1 and 2 respectively.

Table 2 Normal Milk Coulter Control Assay

Channel	Cumulative Population	Differential Population
1	1538	0
2	1538	0
3	1538	0
4	1538	0
5	1538	0
6	1538	703
7	835	386
8	449	198
9	251	137
10	114	59
11	55	32
12	23	7
13	16	6
14	10	1
15	9	9
16	0	0

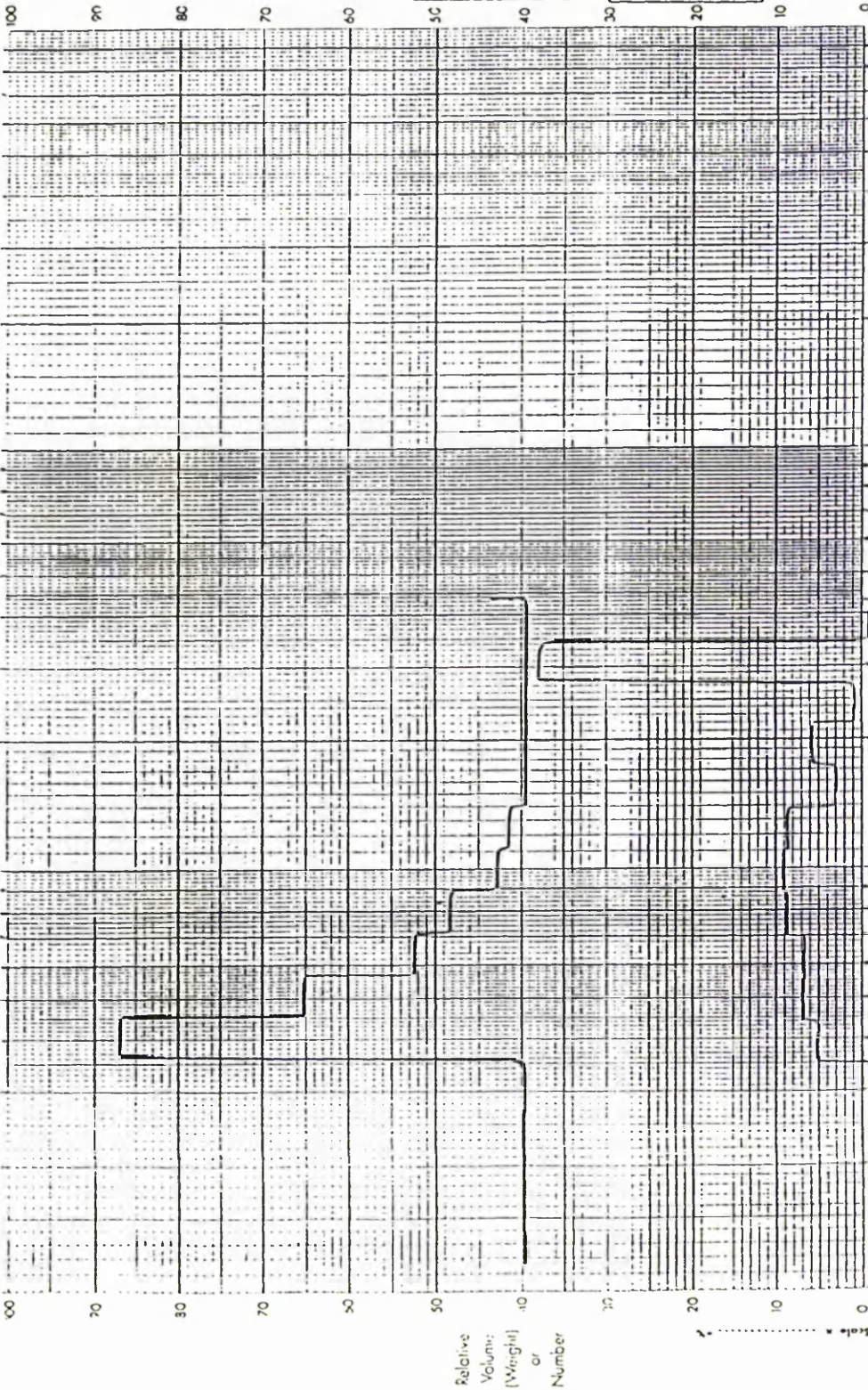
Table 3 Mastitic Milk Coulter Control Assay

