Department of Agriculture, Food
and Rural Development

epa

Investigations of Animal Health Problems
at Askeaton, County Limerick

ANIMAL HEALTH

BÓRD SLÁINTE
AN MHÉAN-IARTHAIR

eagasc

Agriculture and Food Development Authority

Environmental Protection Agency

Establishment
The Environmental Protection Agency Act, 1992, was enacted on 23 April, 1992, and under this legislation the Agency was formally established on 26 July, 1993

Responsibilities
The Agency has a wide range of statutory duties and powers under the Act. The main responsibilities of the Agency include the following:

- the licensing and regulation of large/complex industrial and other processes with significant polluting potential, on the basis of integrated pollution control (IPC) and the application of best available technologies for this purpose,

- the monitoring of environmental quality, including the establishment of databases to which the public will have access, and the publication of periodic reports on the state of the environment;

- advising public authorities in respect of environmental functions and assisting local authorities in the performance of their environmental protection functions;

- the promotion of environmentally sound practices through, for example, the encouragement of the use of environmental audits, the setting of environmental quality objectives and the issuing of codes of practice on matters affecting the environment,

- the promotion and co-ordination of environmental research;

- the licensing and regulation of all significant waste disposal and recovery activities, including landfills and the preparation and periodic updating of a national hazardous waste management plan for implementation by other bodies,

- implementing a system of permitting for the control of VOC emissions resulting from the storage of significant quantities of petrol at terminals;

- implementing and enforcing the GMO Regulations for the contained use and deliberate release of GMOs into the environment,

- preparation and implementation of a national hydrometric programme for the collection, analysis and publication of information on the levels, volumes and flows of water in rivers, lakes and groundwaters, and

- generally overseeing the performance by local authorities of their statutory environmental protection functions

Status
The Agency is an independent public body Its sponsor in Government is the Department of the Environment and Local Government. Independence is assured through the selection procedures for the Director General and Directors and the freedom, as provided in the legislation, to act on its own initiative. The assignment, under the legislation, of direct responsibility for a wide range of functions underpins this independence. Under the legislation, it is a specific offence to attempt to influence the Agency, or anyone acting on its behalf, in an improper manner.

Organisation
The Agency's headquarters is located in Wexford and it operates five regional inspectorates, located in Dublin, Cork, Kilkenny, Castlebar and Monaghan.

Management
The Agency is managed by a full-time Executive Board consisting of a Director General and four Directors The Executive Board is appointed by the Government following detailed procedures laid down in the Act

Advisory Committee
The Agency is assisted by an Advisory Committee of twelve members. The members are appointed by the Minister for the Environment and Local Government and are selected mainly from those nominated by organisations with an interest in environmental and developmental matters The Committee has been given a wide range of advisory functions under the Act, both in relation to the Agency and to the Minister

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Investigations of Animal Health Problems at Askeaton, Co. Limerick.

Animal Health

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Investigations of Animal Health Problems at Askeaton, County Limerick
ANIMAL HEALTH
Errata

The numbers of cows in Tables 2-22, 2-25, and 2-28 are incorrect. The corrected numbers are given below.

Table 2-22: Corrected animal numbers – Index Farm A

Year		Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
	1996	Askeaton			7	9	11	13	12	12	12	11	10	10
		Brought-in			8	10	10	8	12	12	13	11	11	12
1997		Askeaton	7	9	9	10	12	12	12	12	8	7	6	4
		Brought-in	11	12	9	13	17	17	16	16	15	14	11	6

Table 2-25: Corrected animal numbers – Index Farm B

Year		Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
	1996	Askeaton			14	16	18	18	18	20	15	14	12	12
		Brought-in						19	21	18	20	17	14	12
	1997	Askeaton	8	6	5	5	8	9	9	9	9	8	5	5
		Brought-in	8	17	18	27	31	31	31	30	29	29	21	11
	1998	Askeaton	3	5		6	8	8	8	8	8	6		
		Brought-in	9	14		26	33	33	33	32	32	29		

Table 2-28: Corrected animal numbers – Control Farm

Year		Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
	1996	Askeaton				5	6	10	11	13	13	12	12	11
		VRL				9	11	13	14	13	12	10	11	9
	1997	Askeaton	9	9	11	12	16	16	16	15	15	18	17	9
		VRL	7	6	10	16	14	14	14	13	13	18	17	12
	1998	Askeaton	9	15	13	13								
		VRL	7	13	18	18								

In Tables 2-29 and 2-30, the rows titled 'Brought-in' in the 'Origin' columns should read 'VRL'.
On page 63, the reference to 'Farm A(LS)' should read farm LS1.
On page 73, references to farms 'LS6' and 'LS5' should read farms 'LS2' and 'LS1', respectively.
On page 141, references to farms ID08, ID05, etc. should read farms RS08, RS05, etc.

Acknowledgements

CONTENTS

CHAPTER ONE

GENERAL INTRODUCTION

This is the final report of the veterinary investigations into the animal health and production problems on farms in the Askeaton and surrounding area. Details of the events leading up to the initiation of these investigations have been documented in an interim report produced by the Environmental Protection Agency (EPA, 1995). A number of other interim reports on the progress of the investigations have already been published (EPA 1995, 1996, 1997a, 1997b, 1998). The present investigations began in February 1995 when the EPA was invited by the Minister for Agriculture, Food and Forestry to coordinate a general investigation into the reported problems in the area. The veterinary investigations were the responsibility of the Veterinary Laboratory Service (VLS) of the Department of Agriculture and Food (DAF).

The animal health and production investigations, which were carried out in conjunction with Teagasc, comprised the following epidemiological and monitoring studies:

I.   A detailed monitoring, over a two-year period, of the health and production of indigenous and brought-in cows on one of the most severely affected farms (Index Farm A). This study was duplicated on a farm remote from Askeaton (Control Farm) with Askeaton- and non-Askeaton origin cows.

II. A detailed monitoring over a two-year period of the health and production performance of indigenous and brought-in cows on a second severely affected farm (Index Farm B) in Askeaton.

III. A Retrospective Epidemiological Survey of 25 self-identified 'problem' herds in the affected areas together with the two Index Farms.

IV. A two-year Longitudinal Study of animal health and production on five of the self-identified problem farms.

V.   A contemporary investigation of animal health on the remaining self-identified problem farms.

VI. A series of studies to measure the immune function of animals on and from the two Index Farms.

VII. A questionnaire survey of animal health and production in the Askeaton area and in other areas remote from Askeaton.

VIII. A laboratory trial to investigate the effects of feeding soil from one of the Index Farms on laboratory rats.

IX. A field study to investigate a suggested association between concentrations of certain liver enzymes of voles from the Askeaton area and environmental pollution

X.   Pathological and analytical investigations on carcasses of animals from farms in the Askeaton area.

The results of these studies are presented in the following chapters.

Background

Reports of severe animal disease and production problems on farms in the area originated from Index Farm A. This is a medium-sized dairy farm situated about two kilometres from the town of Askeaton (Figure 1-1). Problems on this farm were reported to have commenced around 1988. The main problems reported were infertility, pining and mortality in cows and growing stock, perinatal calf mortality, diarrhoea in calves and skin lesions in cows and growing cattle. Prior to 1993, the only outside involvement in the investigation and control of these problems was the herdowner's private veterinary practitioner.

In 1993, a number of on-farm studies were carried out to investigate claims that the problems were linked with environmental pollution. The main conclusion of the studies was that some of the problems may have been associated with deficiencies of one or more of the elements copper, selenium and iodine. Although animal health and production problems occurred at a lower rate in 1994 and 1995, the herdowner continued to be concerned regarding animal performance.

A second farm in the area (Index Farm B) was reported to have suffered severe animal health problems in 1994 and extending into 1995. These were characterised by a high incidence of illthrift, illness and mortality in cows and growing cattle, abortion, calving difficulty and infertility in cows, and illness and deaths in calves. This was a medium-sized dairy farm situated about 0.5 km from the town of Askeaton and about 1.2 km from Index Farm A (Figure 1-1). The owner of this farm also considered that the problems were associated with environmental pollution. The problems on Index Farm B were first brought to the attention of the VLS and Teagasc in early 1995. A full description and analysis of the reported problems on the two Index Farms is given Chapter Four.

At an early stage of the VLS and Teagasc investigations in 1995 it became clear that concerns regarding an association between animal health problems and environmental pollution in the area were not confined to the two Index Farms. There were reports that other farms in the Askeaton area had experienced an excess of animal disease problems in recent years. In addition, there was widespread local concern regarding the implications for human health of any environmental factors having an adverse effect on animal health.

Following consultations with herdowners and residents in the Askeaton area and surrounds - as well as with the locally-formed Askeaton and Ballysteen Animal Health Committee - 25 farms were identified whose herdowners considered they had an excess of animal disease or production problems. In November 1995, a retrospective questionnaire survey of animal health and production was initiated on these farms in order to assess the nature and extent of the reported disease problems. A summary of the main disease problems identified in this survey has already been reported (EPA, 1997a). The full findings and analysis of the results of the survey are presented in Chapter Five of the present report.

As detailed environmental investigations and monitoring up to and including 1995 had shown no evidence that the area was subject to significant environmental pollution (EPA, 1995), it was decided to initiate a number of prospective animal health studies to monitor the incidence of disease on affected farms in the area over an extended period. These comprised firstly, a two-year monitor of animal health and production on the two Index Farms combined with a comparative study on a control farm remote from Askeaton and secondly, a longitudinal study of animal health and production on five of the 25 self-identified problem farms. The

results of these studies are presented in Chapter Two and Chapter Three, respectively.

Owing to the apparently infectious nature of many of the conditions reported from the two Index Farms, a number of studies were also undertaken to investigate the possibility that animals on these farms had a reduced immune response. These comprised a series of studies to measure the immune responses of animals located on and originating from both of the Index Farms together with parallel studies on a remote Control Farm (Abbotstown). The first of these studies commenced in June 1996. The results of the studies are presented in Chapter Seven.

As part of a Mid-Western Health Board questionnaire survey of 2500 households in the Askeaton and other areas, a short questionnaire on animal health and production was administered to approximately 600 herdowners. The results and analysis of this survey are presented in Chapter Six.

Given that detailed monitoring and analysis had shown that environmental concentrations of a range of potential toxic substances were within acceptable limits, a study was initiated to detect the presence of an unidentified toxic substance in soil from the area. This comprised a laboratory study to compare the effects on rats of feeding soil from one the most severely-affected farms (Index Farm B) with those of soil from a control farm. The results of this study are presented in Chapter Eight.

In a paper reporting the activities of certain enzymes in the livers of voles trapped in the Askeaton and other areas, the authors (Fallon et al., 1997) had suggested that lower activities in voles from the Askeaton area might have been associated with environmental pollution. A further study was commissioned by DAF in order to investigate the possibility that differences were due to differences in mineral (selenium) supply. The results of this study are presented in Chapter Nine.

Approach to Investigations

The central questions to be answered by the Askeaton animal health investigations were:

1. Was there any evidence of an unusually high incidence of animal disease in the Askeaton area?

2. If so, what were the underlying reasons?

A supplementary question to (2) above, though in fact probably the main thrust of the entire investigation, was:

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To deal with the last question first, outbreaks of animal or human disease due to environmental pollution can generally be expected to share a number of essential geographical, temporal and clinical features arising from their common causation. While the geographical and temporal associations of a perceived increase in animal health and productions were instrumental in the initiation of the overall Askeaton investigations, the reported clinical syndromes have been characterised by their diversity rather than their similarity.

Typically, environmental pollution by a single or group of related compounds can be expected to result in the appearance of a common clinical syndrome, for example, bone and teeth changes in fluorosis (Shupe, 1980), or hyperexcitability and sudden deaths in lead poisoning (Hammond and Aronson, 1964). Conversely, knowledge of the identity of a pollutant, and its associated clinical syndrome, can be used to identify the extent of its impact on an affected region. However, neither of these circumstances obtained in relation to the Askeaton investigation - there was neither evidence of a common disease syndrome nor of the identity of a specific pollutant. Rather, there were reports of an increased incidence of a variety of conditions which are commonly seen under normal farming practice. The conditions most often recorded in the Retrospective Survey Report (Chapter Five) as occurring with increased frequency or severity were infertility, illthrift, lameness, and mastitis in cows, and illness and deaths of calves.

With the exception of calf mortality, these conditions are generally referred to as production diseases — so-called because their incidence is a function of production while at the same time their occurrence imposes limits on production. They are endemic to virtually all farms and, rather than referring to their absence as being the norm, acceptable ranges of occurrence are generally set as operational targets. Calf illness and deaths are generally accepted as the most important cause of economic loss on farms in this country (Greene, 1978).

DISEASE INCIDENCE

In the absence either of a clearly identified clinical syndrome or of an identifiable pollutant, the main approach of the veterinary investigations, therefore, had to be to assess the incidences of the reported diseases on affected farms in the Askeaton area and to determine if they were significantly higher than would be expected on comparable farms elsewhere. This involved both retrospective and prospective investigations. The former, which dealt with the historical record of disease in the area is the subject of the Retrospective Survey (Chapter Five). The latter, which dealt with the incidence of disease from 1996 onwards was the subject of a number of prospective studies, i.e. the Monitor Study (Chapter Two), the Longitudinal Study (Chapter Three) and the Immunology Studies (Chapter Seven).

It is difficult, if not impossible, to state what is a normal or acceptable incidence of animal disease. Conditions such as diarrhoea and respiratory disease in calves, gastro-intestinal parasitism in all ages, mastitis and lameness in cows, occur to varying degrees on all farms. In order to measure changes in disease incidence, or to draw comparisons with other areas, it is also necessary to have a baseline from which differences can be measured. Unfortunately, other than scheduled or notifiable diseases, there are few statistics available in this country on the incidence of endemic animal diseases. Although a baseline survey of certain animal health and production indices was carried out in the Aughinish area between 1979 and 1981 (Rogers and Poole, 1984), most of the information collected on disease occurrence related to the presence or absence of specific diseases on a farm rather than to disease incidence at farm level. Farm practices have also altered to such an extent as to render the production data of limited comparative value in the present analysis. A questionnaire survey of animal health and production was carried out as part of the present investigation to partly address this deficit and the results of this are presented in (Chapter Six).

Given the dearth of reliable comparative data on the occurrence of the majority of endemic diseases on Irish farms, the overall approach of the following analysis, therefore, has been to compare values from the Askeaton farms with data on disease incidence from a variety of published sources - both Irish and international. A list of reference ranges, compiled largely from surveys of commercial farm animal populations, is given in Appendix 1. This will be used as the main basis for comparison with rates on farms in the Askeaton area throughout the following analysis. It is important to stress, however, that the quoted rates are not necessarily intended as targets for acceptable levels of performance on individual farms - rather they are an indication of the range of values which could be expected from samples of comparable farm populations.

Two sources of data that will be drawn upon heavily are the Teagasc DairyMIS survey and the

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UK DAISY survey. The DairyMIS survey covers about 340 dairy farms in a moderately intensive dairying region in the south of Ireland and provides information on cow-culling and calf mortality (Crosse, 1991). It has a three-tiered system of data collection. At the most intensive level are about 60 high-producing farms that provide regular records on feeding, fertilizer usage, milk production, cow fertility and culling.

The DAISY survey, which is more comprehensive, is based on data collected from dairy herds situated mostly in Southern England (Esslemont and Spincer, 1993). This survey has been in operation for 20 years. The herds are intensively managed and implement a high level of data recording.

While frequent reference will be made to both of these sources, as they are the most comprehensive collections of information on disease incidence available in dairy herds in these islands, they do, however, have limitations with regard to the comparisons that can be made with farms in the Askeaton area. By definition, farms that participate in formal improvement and recording schemes tend to be better and more intensively managed than average. Herd sizes also tend to be larger. The average DairyMIS herd, for example, comprises 80 cows with an annual average yield of 5,228 kg[1] per cow. DAISY herds average 150 cows with average annual yield of 7,264 kg. In contrast, the estimated average number of milking cows in dairy herds reporting an excess of animal health and production problems in the Askeaton area was 44 with an average yield per cow of about 3,637 kg in 1994. Because of these differences in herd size and productivity, disease incidence rates and targets may not always be comparable. Infertility, for instance, is likely to be more of a problem – either real or perceived - on a large intensive dairy farm than on a medium-sized, less intensive farm (Barr and Anderson, 1993). Cow mortality, on the other hand, is generally higher on small farms (Menzies *et al.*, 1996).

CAUSES OF DISEASE

In addition to determination of disease incidence, the Askeaton investigation also had the task of identifying the causes of disease problems reported in the area over the period of interest, i.e. from approximately the mid-1980s onwards. In the present analysis, this exercise concentrates on the main disease problems reported, i.e. those which are of the greatest animal health and economic significance.

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[1] Conversion: 1 gallon = 4.54 kg.

In general, the approach to identification of the causes of disease has altered radically in recent years. While in the past, the emphasis was on the association of specific diseases with individual aetiological agents, the multifactorial nature of disease is now more widely accepted (Thrusfield, 1995). The basis of this approach is to identify, as far as possible, the primary and secondary factors responsible for the initiation of disease. Together, these are referred to as risk factors for the diseases concerned.

Even in relation to infectious conditions - where primary aetiological agents have been clearly identified - the additional involvement of one or more secondary risk factors is usually instrumental in tipping the balance from health to disease. While recognised pathogens such as rotavirus, cryptosporidia, *Haemophilus* and *Pasteurella*, for example, are widely distributed in the animal population, their presence alone is rarely sufficient to initiate outbreaks of disease. Common secondary risk factors for the development of disease of animals under farm conditions include origin and size of susceptible population, nutrition, housing, and weather (Bruning-Fann and Kaneene, 1992). As these factors are, to a greater or lesser extent, functions of the environment provided by the management system under which the animals are maintained, the pivotal role played by management in determining the health status of farm animals must be recognised.

A number of studies have confirmed that management is by far the most important determinant of variability in disease incidence rates between farms (Hancock and Wikse, 1988; Klerx and Smolders, 1997). However, this is not to say that one system of management is necessarily better, or that one system is good and another bad. It is only to affirm that management is a critical determinant of herd health. Any objective investigation of herds with a reportedly high incidence of a multiplicity of animal diseases must, therefore, in addition to examining the possible immediate causes of disease, include assessment of secondary management factors which are most likely to have been influential in relation to health status.

SPECIFIC DISEASE AND PRODUCTION PROBLEMS

The following paragraphs are intended to provide background information on the occurrence and generally accepted primary and secondary causes of the main animal health and production problems encountered in the Askeaton investigation. The

emphasis throughout is on bovine health and production as most of the problems reported in the Askeaton area were in cattle.

INFERTILITY

Infertility, the most commonly-reported syndrome in the Askeaton investigation, is also the most commonly-reported production problem on dairy farms worldwide (Esslemont and Kossaibati, 1996). The term infertility may be applied at the individual animal or herd level. On an individual animal basis, infertility usually implies inability to reproduce. However, on a herd basis, the term infertility is relative and indicates that the overall performance of the herd is below pre-defined targets.

There is no standard definition of infertility. Fertility performance can range from very good to very poor. The point at which the term infertility can be applied to a herd's performance will vary from herd to herd and will depend on a range of factors such as herd size, breed characteristics, calving season, and most importantly, the herdowner's expectations.

There are a range of measurements which can be used to asses the fertility performance of a herd and to draw comparisons between herds. The most important of these include submission rates, heat detection rates, calving to first service intervals, conception rates and overall pregnancy rates (see Appendix 1). Published target and interference levels for these parameters are given in Appendix 1. Performance targets represent attainable goals for efficient production while the interference levels indicate points at which action should be taken if the herd is not to suffer a significant loss in productivity. The targets and interference levels used in this text are a combination of Teagasc DairyMIS and UK DAISY values which are, themselves, derived from the mean and 95% confidence intervals, respectively, of the surveyed populations. In effect, the 95 per cent confidence intervals can be taken as the 'normal' range while the mean or mid-point of the range is the target. The target for the submission rate, for example, is 80 per cent, while the interference level (lower 95% confidence limit) for a 100-cow dairy herd is 73 per cent.

It is worth noting here, that the 95% confidence limits for small herds are very wide. The reasons for this relate to the small size of the samples on which the ranges are based and to the inherently wider variation of fertility performance in smaller herds (Teagasc, 1987; Allrich, 1993). This has particular relevance to the present investigation as the majority of the herds reporting fertility problems were in the small to medium size-range.

It is also important to stress that the DairyMIS and DAISY performance targets are based on surveys of what would be regarded as progressive or intensive farms. It is very unlikely, therefore, that they are representative of the general population of dairy farms. Unfortunately, baseline data for fertility performance of 'average' farms are not available - either here or in the UK.

Infertility is a major impediment to productivity, worldwide (Maher et al., 1995; Esslemont and Kossaibati, 1996). According to O'Farrell et al (1997), infertility is the single most important reason for culling cows on Irish dairy herds. In the UK, about one third of all cow herds experience subfertility (Lamming et al., 1997). In addition, there is convincing evidence worldwide of a significant decline in fertility in recent years. O'Farrell and Harrington (1999) reported a reduction in DairyMIS calving rates from 53 per cent in 1991 to 48.8 per cent in 1996. A similar reduction in fertility has been reported in the UK (Royal et al., 2000).

This trend is believed to be due both to a direct effect of increased negative energy balance associated with increased production and to selection for high milk production at the expense of fertility (Macmillan et al., 1996). In the UK and Ireland it is believed that the replacement of British Friesian cows by Holstein has been responsible for a significant proportion of the decline in fertility (Lamming et al., 1997; O'Farrell and Harrington, 1999).

While reduced conception rate, early embryonic death, abortion and stillbirths will have a negative impact, the efficiency and accuracy of heat detection are the most important determinants of herd fertility performance (Esslemont and Kossaibati, 1996). Heat detection rates are a function both of the frequency and intensity of heat expression by the cow and the efficiency of observation and recording by the herdowner. Expression of heat by the cow is determined by a variety of intrinsic and extrinsic factors. Intrinsic factors are either physiological or pathological. Reduced expression of heat, and complete absence of heat, are referred to, respectively, as sub-oestrus and anoestrus. Both can occur as part of the normal physiology of the reproductive cycle. Anoestrus obtains throughout pregnancy - and for a short time after calving. The first heat after calving is generally a silent heat, i.e. sub-oestrus (Allrich, 1993).

The main pathological causes of anoestrus and suboestrus are ovarian disease (e.g. cystic ovaries), delayed post-calving uterine involution and metritis and, most importantly, conditions which cause pain. In the latter category, lameness would probably be the most frequent cause of reduced fertility (Oresnik, 1995; Lucey et al., 1986b; Peeler et al., 1994). Lucey et al (1986b) reported increases in calving to service and calving to conception intervals of up to one month associated with certain forms of lameness. These authors also reported a 25 per cent reduction in conception rates in lame cows. Ovarian diseases, on the other hand, are sporadic problems generally affecting only a small number of animals in a herd.

Extrinsic factors which influence expression of heat comprise nutritional and environmental factors. Negative energy balance is the commonest cause of delayed onset of heat post-calving (Butler and Smith, 1989; Ferguson, 1991; Webb et al., 1999). The need for the adequate provision of concentrates to cows on grass in spring, during conditions of poor growth, has been stressed in order to control the degree of negative energy balance which is inevitable at this time (Dillon and Crosse, 1997). While there is abundant evidence that a prolonged or more intense energy deficit post-calving can cause anoestrus or irregular cyclicity (Garverick and Smith, 1993, MacMillan et al., 1996), there is little evidence that it leads to suboestrus or silent heats (Allrich, 1993).

Although mineral (trace element) deficiencies have been widely implicated as causes of infertility, the evidence to support these claims is inconclusive (Ferguson, 1991). In a review of the subject, Whitaker (1999) considered that there was insufficient experimental evidence to conclusively implicate copper, iodine, or cobalt, deficiencies in the genesis of bovine infertility. While a selenium-responsive infertility has been demonstrated in growing heifers, evidence regarding its role in cows is conflicting.

Environmental conditions having a negative effect on fertility include poor (slippery) floor design, overcrowding and competition for space in yards and inclement weather (Allrich, 1993). Signs of oestrus and duration may be reduced during periods of hot weather (Risco et al., 1999). Herd size also has an important impact on heat detection. It is well recognised that expression of heat in cows may be reduced in small herds due to reduced opportunities for interaction between oestrus cows (Allrich, 1993).

While reduced expression of heat by the cow (i.e. anoestrus or suboestrus) is a significant contributor to infertility, the main limiting factor in relation to the overall efficiency of heat detection is the length of time and attention which is given to the task of observation and recording by the herdowner. Heat detection is a repetitive and time-consuming task. Studies have shown that the majority of heats occur between evening and early morning (Sreenan and Diskin, 1984). It is estimated that four observation periods per day of 20 minutes each, commencing at 7.00 am and finishing at 10.00 pm, can detect up to 70 per cent of all heats. A further three observation periods would be required to bring heat detection rates up to 90 per cent. Two observations per day, one morning and one evening, will miss at least 50 per cent of heats.

The incidence of non-detected oestrus (due either to inadequate expression or detection) varies widely. Esslemont and Kossabiti (1996) reported a 46 per cent incidence of treatments for non-detected heats in a survey of 90 DAISY herds. In 10 per cent of the herds, the rate was over 90 treatments per 100 cows. According to these authors, "it is widely recognised that mismanagement of herd fertility is the main reason for cows not being observed in oestrus". According to these authors, analysis of records from the DAISY survey indicated that most animals receiving veterinary treatment for unobserved oestrus were, in fact, cycling normally.

After heat detection, conception rates are the second most important determinant of fertility performance. Factors affecting conception rates include accuracy of heat detection, competency of the inseminator, cow fertility, semen fertility, and early embryonic mortality. Accuracy of heat detection is critical in ensuring that cows are inseminated at the optimum time during oestrus (Sreenan and Diskin, 1992). Risco et al., (1999) have reported that surveys have shown that up to 30 per cent of cows may not be in true heat at insemination. They have also reported that conception rates among inseminators can vary by over 20 per cent.

A variety of factors can influence fertility in the cycling cow. These include retained placenta and metritis (Oltenacu et al., 1990). Nutritional influences affecting conception include inadequate or excess protein in the diet (Lee, 1993). The source of protein in the diet can also have an effect on conception rates (Ferguson, 1991). In addition to delaying the onset of heats post-calving, severe negative energy balance can also have a detrimental effect on conception rates (Butler et al., 1996).

Early embryonic loss is a significant cause of infertility. In a review of the subject, Sreenan *et al.,* (2001), quoted rates of embryonic and foetal mortality of 38 *per cent* – the bulk of which occurs between eight and 16 days post-service. The causes of these losses are poorly understood and probably include genetic, physiological, endocrine and nutritional influences. In relation to the latter, it has been demonstrated that a sharp reduction in energy intake immediately after service results in a significant reduction in embryo survival rate (Dunne *et al.,* 1999).

ILLTHRIFT

Illthrift, the second most commonly-reported problem in the Askeaton investigation, is not a specific disease. It is a descriptive term which can be applied to animals suffering from the effects of a variety of nutritional, environmental and health influences. It implies poor condition, due either to loss of condition or, more commonly, failure to put on condition. It is a chronic condition and can be caused by any factors where energy intake does not meet requirements over a prolonged period.

Factors responsible for illthrift may be intrinsic to the animal or extrinsic. Intrinsic factors are those which lead to a reduction in energy absorption or metabolism. They include diarrhoea, intestinal malabsorption, systemic disease and specific organ dysfunctions, e.g. chronic liver or kidney disease. Extrinsic factors are those which give rise to inadequate energy supply and are generally a function of management and environment - either directly through inadequate provision of feed, or indirectly through failure to adequately compensate for circumstances such as adverse weather or reduced quality of fodder.

Illthrift due to intrinsic factors is more commonly a problem of individual animals suffering specific disease or metabolic problems, e.g. chronic systemic disease, lameness, BVD virus infection. However, more acute illnesses, from which an animal apparently recovers, can also have a significant effect on subsequent thrive. Donovan *et al* (1998) reported that the occurrence of acute disease in dairy calves had a significant effect on subsequent growth rates. Septicaemia and pneumonia, for example, reduced growth rates by 13 to 15 days over the first six months of life. Malabsorption syndrome secondary to acute enteritis is a common cause of chronic illthrift in calves and weanlings (Roy, 1990). In a survey of bovine deaths in Northern Ireland, Menzies *et al* (1995, 1996) reported that illthrift was associated with 10 *per cent* of cow deaths and almost a quarter

of all bovine deaths in the one to 24-month age group.

Illthrift as a result of extrinsic factors, i.e. factors leading to inadequate energy supply, is generally a herd or flock problem, e.g. overstocking, inadequate trough space, poor quality silage, inadequate overwinter feeding. However, in situations of marginal nutritional status, sporadic cases of illthrift affecting individual animals on a farm may occur due to variations in energy requirements (e.g. heavy milkers) or intercurrent disease.

MASTITIS

Mastitis refers to inflammation of the mammary gland. While virtually all cases are caused by infectious organisms, outbreaks are generally classified as either contagious or environmental based on differences in the main sources of infection. In the case of contagious mastitis, the primary source of infection is the udder of an infected cow. The organisms are spread from cow to cow on contaminated hands and milking equipment. Control of infection is effected by improved hygiene and, most importantly, by identification and removal of carrier animals.

The environment in which the cow lives is the source of infection in environmental mastitis and outbreaks are generally associated with a breakdown in hygiene. Control must concentrate on identifying the source of the problem and providing hygienic conditions which reduce the weight of infection to which cows are exposed.

Mastitis is the commonest and most important infectious disease in dairy herds worldwide. Some degree of infection can be expected in all commercial dairy herds. A target level of 10 cases per 100 cows per year is considered attainable. However, in practice most herds will have significantly higher rates of disease (Kossaibati and Esslemont, 1997). In a survey of 90 DAISY herds, the incidence of affected cows per herd ranged from about 8 to 41 *per cent* between the top and bottom 10 *per cent* of herds (Esslemont and Kossaibati, 1995). Gardner *et al* (1990) reported that while the mean mastitis incidence was 30 *per cent* it ranged from 1.3 to 70.0 *per cent* at the extremes. Cow age has a significant effect on incidence with substantially higher rates in older cows (Peeler *et al.,* 1994).

In addition to the immediate welfare implications for the cow, the effects of mastitis on herd productivity are threefold. Firstly, direct loss of milk due to removal from sale during period of

treatment and recovery. Secondly, through the depressive effect of mastitis on milk production (Hortet and Seegers, 1998) and thirdly through the negative effect of a high somatic cell count (SCC) on milk quality and sale price. As affected cows have a high likelihood of repeat cases during the same and subsequent lactations (Rowlands et al., 1986), and will continue to provide a source of infection for other cows, removal from the herd as soon as practicable is generally indicated.

LAMENESS

Lameness is the second most important disease after mastitis in UK (Esslemont and Kossaibati, 1996). It is generally held that it has been increasing in incidence over last 30 years mainly due to the switch from straw yards to cubicle systems of housing (Esslemont and Kossaibati, 1996; Clarkson et al., 1996). A wide range of values have been reported for incidence rates in dairy herds. Arkins (1981) reported that between six and 44 per cent of cows were affected in a survey of 3150 cows on 20 herds in Ireland. Whitaker et al (1983) reported an incidence of from two to 55 per cent of cows on farms in a number of areas in the UK. This study also found that only about 25 per cent of cases were treated by veterinarians. The incidence in the DAISY survey (Esslemont and Kossaibati, 1995) ranged from 1.7 per cent of cows affected in the best herd to 34 per cent in the worst-affected herd. As with mastitis, cows affected by lameness have a high likelihood of relapse during the same lactation (Peeler et al., 1994). The incidence of lameness in cows also increases with increasing age (Peeler et al., 1994).

Lameness is primarily a management-related disease. While infectious agents are generally involved in the pathogenesis, they are, with the possible exception of Mortallero's disease, largely opportunist. The main risk factors for lameness are trauma associated with rough floor and roadway surfaces and the secondary softening effect of wet environments underfoot. Nutrition, hygiene, stress, body conformation and genetic factors also play a role (Blowey, 1992). High starch, low fibre diets causing acidosis and subsequent laminitis are the most important nutritional cause of lameness. Trauma is mainly the result of poor quality floor and roadway surfaces and bad housing design (Faull et al., 1996). Long-term exposure to wet conditions underfoot can predispose to development of foot lesions through a softening effect on horn. Poor hygiene, stress, body conformation and genetic factors can also contribute to the incidence of lameness.

Lameness can be a debilitating condition and, in addition to the welfare implications and direct effect on the cow, it can have a significant negative effect on productivity due to reduced mobility and feed intake (Hassal et al., 1993). Both fertility performance (Lucey et al., 1986b; Collick et al., 1989) and milk production (Kossaibati and Esslemont, 1997) can be significantly impaired in lame cows.

MILK PRODUCTION

The main factors determining a cow's milk yield are genetic potential, energy intake, and overall health. The two main dairy breeds in this country, the Holstein and the British Friesian, vary in terms of genetic potential for milk production. On average, the Holstein is capable of out-producing the British Friesian by about 20 to 25 per cent (Garcia-Muniz et al., 1998). However, the cost of this increased production is that the Holstein is a more demanding cow in terms of feeding and management than the British Friesian and is more prone to a variety of production-related diseases where its requirements are not met. In particular, the Holstein is more likely to suffer from reduced fertility and metabolic disease in response to severe post-calving negative energy balance (O'Farrell, et al., 1997).

While genes determine an animal's potential for milk production, energy intake is the major determinant of actual output. The rising yields of the 1980's, while made possible by an improvement in the genetic status of the national dairy herd (currently about 60 to 70 per cent Holstein), were fueled by increasing concentrate inputs (Dillon, and Crosse, 1997). More recently, quota restrictions have reversed the trend towards increased production and have put more emphasis on efficiency of production. In real terms, this means an increased dependence on grass and reduced usage of expensive concentrates (Murphy and Fitzgerald, 1998).

The amount of concentrate feeding required by a milking cow depends on fodder quality (silage and hay), milk yield, and stage of lactation. With silage quality of dry matter digestibility (DMD) of 70 per cent, which is average, it is recommended that a cow milking 27 kg should receive 7 kg concentrates per day (Teagasc, 1994). However, with silage quality of 60 to 65 per cent DMD, which is poor, this would not be adequate.

The effect of inadequate nutrition on milk production will depend on timing and duration. Cows which are under-supplemented from calving may fail to reach their full potential in the first third

of the lactation and subsequently exhibit an excessively steep decline in yield. Excessive or premature reductions in feed in the first third of lactation may also lead to a sharp fall in production.

Besides inadequate provision, there are a variety of reasons why an animal may not consume the actual amount of ration intended. These include inaccurate measurement, inadequate feeding space, competition between animals, and reduced appetite. Unless an accurate record of actual daily feeding is available - based on measurements of properly calibrated scales and confirmation of intake - it is impossible to ascertain to what extent variations in milk production may be related to variations in feed supply.

At herd level, decreased milk production is more likely to be due to grass management problems than to disease. Deterioration in sward quality due to overgrowth (e.g. as a result of undergrazing), for example, can have an immediate negative impact on milk production (Teagasc, 1994; Stakelum and O'Donovan 1998). A shortfall in grass supply can also rapidly translate to a reduction in milk yield. The average daily yield of milking cows in Index Farm A, for example, dropped by about five kg during June 1997, the second year of the Monitor Study, following a brief period of overgrazing on fields which had not received the correct fertilizer application (*see* page 22).

Many of the common diseases may also have a negative impact on milk production. The most important of these are mastitis and lameness. It has been estimated that mastitis will reduce the yield of an affected cow by between 15 and 40 *per cent* over an entire lactation (Beck *et al.*, 1992), while a case of lameness will reduce yield by between 5 and 10 *per cent* (Kossaibati and Esslemont, 1997). As cows affected with either of these conditions have a high risk of repeat cases in the same lactation, the actual milk loss in individual animals can be substantial. Other acute conditions such as retained foetal membranes and ketosis can also have a significant negative effect on milk production (Rowlands and Lucey, 1986). Likewise, systemic disease accompanied by a rise in body temperature will have a depressive effect on milk production. *Leptospira hardjo* infection is also called 'milk drop syndrome' because of the dramatic effect it can have on milk output (Blood and Radostits, 1990). Even sub-clinical mastitis, i.e. raised SCC, can have a significant negative on milk production (Hortet and Seegers, 1998).

CALF DISEASE AND DEATH

The most important categories of calf disease and deaths worldwide are stillbirths and other calving-related losses in the immediate perinatal period, together with diarrhoea and respiratory disease in growing calves. These were also the main calf problems encountered in the Askeaton investigation.

The greater part of calf losses occur during the first three to four weeks of life (Jenny *et al.*, 1991) – with stillbirths and related periparturient mortality accounting for the single largest proportion within this period (Collery *et al.*, 1996). Menzies *et al.* (1996) reported that in a survey of bovine mortalities in Northern Ireland, stillbirths accounted for 62 *per cent* of all deaths of calves under one month old - with dystocia accounting for over two thirds of the cases. At farm level, reported rates of stillbirth are around 6 *per cent* (Appendix 1). In a survey of 247 dairy herds in the US, Hartman *et al* (1974) reported an incidence of 8.2 *per cent*. McDermott *et al.*, (1992) reported a median stillbirth incidence of 2.8 *per cent* in a survey of 123 herds in Canada. Incidences ranged from 0 *per cent* in over a third of the herds to over 30 *per cent* in a small number.

Trauma and hypoxia associated with difficult calvings are the most-commonly reported causes of stillborn and weak-born calves (Greene 1978; Peeler *et al* 1994; Collery *et al* 1996). Laster and Gregory (1973) reported a 5 *per cent* perinatal mortality rate in calves requiring no assistance at delivery compared to a 20 *per cent* rate in calves requiring assistance. Twinning, due to the associated higher rate of calving complications, is also a risk factor for perinatal mortality (Wells *et al.*, 1996).

After stillbirth and perinatal losses, diarrhoea and respiratory disease are the most common causes of calf losses in the first six months of life. A wide range of incidence rates for post-perinatal calf disease and mortality have been reported. A selection of these is presented in Appendix 1. While infectious agents are ultimately involved in the majority of cases of calf diarrhoea and respiratory disease, environmental and management factors such as weather, housing, hygiene and nutrition invariably play an important contributory role. Greene (1978), for example, reported a 70 *per cent* reduction in calf mortality rates on Agricultural Institute farms following the introduction of improved management techniques. The most commonly-reported infectious causes of calf diarrhoea are bacteria, rota- and coronaviruses, cryptosporidia and coccidia (Roy, 1990). Common

infectious causes of respiratory disease in calves include the *Pasteurella* species of bacteria and the viruses of IBR, RSV and PI$_3$.

According to Roy (1990), a calf mortality rate of around 5 *per cent* for the first six months of life can be taken as normal and acceptable. However, reported rates vary widely (Appendix 1). Losinger and Heinrichs (1997), for example, in a survey 1,685 farms in the US reported a 9.4 *per cent* mortality rate (excluding stillbirths) for dairy heifers in the first three months of life. It should also be noted that in any normal population calf mortality and morbidity rates will be naturally skewed with the majority having low or even zero rates and a small proportion with high rates. For example, in a survey of 48 randomly selected dairy herds in Ohio, USA, Lance *et al* (1992) reported that while 17 herds had a zero mortality rate, the remainder had rates which ranged up to an equivalent of a 25 *per cent* calf mortality rate per month (0.28 deaths per calf month at risk). Similarly, in a survey of 43 herds in California, Gardner *et al* (1990) reported that calf mortality rates varied widely with a few farms contributing the majority of calf deaths. One farm, for example, reported 37 young stock deaths due to an outbreak of pneumonia.

In a review of published rates of post-natal calf mortality, Bruning-Fann and Kaneene (1992) noted that while rates were between 1 and 20 *per cent* on an area basis (i.e. calf-level) rates on individual farms ranged from 0 to 60 *per cent*. Hartman *et al* (1974) reported that calf mortality increased as herd size increased. They reported rates of 16, 19 and 27 *per cent*, in herd size ranges of up to 100, 200 and 300 cows, respectively.

Cow Mortality

Rates for cow mortality are given in Appendix 1. Factors affecting cow mortality rates include herd type (dairy *vs.* suckler), farm size, environment (housing), season, weather. Gardner *et al* (1990) reported a mean dairy cow mortality rate of 2 *per cent*, ranging from zero to just under 5 *per cent* at the extremes. Surveys of animal deaths in Denmark and Northern Ireland (Agger and Willeberg, 1991; Menzies *et al.*, 1995) have reported annual cow losses of between 2.5 and 4.2 *per cent*. The latter authors reported that rates were higher in suckler cows (2.36 *per cent*) than dairy cows (1.55 *per cent*). The higher rate in suckler cows is likely to reflect the more extensive management system, i.e. they are more likely to be out-wintered than dairy cows. Menzies *et al* (1995) also reported consistently higher mortality rates in smaller than larger herds – a finding which they also considered

was associated with the more extensive management systems likely to obtain in the former. This study also reported that highest mortality rates were in the first six months of the year - due to the combined effects of inclement weather and spring calving. There was a clear association between calving and mortality with 29 *per cent* of suckler cow and 45 *per cent* of dairy cow deaths occurring within one month of calving.

In the Northern Ireland study (Menzies *et al.*, 1995), 33 *per cent* of suckler cow and 19 *per cent* of dairy cows were 'found dead'. In these cases, the single most commonly reported sign in animals observed ill before death was illthrift. The commonest reported causes of death were coliform mastitis, calving-associated conditions and hypomagnesaemia. The latter condition was the most frequent cause of death in suckler cows.

ABORTIONS AND PERIPARTURIENT DISORDERS

The incidence of abortions in cows ranges from about 1 to 4 *per cent* (Appendix 1). While the diagnostic rate for abortions is low worldwide – between 23 and 46 *per cent* (Barr and Anderson, 1993) - over 90 *per cent* of all diagnosed abortions are infectious in origin (Murray, 1990). Infectious agents most commonly associated with abortion in cows in Ireland are bacterial and protozoal in nature.

The main periparturient disorders seen in cows in Ireland - and also the most commonly reported in the Askeaton investigation - are dystocia, parturient paresis (downer cow), milk fever, retained foetal membrane and metritis. Internationally-reported incidence rates for these conditions are given in Appendix 1. In a survey of 123 herds in Canada, McDermott *et al.*, (1992) reported a median dystocia incidence of 5.8% with some herds having incidences of over 30 *per cent*.

The size of the calf is considered to be the single most important determinant regarding ease of calving (McDermott *et al.*, 1992). The clinical significance of this can also be compounded by factors such as unattended calvings, inadequate, excessive, or premature intervention, and cow condition and parity. According to Peeler *et al* (1994), who reported an incidence of dystocia from 2 to 36 *per cent*, the wide range in incidence can be at least partly explained by variations in the willingness of herdsman to assist, the necessity to do so, and the criteria used by the herdsman to define a case. Excessive condition (i.e. overfat) of cows pre-calving is a recognised risk-factor for dystocia (Gearhart *et al.*, 1990). The incidence of dystocia is also closely correlated with parity (i.e.

number of calvings per cow). Busato *et al* (1997) reported a 2.9 *per cent* incidence in cows compared to 13.6 *per cent* in heifers. According to Crosse and Soede (1988), the incidence of dystocia and stillbirths are approximately two to four times higher in heifers than in older cows.

There is a strong inter-relationship between the incidences of the main periparturient disorders. Downer-cow syndrome, for example, is a common outcome of dystocia, i.e. due to exhaustion and traumatic damage to the pelvis. Dystocia and periparturient mastitis, on the other hand, are risk factors for retained placenta (Emanuelson and Oltencau, 1998). The incidences of dystocia and retained placenta are closely associated with the subsequent development of metritis. It is clear, therefore, that a herd with a dystocia problem, possibly due to management decisions relating to cow breed or sire selection, could be expected to suffer from an increased incidence of several periparturient conditions. As the incidence of dystocia also has an important bearing on subsequent calf health and survival (Waltner-Toews *et al.*, 1986) as well as cow fertility (Oltenacu *et al.*, 1990), the implications of calving-related problems can extend well beyond the immediate post-calving period and result in an increased incidence of a range of cow and calf conditions on an affected farm.

TWINNING AND CONGENITAL DEFORMITIES

Twinning and congenital deformities were also reported to have occurred at an increased incidence on some of the Askeaton farms - though it should be noted that the incidence of twins on the original Index Farm (A) was low. According to the report of an independent investigation of the farm in 1993 *"only two sets of twins* (were reported) *in 30 years"* (Dowding and Dowding, 1994). Internationally reported rates for twinning are given in Appendix 1. Worldwide, rates range from about 1 to 6 *per cent*. In a UK survey, Esslemont and Kossaibati (1996) reported a range in incidence of twin calvings from 0 to 9 *per cent* and noted that there had been a trend of increasing incidence (of twins) over the past 25 years associated with increasing milk production.

Mee (1991b) reported that about 11 *per cent* of calves that were stillborn or died shortly after birth had one or more congenital defects. In 5.4 *per cent* of cases the defects were considered lethal. In another Irish study, Greene (1978) reported that calves with congenital defects usually had multiple abnormalities. Intestinal malformations occur frequently in cattle (Steenhaut *et al.*, 1976). The commonest, atresia ani/recti, is genetic in origin.

MINERAL DEFICIENCIES

For a number of reasons, mineral nutrition of farm animals in the Askeaton area has had a particular significance in relation to the reports of an increased incidence of disease in the area. Firstly, baseline studies carried out in the area prior to the development of the Aughinish bauxite refinery identified the marginal or deficient status of pastures and livestock on many farms in the area in relation to copper, selenium and iodine nutrition (Fleming and Parle, 1983; Rogers and Poole, 1984). A further survey carried out in 1993 confirmed these findings and reported that there was no evidence of a significant deterioration in the herbage status of these elements for animal nutrition (Coulter, *et al.*, 1994). Secondly, it has been widely speculated that industrial emissions of sulphur in the area – in the form of SO_2 – could interfere with the availability or uptake of one or more of these elements. The authors of the Aughinish Baseline study (Rogers and Poole, 1984) speculated that increased deposition of sulphur from industrial sources in the area could tip the balance from marginal supply to deficiency on some farms. Thirdly, some of the problems reported in the Askeaton investigation were, or were said to have been, consistent with the effects of deficiencies of one or more of these minerals.