Channel	Cumulative Population	Differential Population
1	12528	0
2	12528	0
3	12528	0
4	12528	0
5	12528	0
6	12528	4237
7	8291	7648
8	643	549
9	94	54
10	40	20
11	20	11
12	9	7
13	2	1
14	1	1
15	0	0
16	0	0

COULTER COUNTER © MODEL TA II

SAMPLE C-391  
SOURCE Calcium

DATE 9.11.53  
OPERATOR fs



W	2 <sup>w</sup>
1	2
2	4
3	8
4	16
5	32
6	64
7	128
8	256
9	512
10	1024
11	2048
12	4096
13	8192
14	16384
15	32768
16	65536
17	131072

Calibration	
Particle size dμm	
A	
W	
K	

$$d\mu m = K \sqrt{\frac{2^w}{A}}$$

$$A_i = \left(\frac{d_i}{d_0}\right)^3 \left(\frac{K_i}{K_0}\right)^2 \left(\frac{w_i}{w_0}\right)$$

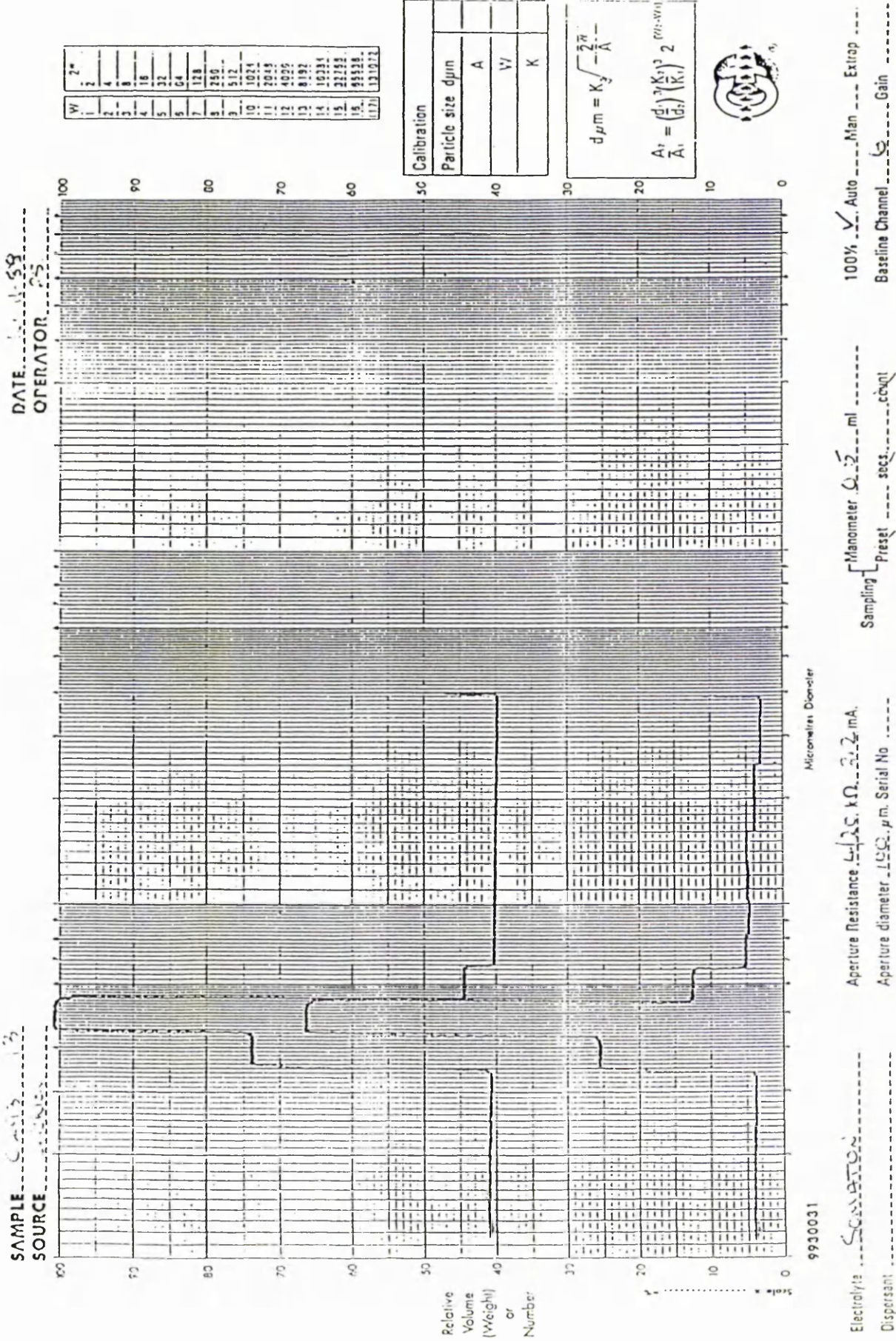


9930031

Electrolyte SOMATON  
 Dispersant \_\_\_\_\_  
 Aperture Resistance 420 kΩ  
 Aperture diameter 100 μm  
 Serial No. \_\_\_\_\_  
 Manometer 0.5 ml  
 Sampling  Preset  Count  
 100%  Auto  Man  Extrap  
 Baseline Channel 6 Gain \_\_\_\_\_



COULTER COUNTER ③ MODEL TA II



## APPENDIX 6

### SERUM PEPSINOGEN EVALUATION

#### Principle

The presence of Ostertagia ostertagi in significant numbers in the bovine abomasum gives rise to extensive biochemical and pathological changes and severe clinical signs. The plasma or serum pepsinogen test (Porter, 1977) is dependent upon 2 major consequences of these various processes. Firstly there is an elevation of abomasal pH from 2 to 7 this reduction in the acidity of the abomasal fluid prevents pepsinogen being activated into the proteolytic enzyme pepsin and thus pepsinogen levels rise. Secondly there is an enhanced permeability of the abomasal wall to macromolecules, This allows inactivated pepsinogen to pass from the lumen via the open epithelial junctions into the circulation. Thus serum pepsinogen levels rise in response to the abomasal damage (Armour, 1974) and this elevation is now the biochemical basis of the diagnosis of blood.