The following sections give a summary of the main roles played by each of the three minerals copper, selenium, and iodine in relation to animal health - as well as outlining some of the most important effects of deficiency and the significance of the latter in relation to the Askeaton investigation. More specific analyses of the possible roles of primary or sulphur-induced deficiencies are discussed elsewhere in relation to specific syndromes or individual farm case-histories. The suggested association between iodine deficiency and some of the problems reported on Index Farm A, for example, is discussed in detail in Chapter Four.

It should also be noted from the following discussion that a large proportion of Irish pastures contain concentrations of these elements below the recommended minimum for animal nutrition. However, the fact that clinical disease is not more widespread in unsupplemented animals suggests either an overestimation of requirements or failure to adequately characterise or recognise the full spectrum of signs attributable to deficiency.

Copper

Copper is a component of many essential enzyme systems in the body and the signs of deficiency are varied and can effect all stages of growth and

production (Graham, 1991). Signs of copper deficiency include illthrift, alteration of coat color, diarrhoea, anaemia and infertility (Blood and Radostits, 1990). Copper also has an important role in relation to immune function. Reductions in cytokine production (chemical messengers involved in immune inflammatory reactions) and the humoral immune response have been reported in copper deficient calves (Gengelbach and Spears, 1998). Copper deficiency can also depress the phagocytic activity of neutrophils (Babu and Failla, 1990).

Copper requirements for cattle range from about 5 to 15 mg/kg dry matter depending on age, production status, feed type and the concentration in the feed of certain other elements (ARC, 1980). Herbage concentrations of copper above about 10 mg/kg dry matter are generally considered optimal for grazing livestock. As the majority of Irish pastures have values below this level (Fleming, 1962), the risk of clinical or sub-clinical copper deficiency in unsupplemented cattle is high on farms in this country.

In addition to inadequate supply, copper deficiency can also be induced by the presence of excess of elements such as molybdenum and sulphur in the diet. Any pasture molybdenum concentrations above 5.0 mg/kg, for example, are likely to lead to problems with copper availability. Values between 2.0 and 5.0 mg/kg, where the copper-molybdenum ratio is less than 2.0, may also be significant.

Under Irish grazing conditions, molybdenum is the element most likely to interfere with the bio-availability of copper. Herbage concentrations of molybdenum above about 2 - 5 mg/kg can lead to the formation of stable copper-thiomolybdates and consequent reduction in copper availability. About 40 *per cent* of Irish soils are known to supply excess molybdenum (Fleming, 1962). These include soils in the Askeaton area (Coulter, *et al.*, 1994). In a survey of farms in the south-west of Ireland, Poole and Rogers (1984) reported clear evidence of a negative correlation between herbage molybdenum and blood copper concentrations – which conforms with clinical experience regarding the higher probability of copper deficiency on high-molybdenum pastures.

Copper availability can also be reduced in the presence of high sulphur concentrations due to the formation of insoluble copper-sulphur compounds in the digestive tract. The possible significance of this interaction in the context of the Askeaton investigations is discussed further below.

Selenium

Selenium is a component of the antioxidative enzyme glutathione peroxidase. This enzyme prevents cellular injury by removing reactive peroxide by-products of cell metabolism (Wichtel, 1998). Classical selenium deficiency is characterised by myopathy and its attendant clinical signs, i.e. stiffness, muscular weakness, and sudden deaths. Other conditions which have been attributed to selenium deficiency include abortion, stillbirths, weak calves, retained placenta and infertility (Blood and Radostits, 1990). However, the role of selenium in relation to these latter, and its possible mode of action, remains to be clarified (Wichtel, 1998).

Laboratory studies have also demonstrated an association between selenium deficiency and impaired immune functions (Wichtel, 1998). However, there is little field evidence as yet to confirm these findings. The results of an animal study on Index Farm B, carried out as part of the present investigations, showed no evidence of a significant reduction in KLH-responsiveness (a measure of cell-mediated immune function) in selenium-deficient cattle when compared to cattle receiving selenium supplementation. These findings are discussed in Chapter Seven.

Recent studies which have demonstrated the involvement of selenium-containing enzymes in the production of thyroid hormones (Arthur *et al.*, 1991) have raised the possibility that some of the conditions attributed to selenium deficiency may, in fact, be due to a combined selenium-iodine deficiency (Wichtel *et al.*, 1996).

Recommended dietary concentrations of selenium range from about 0.1 to 0.24 mg/kg dry matter (Graham, 1991). Teagasc archival data indicates that 75 *per cent* of Irish herbage samples have concentrations below 0.1 mg/kg which suggests that the majority of unsupplemented cattle in this country receive less than the recommended minimum quantity of selenium.

Selenium toxicity is characterised by a range of signs including stiffness, lameness, hoof deformities, dullness and emaciation, and may occur in areas of high natural geochemical concentrations of the element (Blood and Radostits, 1990). High selenium concentrations were recorded in blood samples collected from animals on two farms in the Askeaton investigation which were located on a previously-identified outcrop of seleniferous soils.

Iodine

The main role of iodine in relation to animal metabolism is as a component of thyroid hormones that are necessary for normal growth and development. The primary signs of iodine deficiency are goitre (enlarged thyroid), hair loss, abortion, and stillbirths (Blood and Radostits, 1990). Iodine deficiency has also been associated with infertility, immune suppression, and retained placenta (Graham, 1991). A possible contributory role of selenium deficiency in relation to some of these conditions remains to be clarified (Wichtel, 1998).

Nutritional requirements of iodine for livestock have not been clearly defined. The UK Animal Research Council (ARC, 1980) and the US National Research Council (Anon, 1989) recommendations state that diets containing from 0.15 (summer) to 0.5 mg/kg (winter) iodine are adequate for ruminants − with a maximum total intake of 12.0 mg iodine per day.

Confirmed clinical iodine deficiency is rare in Ireland − probably because of the relatively widespread availability of the element. Although normal Irish pasture concentrations of between 0.1 and 0.5 mg/kg would be unlikely to provide the recommended daily intake of 1.5 mg, grazing animals receive most of their iodine from soil which generally has a concentration in excess of 5.0 mg/kg (McGrath and Fleming, 1988). Even relatively small quantities of concentrate supplementation will also provide adequate iodine.

Phosphorus

Phosphorus is an essential element for bone and tooth development and is also involved in carbohydrate metabolism. Clinical deficiency, which used to be common in low-phosphorus areas of this country, e.g. bog-lands, is now rare due to the widespread use of phosphatic fertilizers. Mild deficiency may occur in high-yielding cows due to the high phosphorus requirements of milk production. The main significance of phosphorus in the Askeaton investigation is in relation to a possible aluminium toxicity. The latter exerts its main effects through the formation of insoluble phosphorus compounds that are then unavailable for absorption. The result is hypophosphataemia and an induced phosphorus deficiency (Allen et al., 1986; Allen et al., 1991; Crowe et al., 1990). The clinical signs of phosphorus deficiency include weak bones, joint-lameness ('bog lameness'), abnormal hoof growth, depraved appetite or anorexia, and illthrift (Blood and Radostits, 1990).

Potential for sulphur-induced mineral deficiencies

Sulphur, possibly in the form of sulphates, can interfere with the bioavailability of copper and possibly also selenium (Suttle, 1974; Langlands et al., 1981; Rogers, 1990). Because of the presence in the Askeaton area and surrounds of industrial sources of significant quantities of sulphur dioxide (EPA, 1995), the potential exists for the occurrence of sulphur-induced mineral deficiencies in animals consuming contaminated pastures and preserved fodder. The biochemical basis for these interactions, and the environmental and nutritional implications of the likely rates of deposition of industrial sulphur on grassland in the Askeaton area, are discussed in depth in the external assessment by N.F. Suttle (Appendix 15). The following paragraphs give a brief overview of the topic.

One of the mechanisms whereby sulphur could interfere with the uptake of copper is thought to involve the formation of insoluble copper sulphides (Suttle, 1975) and depends not only on the concentrations of each of these elements, but also of molybdenum. Thiomolybdates, in addition to specifically reducing the availability of copper in their own right, can have an enhanced effect in the presence of dietary sulphur above certain concentrations. However, most of the information regarding this interaction is based on the results of laboratory trials and the circumstances under which sulphur-copper interactions are of nutritional significance for livestock under commercial farming conditions have not been clearly defined.

While Suttle (1986) reported that a sulphur concentration 0.4 *per cent* retarded the replenishment of liver copper reserves in sheep with artificially-induced copper deficiency − and replenishment was further retarded by addition of molybdenum to the diet − Poole and Rogers (1984) reported no evidence of a correlation between blood copper concentrations and herbage sulphur levels of between 0.1 and 0.7 *per cent* in a survey of farms in the south-west of Ireland.

There is, however, little evidence to suggest that herbage sulphur concentrations in the Askeaton area are significantly different from those found elsewhere in the country. The normal range for herbage sulphur concentrations on Irish farms is from 2.7 to 6.7 g/kg (Whitehead, 1966). Similar values have been reported for samples collected on farms in the Askeaton area between 1994 and 1997 (EPA, 1997a, 1997b, 1998; Soil, Herbage, Feed and Water Volume). Re-sampling of the original baseline farms also failed to show any evidence of

an overall increase in herbage sulphur in the Askeaton area between 1980 and 1993 (Coulter, *et al.*, 1994). Environmental monitoring has also failed to show any evidence of significant pollution of the area with industrial-origin sulphur. Monitoring by the EPA since 1994 (EPA, 1995, 1997a, 1998) has shown that sulphur deposition of industrial origin was negligible in comparison to other agricultural and environmental sources. Monitoring in earlier years also showed that non-marine deposition of sulphur in the area was low and comparable to other regions in the west of Ireland (Bowman, 1991). Overall, these findings would indicate that it is highly unlikely that

sulphur-induced mineral deficiencies - regardless of the source of the sulphur - had a significant impact on animal health or production in the Askeaton area over the past 10 or 15 years.

The possible nutritional significance of sulphur-selenium interactions is less clear. Although a theoretical basis exists for the inhibition of selenium absorption by animals, it appears that this is unlikely to be of significance under normal grazing conditions (Wichtel, 1998). Kahn *et al* (1987) reported no evidence a significant reduction in the selenium status of cattle fed a diet containing 0 75 *per cent* sulphur compared to cattle on a diet of 0 2 *per cent*.

Figure 1-1: Location of two Index Farms (A and B on map) and 25 other farms reporting animal health problems in the Askeaton area.
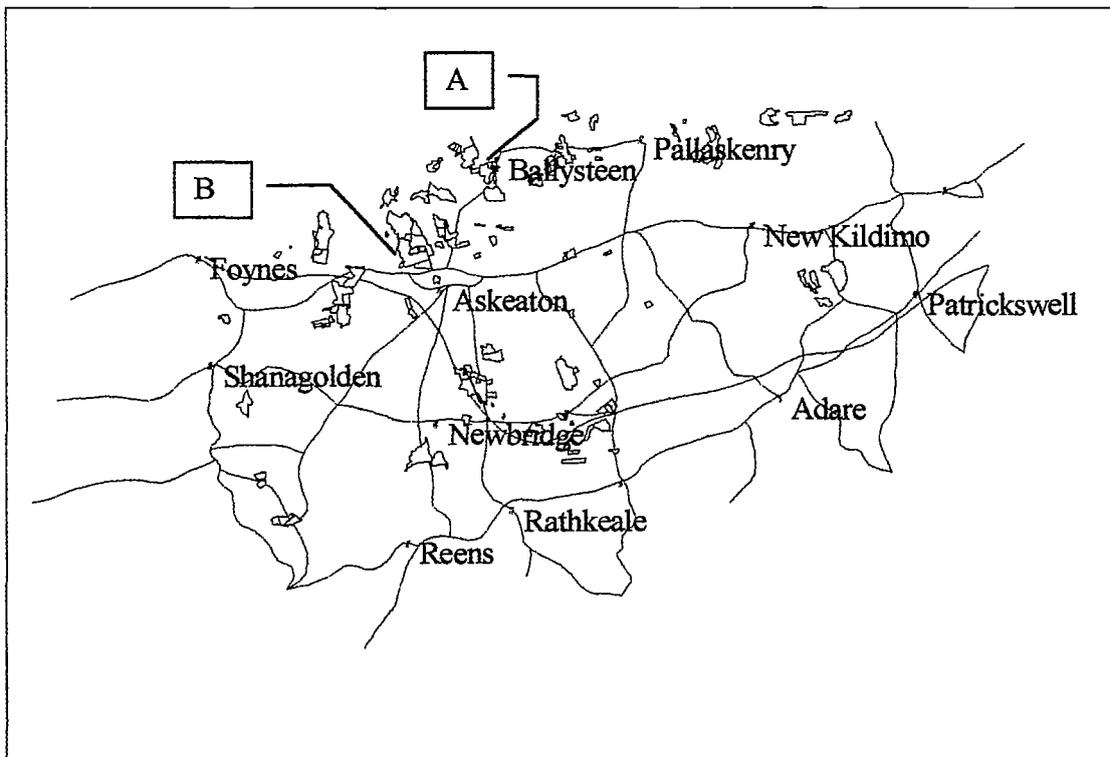
CHAPTER TWO

MONITOR STUDY ON THE TWO INDEX FARMS AND A CONTROL FARM

A Monitor Study of animal health and production was carried out on the two Index Farms in Askeaton and a Control Farm at the Central Veterinary Laboratory (CVL) in Abbotstown, Co. Dublin. The purpose of the study was to determine if animals (cows and growing stock) on and from the Index Farms continued to exhibit production and health problems outside accepted normal limits following the implementation of a standardised regime of management and nutrition based on published Teagasc guidelines (see Study Protocol). In the event that significant problems, for which an underlying cause could not be identified, continued to occur, then it was hoped that the availability of contemporaneous clinical samples and measurements would help to indicate the most appropriate areas for further analytical or other investigations.

Details of the study design have already been reported (EPA 1997a). Briefly, Index Farm A was leased to the DAF in October, 1995 following which essential refurbishments to farm buildings and effluent management facilities were carried out. The herdowner continued to be responsible for the day-to day running of the trial according to the Study Protocol (EPA 1996, unpublished). An assistant farm manager was also appointed by DAF in June 1996. For the purpose of the Monitor Study, the herd comprised about 30 dairy cows, approximately half of which were indigenous to the farm (Askeaton-origin) and half of which were from the Control Farm at CVL Abbotstown (Abbotstown-origin). A herd of similar size and composition (i.e. Askeaton- and Abbotstown-origin cows) was maintained at the Control Farm.

The original exchange of cows between Abbotstown and Index Farm A took place in December 1995. A further group of Abbotstown-origin cows was sent to Askeaton in July 1996 to replace some of the Abbotstown-origin animals transferred in December 1995 which had to be culled following housing-related health problems which developed over the winter of 1995 (EPA 1997a). The Askeaton-origin cows from Index Farm A were British Friesian type of unknown genetic merit. The Abbotstown-origin cows

brought onto Index Farm A were Holstein-type of medium genetic merit

The Monitor Study on Index Farm A and the Control Farm commenced on 1 April 1996 and continued until 31 March 1998. In order to facilitate the herdowners' wish to recommence full-time farming at the earliest possible date, the DAF-appointed assistant farm manager was withdrawn from the farm at the end of 1997 after all cows had been dried off. As a result, the DAF had no involvement in the day-to-day management of the farm during the 1998 calving season, i.e. the final three months of the project. However, monthly blood-samplings and animal inspections continued up to the end of March, 1998.

A second Monitor Study was designed for Index Farm B. Arrangements for the purchase of the farm concluded in November, 1995. Following that, some of the older animals were culled on grounds of advanced age (EPA, 1997a). Essential refurbishment of farm buildings and effluent management facilities was also carried out. In spring 1996, a farm manager was appointed and the herd was augmented by the purchase of pregnant and calved Holstein cows of known pedigree (RBI average 106, range 89 – 118). Indigenous cows on Index Farm B were British Friesian type of unknown genetic merit.

The objective of the study on Index Farm B was to maintain a commercial-type dairy herd of approximately 50 cows according to the management and nutrition procedures outlined in the Study Protocol (EPA 1997a). The study commenced in May 1996. Although the study formally ended in April 1998, monthly animal health and production monitoring continued until October 1998.

As outlined in the Study Protocol, assessment of performance of the Askeaton herds was to be made by reference to published normal ranges for the type and age of animal concerned (Appendix 1). The study design did not envisage direct comparison between the Abbotstown and Askeaton farms. Although a standard of management was laid down for all three farms, the main purpose of this was to minimise the possible contribution of

management or nutrition to deficiencies in performance - rather than to attempt to implement an identical standard of management on all three farms. Differences in weather, grass quality and the actual implementation of management regimes, as well as differences in genetic merit, reproductive status, and age of cows, were such that it would have been inappropriate to assume that observed differences in performance could be attributed solely to location. However, where clear and consistent differences were found, these will be discussed and, where possible, explanations offered.

The main purpose of locating a control herd in Abbotstown was to determine if Askeaton-origin cows from Index Farm A would exhibit acceptable performance when removed from the Askeaton environment. If not, then the possibility that they possessed some intrinsic or long-term attribute, either genetic, or acquired (e.g. toxic) prior to the move to Abbotstown, and which limited performance, would have to be investigated. In the event that either or both of the groups in Askeaton performed poorly, the Abbotstown groups could also provide information of value in any analysis of such a result.

While a pairing of animals was envisaged in the original study design, this had to be abandoned owing to udder health problems and cullings arising from the overwinter housing difficulties of 1995 as well as to variations in fertility performance between the farms and resultant loss of synchronisation in calving dates (EPA, 1997a). In addition, owing to the limited supply of Askeaton-origin dairy cows (34 in December 1995 – over half of which were over six years of age), problems also arose in relation to matching when replacements were needed for animals culled on health or welfare grounds.

ANIMAL HEALTH

The present study was set up to monitor the incidence of animal disease on the two Askeaton Index Farms - as well as the Control Farm in Abbotstown - and to investigate the immediate and underlying causes of any disease outbreaks which occurred. The results of the study must be viewed in the context of the' serious animal health and production problems which had been reported on these farms in the years immediately preceding its commencement. As described elsewhere (Chapter Four), mortality in cows, yearlings and calves, as well as pining, skin lesions and abortions in cattle were reported to have occurred at an abnormally high rate on Index Farm A between 1992 and 1995. On Index Farm B, up to 14 cows were reported by

the herdowner to have died in 1994 and up to 18 in 1995. Lameness, ill thrift, skin diseases and parturient paresis in cows, as well as calf deaths and illthrift in growing cattle were also reported to have been problems.

Details of the methods used to monitor animal health and disease for the duration of the Monitor Project are described in the Study Protocol (EPA, 1997a). Briefly, all cattle were subjected to regular veterinary clinical examinations. These were complemented by monthly weighings and body condition scoring of cows. All incidents of animal disease, together with a description of clinical signs and details of treatment, were recorded either by the farm manager/herdowner or by the attending veterinarian. Where necessary, clinical pathology samples were submitted for laboratory examination. Where deaths occurred, carcasses were submitted for laboratory post mortem examination. The results of periodic (monthly in the case of cows) blood sample analyses were also monitored for any evidence of abnormalities.

The Monitor Study on the three farms formally ran from April 1996 to March 1998. However, as animal health data was available from the start of 1996, and in order to make the analysis more meaningful, results for each farm are presented for the full calendar years of 1996 and 1997. Morbidity and mortality data are also reported for the first six months of 1998 for Index Farm A and up to the October 1998 for Index Farm B. Recording of animal health events concluded on the Control Farm at the end of March 1998.

Morbidity and mortality rates for the three Monitor farms are given in Table 2-1 to Table 2-18 at the end of this Chapter. Incidence is calculated as the number of new cases (i.e. only the first occurrence of a disease is counted in each animal) as a proportion of the population at risk. However, the population at risk varies depending on the event being measured. For death or disease related to a specific event or a limited time period, e.g. calving, lactation, the perinatal period, the population at risk is defined as all exposed animals, e.g. all calved cows. For events which can occur at any time during the year, e.g. lameness, respiratory disease, etc., the population at risk is the average number of animals present during the period.

INDEX FARM A

Morbidity and mortality rates for Index Farm A for the period January 1996 to June 1998 are given in Table 2-1 to Table 2-6. Animal health was generally good during the period of the Monitor Project. The main disease problems encountered in

1996 were mastitis and lameness - both of which were largely secondary to the housing problems of the winter of 1995/96. The overall lactational incidence of mastitis in 1996 was 46 *per cent* which is high. However, the cause in the majority of cases was identified as environmental (i.e. housing) and the response to control measures was good. In 1997, the rate had fallen to 14 *per cent* (three cases) which is low.

Two cases of acute toxic coliform mastitis occurred in the first three months of 1998. Although clinically severe - and one cow died – the incidence is not unusual. According to Menzies *et al.*, (1995), toxic mastitis was the single most commonly diagnosed cause of cow death in Northern Ireland.

One cow also developed severe udder oedema post-calving in 1998. She had also developed this condition after calving in 1997 and recovered uneventfully. However, in 1998, she responded poorly to treatment by the private veterinary practitioner and, after developing limb oedema and a discharging abscess, lost considerable condition and was eventually dried off.

Seven cows were affected by lameness in 1996. This represents a lactational incidence of 21 *per cent*. Although relatively high, and could not be classified as a good performance, it was largely secondary to the housing problems referred to above and was, at any rate, within reported ranges for commercial dairy herds (Appendix 1). The incidence of lameness was low in 1997 and 1998 (half-year) at two and one cases, respectively.

The incidence of perinatal calf mortality was low in 1996 and 1997. Only one stillbirth was recorded in each of these years. However, five perinatal losses were recorded in the 1998 calving season (extended past end of Monitor Study). While the incidence rate at 18 *per cent* is high, clear-cut diagnoses were made in most cases, i.e. trauma, twin delivery and haemorrhage. The fact that no diagnosis was made in two cases, again, is not unexpected. Hypoxia as a result of prolonged or difficult calving, for example, which is probably the commonest cause of stillbirth/perinatal mortality (McDermott *et al.*, 1992), leaves few diagnostic lesions.

An outbreak of calf diarrhoea occurred in 1997. Eight cases (five in calves under two weeks of age, three in calves over two weeks) were recorded over a three week period in May. Most were relatively mild and there were no deaths. While the overall incidence of calf diarrhoea in 1997 was 50 *per cent* as a result of this outbreak, this is within reported ranges and, given the zero mortality rate, cannot be

considered as a serious problem. The incidence of calf diarrhoea was low in 1996 and 1998 at two and one cases, respectively.

Incidence rates for the other main disease conditions were also low and well within acceptable ranges throughout the entire period of the Monitor Project. No cases of significant respiratory disease were recorded in calves or weanlings throughout.

Comparison with previous years

Reported mortality rates for cows, yearlings and calves were high on Index Farm A for the period 1986 to 1995 (*see* Chapter Four). These peaked in 1993 when 27 cattle, including seven cows and 16 calves, were reported to have died. In contrast, mortality rates for all categories of cattle were low during the period of the Monitor Project and, with the exception of stillbirths in 1998, was well within normal ranges. Other conditions which were reported to have occurred at a high incidence on the farm in previous years were pining, skin lesions and abortions. These were not a problem during the period of the Monitor Project. The only clinically significant occurrence of skin lesions was a single case of photosensitization – possibly secondary to liver dysfunction - in early 1996. No cases of pining or abortion were recorded.

INDEX FARM B

Morbidity and mortality rates for Index Farm B for the period January 1996 to (October) 1998 are given in Table 2-7 to Table 2-12. Animal health was also generally good throughout the period of observation and no serious disease outbreaks were encountered. The only diseases of any significance were mastitis and lameness.

The lactational incidence of mastitis in 1996 was 21 *per cent*. While relatively high, this is still within reported ranges (Appendix 1). In contrast to Index Farm A, the bulk of cases were infectious rather than environmental in origin and it became clear during the course of investigations that this problem had probably been endemic to the herd for some time prior to the commencement of the Monitor Study. A number of the older indigenous cows were identified with persistently high somatic cell counts (SCC) and, when individual-cow sampling was carried out, 10 were found to be carriers for *Staphylococcus aureus* – a common primary mastitis pathogen. As these animals were providing a focus of infection for younger and newly introduced cows, they were culled from the milking herd and put suckling calves. The response to this and other mastitis control measures is

indicated by the drop in incidence to 18 *per cent* in 1997 and only 4 *per cent* in 1998.

Eight cases of lameness were recorded in 1996 – six in indigenous cows and two in brought-in cows. The higher incidence in the indigenous cows largely reflects their age – mean 8.0 years compared to mean 3.5 years for brought-in cows. The overall annual incidence rate at 21 *per cent* is well within reported normal ranges (Appendix 1). The incidence of lameness was higher in 1997 at 32 *per cent*. The increase was mainly due to two separate outbreaks which occurred in cows at grass. These were probably due to Mortellaro's disease which is an infectious condition (David, 1993). Rough ground around the drinking troughs was also considered to be a contributory factor in the first outbreak. Although high, this incidence is within reported ranges and would be consistent with an outbreak of a moderately infectious condition. A further outbreak of Mortellaro's disease occurred in January 1998 in which 10 animals were affected. Most of the cases of Mortallero's disease in 1997 and 1998 responded rapidly to foot-bathing.

Three calves were stillborn in 1996. This represents an incidence of 8.6 *per cent* and is within published ranges (Appendix 1). While three abortions were reported for 1996 (EPA, 1997a), only one was actually confirmed by the finding of a foetus. In the case of the other two cows, abortions were presumed to have occurred on the basis that they had been previously diagnosed as pregnant on scanning. However, as the possibility of a scanning misdiagnosis cannot be ruled out owing to the early stage of (presumed) pregnancy, the incidence of confirmed abortion was under 6 *per cent*. No stillbirths or abortions were recorded in 1997. There were two perinatal calf deaths in 1998 - one a stillbirth and the other shortly after birth. Both were associated with difficult calvings. No abortions were recorded in 1998.

Incidence rates for metritis of 15 to 17 *per cent* in 1996 and 1997 were comparable to the DAISY average rate of 15 *per cent*. Other incidence rates for disease in cows and young stock in 1996, 1997 and 1998 were comparable to or lower than published rates. No cases of respiratory disease were recorded in young stock in 1996, 1997 or 1998.

Mortality rates for all categories of cattle were low throughout the period of the Monitor Study. Two cows died - one in 1996 and one in 1997. The former was following a difficult calving and the latter as a result of hypomagnesaemic tetany. No cows died in 1998. No post-perinatal calf deaths (i.e. excluding stillbirths and calving-associated losses) were recorded in 1996, 1997 or 1998.

Comparison with previous years

Fourteen cows were reported to have died on Index Farm B in 1994 and up to 18 in 1995 (*see* Chapter Four). This contrasts with one cow death in 1996, one in 1997 and none in 1998. Lameness was also reported to have been a severe problem up to and including 1995. While it is not possible at this remove to draw accurate conclusions regarding causation - information supplied in the Retrospective Survey Report (Chapter Four) suggests that many cases were environmental (i.e. housing-related) in origin. On the basis of expert advice regarding the unsuitability of the existing cubicle house, a new unit was installed following the DAF take-over of the farm. The benefits of this are reflected in the fact that the majority of cases of lameness recorded during the Monitor Project were infectious rather than environmental in origin.

Severe illthrift in growing cattle and cows, which was a problem on Index Farm B in 1995, and reportedly in 1994, did not occur during the three years when the farm was under DAF control. Calves and growing stock throve well. While loss of condition post-calving was a problem in some of the brought-in Holstein cows in 1997, this was a breed-related metabolic response to post-calving energy deficit and is discussed in detail elsewhere (*see page 30*).

Skin diseases, downer cows, prolonged calvings and twin calvings, which were reported to have occurred with an abnormally high incidence up to and including 1995, either did not occur, or were well within reported ranges during the period of the Monitor Project.

CONTROL FARM

Morbidity and mortality rates for the Control Farm for the period January 1996 to March 1998 are given in Table 2-13 to Table 2-18. With the exception of mastitis, the health of Askeaton- and Abbotstown-origin cows and their offspring was good throughout the period of the Monitor Study. Mastitis, however, was a significant problem. In 1996, eight of 20 Abbotstown-origin cows and seven of 20 Askeaton-origin cows experienced one or more cases of mastitis (overall incidence 40.5 *per cent*). As with Index Farm B in Askeaton, the problem was primarily infectious (*Staphylococci* and *Streptococci*) rather than environmental mastitis and had been recognised on the Control Farm prior to the start of the Monitor Study. It is likely that most of the cases in Askeaton-origin

cows were new infections acquired following introduction to the herd. The incidence remained high in 1997 (47 *per cent*) largely owing to the restrictions imposed on culling by the requirements of the Project.

It is unlikely that the mastitis problem had a crucial impact on the central objective of the Monitor Study, i.e. monitoring for evidence of an abnormal incidence of disease in Askeaton-origin cows which could be related to prior exposure to an environmental pollutant in Askeaton. Its significance in relation to the Immunology Project is discussed elsewhere (Chapter Seven).

The incidence of other diseases in cows - including lameness - was low throughout the period. The low incidence of lameness undoubtedly reflects the generally good condition of farm surfaces and roadways. No cow deaths occurred in 1996 or 1998. Two cows died in 1997 - both due to acute vulvo-vaginal haemorrhage post-calving.

The recorded incidence of diarrhoea in calves and weanlings was relatively high in 1996 (31 and 58 *per cent*, respectively). However, most of these were mild cases of nutritional diarrhoea (milk scour) and of little clinical significance. Veterinary intervention was only sought on two occasions. The incidence of other disease conditions in calves and yearlings was low throughout the period of the Study.

CONCLUSION

The overall incidence of disease and mortality was low on the three farms during the period of the Monitor Study and was well within reported ranges from Irish and international sources (Appendix 1). There was no evidence of a recurrence of the severe animal health problems of previous years on the two Index Farms and no outbreaks of unusual or undiagnosed disease were encountered. While moderately high incidences of mastitis and lameness were recorded, these were associated with risk factors which are common to many commercial farming operations and rates were, in any case, close to or within reported ranges from elsewhere.

Given the severity of the reported problems on the two farms in the years preceding the present study, in particular the exceptionally high reported loss of 32 cows on Index Farm B in 1994-95, the results represent a dramatic improvement in animal health performance. It must be concluded, therefore, that whatever factors had contributed to the unusually high incidence of disease on the two Askeaton

farms in previous years, they were no longer present by early 1996.

CULLING

Culling is defined as the considered removal of animals from the herd and may be voluntary or involuntary (Stevenson and Lean, 1998). Culling is an essential part of any commercial farming enterprise. Its purpose is to remove animals which have reduced productivity due to infertility, age, illness or poor conformation and to allow replacement with younger home-reared or brought-in animals of improved genetic merit. It is also used as a means of controlling infectious diseases such as mastitis, by removing persistently affected animals which act as a reservoir of infection for the herd. Average culling rates vary depending on herd type and management policy. Rates are highest in intensively managed farms where culling is actively used to promote health and production. The Teagasc DairyMIS Farm survey, for example, recorded an overall culling rate of 15.2 *per cent* for the entire 340-herd sample (O'Farrell *et al.*, 1997). However, the culling rate for a sub-group of more intensively-managed farms was over twice that at 30 *per cent* (Teagasc, 1996). Similarly, the UK DAISY survey reported an average culling rate of 24 *per cent* with a range of 18 to 29 *per cent* (Esslemont and Kossaibati, 1995). Whitaker *et al.*, (2000) reported a culling rate of 22 *per cent* in 340 intensively managed dairy herds in southern England. A culling rate of 18 *per cent* per annum is generally considered the maximum consistent with efficient production.

In the present study, culling pressures on the Index Farms were higher than normal from the start of the Project. Several Abbotstown-origin cows on Index Farm A qualified for culling from the start of the project as a result of mastitis and lameness secondary to the 1995/96 overwinter housing problems (EPA, 1997a). On Index Farm B, the age structure was highly unbalanced at the start of the study with many cows over 10 years old. As older cows tend to have a higher incidence of udder and reproductive disorders (Stevenson and Lean, 1998), all of these would have qualified for immediate culling if normal commercial criteria were applied. Selective culling would also have been the method of choice to control the serious mastitis problem which existed on this farm.

However, because of the nature of the Monitor Study, considerations other than purely commercial ones had to be taken into account in determining culling policy. In the first case, the cows of primary interest, i.e. Askeaton-origin, were a finite resource and could not be replaced on culling. In the second,

the Askeaton and Ballysteen Animal Health Committee had repeatedly voiced concerns regarding removal of animals which had been exposed to the local environment for a number of years. In order to meet these conflicting pressures, a culling policy had to be implemented which represented a balance between commercial and non-commercial considerations. Where possible, therefore, culling of study cows due to chronic disease was restricted to cases where it was indicated on welfare grounds. Inevitably, this compromise had certain negative implications for the overall efficiency and productivity of the farms. On Index Farm B, a secondary suckler herd was maintained to hold cows culled from the dairy herd on grounds of chronic mastitis.

INDEX FARM A

Total annual culls for Index Farm A for 1996 to 1998 are given in Table 2-19. Culling in 1996 was high at 40 *per cent*. Most of the culls were Abbotstown-origin cows with chronic mastitis infections arising from the overwinter housing problems. Culling in 1997 was also high at 26 *per cent*. Over half of this was for infertility. The significance of this result is discussed in the section on fertility performance (*see* page 31). Only three cows were culled for disease - two for mastitis and one for lameness. This is an acceptable culling rate for disease on a farm of this size. There were no culls during the last three months of the Project (January to March, 1998).

INDEX FARM B

Total annual culls for Index Farm B for 1996 to 1998 are given in Table 2-20. Culling in 1996 was 23 *per cent*. All cows culled were Askeaton-origin. The relatively high rate of culling was largely due to the need to reduce the average age of the herd. Seven of the cows culled were over 11 years old. No brought-in cows were culled in 1996. Culling at 9 *per cent* in 1997 was within target. All cullings were due to infertility (*see* page 32). No cows were culled from the farm due to disease -- though some were transferred to the suckler herd. The culling rate in 1998 (up to October) was artificially high due to depopulation of the suckler herd at the end of the Project. Culling of brought-in cows at 10 *per cent*, prior to total de-stocking in October 1998, was well within target.

CONTROL FARM

Total annual culls for the Control Farm for 1996 to 1998 are given in Table 2-21. Culling was low throughout the Project. Two Askeaton-origin cows were culled in 1996 - one for mastitis and one for

behavioral faults. Six Abbotstown-origin cows were transferred to Askeaton for inclusion in the Immunology Project in the same year. Culling for infertility and disease were low and well within target in 1996 and 1997. Two Abbotstown-origin cows were culled for mastitis and one for lameness in 1997. Culling in 1998 was associated with the termination of the Project.

CONCLUSION

The main causes of culling on the three farms, i.e. age, infertility, mastitis and lameness, were consistent with normal commercial experience (Esslemont and Kossaibati, 1995; Teagasc, 1997). Although rates were high at times, these were influenced by factors which pre-dated the start of the Project, e.g. cow age and mastitis on Index Farm B; mastitis and lameness in Abbotstown-origin cows on Index Farm A. Culling for infertility, on the other hand, was restricted by the need to retain animals of Askeaton-origin for purposes of the Project.

MILK PRODUCTION

The actual expression of a cow's genetic potential in relation to milk production is largely determined by her health and nutritional status. Although not specific as to cause, a significant shortfall in production can be taken to indicate problems in relation to one or both of these areas. Variations in the health and nutritional status of a cow can also affect milk quality. The main constituents routinely measured in milk are protein, fat and somatic cell count. Normal ranges for these are given in Appendix 1. While milk protein and fat concentrations respond to changes in a range of feed-related variables, in broad terms, protein concentrations at the lower end of the scale, and fat concentrations at the upper end of the scale, are associated with rations which are inadequate in quantity (i.e. energy) and/or quality to meet production demands (Duffield *et al.*, 1997). As a guideline, milk protein concentrations of 2.9 g/l or less are regarded as indicative of inadequate energy supply.

Somatic cell counts (SCC) give an indication of the cellular content of milk. High SCCs are associated with an inflammatory response in the udder, i.e. mastitis. Monthly SCCs can, therefore, be used to monitor both the overall mastitis status of a herd and that of individual cows within the herd. At the individual cow level, counts of between 200,000 and 400,000 cells/ml are generally taken as the cut-off range in relation to infection. The majority of cows with counts of above 400,000 can be regarded as actively or recently infected while

counts below 200,000 are not indicative of infection.

In Askeaton, milk production was reported to have been poor on the two Index Farms – as well as on other farms in the area (*see* Chapter Five) - in the years leading up to the present investigation. Concern had been expressed locally that this was due to environmental pollution. One of the objectives of the present investigation, therefore, was to monitor milk production of indigenous and brought-in cows on the two Index Farms over an extended period. In the event that production continued at an unacceptably low level then investigations could be instituted to ascertain the causes. Milk production of Askeaton-origin (Index Farm A) and non-Askeaton-origin cows was also monitored on the Control Farm at Abbotstown. The main purpose of this was to determine if the former showed evidence of a similar negative effect on milk yield in the event that their comrades in Askeaton seriously under-performed.

Mean monthly milk yield, protein concentration, and cell counts for all groups on the three farms are given in Table 2-22 to Table 2-30. However, owing to the natural spread of calving dates, and therefore variations between groups in terms of average stage of lactation, these are of limited value in analysis of production. Individual annual yields (1996 and 1997) for each cow are given in Appendix 2. These are the main basis for the analysis and assessment of production performance which follows.

Note: cows with lactations under 200 days are not included in the assessments Where lactations extended beyond 340 days, the projected 305-day yields are used for purposes of comparison.

INDEX FARM A

1996 - Milk Yield and Quality

Mean monthly milk production data for the two groups of cows on Index Farm A are given in Table 2-22 to Table 2-24. Average lactational yield for the 11 Askeaton-origin cows (average seventh lactation) which were milked during the year was 4,355 kg. This is a good result for the type and age of cow and compares favorably with the estimated yield per cow of around 3,410 kg in 1987 before the problems were reported to have begun (*see* page 89).

Analysis of results for the 17 Abbotstown-origin cows which were milked in 1996 is complicated by a number of factors. Firstly, seven cows were calved too long before milk recording commenced in April 1996 to allow reliable estimation of actual or projected yields. Secondly, three of six animals

moved to Askeaton in July 1996 as replacements for animals culled on health grounds, had calved too long before transfer (three to five months) to allow meaningful analysis of yields in Askeaton. Thirdly, at least five cows had chronic health problems (i.e. mastitis, lameness, ill thrift, teat injury) arising from the 1995-96 overwinter housing problems (EPA 1997a) which would have had a significant negative impact on milk yield.

Only two Abbotstown-origin cows calved in Askeaton late enough in 1996 to provide reliable milk recording data. These had 305-day yields of 5,387 and 5,637 kg. Mean yield for four first-calvers moved to Askeaton in July (within one or two months of calving) was 4,146 kg. These are acceptable performances.

Mean milk protein and fat values were within normal ranges for both Askeaton- and Abbotstown-origin groups throughout the year. Five of 15 milk samples collected in March had protein concentrations below 3.0 g/l. However, as three of these were calved since 1995, the results have little significance in relation to post-calving energy balance.

Note: accurate production values cannot be calculated for Abbotstown-origin cows brought onto Index Farm A in 1995 and which calved in 1995 as milk recording did not commence until March, 1996 In addition, it is clear that production in this group was significantly affected by the subsequent overwinter health problems.

1996 - Somatic Cell Counts

Mean SCCs in the Askeaton-origin group were below 400,000 throughout. However, individual high counts were recorded. Details of these have already been reported (EPA, 1997b).

Mean SCCs were over 500,000 in the Abbotstown-origin group from March to June, inclusive. This was largely due to the cases of mastitis which had developed as a result of the over-winter housing problems. Counts in the second half of the year were substantially lower – partly due to control measures and selective culling and partly a normal lactation effect.

1997 - Yield and Quality

Four Askeaton-origin cows which did not go in calf in 1996 are not included in the analysis of production results for 1997. One cow in the Abbotstown-origin group which had last calved in January 1996 has also been excluded.

Three first-calvers in the Askeaton-origin group had an average total milk yield of 3,237 kg for the year. This is an acceptable performance given that the heifers were light and under two years of age at

calving. The five older cows (average six to seventh lactation) which calved in 1997 had a mean yield of 4,491 kg which is also an acceptable performance. One of these, No. 303 (lactation length under 200 days), showed a drop in yield around three months after calving from an average of 26 kg per day in April to 12 kg in May. Although she was not recorded as having been ill during the period, falling body weight and condition, milk protein of 2.7 g/l, and raised serum ßHB concentration are all consistent with sub-clinical ketosis due to a prolonged negative energy balance post-calving. The remaining four cows had a mean yield of 4,682 kg which is a good performance.

The five Abbotstown-origin first-calvers had a mean yield of 3,660 kg in 1997. This included one cow, No. 111, which had a yield of only 2,596 kg. This animal had delivered a stillborn calf on 2/2/97. Although she was not recorded as showing clinical signs of illness, her body condition score fell from 3.5 shortly before calving to 1.5 at five weeks post calving. It is likely, therefore, that she had suffered from an unidentified sub-clinical condition – possibly related to parturition and the delivery of a dead calf. Excluding cow 111, mean production for the other four heifers was 3,923 kg which is an acceptable performance.

Mean yield for the remaining 11 Abbotstown-origin cows (average third lactation) was 4,410 kg. While this was only a moderate performance, five cows had lactations of under 240 days due to the combined effect of late calving and a management decision to dry off all cows before the end of 1997. Some of these animals also suffered residual udder health problems from the winter of 1995/96 and one was milking on three quarters.

A number of factors relating to non-implementation of pasture management and feeding recommendations are also likely to have had a negative impact on milk production in 1997. These include infrequent pasture topping, inadequate nitrogen application in the spring and a period of overgrazing in June. In relation to the latter, during the two-week period 16/6/97 to 2/7/97, the average yield per cow dropped by over five kg per day. While this also coincided with the withdrawal of concentrate feeding, grass supply at the time should have been adequate to maintain production. Withholding of meal feeding in the autumn due to anticipated quota problems (which did not materialise), is also estimated to have reduced average yield by about 318 kg per cow.

Mean milk protein and fat values were within normal ranges for both groups throughout the year.

However, eight of 21 milk samples had protein concentrations below 3.0 g/l in February 1997 - five of which were below 2.9 g/l - which is consistent with post-calving negative energy balance. The significance of this is discussed further below.

1997 - Somatic Cell Counts

Mean SCCs for the Askeaton-origin group cows were below 400,000 for most of the year. High values in February and May in the Askeaton-origin group were due on each occasion to individual animals – cow 307 in February and cow 304 in May. The latter animal had blood clots in the milk of two front quarters following calving and had calved down with a large, oedematous udder (this animal also developed severe udder oedema post-calving in 1998 and subsequently became severely ill – see page 16). Otherwise, generally low individual values throughout the year reflected the low incidence of mastitis. Mean SCCs for the Abbotstown-origin cows were below 250,000 throughout the year.

1998

Individual-cow milk recording concluded with the full transfer of management to the herdowner at the end of 1997. Although data are available for bulk milk collection for the first three months of 1998, it is not possible to estimate average daily yield as some milk was withheld for feeding to calves. SCC values for the twice monthly tests ranged from 100,000 in February to 357,000 in March.

INDEX FARM B

1996 - Yield and Quality

Mean monthly milk production data for the two groups of cows on Index Farm B are given in Table 2-25 to Table 2-27. Twenty six indigenous cows calved on the farm in 1996. A number of these had short lactations due to subsequent culling (see page 20). Excluding cows which had lactations of less than 200 days, and using 305-day yields for two cows which had extended lactations, the average annual yield for the remaining 13 indigenous cows (average sixth lactation) was 4,432 kg which is a good performance.

Twenty one cows (average second lactation) were brought in to the dairy herd in 1996. Eight of these calved on the farm. The remainder had calved between one and two months before arrival. Excluding two short lactation cows, mean yield per cow was 4,951 kg. This is also a good performance.

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Mean milk protein and fat values were within normal ranges for both groups throughout the year. Six milk samples collected from indigenous cows in April had protein concentrations below 3.0 g/l. Milk sample collection from the brought-in cows commenced in June 1996.

1996 - Somatic Cell Counts

Mean SCCs in monthly samplings for indigenous cows were above 400,000 on six occasions during the year. Most of the high counts were in the first half of the year and reflected the recognised chronic mastitis problem (see page 17). With the exception of December 1996, counts for indigenous cows were lower in the second half of the year as a result of control measures introduced. Mean counts for the brought-in cows were below 200,000 in four of the seven months for which recordings were taken. Mean counts of around 400,000 in two months were largely due to very high values in two cows.

1997 - Yield and Quality

Eleven indigenous cows calved into the dairy herd in 1997. Five had short lactations (< 200 days) due to subsequent culling. Mean yield for the remaining six cows (average seventh lactation) was 4,346 kg which is a good performance for age and breed.

Mean yield for the 13 brought-in cows (average third lactation) which calved on the farm in 1997 was 5,237 kg. Mean yield for a further 13 first-calvers purchased after calving in 1997 was 4,464 kg. These are satisfactory production results.

Mean milk protein and fat values were within normal ranges for brought-in and indigenous cows throughout the year. A total of 14 cows had milk protein values below 3.0 g/l – seven below 2.9 g/l – at some time in the first three months of 1997. The significance of this in relation to post-calving energy balance is discussed further below.

1997 - Somatic Cell Counts

Mean SCCs for the indigenous and brought-in cows in the dairy herd were around 200,000 throughout the year. The improvement in counts for the indigenous cows compared to 1996 was also reflected in a significantly lower incidence of clinical mastitis (see page 17) and was largely the result of control measures introduced in 1996.

1998

Although the Monitor Study on Index Farm B formally ended in March 1998, monitoring of production of the dairy herd continued up to its disposal in October. Forty two cows were milked during the year - 33 brought-in, eight indigenous and one cow transferred from Index Farm A in December 1997. All of the dairy cows calved in 1998, the first on 9 January and the last on 5 May. One cow was culled in July due to severe lameness.