Blood obtained by venepuncture into 7 ml vacutainer (Becton Dickinson Vacutainer Systems, Europe) (no anticoagulant added). Blood was allowed to coagulate and serum drawn off (plasma can be used). Each serum sample was labelled and frozen  $-30^{\circ}\text{C}$  until the sampling period was over. The serum samples were thus analysed in one batch.

Test procedure

Serum is incubated at 37°C with albumin substrate pH 2.0 producing soluble peptides with a terminal tyrosine residue. These peptides are measured colorimetrically using Folin and Ciocalteu Reagent (BDH, chemicals).

(a) Reagents

1. Substrate - 2.5% bovine serum albumin in 0.1 N Hydrochloric Acid (HCl) to a pH 2.0.
2. 10% trichoroacetic acid (TCA).
3. 0.6 N Sodium Hydroxide (NaOH)
4. Folin and Ciocalteu reagent as per manufacturers instructions.
5. Stock Tyrosine standard - 2500 ug/ml  
Dissolve 0.250 g of L-tyrosine in 100 ml of 1M HCl (Stable 4 weeks at 4°C).
6. Dilute tyrosine standard  
2.0 ml of stock standard made up to 100 ml

Technique

1. The tests are prepared in duplicate, tube A being the test and tube b the test blank, tubes C & D being substrate blanks.

Tube	A	B	C	D
Serum	0.25	0.25	-	-
Dist H <sub>2</sub> O	0.75	0.75	0.75	0.75
2.5% Substrate	2.00	2.00	2.00	2.00
10% TCA	-	2.00	-	2.00

Mix and incubate A and C at 37°C for 18 hours

Mix and leave B and D at Room Temp for 18 hours

The TCA added to tube B and D stops the reaction.