1998 - Milk Yield and Quality

Mean yield for the six indigenous cows which had lactations of 200 days or more was 4,964 kg with an average lactation length of 251 days. Mean yield for the 24 brought-in cows with 200-day plus lactations was 4,469 kg with an average lactation length of 242 days. This is a good performance for both groups.

1998 - Somatic Cell Counts

Mean cell counts in both indigenous and brought-in groups were below 200,000 throughout most of the year – a fact which reflects the very low incidence of mastitis in 1998 (three cases). A count of 600,000 in the brought-in group in January was largely due to very high counts in two cows which had calved two days prior to sampling. One subsequently developed clinical mastitis in February. Mean counts of between 500,000 and one million in October 1998 were due to the combined effects of high counts in a small number of cows together .with an expected increase in counts at the end of lactation.

CONTROL FARM

1996 - Milk Yield and Quality

Mean monthly milk production data for the two groups of cows on the Control Farm are given in Table 2-28 to Table 2-30. Fourteen Askeaton-origin cows were milked during the year. Ten of these with full lactations (average fourth lactation) had a mean annual yield of 4,955 kg per cow. Fourteen Abbotstown-origin cows were milked – 10 of which had full lactations. Five first calvers had a mean annual yield of 5,428 kg per cow while five cows of average fourth lactation had a mean yield of 6,233 kg per cow. This is a good performance.

Mean milk protein and fat values were within normal ranges for both groups throughout the year. No milk protein concentrations below 3.0 g/l were recorded in April and May, the first two months of milk recording on the Control Farm.

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1996 - Somatic Cell Counts

High SCCs were a problem in both Askeaton- and Abbotstown-origin groups in Abbotstown throughout the year. Mean counts above 400,000 were recorded on four occasions for the Askeaton-origin group and on five occasions for the Abbotstown-origin group. High counts were reflected in the high incidence of clinical mastitis cases (*see* page 18).

1997 - Milk Yield and Quality

Eighteen Askeaton-origin cows were milked during the year – three heifers and one cow had been transferred from Index Farm A in July 1996. The three first-calvers had a mean annual yield of 4,478 kg per cow which is a good performance. Eleven Askeaton-origin cows (average fifth lactation) with full lactations had a mean annual yield of 5,219 kg per cow. Seventeen Abbotstown-origin cows were milked during the year. Four first-calvers had a mean annual yield of 5,246 kg per cow. Eleven cows average fourth lactation had a mean yield of 5,382 kg per cow. These are good production results for both groups.

Mean milk protein and fat values were within normal ranges for both groups throughout the year. No milk protein concentrations below 3.0 g/l were recorded in January, one in February and five in March, 1997.

1997 - Somatic Cell Counts

High cell counts continued to be a problem in both groups in 1997. This was mainly due to the retention of persistently affected animals - of 15 animals affected with clinical mastitis, eight were affected on more than one occasion.

1998 - Milk Yield and Quality

A total of 35 cows – 16 Askeaton-origin and 19 Abbotstown-origin - were milked in the last three months of the project on the Control Farm, i.e. January to March 1998. Nineteen of these calved during the period while the remainder had calved in the latter part of 1997. For cows which calved in 1998, average daily yields in the Askeaton-origin group ranged from 15 to 31 kg. Mean daily yield for the group for March was 24 kg.

Average daily yields for 1998-calved cows in the Abbotstown-origin group ranged from 16 to 33 kg with a mean daily yield for all 1998-calved cows in the group in March of 27 kg.

1998 - Somatic Cell Counts

Mean cell counts for both groups of cows were below 200,000 throughout the last three months of the project. The reduction from 1997 values was largely due to the culling of high cell count cows at the end of 1997.

CONCLUSION

Milk production on the three farms ranged from moderate to good. Mean lactational yields for the Askeaton-origin cows in Askeaton were between 4,091 and 4,546 kg which is an acceptable performance for the type and age of animal. Mean yields for the higher genetic merit brought-in cows on Index Farm B were around 5,000 kg.

While most of the Abbotstown-origin cows on Index Farm A in 1996 had calved too long before milk recording commenced to allow accurate analysis, it is clear that the health problems which developed over the winter of 1995-96 had long-term effects on milk production. This is not surprising as some animals went dry in individual quarters. Residual udder health problems, as well as short lactations, contributed to an only moderate performance of average 4,410 kg for this (Abbotstown-origin) group in 1997.

Periods of high milk cell counts (SCCs) in the three herds were due to intermittent mastitis outbreaks and the presence of persistently infected carrier animals. These outbreaks, which were not considered unusual for a commercial dairy herd, were controlled by implementation of standard preventative measures. Milk quality was also satisfactory on the three farms. Group mean protein and fat concentrations were within acceptable limits throughout. Protein concentrations at the lower end of the normal range in individual cows were associated with changes in post-calving energy balance.

While the purposes of the study did not include between-farm comparisons of similar-origin groups, it is clear that milk production was higher on the Control Farm in Abbotstown than on Index Farm A for both the Askeaton- (1996) and Abbotstown-origin (1997) groups. Milk protein concentration also tended to be higher on the Control Farm than on either of the Askeaton farms in 1997. Only 10 *per cent* of milk samples collected in the first three months post-calving on the Control Farm were below 3.0 g/l. This compares to 40 *per cent* on Index Farm A, and 23 *per cent* on Index Farm B for similar periods (milk sampling dates matched for lactational rather than calendar month). Mean milk protein concentrations for all cows on the Control Farm were significantly

higher (p < 0.05) than for cows on Index Farm A for five of the first eight (lactational) months post-calving.

There are a number of factors which are likely to have accounted for these differences in milk quality and yield. In relation to the Abbotstown-origin cows in Askeaton, yields would have been substantially reduced as a result of the disease problems experienced over the winter of 1995/96. The significantly shorter lactation lengths on Index Farm A than on the Control Farm in 1997 would also have reduced yields for both groups of cows in the former location. Differences in nutrition must also have played a role. The generally lower protein concentrations on the Index Farms in 1997 indicate that energy supply was better matched to production on the Control Farm than in Askeaton at that time. It is likely that the better quality silage and grass in Abbotstown was a major influence in this regard (Table 2-31). Studies in Moorepark, for example, have shown that an increase in silage DMD is directly reflected by an increase in milk yield and protein content (Murphy and O'Brien, Moorepark, 1997). These issues are discussed in more detail below.

The overall conclusion of this aspect of the study is that milk production - both in terms of quantity and quality - was acceptable at the three locations and shortfalls, where they occurred, could be accounted for by factors related to breed, age, nutrition and health status. There was no evidence to suggest that other unidentified environmental influences had adversely affected production at any of the three locations.

NUTRITION, BODY WEIGHT AND CONDITION

Estimation of average daily weight gain (ADG) is a good indicator of the general health and productivity of growing animals. Any significant check in growth rates will be reflected in failure to reach target weights, and, in more serious cases, chronic ill-thrift. Weight changes in cows, on the other hand, largely mirror the reproductive cycle, i.e. a gradual increase during pregnancy followed by a sharp drop at calving and a continued more gradual decline up to about two months post-calving. While the latter decline is a physiological energy deficit associated with milk production, excessive or prolonged weight loss post-calving is associated with a variety of reproductive and metabolic disorders (see page 7).

In cows, body condition scoring is a more sensitive indicator of energy balance than weight change. Because of shifts in body water associated with fat

mobilization, cows losing condition may actually be gaining weight. According to Ferguson (1991), reductions in body condition score, on the other hand, are highly correlated with adipose tissue mobilization and negative energy balance. The method of condition scoring is based on visual or manual assessment of fat coverage of pre-defined areas of the body, e.g. tail-head, backbone, loins (Edmonson et al., 1989). It is measured on a scale zero to five. Animals of condition score one are in poor condition while those with a score of four are overfat. Published values are available for recommended optimal condition scores for cows at different stages of their productive cycle (Sreenan and Diskin, 1992). In general, cows should have a condition score of between 2.5 and 3.5 at calving and 2.0 to 3.0 at breeding. It is recommended that cows should not loose more than a half point between calving and breeding.

When carried out by the same operator, condition-scoring is a sensitive indicator of an animal's overall health and nutritional status over time and is potentially of greater value than the recording of body weight changes. However, because of its subjective nature, allowance must be made when comparing results of scorings carried out by different operators. To illustrate this point, it has already been reported (EPA, 1997a) that most of the reductions in condition scores between September and December 1996 on the Index Farms were probably due to the change of operator as all cows gained in body weight during this period.

Operator variation is also likely to have accounted, in part at least, for the differences in scores recorded between Abbotstown and the two Askeaton farms in 1997. Up to September 1996, scoring at the three locations was carried out by the one operator. From December 1996 onwards, scoring at the Askeaton farms was carried out by a different operator to the Control Farm. Even where comparison is being made between animals at the same location, allowance must be made for the effect of breed differences on condition performance. The differences between British Friesian and Holstein cows is discussed below (see page 30).

The overall approach to feeding and supplementation is outlined in the Study Protocol. Concentrate supplementation was carried out according to Teagasc guidelines for efficient commercial dairying (EPA 1996, unpublished). These recommendations take into account both silage quality and milk yield potential of the cows. Pre-calving, it is considered that cows with a condition score of 2.5 at dry-off should reach target condition at calving of 3.0 to 3.5 on good quality

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silage alone (DMD 70 *per cent* or better). Post-calving, it would be economic to feed 7 to 8 kg concentrates per day to average genetic merit cows on 70 *per cent* DMD silage (Dillon and Crosse, 1997). However, it should be noted that silage quality on the Askeaton farms was generally below 70 *per cent* DMD (Table 2-31).

The results of weighings and body condition scorings carried out on animals on the three monitor farms are presented in the following sections. Details are also given of feeding for all stock during the Project. Cows and growing stock were weighed monthly. In addition, cows and their newborn calves were weighed immediately after calving. In 1996, body condition scoring was carried out quarterly on the three farms. This was changed to monthly in 1997.

The results of silage analyses carried out on the three farms are given in Table 2-31.

INDEX FARM A – COWS

1996

Group mean monthly weights and condition scores for Askeaton- and Abbotstown-origin cows in 1996 are given in Table 2-32. Dry cows were fed silage alone. From calving to shortly before turnout on 16/4/96, cows were fed 6.7 kg concentrates per head per day. This was reduced to 4.5 kg from 12/4/96, to 3.4 kg from 22/4/96, to 2.2 kg from 14/5/96 and to 1.2 kg from 1/6/96. Concentrate feeding was discontinued on 10/06/96. From 12/10/96 all milkers were put back on 2 kg per day. Two freshly calved cows were given 4 kg per day. Total feed usage per cow is difficult to estimate owing to cow movements and partially recorded lactations. However, average input for Askeaton-origin cows in 1996 was estimated at about 569 kg concentrate per head. This was less than originally intended owing to inaccurate parlour feed dispensers (EPA, 1997a).

As condition scoring was only carried out quarterly in 1996, it is not possible to make a full assessment of performance throughout the year. Four of nine Askeaton-origin cows scored before calving were below the recommended minimum of 2.5. Six of 10 cows scored post-calving had scores below 2.0. No spring-calving Abbotstown-origin cows were condition-scored pre-calving. All eight cows scored between one and three months post-calving were below 2.5. Condition improved throughout the summer and by September, only seven cows were under 2.5.

The low condition scores in the Abbotstown-origin animals during the spring were probably largely associated with the overwinter housing problems. The latter also contributed to the low scores in some Askeaton-origin animals. The net effect was that about a third of all cows had condition scores under the recommended minimum 2.0 in May 1996 which was in the middle of the breeding season. The possible impact of this on fertility performance is referred to elsewhere (*see page 31*). However, it is worth noting that the major biochemical indicators of negative energy balance - milk protein and serum ßHB - were generally within normal ranges at these times.

1997

Group mean monthly weights and condition scores for Askeaton-origin and Abbotstown-origin cows in 1997 are given in Table 2-33. Dry cows were housed from 10/1/97 on a diet of hay. Six thin cows were fed approximately 3 kg beef fattener concentrate per head per day. All dry cows were put on pit silage and 2.5 kg concentrates per head per day from 20/2/97. After calving, cows were fed 7 kg concentrates until shortly after turnout. On 27/3/97 this was reduced to 4 kg daily until 24 April when it was further reduced to 2 kg Concentrate feeding ceased on 19 June. The cows were re-housed in November and fed silage *ad-lib*.

Despite supplementation, about half of the cows had condition scores below the recommended 2.0 to 2.5 condition score at calving. Condition remained below optimum in a number of animals post-calving and in April, at the start of the breeding season, five Askeaton-origin and ten Abbotstown-origin cows had condition scores below 2.0. In contrast to 1996, a proportion of cows in both groups also had low milk protein values (<3.0 g/l) and raised serum ßHB values (> 0.9 mmol/l) in the April - June period. This is further indication of a negative energy balance at this time. The significance of these findings in relation to nutrition and management is discussed below. Body condition improved throughout the summer and by September, only one animal had a score of under 2.0.

1998

Group mean monthly weights and condition scores for Askeaton-origin and Abbotstown-origin cows for the first three months of 1998 are given in Table 2-34. Cows were over-wintered in the cubicle house on silage *ad-lib*. No concentrate supplementation was given to dry cows. Following calving, cows were fed approximately 7 kg concentrates per head per day in addition to silage. The condition of cows in both groups was satisfactory in the first three months of 1998. Only

three cows in each of the two groups had scores under 2.0 in March. One of the Askeaton-origin cows involved had experienced a difficult calving (twins) and the second was diagnosed with metritis post-calving.

INDEX FARM A - GROWING STOCK

Mean monthly weights for calves born and reared on the Index Farm A in 1996 and 1997 are given in Table 2-35. Thirteen calves were reared on the farm in 1996. They were fed between 0.5 and 1.0 kg concentrates per head per day up to the first week of July when feeding was stopped. They were out-wintered in 1996/97 on a diet of hay and approximately 2 kg concentrates per head per day.

Thirteen calves were kept on the farm throughout most of 1997. They were fed 1 to 2 kg concentrates per head per day throughout the summer and autumn. They were out-wintered over the winter of 1997/98 on hay *ad-lib* and approximately 2 kg concentrates per head per day.

Average daily weight gain for calves was close to 1 kg during 1996 and 1997. They had reached target weight of 220 kg (Teagasc, 1994) by the autumn of both years. This is a good performance and reflects the overall good health of calves on the Index Farm A in 1996 and 1997.

Mean monthly weights for yearlings for 1997 are given in Table 2-36. Thirteen yearlings were out-wintered in 1996/97. They were fed hay *ad-lib* and approximately 1 kg Concentrates per head per day. Concentrate feeding was increased to 2 kg from 24/1/97 until 18/3/97, after which time it was withdrawn. They were on grass without supplementation for the remainder of 1997. The yearlings performed well during the year with average daily growth rates of around 1 kg from April to September. Average weight of the nine remaining animals was 460 kg in October which is a good performance (target for yearlings at start of second winter = 470 kg; Teagasc, 1994).

INDEX FARM B - COWS

1996

Group mean monthly weights and condition scores for indigenous and brought-in cows in 1996 are given in Table 2-37. Due to the high risk of hypomagnesaemia after the take-over of the farm in November 1995, all cows were housed on 24/11/95 and fed on a diet of hay, 2 kg rolled barley and 2 oz mineral supplement (calcined magnesite) per head per day. After a week, the hay was replaced by silage and mineral supplementation was increased to 3 oz per head per

day. In late December, the cows were divided into 'fat' and 'thin' groups. The fat group were supplemented with 2 kg rolled barley per head per day while the thin group were fed 4 kg On 4/2/96, rolled barley was changed to 3 kg of a 16 % dairy ration. Following condition scoring on 26/2/96, some of the fat cows were reduced to 1 kg per head per day.

A ration of 9 kg dairy concentrate had been intended for calved cows to compensate for the poor quality of the silage (*see* Table 2-31). However, owing to incorrect calibration of the feed hoppers that had been installed, the amount actually fed was estimated at 6.7 kg per head per day (EPA, 1997a). Concentrate feeding was reduced in stages so that by 22/4/96 – six days after turnout - they were on 3.4 kg per head per day. On 28/5/96 this was further reduced to 1.2 kg per head per day. Concentrate feeding ceased on 3/6/96.

Feeding of dairy ration restarted on 27/09/96. Eight good milkers were fed 2 kg dairy ration per head per day and the remaining 24 were fed 1 kg per day. At the end of October, feeding levels were raised (15 cows on 4 kg. per day, 12 cows on 1 kg per day). After housing on 19/11/96, the lactating cows were fed round-bale silage. Total concentrate feeding for cows on a full lactation in 1996 was estimated at about 508 kg per cow.

Condition-scoring was carried out quarterly in 1996. All indigenous cows were in good condition pre-calving (score 2.5 to 3.5) and held condition well post-calving. Only one cow had a score of below 2.0 in the two months post-calving and the majority had scores of 3.0 to 3.5.

All except one of the brought-in cows were calved before arrival on Index Farm B in 1996. Their performance in 1996 was satisfactory - though condition scores tended to be lower than indigenous cows. Five of the brought-in cows had scores below 2.0 on at least one occasion in 1996. These reflect breed differences in relation to post-calving energy metabolism and are discussed elsewhere (see page 30).

1997

Group mean monthly weights and condition scores for indigenous and brought-in cows in 1997 are given in Table 2-38. Cows were over-wintered on big-bale silage. Dry cows were supplemented with 1 kg concentrates per head per day, plus 100 g pre-calver mineral mix, from 13/1/97 to calving. After calving, cows were fed 8 kg concentrates until shortly after turnout. On 24/03/97 concentrate supplementation was reduced to 6 kg per day. It

was further reduced to 4 kg per day from 23/04/97 and to 2 kg from 17/05/97. Concentrate feeding ceased on 12/07/97. From 23/8/97 until dry-off, lactating cows were given 2 kg per day. Dry cows were housed at the end of November and fed silage *ad-lib*. Eighteen thin cows were also fed 2 kg per head per day rolled barley. Total concentrate feeding for cows on a full lactation in 1997 was estimated at about 757 kg per cow.

Only three cows (brought-in) had condition scores below the recommended minimum of 2.5 pre-calving. Post-calving, there was a noticeable difference in performance between indigenous and brought-in cows. As a group, the latter suffered a more marked and more prolonged loss in condition than the indigenous cows. While four indigenous cows had condition scores below 2.0 in the first two months post-calving, 17 brought-in cows had scores of below 2.0 during the same period. The significance of these findings in relation to nutrition and management is discussed below (*see* page 30 *et seq.*).

The indigenous cows gained condition throughout the summer - to the extent that they had to be moved to poorer grass for the autumn and early winter to avoid problems associated with excess condition. The brought-in cows performed less well and in September, 13 still had scores below 2.0.

1998

Group mean monthly weights and condition scores for indigenous and brought-in cows on Index Farm B for January, February, and May, 1998 are given in Table 2-39. Cows were over-wintered on big-bale silage. Dry cows were supplemented with 1 kg concentrates per head per day, plus 100 g pre-calver mineral mix to calving. After calving, concentrate supplementation was introduced at the rate of 7 kg per head per day. This was gradually reduced to 3 kg by the end of April and to 1 kg in May. It was increased to 2 kg in June to meet quota as milking was to cease in October. Body condition was generally improved in 1998 when compared to 1996 and 1997. The overall average condition score of 2.2 during the month of May was within Teagasc guidelines for breeding.

INDEX FARM B - GROWING STOCK

Calves were not reared on Index Farm B in 1996. Mean monthly calf weights for 1997 are given in Table 2-40.

Nineteen calves were reared in 1997; the remainder were sold - most within a few weeks of birth. Nine calves, five from indigenous cows, were reared on

cows until the autumn. The remainder were fed on whole milk to weaning at six to eight weeks of age. A calf starter ration was introduced in the first week of life. This was changed to a calf-rearer ration after a few weeks. A calf fattener ration was fed at grass from late August at a rate of 1 kg per head per day. This was increased to 2 kg per head per day after two weeks. Six weeks later it was reduced again to 1 kg per head per day. Supplementation was stopped when the animals were housed in December.

Calves performed well throughout the year. Average daily gain was around 1 kg from May to October - by which time calves had reached target weight for the year of 230 kg.

Forty eight calves were born alive in 1998. Ten of these (heifers) were reared on the farm. The remainder were sold within a few weeks of birth. Eighteen yearlings (1997 calves) were held on the farm and used in the Immunology (selenium) Project. They were housed up to 16/3/98 on *ad lib* round-bale silage. They received no concentrates over the winter of 1997-98 and throughout the remainder of 1998. Average daily liveweight gain for the group was 0.67 kg for the first eight months of 1998. Twenty yearlings and two-year-old animals from another farm in the area were also maintained on the farm for a period of about six months from the end of March to September.

Growth rates for the 12 steers in the Immunology Project, purchased at about six months of age in October 1996, are shown in Table 2-41.

The steers were maintained outdoors over the winter of 1996-97. They were fed silage *ad-lib* and approximately 1.5 kg beef concentrate per head per day and had reached 385 kg by February 1997. They received no concentrate feeding while at grass during 1997 and had reached slaughter weight by the autumn. Growth rates fell at the end of the year when they were fed hay for a short period prior to moving to an over-wintering paddock where they were fed silage. They were out-wintered in 1997-98 and removed in March 1998 after the completion of the Immunology Project.

CONTROL FARM – COWS

1996

Group mean monthly weights and condition scores for Askeaton-origin and Abbotstown-origin cows in Abbotstown in 1996 are given in Table 2-43. Cows were over-wintered in a cubicle house and fed pit silage. No concentrate was fed pre-calving. After calving, cows were fed 5.5 kg concentrates

per day until turnout on 16/4/96. At pasture, they were fed 2 kg per day. It is not possible to provide an accurate figure for total feed per cow in Abbotstown owing to the number of cow groups maintained on the farm, and to the year-round milking pattern. However, overall concentrate usage/cow in 1996 is estimated to have been between 750 and 900 kg depending on milking status and yield. The relatively higher level of feeding on the Control Farm than at Askeaton reflects the number of cows in winter milk production.

Both groups of cows performed well during the year. As condition scoring was only performed in February, May and September, detailed analysis of condition change in the immediate post-calving period cannot be made. However, none of the animals in either group showed excessive loss of condition post-calving in 1996 - all except two were within the recommended range at breeding.

1997

Group mean monthly weights and condition scores for Askeaton-origin and Abbotstown-origin cows in 1997 are given in Table 2-44. Dry and lactating cows were over-wintered in a cubicle house and fed pit silage. No concentrate was fed pre-calving. Cows were fed approximately 5.5 kg concentrates from calving to shortly after turnout. On 11/4/97 concentrate supplementation was reduced to 2.25 kg per day. On 5/5/97 this was further reduced to 2.0 kg per day and continued at this level throughout the summer and autumn. From 5/11/97 all cows were fed silage *ad lib*. Milking cows were supplemented with 4.5 kg of concentrates per day. Total concentrate supplementation averaged about 700 kg/cow in 1997.

Cow condition in both groups was good throughout the year. In general, Askeaton-origin cows had higher scores post-calving than Abbotstown-origin cows. No cows had condition scores below the recommended range during the breeding season.