2. Add 2 mls TCA to A and C

Remix all tubes and leave to stand for 10 mins

Centrifuge 15 mins

Pipette 1 ml supernatant into fresh tubes and add the following

Tube	A-D
0.6M NaOH	2.5
Folins Reagent (Diluted 1:1 Dist H <sub>2</sub> O)	0.75

Mix and allow 5-10 minutes for colour development

Read at 560 nm within 10 minutes on a spectrophotometer (Phillips PU 8610)

**NB** A quality control sera with known pepsinogen value (7000 units) should be included with each batch as a standard (Sigma).

### 3. Preparation of Standards

Prepare the following in duplicate

	Blank	10 mg/ml standard	25 mg/ml standard
Dist H <sub>2</sub> O	3.0	2.0	0.5
10% TCA	2.0	2.0	2.0
*50 mg/ml Tyrosine	-	1.0	2.5

\*2 ml stock Tyrosine made up to 100 ml

Mix tubes and take 1 ml of fluid and treat as per supernatant as before (2).

Calculation of results

F20  
mean absorption of 10 ug standard = X1

50  
mean absorption of 50 ug standard = X2

$\frac{X1+X2}{2}$  = F where F = the ratio of standard concentration  
and optical density at absorption  
of unity.

Standard Factor (ST) =  $F \times 1000 \times 1000$   
 $0.1 \times 18 \times 60 \times 181.2$

where 1000 = conversion to milli units  
1000 = per litre  
0.1 = vol of serum equivalent in final colorimetry tube  
18 = hours  
60 = minutes  
181.2 = molecular weight of tyrosine

. . . ST = F x 51.1

Results are calculated

(A - B) - (C - D) x ST

Express as milliunits/ml at 37°C to nearest 10 units

Interpretation

Normal < 1500 units

Significant Elevation > 2500 units

## APPENDIX 7

### NEMATODE FAECAL EGG COUNTS USING A MODIFIED McMASTER METHOD

#### Principle

Female adult worms lay eggs, the more adult females the more eggs. A solution of specific gravity sufficiently high to allow the ova or nematode eggs to float and faecal debris to sediment is used in this technique (Gordon and Whitlock, 1939).

#### Materials and Methods

1. Weigh out 2 grams faeces (sample obtained by snatch method).
2. Measure out 60 ml saturated solution of sodium chloride.
3. Put faeces in a seive with a 0.15 mm aperture and sit in an evaporating dish.
4. Pour half the saline into the sieve and macerate faeces with a spatula through the sieve to form a suspension in the dish.
5. Rinse residue from sieve with remaining salt solution.
6. Stir faecal suspension thoroughly; immediately fill pipette and transfer to first chamber of McMaster slide.
7. Restir faecal suspension, fill pipette and transfer to second chamber of slide.
8. Place slide under microscope and view using x 10 objective.
9. Count all eggs present inside both grids marked on counting chamber.



Results

No eggs seen x 100 = no. eggs/1 g of faeces.

Significance

The presence of eggs in faeces indicates the presence of egg laying females in the gut. The number, however, is not proportional to the number of egg laying females in the gut and does not give any indication of male worm presence.

## APPENDIX 8

### LARVAL COUNTS IN FAECES USING THE BAERMANN APPARTUS

#### Principle

The egg of Dictyocaulus viviparus is unusual in that it hatches after a very short period of time and thus unlike sheep the infective L<sub>3</sub> stage is present in faeces. These larvae can be collected using their tendency to migrate into warm water whereupon they sink.

#### Materials and Methods

##### 1. Baermann Apparatus

A large glass or plastic funnel is clamped in a retort stand. A length of rubber tube is fitted to the lower end of the funnel, this is closed by a spring clip.

A metal sieve (0.15 mm gauge) is placed at the top of the funnel. A collecting tube is placed under the funnel.

##### 2. Technique

1. Place 20-30 g faeces on cotton gauze and position of top of sieve.
2. Fill funnel with warm water (less than 37°C) until faeces are covered with 25 mm water.
3. Leave for 18 hours.
4. Collect first 10 ml of fluid in collecting tube by releasing spring clip.
5. Centrifuge at 1500 rpm, discard supernatant.
6. Transfer sediment to a slide and view through microscope using x 10 objective.

Results

Record as positive or negative for the presence of Dictyocaulus  
sp which are distinctly recognisable by their size and shape.

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