1998

Group mean monthly weights and condition scores for Askeaton-origin and Abbotstown-origin cows on the Control Farm for the first three months of 1998 are given in Table 2-45. Cows were over-wintered in the cubicle house on silage *ad-lib* for the winter of 1997-98. No concentrate supplementation was given to dry cows. Following calving, cows were fed approximately 7 kg concentrates per head per day in addition to silage. The condition of cows in both groups was satisfactory in the first three months of 1998. Only

one cow in each of the two groups had a score under 2.0 in March. One, an Abbotstown-origin cow, had produced twins in February and the other, an Askeaton-origin cow, had a chronic hock lameness and was sold to the abattoir a month after calving.

CONTROL FARM - GROWING STOCK

Mean monthly weights for calves born and reared on the Control farm in 1996 and 1997 are given in Table 2-46. Figures for average daily gain are not given owing the varied age ranges of calves as a result of the near all-year calving pattern. Calves were turned onto grass at around 10 weeks of age in 1996 and 1997. At grass, they were fed 1 kg concentrates. Average calf weight of 170 kg in December 1996 was lower than on the two Askeaton farms owing to the more widely-spread calving pattern. Average calf weight in December 1997 was 236 kg which is a good weight for spring-born calves.

Mean monthly weights for yearlings on the Control farm in 1997 are given in Table 2-47. Average daily gain for the year was close to 1 kg

Growth rates for the 12 steers in the Immunology Project, purchased at about six months old in October, 1996. are shown in Table 2-48. The steers were out-wintered in 1996-97 and fed hay and silage *ad-lib*. They did not receive any concentrate supplementation over the winter of 1996-97 or throughout 1997. While not as good as the matching group on Index Farm B in Askeaton, growth rates on the Control Farm were acceptable throughout the year.

CONCLUSION

Growing animals, i.e. calves, weanlings, yearlings and two-year-olds, all performed well on the two Index Farms in Askeaton and on the Control Farm in Abbotstown throughout the two-year Project. Target weights were reached or exceeded in acceptable time periods. Ill-thrift was not a problem on any of the three farms and there was no evidence of depressed growth rates or feed conversion efficiency. Age for age, the Askeaton groups of growing animals generally out-performed their Abbotstown counterparts. These results, in Askeaton and Abbotstown, were achieved on feeding levels recommended by Teagasc for commercial farming.

Cow performance overall was satisfactory on the three farms. In general, body condition and weight changes were consistent with normal responses to the varying energy demands of the reproduction and lactation cycles. While post-calving condition

losses were, at times, in excess of what is generally recommended, these were clearly physiological or nutritional in origin and were well within the normal range for commercial farming.

The study has clearly demonstrated a difference between the Askeaton and Abbotstown herds in relation to periparturient energy balance in cows. This difference was particularly apparent in 1997 - the second year of the Project - when cows were monitored throughout an entire production cycle from pre-calving to dry-off. These differences are illustrated in Figure 2-1 where it can be seen that mean body condition scores on the two farms in Askeaton were near or below the recommended minimum of 2.0 points for up to three months post-calving – while mean scores on the Control farm remained above 2.5 throughout the equivalent period. Post-calving weight loss was also more marked on the Askeaton farms - in particular on Index Farm A.

That these changes were a reflection of differences in post-calving energy balance is illustrated by the significantly higher serum βHB, and lower glucose concentrations (both indicators of negative energy balance - Lomax, 1992; Whitaker *et al.*, 1993), in the Askeaton cows than the Abbotstown cows in blood samples collected between 30 and 60 days after calving (Table 2-49). Milk protein-to-fat ratios (Figure 2-2), which are also a measure of energy supply (Duffield *et al.*, 1997), were significantly lower (i.e. more severe negative energy balance) on the two Askeaton farms for the first four months of lactation (p < 0.05) in 1997.

While significant differences in performance were to be expected due to differences in location, pasture, and weather, the actual extent of the differences is surprising given that the study design was intended to minimise both inter-farm variation in nutrition and the possible contribution of nutrition to production or health problems. However, it is clear from the results of the study that the actual implementation of nutritional management on the two Askeaton farms, was, at times less successful than on the Control farm at Abbotstown in meeting the demands of post-calving production - though it should be stressed that the results on the Askeaton farms in terms of cow condition were by no means unusual or unacceptable in the context of normal commercial dairy farming.

There are a number of factors which may have contributed to the poorer condition performance on the Askeaton farms. One relates to pasture management. As reported elsewhere (Soil, Herbage, Feed and Water Volume), grass and silage quality were significantly better in Abbotstown than in Askeaton. This was partly due to the better quality of existing pastures and partly to closer adherence to grass management recommendations in Abbotstown. In relation to the latter, the effect of grass overgrowth in June, 1997 on Index Farm B can be seen by comparison of yields at this time with the same period in 1998 when no problems with grass supply occurred. In 1997, the cows were giving 20 kg on 4 kg of concentrates while in 1998 they gave 25 kg on only 2 kg concentrates.

As some differences in performance were observed before animals were turned out to grass, it is also likely that the actual amount of concentrate supplement consumed by the cows in Askeaton was, at times, inadequate to compensate for the poorer quality silage than in Abbotstown. The under-feeding of concentrate supplementation on Index Farm B in 1996 due to faulty scales has been referred to elsewhere (EPA, 1997a). Differences between the two locations in terms of supplementation may also have occurred at times due to the fact that animals in Abbotstown were milked alongside a winter-milking herd.

Besides the overall better performance of the Abbotstown farm, the study also demonstrated that the indigenous cows on the Askeaton farms tended to maintain body condition better post-calving than the brought-in cows. This probably largely reflects the breed differences between the two groups of cows. The Askeaton-origin cows on both Index Farms were British Friesian type. The Abbotstown-origin cows, and the cows brought onto Index Farm B, on the other hand, were Holstein-type (Abbotstown) or pure Holstein (Index Farm B; average RBI 107). Holstein cows are higher milk yielders, and are known to be more sensitive to post-calving condition loss, than the British Friesian. According to Teagasc (J. Sreenan, *personal communication*) it is difficult to make a direct comparison in condition performance between British Friesians and Holsteins due to physiological differences in energy partitioning. In general, the British Friesian tends to convert energy to fat while the Holstein preferentially converts it to milk - even at the expense of body condition (Webb *et al.*, 1999).

On both of the Askeaton farms, the cows were fed a fixed amount of concentrates rather than according to milk yield. This undoubtedly meant that many of the higher yielding Holstein cows suffered a more severe negative energy balance post-calving than the lower yielding British Friesians. This conclusion is supported by blood analysis results which showed that a number of the

brought-in cows on Index Farm B in March 1997, for example, had sub-clinical ketosis (i.e. raised βHB concentrations). The implications of this on fertility performance are discussed below.

In summary, condition performance was satisfactory on the three farms during the two-year Project. Variations in cow condition reflected normal physiological changes in energy balance associated with changes in productive status and time of year. Differences in performance between the Abbotstown and Askeaton herds was attributed to differences in location, grass supply and management. The fact that growing stock on the two Askeaton farms, which spent the greater part of their time outdoors on grass, tended to outperform their counterparts in Abbotstown in 1996 and 1997, provides convincing evidence of the wholesomeness of pastures in the area.

FERTILITY

Fertility performance is the ultimate determinant of a dairy herd's economic viability. Its success or otherwise determines the number of calves which will be born into the herd and the quantity of milk produced. In a seasonal-calving herd, the optimum target for a successful fertility program is to get as many cows in calf as possible in as short a time as possible. However, for a variety of reasons, the actual implementation of these goals varies widely in commercial farming practice. In the Teagasc DairyMIS survey, for example, which comprises herds generally considered to have a relatively high standard of management, average heat detection results are between 50 and 60 *per cent*. These are well short of the target 80 *per cent*. Assessment of fertility performance, therefore, is generally made by comparison with accepted reference ranges rather than attainment of a specific target figure. The reference ranges published by Teagasc, and which are based on the DairyMIS survey, are given in Appendix 1 and are used for comparative purposes throughout the following analysis.

In the present study, fertility on the three farms was monitored as part of the overall assessment of herd performance. In addition, as infertility was the most commonly reported syndrome on problem farms in the Askeaton area, it was also envisaged that the availability of comprehensive records from the two-year study would allow in-depth investigation of factors contributing to any significant performance shortfalls which might occur. In the event that fertility performance was below target to an extent that could not be accounted for by analysis of management and animal records, it was intended that further lines of

investigation could have been pursued to identify other contributory factors.

Details of fertility management are described in the Monitor Study Protocol (EPA, 1997a). Briefly, the program specified that heat detection during the breeding season was to be carried out by four daily periods of visual inspection of cows between 7.00 am and 9.00 pm. Tail-painting was used as an aid to heat detection. In 1997, a vasectomised bull with chin-ball marker was also used on Index Farm B to help identify cows in heat. On Index Farm A and the Control Farm, cows were served by artificial insemination (AI) throughout. On Index Farm B, all cows were served by artificial insemination in 1996. In 1997, all dairy cows were served by artificial insemination up to mid-June. All suckler cows – as well as any dairy cows remaining open after mid-June - were served by the stock bull. Records were kept of all calvings, heats, services and pregnancy examinations on the three farms.

The main fertility indices for the three herds are presented in the following sections. More detailed fertility records have already been published in Interim Reports (EPA 1997a and 1998). The Monitor Project formally extended from 1 April 1996 to 31 March 1998. However, owing to the introduction of cows which had calved and completed part of their lactation before arrival, a full assessment of fertility performance could not be made for Index Farm B in 1996. In order to provide a full two years data, it was decided to continue monitoring fertility performance on Index Farm B up to the end of the 1998 lactation period.

INDEX FARM A

Fertility performance indicators for Index Farm A in 1996 and 1997 are given in Table 2-50. Fertility performance was poor in 1996. The main problems were a poor submission rate at the start of the season and a poor overall conception rate. The poor submission rate was clearly a problem of heat detection. Although over two-thirds of the herd had calved by March, only one cow had been seen on heat by early May. That the problem was not due to anoestrus at this time was demonstrated by the results of uterine scanning carried out which indicated that most cows were cycling normally.

Although it is not possible to determine to what extent the problem was due to inadequate observation on the one hand, and to reduced or non-expression of heat on the other, it is unlikely that the latter can have accounted for a significant proportion of the missed heats. According to Allrich (1993), suboestrus is mainly a problem of heifers. Although excessive negative energy

balance is known to delay the onset of heats post-calving – and this may have contributed to anoestrus in some cows on the farm at an earlier stage in the season (see page 26) - there is little evidence to specifically associate energy deficit with suboestrus once heats have commenced (Allrich, 1993).

Neither was there any evidence that reproductive or other disease could account for the problem. The cows were generally in good health and had normal appetites. Other than changes associated with post-calving energy balance, biochemistry and haematology analyses showed no evidence of changes which should have affected fertility.

A poor overall conception rate of 34 per cent was largely due to poor results with hormonal induction of heats in June. The latter was introduced to deal with the earlier poor submission rate. These results have been discussed in detail elsewhere (EPA, 1997a). Conception rates in May and July were 71 and 50 per cent, respectively which represent a good performance.

Fertility performance improved in 1997. Average calving to first service and calving to conception intervals, as well as submission rates, were all close to target. Though below target at 42 per cent, overall conception performance was within the reported range for herds of this size (Appendix 1). Factors contributing to reduced performance included missed returns to service and repeat-breeders. As previously reported (EPA, 1997a), milk progesterone analysis also indicated that while accuracy of heat detection was good, the efficiency of heat detection was less so – leading to a relatively high incidence of missed heats.

It is also likely that the decision on scientific grounds to retain cows which were identified as having a poor fertility record in 1996, and which on a purely commercial basis would have been culled, had a negative impact on conception rates. At least two of the animals classified as repeat-breeders would normally have been culled based on their performance in 1996.

INDEX FARM B

Fertility performance indices for Index Farm B in 1996, 1997 and 1998 are given in Table 2-51. Performance was only moderate in 1996. Submission rate was low due to poor heat detection results at the start of the season. Although calving to first service and calving to conception were within target, overall conception performance was poor.

Part of the reason for the latter was a management decision to serve cows as early as possible after calving in order to tighten up the calving pattern for the following year (EPA, 1997a). This would have resulted in some cows being served at a time when fertility was less than optimal. However, as already reported (EPA, 1997a), there was also evidence that both accuracy and efficiency of heat detection were below expectations. Missed heats were a particular problem later in the season.

Heat detection results were poor on Index Farm B at the start of the 1997 breeding season. Because of this, a decision was made to increase the use of artificial heat induction with progesterone implants (PRID coils, Interpharm) where necessary (EPA, 1998). Subsequent investigation of the heat detection problems, based on the result of milk progesterone assays, indicated that while accuracy of heat detection was good, efficiency was less so - i.e. only about 50 per cent of returns to service were being detected. Despite these problems, overall fertility performance on Index Farm B in 1997 was good. Conception rate to all services was over 50 per cent and the fertility rate for the year was 90 per cent.

As outlined above, monitoring of fertility performance on Index Farm B continued throughout the 1998 breeding season. As no new breeding stock were introduced, the herd was essentially the same as that in 1997. Fertility performance in 1998 was good. Submission rate, heat detection rate, calving to first service and calving to conception were all close to or better than target. Only nine of a total of 69 services in 1998 were artificially induced (PRID). Five of these resulted in pregnancies which is a comparable conception performance to natural heats.

A number of factors can be identified which contributed to the improved fertility performance on Index Farm B in 1998. Firstly, the calving spread had been reduced from eight to four months over the two previous years. This facilitated the implementation of a more intensive heat detection regime. Secondly, a number of changes in relation to fertility management and heat detection were introduced based on experience in previous years. Thirdly, a tighter control over post-calving nutrition and grass management ensured that cows were in better condition at breeding than in previous years. Mean condition score for the brought-in cows in May 1998, for example, was 2.2 compared to 1.9 in 1997. This difference was highly significant (paired t-test - 1997 vs 1998 BCS: $n = 30$ cows, $p = 0.0007$). Biochemical markers of negative energy balance, i.e. serum

βHB and glucose concentrations, were also consistent with a less severe post-calving energy deficit in 1998 than in 1997 (Table 2-42) and *see* discussion below re three farms.)

CONTROL FARM

Fertility performance indicators for the Control Farm in 1996 and 1997 are given in Table 2-52. Calving was essentially year-round - though with a peak in spring and a smaller peak in autumn. As previously reported (EPA, 1997a), hormonal induction of heats was used routinely on the Control Farm to accommodate staff rostering arrangements. Analysis of heat detection performance is, therefore, of little value. Overall fertility performance was good in 1996 and 1997. Conception rates were at or above target. Fertility rate was over 90 *per cent* in both years.

CONCLUSION

Fertility performance of indigenous and brought-in cows on the two Askeaton farms was only moderate for the two years of the study – 1996 and 1997. The main problems encountered on both farms related to heat detection. This is not an unexpected finding as failure to detect heat (non-detected oestrus) is regarded as the commonest cause of infertility in dairy farming (Esslemont and Kossaibati, 1995; Esslemont and Kossaibati, 1996). The causes of non-detected oestrus are generally considered to be a function of fertility management (Sreenan and Diskin, 1992; Esslemont and Kossaibati, 1996) rather than reproductive pathology.

In the present study, there was no evidence that reproductive or systemic disease was a significant contributory factor to the fertility problems at herd level. The animals on the two Askeaton farms were generally in good health throughout. However, there was evidence that both the implementation of heat detection and nutrition management played a role in relation to reduced performance. Analysis of fertility records, as well as the results of ultrasound scanning and milk progesterone analysis showed evidence of a relatively high incidence of missed heats at times on the two farms.

While it is not possible to precisely quantify the role of nutrition in relation to performance on these farms, there is an accepted association between excessive condition loss post-calving and delayed onset of heat (Beam and Butler, 1999). Condition-scoring results in 1996 and 1997 demonstrated a more severe post-calving negative energy balance on the two Askeaton farms than the Control Farm. The effect of this on fertility may have been

mediated through delayed resumption of post-calving oestrus cyclicity, reduced conception rates, or both. On the other hand, the significantly better body condition of cows on Index Farm B during the 1998 breeding season, as well as the management changes introduced before the start of the breeding season, probably contributed to the greatly improved fertility performance in that year (*see above*).

The results of fertility performance on the Askeaton farms in 1996 and 1997 must also be viewed in the context of the fertility problems which had built up in preceding years. The calving season on Index Farm B in 1996, for example, extended over eight months making heat detection difficult. However, despite the fact that changes in breeding management can take several years to implement, there was evidence of a definite improvement in performance over the period of the study. This was particularly so on Index Farm B where fertility in 1998 was very good. This improvement reflected the cumulative effect of management changes which had been introduced over the period of the two-year study. Although conception performance data are not available for Index Farm A in 1998, the calving spread had been reduced to four months and, based on herdowner records, the submission rate, calving to first service, and heat detection rates were all close to target in 1998.

Fertility performance of both Askeaton- and Abbotstown-origin cows was good on the Control Farm in 1996 and 1997.

In conclusion, while fertility performance was below target at times on the two Askeaton farms, the results of the present study provide no evidence that shortfalls in performance could be attributed to environmental pollution. The main problems encountered related to heat detection and conception performance. These are also the most-commonly-reported causes of fertility under-performance on commercial farms elsewhere. On the other hand, the results of the study clearly demonstrate the importance and inter-relationship of nutrition, grass management and heat detection regimes in relation to fertility performance – and the gap which may exist at farm level between recommendations and their actual implementation.

BLOOD ANALYSIS

The present section presents the results of laboratory tests performed on blood samples collected from animals on the three Monitor Farms. Samples were collected from all study cows at approximately monthly intervals throughout the

project. Blood samples were also collected from growing stock on the two Index Farms in 1997 and 1998. Other than the Immunology Project steers, growing stock were not sampled on the Control Farm.

Details of the range of tests carried out on samples collected on the three farms have already been reported (EPA, 1995). A standard test profile (Standard Blood Profile) was applied monthly involving haematology and biochemistry analysis. The latter comprised albumin, A/G ratio, AST, ßHB, copper, γGT, GLDH, globulin, magnesium, phosphorus, total protein, urea, and zinc analyses. A more extensive biochemistry profile (Enhanced Blood Profile) was applied at approximately every third monthly sampling. This included the following additional biochemical analyses: calcium, chloride, CK, selenium and thyroxin.

A full range of haematology analyses, with the exception of differential white cell counts, was performed throughout. Differential white cell counts were carried out on individual samples as indicated by total counts and clinical history. Standard methodology was used for all blood and clinical pathology analyses throughout the study.

Additional tests were performed as required, e.g. bacterial and viral serology, non-standard mineral analyses.

Reference ranges for all measured parameters are given in Appendix 1. These are from published sources and are used as a general guideline to assess the normality or otherwise of analysis results. They should not, however, be regarded as defining normality in all cases or under all circumstances. In the case of some minerals, for example, opinions are divided among experts regarding the point at which a value should be regarded as deficient. This particularly applies to selenium and iodine. Blood white cell counts can also vary widely depending on breed, sex, age, pregnancy status, exercise, and time of sampling. No particular significance, therefore, can usually be given to values which are marginally above or below the range — unless they are associated with specific clinical signs of illness.

Conversely, values within the reference ranges do not necessarily indicate 'normality' of the animal from which the samples were taken. This particularly applies in relation to white cell counts where, for example, a total count may mask abnormal values in the underlying differential (e.g. a reduced neutrophil count and raised lymphocyte count could add up to a normal total count).

HAEMATOLOGY

Mean haematology results for all monthly samplings of study cows on the three Monitor Farms for the period of the two-year study are given in Appendix 3. Analysis of results in the present report is primarily concerned with changes at group and farm level. Discussion of individual values outside normal ranges for the period April 1996 to December 1997 has already been included in previous interim reports (EPA 1996, 1997a, 1998). Briefly, occasional cases of mild anaemia were noted. These were probably largely physiological in nature and relating to stage of lactation. Total or differential white cell counts above normal were generally associated with intercurrent clinical or sub-clinical infections, e.g. mastitis, lameness, and upper respiratory tract infections. Occasional total white cell counts below the reference range were not associated with any signs of illness and were not regarded as being of clinical significance.

The only significant haematology findings at individual-animal level in the last three months of the Project, i.e. January to March 1998, – and which have not previously been reported - were occasional cases of mild anaemia and raised total white cell counts. One raised white cell count in a cow on Index Farm B in February 1998 was associated with a case of lameness. In the same month, two packed cell volume (PCV) values below the lower end of the reference range (20.6 and 23.6 g/l) were recorded in two recently-calved cows which were otherwise healthy. On Index Farm A, a PCV of 22.7 was recorded in February, 1998 in a cow which developed metritis after calving in January. Also on Index Farm A, a WBC of 12.0 x 10^9 cells/l was recorded in a cow which had delivered one live and one dead twin in February, 1998. A WBC of 23.5 x 10^9 cells/l was recorded in a cow with metritis on the Control Farm in February, 1998.

Summary statistics for all haematology parameters for samples collected from animals on the three farms, grouped according to location and origin, are given in Appendix 3. Group mean values for all red cell parameters were within accepted reference ranges throughout the two-year period. Overall, red cell counts show evidence of a seasonal trend which probably reflects the calving and lactation cycles, together with changes in grass supply and quality. Counts tended to increase during the first half of the year peaking around June and declining gradually thereafter. This pattern is most apparent for Index Farms A and the Control Farm in 1996, and for Index Farms A and B in 1997 (Figure 2-3). The more level pattern for the Control Farm in

1997 may reflect the more extended calving pattern in that year.

Group mean values for total white cell counts were within accepted reference ranges throughout the two-year period.

Although statistically significant differences for haematology parameters ($p < 0.05$) were noted at times between cow groups on all three farms, mean values were generally within normal reference ranges. As the differences were not associated with specific differences in group health status, no particular clinical significance can be attached to these findings.

BIOCHEMISTRY

Mean biochemistry results for all monthly samplings of study cows on the three Monitor farms for the period of the two-year study are given in Appendix 4. Detailed analyses of individual-animal deviations from reference ranges have already been reported for the period April, 1996 to December, 1997. Briefly, occasional low phosphorus concentrations were seen in aged cows and also in high yielders at times on all three farms. They were most prevalent at peak milk yield and were noted more often on the Control Farm than either of the Askeaton farms. Low phosphorus in aged or high-yielding cows is a normal nutritional-metabolic response (Puls, 1994).

Individual-animal increases in globulin concentrations consistent with nutritional or inflammatory influences were also observed on occasions over the two-year period. The only significant biochemical findings at individual-animal level in the final three months of the project related to a number of cases of illness in cows on Index Farm A details of which have been outlined elsewhere (see page 16). A raised βHB concentration was recorded in one cow which developed E. coli mastitis shortly after calving in January 1998. This was most likely due to inappetance as blood glucose was also low. Raised globulin concentrations in a number of blood samples from cows in the first three months of 1998 were associated with cases of mastitis.

The most significant biochemistry changes throughout the Project were at group-level. They were largely seasonal in nature and were associated with variations in nutritional and production status. Raised blood urea concentrations (reference range 2.65-6.89 mmol/l) on all three farms during the summer-autumn grazing periods were probably associated with changes in herbage supply and quality (Figure 2-4). Low urea and phosphorus

concentrations in the majority of cows in July 1997 on Index Farm A may have been associated with a transient insufficiency of grass supply due to delayed fertilizer application. The marked fluctuations in mean urea values on this farm - in contrast to the other two farms - are also suggestive of a less consistent grass management program.

Seasonal changes in copper and magnesium concentrations consistent with changes in herbage composition were also noted. Copper concentrations tended to fall over the summer months. This was most obvious on Index Farm A. A similar finding has already been reported by O'Farrell et al., (1986) in relation to grazing cattle in Ireland. Low blood magnesium in November 1996 and 1997 on Index Farm A was consistent with reduced dry matter intake on grass. This is a not uncommon occurrence in cows on grass at this time of the year and was corrected by housing animals and by provision of silage. Copper concentrations marginally below the reference ranges were observed in about a third of the cows on the Control Farm and two-thirds on Index Farm A in February 1998. They were not associated with any signs of ill-health and are not regarded as having been of clinical significance.

βHB concentrations above the reference range (0 - 0.9 mmol/l) were recorded on all three farms on a number of occasions (Figure 2-5). Samples were generally from recently-calved cows and the finding is consistent with post-calving negative energy balance. It was most marked on the two Askeaton farms in 1997 when about a third of cows had raised values. Both indigenous and brought-in animals were affected. The presence of a more pronounced post-calving negative energy balance on the two Askeaton farms has been discussed elsewhere (see page 30). In this context, it is also worth noting that few raised βHB values were recorded on Index Farm B in the spring of 1998 when fertility performance was also good.

It is difficult to account for the raised βHB concentrations which were observed in the majority of the Askeaton-origin cows on the Control Farm in June 1997. As about half of the cows in question were three or more months calved, it is unlikely that the response was due to post-calving negative energy balance. The fact that only one of the Abbotstown-origin cows - which were on the same feeding regime - had a raised value rules out the possibility of a feed-related problem. Neither was there any evidence of ill-health or an unexpected drop in body condition or milk production in the group at the time.

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Raised concentrations of the liver enzyme GLDH were also noted in a large proportion of cows on the Control Farm in September 1997 (Figure 2-6). Eighty *per cent* of samples had GLDH concentrations above the reference range of 0 - 25 iu/l. Although both brought-in and indigenous cows were affected, values were significantly higher (p < 0.05) in the latter. As the finding was not associated with specific signs of ill-health, and the majority of the cows had a higher milk yield in that month than in the previous one, they were probably production-related.

About a third of the cows on Index Farm A had raised GLDH concentrations in November 1996. As this change was accompanied by raised serum urea and reduced magnesium concentrations, it may have been associated with a reduction in grass quality and supply at the time.

Changes in blood selenium concentrations on the three farms reflected their differing selenium status. Index Farm B has a low to marginal selenium status with herbage concentrations generally below 0.1 mg/kg throughout the grazing season (EPA 1997b). All of the Control Farm and parts of Index Farm A, on the other hand, would be classified as high selenium status with herbage concentrations on the former between 0.12 and 1.85 and the latter from about 0.03 to 0.45 mg/kg (EPA 1997b).

Blood selenium concentrations below the normal range of 0.75 - 3.0 µmol/l were recorded in some grazing animals in the autumn of 1997 on Index Farm B. Mean values for blood samples from steers in the Immunology Project on the same farm were also low in September and December 1997. In contrast, selenium concentrations in blood samples from cows on the Control Farm rose throughout the grazing period in both 1996 and 1997 so that by the autumn of each year almost all cows had values above 3.0 µmol/l. Mean blood selenium concentrations on Index Farm A ranged from a high of 3.4 µmol/l in September 1996 to a low of 1.2 µmol/l in March 1998. Over a half of the 35 cows sampled in September 1996 had concentrations above 3.0 µmol/l.

With the exception of selenium, mean biochemistry values for blood samples collected from growing stock on the two Askeaton farms in 1997 and 1998 were generally within normal ranges.

CONCLUSION

Haematology and biochemistry findings on cows on the three farms were consistent with animals in overall good health and there was no evidence of changes in blood values which would suggest that unidentified factors had a significant negative effect on herd health. Group fluctuations in a number of parameters were mainly due to seasonal changes in productive or nutritional status. Individual-animal values outside normal ranges were consistent with the type and range of conditions which would be expected in cows and growing stock managed under normal farm conditions.

GENERAL CONCLUSION

The results of this two-year study have shown no evidence of a continuation of the severe animal health and production problems which had previously been reported on the two Index Farms in Askeaton. Neither was there any evidence that cows from one of the Index Farms (Farm A), when moved to a distant Control Farm, performed less well than might have been expected given breed and type. Animal health was generally good throughout the period of the study. Diseases encountered generally comprised conditions which are common on farms elsewhere in Ireland and incidence rates were within normal limits. The main disease conditions recorded in cows were lameness and mastitis. The health of calves and growing stock was generally good and no major disease incidents were encountered. Although a number of disease incidents in cows and calves were recorded on Index Farm A in the last three months of the project, straightforward diagnoses were made in most cases and there was no evidence to suggest that environmental pollution or other unusual factors were involved.

Animal production was also generally satisfactory on the three farms. Milk production and body condition performance of both Askeaton- and non-Askeaton-origin cows ranged from moderate on the two Askeaton farms to good on the Control Farm. While fertility performance was below target at times, mainly on the two Askeaton farms in the first year of the project, problems largely related to heat detection and there was no evidence to suggest that other unusual factors were involved.

In conclusion, performance on the three study farms was generally satisfactory and there was no evidence to suggest that health or production were adversely affected by environmental pollution or other unidentified factors throughout the duration of the project.

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Tables

Table 2-1: Disease incidence rates Index Farm A 1996 - Cows

	Askeaton-origin cows		VRL-origin cows		All cows	
Parturient/lactation Conditions						
Population at risk[1]	13		10		23	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	0	0.000	0	0.000	0	0.000
Milk Fever	1	0.077	1	0.100	2	0.087
Downer cow	2	0.154	0	0.000	2	0.087
Retained Placenta	3	0.231	1	0.100	4	0.174
Vulval discharge/metritis	6	0.462	0	0.000	6	0.261
Ketosis	0	0.000	0	0.000	0	0.000
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	6	0.462	5	0.500	11	0.478
Udder Oedema	0	0.000	0	0.000	0	0.000
Miscellaneous conditions						
Population at risk[3]	13		12		25	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	0	0.000	0	0.000	0	0.000
Respiratory disease	0	0.000	0	0.000	0	0.000
Lameness	1	0.006	6	0.042	7	0.023
Haematoma	0	0.000	0	0.000	0	0.000
'Pink eye'	0	0.000	0	0.000	0	0.000
Redwater	0	0.000	0	0.000	0	0.000
Cystic ovary	2	0.013	0	0.000	2	0.007
All other	2	0.013	2	0.014	4	0.013

[1]Total calved. [2]Cumulative incidence. [3]Monthly average numbers [4]Incidence rate - cases per animal month at risk.

Table 2-2: Disease incidence rates Index Farm A 1996 – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 24[3])	1	0.042
1 day – 1 month (PAR = 23[4])		
Deaths	0	0.000
Diarrhoea	2	0.087
Respiratory disease	0	0.000
Navel/Joint Ill	0	0.000
All other	1	0.043
1 month – 1 year (PAR = 10.6[5])		
	Cases	IR[6]
Deaths	0	0.000
Umbilical abscess	1	0.008
All other	0	0.000
Over 1 year	None in 1996	

[1]Cumulative incidence. [2]PAR = population at risk. [3]Total calved. [4]Calved and alive at six hours [5]Monthly average numbers. [6]Incidence rate - cases per animal month at risk.

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Table 2-3: Disease incidence rates Index Farm A 1997 - Cows

Parturient/lactation Conditions	Askeaton-origin cows		VRL-origin cows		All cows	
Population at risk[1]	13		· 16		29	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	0	0.000	0	0.000	0	0.000
Milk Fever	0	0.000	0	0.000	0	0.000
Downer cow	0	0.000	0	0.000	0	0.000
Retained Placenta	0	0.000	0	0.000	0	0.000
Vulval discharge/metritis	2	0.154	1	0.063	3	0.103
Ketosis	0	0.000	0	0.000	0	0.000
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	1	0.077	2	0.125	3	0.103
Udder Oedema	1	0.077	0	0.000	1	0.034
Miscellaneous conditions						
Pop. At risk[3]	8		13		21	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	0	0.000	0	0.000	0	0.000
Respiratory disease	0	0.000	0	0.000	0	0.000
Lameness	2	0.021	0	0.000	2	0.008
Haematoma	0	0.000	2	0.013	2	0.008
'Pink eye'	0	0.000	0	0.000	0	0.000
Redwater	0	0.000	0	0.000	0	0.000
Cystic ovary	0	0.000	0	0.000	0	0.000
All other	2	0.021	1	0.006	3	0.012

[1]Total calved [2]Cumulative incidence. [3]Monthly average numbers. [4]Incidence rate - cases per animal month at risk

Table 2-4: Disease incidence rates Index Farm A 1997 – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 20[3])	1	0.050
1 day – 1 month (PAR = 19[4])	Cases	CI
Deaths	0	0.000
Diarrhoea	5	0.263
Respiratory disease	0	0.000
Navel/Joint Ill	0	0.000
All other	0	0.000
1 month – 1 year (PAR = 14.4[5])	Cases	IR[6]
Deaths	0	0.000
Diarrhoea	4	0.023
Respiratory disease	0	0.000
Umbilical abscess	0	0.000
'Pink eye'	0	0.000
Lameness	1	0.006
All other	0	0.000
Over 1 year (PAR = 8.7[5])	Cases	IR
Deaths	0	0.000
All other	0	0.000

[1]Cumulative incidence. [2]PAR = population risk. [3]Total calved. [4]Calved and alive at six hours. [5]Monthly average numbers. [6]Incidence rate - cases per animal month at risk.

Table 2-5: Disease incidence rates Index Farm A 1998 - Cows

	Askeaton-origin cows		VRL-origin cows		All cows	
Parturient/lactation Conditions						
Population at risk[1]	10		12		22	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	1	0.100	0	0.000	1	0.045
Milk Fever	0	0.000	0	0.000	0	0.000
Downer cow	0	0.000	0	0.000	0	0.000
Retained Placenta	1	0.100	1	0.083	2	0.091
Vulval discharge/metritis	1	0.100	1	0.083	2	0.091
Ketosis	0	0.000	0	0.000	0	0.000
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	2	0.200	0	0.000	2	0.091
Udder Oedema	1	0.100	0	0.000	1	0.045
Miscellaneous conditions						
Population at risk[3]	10		12		22	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	1	0.008	0	0.000	1	0.004
Respiratory disease	0	0.000	0	0.000	0	0.000
Lameness	2	0.017	0	0.000	2	0.008
All other	0	0.000	0	0.000	0	0.000

[1]Total calved. [2]Cumulative incidence. [3]Monthly average numbers. [4]Incidence rate - cases per animal month at risk.

Table 2-6: Disease incidence rates Index Farm A 1998 – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 28[3])	5	0.179
1 day – 1 month (PAR = 23[4])	Cases	CI
Deaths	0	0.000
Diarrhoea	1	0.043
Respiratory disease	0	0.000
Navel/Joint Ill	1	0.043
All other	1	0.043
1 month – 1 year (PAR = 5.7[5])	Cases	IR[6]
Deaths	0	0.000
Diarrhoea	0	0.000
Respiratory disease	0	0.000
Lameness	2	0.029
All other	0	0.000
Over 1 year (PAR = 13[5])	Cases	IR
Deaths	0	0.000
All other	0	0.000

[1]Cumulative incidence [2]PAR = population at risk. [3]Total calved [4]Calved and alive at six hours [5]Monthly average numbers. [6]Incidence rate - cases per animal month at risk.

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Table 2-7: Disease incidence rates Index Farm B 1996 - Cows

	Askeaton-origin cows		Bought-in cows		All cows	
Parturient/lactation Conditions						
Population at risk[1]	26		8		34	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	1	0.125	1	0.029
Difficult calving	2	0.077	0	0.000	2	0.059
Milk Fever	1	0.038	0	0.000	1	0.029
Downer cow	1	0.038	0	0.000	1	0.029
Retained Placenta	0	0.000	1	0.125	1	0.029
Vulval discharge/metritis	4	0.154	1	0.125	5	0.147
Ketosis	0	0.000	0	0.000	0	0.000
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	8	0.308	4	0.500	12	0.353
Udder Oedema	0	0.000	0	0.000	0	0.000
Miscellaneous conditions						
Population at risk[3]	26		14		40	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	1	0.003	0	0.000	1	0.006
Respiratory disease	0	0.000	0	0.000	0	0.000
Lameness	6	0.019	2	0.012	8	0.048
Haematoma	0	0.000	0	0.000	0	0.000
'Pink eye'	0	0.000	2	0.012	2	0.012
Redwater	0	0.000	1	0.006	1	0.006
Cystic ovary	1	0.003	0	0.000	1	0.006
All other	1	0.003	1	0.006	2	0.012

[1]Total calved [2]Cumulative incidence. [3]Monthly average numbers.[4]Incidence rate - cases per animal month at risk.

Table 2-8: Disease incidence rates Index Farm B 1996 – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 34[3])	3	0.088
1 day – 1 month (PAR = 31[4])	**Cases**	**CI**
Deaths	0	0.000
Diarrhoea	4	0.129
Respiratory disease	0	0.000
Navel/Joint Ill	1	0.032
All other	0	0.000
1 month – 1 year (None in 1996[5])		
Over 1 year (PAR = 3[5])	**Cases**	**IR[6]**
Deaths	0	0.000
Lameness	1	0.028
Transit Fever (12 steers on arrival)	12	0.333
All other	0	0.000

[1]Cumulative incidence [2]PAR = population at risk.[3]Total calved. [4]Calved and alive at six hours [5]Monthly average numbers.[6]Incidence rate - cases per animal month at risk.

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Table 2-9: Disease incidence rates Index Farm B 1997 - Cows

	Askeaton-origin cows		Bought-in cows		All cows	
Parturient/lactation Conditions						
Population at risk[1]	11		25		36	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	0	0.000	2	0.080	2	0.056
Milk Fever	0	0.000	0	0.000	0	0.000
Downer cow	0	0.000	0	0.000	0	0.000
Retained Placenta	0	0.000	0	0.000	0	0.000
Vulval discharge/metritis	1	0.091	6	0.240	7	0.194
Ketosis	0	0.000	1	0.040	1	0.028
Tetany	0	0.000	1	0.040	1	0.028
Mastitis	3	0.273	4	0.160	7	0.194
Udder Oedema	0	0.000	0	0.000	0	0.000
Miscellaneous conditions						
Population at risk[3]	17		31		48	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	0	0.000	1	0.003	1	0.002
Respiratory disease	1	0.005	0	0.000	1	0.002
Lameness	6	0.029	13	0.035	19	0.033
Haematoma	0	0.000	0	0.000	0	0.000
'Pink eye'	0	0.000	3	0.008	3	0.005
Redwater	0	0.000	0	0.000	0	0.000
Cystic ovary	0	0.000	1	0.003	1	0.002
All other	0	0.000	2	0.005	2	0.003

[1]Total calved [2]Cumulative incidence. [3]Monthly average numbers. [4]Incidence rate - cases per animal month at risk.

Table 2-10: Disease incidence rates Index Farm B 1997 – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 36[3])	1	0.028
1 day – 1 month (PAR = 35[4])	Cases	CI
Deaths	0	0.000
Diarrhoea	7	0.200
Respiratory disease	0	0.000
Navel/Joint Ill	0	0.000
All other	0	0.000
1 month – 1 year (PAR = 18[5])	Cases	IR[6]
Deaths	0	0.000
Diarrhoea	1	0.005
Respiratory disease	0	0.000
Umbilical abscess	1	0.005
Lameness	0	0.000
All other	0	0.000
Over 1 year (PAR = 12.5[5])	Cases	IR
Deaths	1	0.007
Respiratory disease	1	0.007
Diarrhoea	0	0.000
Lameness	2	0.013
All other	0	0.000

[1]Cumulative incidence. [2]PAR = population at risk. [3]Total calved [4]Calved and alive at six hours. [5]Monthly average numbers [6]Incidence rate - cases per animal month at risk.

Table 2-11: Disease incidence rates Index Farm B 1998 (January – October) – Cows

	Askeaton-origin cows		Brought-in cows		All cows	
Parturient/lactation Conditions						
Population at risk[1]	15		34		49	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	1	0.067	2	0.059	3	0.061
Milk Fever	0	0.000	1	0.029	1	0.020
Downer cow	0	0.000	0	0.000	0	0.000
Retained Placenta	0	0.000	0	0.000	0	0.000
Vulval discharge/metritis	0	0.000	0	0.000	0	0.000
Ketosis	0	0.000	1	0.029	1	0.020
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	1	0.067	2	0.059	2	0.041
Udder Oedema	0	0.000	0	0.000	0	0.000
Miscellaneous conditions						
Population at risk[3]	11		35		47	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	0	0.000	0	0.000	0	0.000
Respiratory disease	0	0.000	0	0.000	0	0.000
Lameness	6	0.045	7	0.017	13	0.023
Cystic ovary	0	0.000	0	0.000	0	0.000
All other	0	0.000	0	0.000	0	0.000

[1]Total calved. [2]Cumulative incidence. [3]Monthly average numbers. [4]Incidence rate - cases per animal month at risk

Table 2-12: Disease incidence rates Index Farm B 1998 (January – October) – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR = 48[2])	2	0.042
1 day – 1 month (PAR = 46[3])	Cases	CI
Deaths	0	0.000
Diarrhoea	6	0.130
Respiratory disease	0	0.000
Navel/Joint Ill	2	0.043
All other	0	0.000
1 month – 1 year (PAR = 10[4])	Cases	IR[5]
Deaths	0	0.000
Diarrhoea	3	0.030
Respiratory disease	0	0.000
Umbilical abscess	0	0.000
Lameness	0	0.000
All other	1 (Ringworm)	0.010
Over 1 year (PAR = 16[4])	Cases	IR
Deaths	0	0.000
Respiratory disease	0	0.000
Diarrhoea	0	0.000
Lameness	1	0.006
All other	0	0.000

[1] Cumulative incidence. [2] Total calved. [3] Calved and alive at six hours. [4]Monthly average [5]Incidence rate - cases per animal month at risk

Table 2-13: Disease incidence rates Control Farm 1996 - Cows

Parturient/lactation Conditions	Askeaton-origin cows		VRL-origin cows		All cows	
Population at risk[1]	17		20		37	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	0	0.000	0	0.000	0	0.000
Milk Fever	2	0.118	2	0.100	4	0.108
Downer cow	0	0.000	0 .	0.000	0	0.000
Retained Placenta	1	0.059	2	0.100	3	0.081
Vulval discharge/metritis	0	0.000	0	0.000	0	0.000
Ketosis	2	0.118	2	0.100	4	0.108
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	7	0.412	18	0.900	25	0.676
Udder Oedema	0	0.000	0	0.000	0	0.000
Miscellaneous conditions						
Population at risk[3]	27		23		50	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	0	0.000	0	0.000	0	0.000
Respiratory disease	1	0.003	1	0.004	2	0.003
Lameness	0	0.000	4	0.014	4	0.007
Haematoma	0	0.000	0	0.000	0	0.000
'Pink eye'	0	0.000	0	0.000	0	0.000
Cystic ovary	0	0.000	0	0.000	0	0.000
All other	2	0.006	2	0.007	4	0.007

[1]Total calved. [2]Cumulative incidence [3]Monthly average numbers. [4]Incidence rate - cases per animal month at risk.

Table 2-14: Disease incidence rates Control Farm 1996 – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 37[3])	3	0.081
1 day – 1 month (PAR = 34[4])	Cases	CI
Deaths	0	0.000
Diarrhoea	13	0.382
Respiratory disease	0	0.000
Navel/Joint Ill	0	0.000
All other	1	0.029
1 month – 1 year (PAR = 12)[5]	Cases	IR[6]
Deaths	0	0.000
Diarrhoea	7	0.049
Respiratory disease	1	0.007
Umbilical abscess	0	0.000
'Pink eye'	1	0.007
Lameness	0	0.000
All other	0	0.000
Over 1 year (PAR = 3)[5]	Cases	IR
Deaths	0	0.000
Transit Fever (12 steers on arrival)	0	0.000
All other	2	0.056

[1]Cumulative incidence. [2]PAR = population at risk [3]Total calved. [4]Calved and alive at six hours [5]Monthly average – 12 steers arrived October. [6]Incidence Rate - cases per animal month at risk.

Table 2-15: Disease incidence rates Control Farm 1997 - Cows

Parturient/lactation Conditions	Askeaton-origin cows		VRL-origin cows		All cows	
Population at risk[1]	19		17		36	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	4	0.211	0	0.000	4	0.111
Milk Fever	0	0.000	0	0.000	0	0.000
Downer cow	0	0.000	0	0.000	0	0.000
Retained Placenta	0	0.000	1	0.059	1	0.028
Vulval discharge/metritis	0	0.000	0	0.000	0	0.000
Ketosis	0	0.000	0	0.000	0	0.000
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	9	0.474	8	0.471	17	0.472
Udder Oedema	1	0.053	0	0.000	1	0.028
Miscellaneous conditions						
Population at risk[3]	23		17		40	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	1	0.004	1	0.005	2	0.004
Respiratory disease	0	0.000	1	0.005	1	0.002
Lameness	3	0.011	1	0.005	4	0.008
'Pink eye'	1	0.004	0	0.000	1	0.002
Cystic ovary	0	0.000	0	0.000	0	0.000
All other	3	0.011	2	0.010	5	0.010

[1]Total calved. [2]Cumulative incidence. [3]Monthly average numbers. [4]Incidence rate - cases per animal month at risk.

Table 2-16: Disease incidence rates Control Farm 1997 – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 38[3])	2	0.053
1 day – 1 month (PAR = 36[4])	Cases	CI
Deaths	0	0.000
Diarrhoea	1	0.028
Respiratory disease	0	0.000
Navel/Joint Ill	0	0.000
All other	0	0.000
1 month – 1 year (PAR = 27)[5]	Cases	IR[6]
Deaths	0	0.000
Diarrhoea	6	0.019
Respiratory disease	0	0.000
Lameness	0	0.000
All other	0	0.000
Over 1 year (PAR = 34)[5]	Cases	IR
Deaths	0	0.000
Respiratory disease	0	0.000
Diarrhoea	1	0.002
Lameness	1	0.002
All other	0	0.000

[1]Cumulative incidence. [2]PAR = population at risk. [3]Total calved [4]Calved and alive at six hours. [5]Monthly average. [6]Incidence Rate - cases per animal month at risk.

Table 2-17: Disease incidence rates Control Farm 1998 (January – March) - Cows.

Parturient/lactation Conditions	Askeaton-origin cows		VRL-origin cows		All cows	
Population at risk[1]	6		11		17	
	Cases	CI[2]	Cases	CI	Cases	CI
Abortion	0	0.000	0	0.000	0	0.000
Difficult calving	0	0.000	0	0.000	0	0.000
Milk Fever	0	0.000	0	0.000	0	0.000
Downer cow	0	0.000	0	0.000	0	0.000
Retained Placenta	0	0.000	0	0.000	0	0.000
Vulval discharge/metritis	1	0.167	0	0.000	1	0.059
Ketosis	0	0.000	0	0.000	0	0.000
Tetany	0	0.000	0	0.000	0	0.000
Mastitis	4	0.667	4	0.364	8	0.471
Udder Oedema	0	0.000	0	0.000	0	0.000
Miscellaneous conditions						
Pop. At risk[3]	23		15		38	
	Cases	IR[4]	Cases	IR	Cases	IR
Deaths	0	0.000	0	0.000	0	0.000
Respiratory disease	0	0.000	0	0.000	0	0.000
Lameness	0	0.000	0	0.000	0	0.000
Cystic ovary	0	0.000	0	0.000	0	0.000
All other	1	0.010	0	0.000	1	0.005

[1]Total calved. [2]Cumulative incidence. [3]Monthly average numbers. [4]Incidence rate - cases per animal month at risk.

Table 2-18: Disease incidence rates Control Farm 1998 (January – March) – Calves and growing stock

	Cases	CI[1]
Perinatal deaths (PAR[2] = 19[3])	1	0.053
1 day – 1 month (PAR = 18[4])	Cases	CI
Deaths	0	0.000
Diarrhoea	0	0.000
Respiratory disease	0	0.000
Navel/Joint Ill	0	0.000
All other	0	0.000
1 month – 1 year (PAR = 9)[5]	Cases	IR[6]
Deaths	0	0.000
Diarrhoea	0	0.000
Respiratory disease	0	0.000
Lameness	0	0.000
All other	0	0.000
Over 1 year (None in 1998)	NA[7]	NA

[1]Cumulative incidence [2]PAR = population at risk.[3]Total calved [4]Calved and alive at six hours.[5]Monthly average.[6]Incidence Rate - cases per animal month at risk. [7]Not available.

Table 2-19: Cow culling rates - Index Farm A 1996 - 1998

		Askeaton-origin		Abbotstown-origin		All	
1996							
Cow Population[1]		14		13		27	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	0.0	4[2]	30.8	4	14.8
Disease		2	14.3	3	23.1	5	18.5
Other		2[3]	14.3	0	0.0	2	7.4
	Total	4	28.6	7	53.8	11	40.7
1997							
Cow Population[4]		13		18		31	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	0.0	4	22.2	4	12.9
Disease		1	7.7	2	11.1	3	9.7
Other		1	7.7	0	0.0	1	3.2
	Total	2	15.4	6	33.3	8	25.8
1998 (Jan – Jun)							
Cow Population[4]		12		12		24	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	0.0	0	0	0	0.0
Disease		1	8.3	0	0	1	4.2
Other		0	0.0	0	0	0	0.0
	Total	0	0.0	0	0	0	0.0

[1]No. cows on farm on 1 April 1996 [2]All of these Abbotstown-origin cows also had a history of mastitis [3]One cow transferred to Abbotstown for pairing in Immunology Project [4]No cows on farm on 1 January 1997 or 1998.

Table 2-20: Cow culling rates - Index Farm B 1996 - 1998

		Indigenous cows		Brought-in cows		All cows	
1996							
Cow Population		31[1]		21[2]		52	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	0.0	0	0	1	1.9
Disease		2	6.5	0	0	9	17.3
Other		10	32.3	0	0	2	3.8
	Total	12	38.7	0	0	12	23.1
1997							
Cow Population		19[3]		36[2]		55	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		2	10.5	3	8.3	5	9.1
Disease		0	0.0	0	0.0	0	0.0
Other		0	0.0	0	0	0	0
	Total	2	10.5			5	
1998 (Jan – Jun)							
Cow Population		17[3]		37[2]		54	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	-	2	5.4	-	-
Disease		0	-	1	2.7	-	-
Other		10[4]	-	1	2.7	-	-
	Total			4	10.8	-	

[1]Number of indigenous cows on farm on 1 April 1996 [2]All cows brought-in in 1996 or 1997. [3]Number of indigenous cows on farm on 1 January 1997 or 1998. [4]De-stocking at end of Project

Table 2-21: Cow culling rates – Control Farm 1996 - 1998

		Indigenous cows		Brought-in cows		All cows	
1996							
Cow Population		23[1]		26[2]		49	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	0.0	0	0	0	0.0
Disease		1	4.3	0	0	1	2.0
Other		1	4.3	72	26.9	8	16.3
	Total	**2**	**8.7**	**7**	**26.9**	**9**	**18.4**
1997							
Cow Population		23[3]		17[2]		40	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	0	0	0	0	0
Disease		0	0	3	17.6	3	17.6
Other		0	0	1	5.8	1	5.8
	Total	**0**	**0**	**4**	**23.5**	**4**	**23.5**
1998 (Jan – Mar)							
Cow Population[4]		23		15		38	
		Cows	Rate (%)	Cows	Rate (%)	Cows	Rate (%)
Infertility		0	0	0	0	0	0
Disease		0	0	0	0	0	0
Other		0	0	0	0	0	0
	Total	**0**	**0**	**0**	**0**	**0**	**0**

[1] Number of indigenous cows on farm on 1 April 1996. [2] All cows brought-in in 1996 or 1997. [3] Number of indigenous cows on farm on 1 January 1997. [4] Number of indigenous or brought-in cows on farm on 1 January 1998.

Table 2-22: Monthly mean milk production (kg/day) – Index Farm A

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton			16	22	20	19	17	16	13	12	13	12
	No. cows			7	41	51	60	55	55	55	51	46	46
	Brought-in			2.5	18	16	15	16	16	14	14	13	11
	No. cows			8	46	46	37	55	55	60	51	51	55
1997	Askeaton	10	13	16	18	17	14	14	12	12	12	8.7	9.7
	No. cows	32	41	41	46	55	55	55	55	37	32	28	18
	Brought-in	11	14	18	24	21	15	16	13	11	11	8.7	7.8
	No. cows	51	55	41	60	78	78	74	74	69	64	51	28

Table 2-23: Mean monthly milk protein concentration (g/l) – Index Farm A

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton			3.3	3.1	3.2	3.1	3.1	3.3	3.7	3.5	3.5	3.2
	No. cows			7	9	11	13	12	12	12	11	10	10
	Brought-in			3.3	3.3	3.5	3.4	3.1	3.3	3.6	3.3	3.4	3.3
	No. cows			8	10	10	8	12	12	13	11	11	12
1997	Askeaton	3.3	3.1	3.2	3.1	3.6	3.2	3.2	3.3	3.6	3.3	3.2	3.1
	No. cows	7	9	9	10	12	12	12	12	8	7	6	4
	Brought-in	3.4	3.3	3.2	3.2	3.1	3.2	3.2	3.4	3.6	3.6	3.3	3.3
	No. cows	11	12	9	13	17	17	16	16	15	14	11	6

Table 2-24: Mean monthly milk somatic cell counts (x10³/l) – Index Farm A

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton			182	264	177	203	319	389	317	186	343	241
	No. cows			7	9	11	13	12	12	12	11	10	10
	Brought-in			585	991	811	571	162	284	441	147	419	339
	No. cows			8	10	10	8	12	12	13	11	11	12
1997	Askeaton	217	501	110	115	409	229	218	253	205	179	110	191
	No. cows	7	9	9	10	12	12	12	12	8	7	6	4
	Brought-in	167	113	47	45	169	148	102	171	151	170	154	228
	No. cows	11	12	9	13	17	17	16	16	15	14	11	6

Table 2-25: Monthly mean milk production (kg/day) – Index Farm B

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton			19	19	22	18	18	15	14	13	12	10
	No. cows			64	74	83	83	83	92	69	64	55	55
	Brought-in						18	17	14	11	10	11	12
	No. cows						87	97	83	92	78	64	55
1997	Askeaton	12	13	20	24	23	22	20	15	13	13	13	12
	No. cows	37	28	23	23	37	41	41	41	41	37	23	23
	Brought-in	15	19	23	24	22	19	18	13	13	12	11	10
	No. cows	37	78	83	124	143	143	143	138	133	133	97	51
1998	Askeaton	25	26		26	22	25	20	20	13	6		
	No. cows	14	23		28	37	37	37	37	37	28		
	Brought-in	21	25		23	24	24	18	18	12	7.8		
	No. cows	41	64		120	152	152	152	147	147	133		

Table 2-26: Mean monthly milk protein concentration (g/l) – Index Farm B

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton			3.2	3.1	3.2	3.2	3.0	3.3	3.4	3.5	3.7	3.5
	No. cows			14	16	18	18	18	20	15	14	12	12
	Brought-in						3.2	3.1	3.4	3.6	3.8	3.9	3.5
	No. cows						19	21	18	20	17	14	12
1997	Askeaton	3.4	3.6	3.3	3.4	3.3	3.3	3.2	3.3	3.7	3.7	3.4	3.5
	No. cows	8	6	5	5	8	9	9	9	9	8	5	5
	Brought-in	3.6	3.3	3.1	3.2	3.2	3.2	3.2	3.2	3.6	3.7	3.5	3.6
	No. cows	8	17	18	27	30	31	31	30	29	29	21	11
1998	Askeaton	3.3	3.0		3.1	3.6	3.3	3.3	3.3	3.9	4.5		
	No. cows	3	5		6	8	8	8	8	8	6		
	Brought-in	3.7	3.1		3.3	3.3	3.3	3.3	3.4	3.7	4.2		
	No. cows	9	14		26	33	33	33	32	32	29		

Table 2-27: Mean monthly milk somatic cell counts (x10³/l) – Index Farm B

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton			532	438	817	826	398	682	282	335	76	507
	No. cows			14	16	18	18	18	20	15	14	12	12
	Brought-in						125	123	428	270	198	102	413
	No. cows						19	21	18	20	17	14	12
1997	Askeaton	184	131	184	179	197	196	342	253	246	355	97	92
	No. cows	8	6	5	5	8	9	9	9	9	8	5	5
	Brought-in	548	265	212	118	120	179	229	205	198	416	184	185
	No. cows	8	17	18	27	31	31	31	30	29	29	21	11
1998	Askeaton	62	24		129	100	57	71	109	179	990		
	No. cows	3	5		6	8	8	8	8	8	6		
	Brought-in	671	155		141	180	169	133	194	198	521		
	No. cows	9	14		26	33	33	33	32	32	29		

Table 2-28: Monthly mean milk production (kg/day) – Control Farm

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton				20	22	23	20	17	16	14	13	13
	No. cows				23	28	46	51	60	60	55	55	51
	Brought-in				23	20	23	21	15	16	16	15	16
	No. cows				41	51	60	64	60	55	46	51	41
1997	Askeaton	12	16	18	22	23	21	18	16	16	12	13	15
	No. cows	41	41	51	55	74	74	74	69	69	83	78	41
	Brought-in	16	21	22	25	26	23	19	17	17	13	15	18
	No. cows	32	28	46	74	64	64	64	60	60	83	78	55
1998	Askeaton	15	18	19	16								
	No. cows	41	69	60	60								
	Brought-in	18	23	24	23								
	No. cows	32	60	83	83								

Table 2-29: Mean monthly milk protein concentration (g/l) – Control Farm

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton				3.5	3.5	3.4	3.2	3.2	3.4	3.7	3.4	3.3
	No. cows				5	6	10	11	13	13	12	12	11
	Brought-in				3.4	3.5	3.5	3.3	3.3	3.5	3.7	3.4	3.2
	No. cows				9	11	13	14	13	12	9	11	9
1997	Askeaton	3.2	3.3	3.2	3.3	3.3	3.3	3.3	3.3	3.6	4.1	3.4	3.2
	No. cows	9	9	11	12	16	16	16	15	13	18	17	9
	Brought-in	3.3	3.1	3.2	3.3	3.3	3.3	3.4	3.3	3.6	3.9	3.4	3.2
	No. cows	7	6	10	16	14	14	14	13	11	18	17	12
1998	Askeaton	3.2	3.3	3.1	3.4								
	No. cows	9	15	13	13								
	Brought-in	3.3	3.3	3.3	3.2								
	No. cows	7	13	18	18								

Table 2-30: Mean monthly milk somatic cell counts (x10^3/l) – Control Farm

Year	Origin	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	Askeaton				1570	1654	394	373	992	576	310	211	163
	No. cows				5	6	10	11	13	13	12	12	11
	Brought-in				1634	683	844	427	710	373	245	387	399
	No. cows				9	11	13	14	13	12	10	11	9
1997	Askeaton	193	236	385	194	289	423	662	693	985	935	1052	198
	No. cows	9	9	11	12	16	16	16	15	15	18	17	9
	Brought-in	618	414	1529	400	242	355	227	321	351	663	633	227
	No. cows	7	6	10	16	14	14	14	13	13	18	17	12
1998	Askeaton	90	107	125	441								
	No. cows	9	15	13	13								
	Brought-in	126	89	145	199								
	No. cows	7	13	18	18								

Table 2-31: Silage analysis results for Monitor Farms – 1996/97

			Farm A	Farm B	Control
Year	Cut	Type	DMD	DMD	DMD
1996	1st cut	pit	65	71	74
	2nd cut	bag	70		
1997	1st cut		67	64	74
	1st cut			74	
	2nd cut	bag	66	66	

Table 2-32: Cow group mean monthly weights (kg) and condition scores - Index Farm A 1996.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Ask-origin												
Weight (n)					490 (4)	474 (13)	495 (14)	522 (16)	495 (15)	535 (16)		533 (30)
Condition (n)		2.3 (13)			2 2 (12)				2.7 (15)		2.5 (115)	
VRL												
Weight (n)						527 (10)	604 (11)	602 (22)	559 (20)	595 (22)		583 (35)
Condition (n)		2.2 (10)			2.2 (10)				3 2 (20)		2.3 (18)	

Table 2-33: Cow group mean monthly weights (kg) and condition scores - Index Farm A 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Ask-origin												
Weight (n)	526 (13)	520 (13)	518 (13)	494 (13)	496 (13)	502 (13)		493 (12)	545 (11)	569 (12)	557 (12)	567 (12)
Condition(n)	2.3 (13)	2.1 (13)	2 2 (13)	2 3 (13)	2.3 (13)	2 4 (13)		2 4 (12)	2.6 (12)	2.6 (12)	2.6 (12)	2 7 (12)
VRL												
Weight (n)	571 (18)	585 (18)	587 (18)	545 (18)	533 (18)	547 (18)		545 (20)	581 (18)	602 (18)	577 (17)	591 (17)
Condition(n)	2.0 (18)	1.9 (18)	1.9 (18)	2.1 (18)	2.0 (18)	2.0 (18)		2.0 (20)	2.3 (18)	2 3 (18)	2 2 (17)	2.4 (17)

Table 2-34: Cow group mean monthly weights (kg) and condition scores - Index Farm A 1998.

	Jan	Feb	Mar
Ask-origin			
Weight (n)		579 (10)	532 (10)
Condition (n)	2.9 (10)	3.1 (10)	2.7 (10)
VRL			
Weight (n)		617 (12)	602 (11)
Condition (n)	2.8 (12)	2 9 (12)	2 6 (11)

Table 2-35: Mean monthly calf weights (kg) and average daily gain - Index Farm A 1996 & 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996												
Weight (n)				57 (6)	92 (13)		134 (13)	155 (13)	175 (13)	200 (13)	230 (13)	232 (13)
ADG (kg)					0 9		1.6	0 7	0 6	0 8	1.0	0.1
1997												
Weight (n)		52 (8)	63 (9)	72 (13)	82 (16)	123 (13)		171 (26)	206 (13)	228 (13)	230 (13)	254 (11)
ADG (kg)			0.5	0.8	0.9	1.1		0 8	1.2	0.7	0.1	1.2

Table 2-36: Yearling mean monthly weights (kg) and average daily gain - Index Farm A 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Weight (n)	256 (13)	276 (13)	299 (13)	334 (13)	360 (13)	399 (13)		424 (26)	455 (22)	460 (29)		
ADG (kg)		0 7	0 8	1.2	0.9	1 3		0 8	1 2	0 4		

Table 2-37: Cow group mean monthly weights (kg) and condition scores - Index Farm B 1996.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Indigenous												
Weight (n)		575 (7)	571 (22)	572 (10)	532 (19)		541 (21)	548 (29)	554 (21)			589 (21)
Condition (n)		3 5 (30)			3.2 (30)				3.0 (21)		2.5 (21)	
Brought-in												
kg (n)					538 (17)		522 (21)	522 (21)	552 (20)			609 (25)
Score (n)					2 9 (7)				2 5 (21)		2 2 (21)	2 4 (17)

Table 2-38: Cow group mean monthly weights (kg) and condition scores - Index Farm B 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Indigenous												
Weight (n)	552 (3)	621 (19)	591 (16)	576 (17)	573 (17)	568 (16)	575 (15)	576 (17)	585 (16)	592 (17)	606 (17)	612 (17)
Condition (n)	2.9 (19)	2.9 (17)	2 9 (16)	2.9 (17)	2.7 (17)	2.9 (17)	3.0 9 17)	2 9 (17)	2 9 (17)	2 7 (17)	2 8 (17)	2.9 (17)
Brought-in												
kg (n)	579 (3)	624 (29)	591 (25)	528 (33)	543 (33)	527 (30)	542 (21)	551 (33)	566 (32)	574 (32)	606 (32)	610 (32)
Score (n)	2.5 (28)	2.5 (24)	2 2 (26)	2 1 (33)	1 9 (33)	2 1 (32)	2.0 (33)	2.1 (33)	2.2 (32)	2 2 (32)	2.3 (32)	2 4 (32)

Table 2-39: Cow group mean monthly weights (kg) and condition scores - Index Farm B 1998.

	Jan	Feb	May
Indigenous			
Weight (n)	598 (17)		577 (12)
Condition (n)	3 0 (17)	3.0 (12)	3 0 (12)
Brought-in			
kg (n)	588 (37)		536 (35)
Score (n)	2.6 (37)	2.6 (32)	2 2 (35)

Table 2-40: Mean monthly calf weights (kg) and average daily gain - Index Farm B 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Weight (n)					96 (21)	117 (22)	140 (20)	185 (18)	184 (18)	233 (14)	240 (34)	
ADG (kg)					1.3	0 8	0 8	0 9	0.9	1.3	0.5	

Table 2-41: Mean monthly steer weights (kg) and average daily gain - Index Farm B 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Weight (n)	385 (12)	400 (12)	412 (12)	446 (12)	476 (12)	503 (12)	547 (12)	579 (12)	603 (12)	607 (12)	597 (12)	
ADG (kg)		0.6	0.5	0.3	1.1	1.2	1.0	0.9	1 1	0.6	0.1	-0.8

Table 2-42: Comparison of mean serum βHB (mmol/l) and glucose (mmol/l) concentrations for Index Farm B for 1997 vs 1998 at 30-60 days post calving.

	βHB	p[1]	Glucose	p[1]
1997	1.13 ± 0 70[2] (23)[3]		3 15 ± 0 44 (23)	
1998	0.63 ± 0.17 (24)	0.002	3.47 ± 0.32 (24)	0 008

[1]Significance of difference between Farm A or B and Control. [2]Standard deviation. [3]Number samples.

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Table 2-43: Cow group mean monthly weights (kg) and condition scores – Control Farm 1996.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Ask-origin												
Weight (n)				511 (14)	494 (17)	467 (15)		550 (17)	565 (16)	561 (18)	573 (18)	572 (19)
Condition (n)		2.9 (11)			3.1 (12)				3 7 (19)			
VRL												
Weight (n)				631 (20)	622 (16)	617 (14)		634 (19)	638 (20)	644 (18)	663 920)	631 (20)
Condition (n)		3.2 (7)			3.2 (12)				3.5 (19)			

Table 2-44: Cow group mean monthly weights (kg) and condition scores - Control Farm 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Ask-origin												
Weight (n)				511 (14)	494 (17)	467 (15)		550 (17)	565 (16)	561 (18)	573 (18)	572 (19)
Condition (n)	3.6 (18)		3.4 (18)	3 8 (18)	3.6 (18)				3.6 (18)		3.3 (18)	
VRL												
Weight (n)				631 (20)	622 (16)	617 (14)		634 (19)	638 (20)	644 (18)	663 (20)	631 (20)
Condition (n)	3.4 (17)		2.8 (17)	3.1 (17)	3.2 (14)				3.3 (20)		2.8 (14)	

Table 2-45: Cow group mean monthly weights (kg) and condition scores - Control Farm 1998.

	Jan	Feb	Mar
Ask-origin			
Weight (n)	535 (17)	517 (18)	487 (17)
Condition (n)			2 8 (18)
VRL			
Weight (n)	604 (14)	560 (17)	488 (16)
Condition (n)			2 4 (17)

Table 2-46: Mean monthly calf weights (kg) – Control Farm 1996 and 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996												
Weight (n)				31 (7)	47 (15)	58 (16)	52 (3)		96 (5)			170 (21)
1997												
Weight (n)	77 (8)	67 (17)		75 (36)		111 (41)	134 (41)		199 (41)	181 (8)		236 (43)

Table 2-47: Mean monthly yearling weights (kg) and average daily gain – Control Farm 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Weight (n)	200 (21)	220 (22)		268 (22)		305 (22)	NA[1]	345 (10)	363 (23)	401 (11)		375 (8)
ADG (kg)		0.66		0 82		0.63		1.5	0 58	1.29		-0.4

Table 2-48: Mean monthly steer weights (kg) and average daily gain – Control Farm 1997.

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Weight (n=12)	372	377		435	497	NA[1]	NA[1]		537	556		577
ADG (kg)	1.4	0.1		1.0	2.1				1.1	0.6		0.3

Table 2-49: Comparison of mean serum βHB (mmol/l) and glucose (mmol/l) concentrations for the three Monitor Farms at 30-60 days post calving 1997.

	βHB	p[1]	Glucose	p[1]
Farm A	$0.87 \pm 0.51^2 (24)^3$	0.02	3.25 ± 0.39 (24)	0.004
Farm B	0.98 ± 0.62 (20)	0.005	3.20 ± 0.37 (20)	0.001
Control	0.61 ± 0.23 (29)		3.53 ± 0.30 (29)	

[1] Significance of difference between Farm A or B and Control. [2] Standard deviation. [3] Number samples.

Table 2-50: Index Farm A fertility - 1996 to 1998

	1996	1997	1998[1]
No. of Calvings	24	21	25
Calving spread (months)	12[2]	4[3]	4[3]
Submission rate (%)	40	79	69
Non-detected oestrus (%) (60-days)	82	70	52
Heat detection rate (%)[4]	53	68	86
Calve to first service (days)	108	67	60
Calve to conception (days)	114	92[5]	NA[6]
No. cows served	19	32	NA
Conception rate to all services (%)	34	42	NA
Services per conception	2.9	2.4	NA
No. conceived	11	26	NA
Fertility rate (%)	58[7]	81	NA

[1]Herdowner-supplied data [2]Includes cows moved from year-round calving herd (Abbotstown). [3]All cows. [4]21 divided by average interval between heats x 100. [5]Cows calved 1997. [6]Not available [7]For cows served in Askeaton in 1996

Table 2-51: Index Farm B fertility - 1996 to 1998

	1996	1997	1998
No. of calvings	34	36	49
Calving spread (months)	8	5	4
Submission rate (%)	44	NA[1]	79
Non-detected oestrus (%) (60-days)	56	NA[1]	29
Heat detection rate (%)[2]	56	34	70
Calve to first service (days)	47	NA[1]	64
Calve to conception (days)	66	NA[1]	84
No. cows served	33[3]	40	42
Conception rate to all services (%)	32	52	54[4]
Services per conception	3.2	1.8	1.9
No. conceived	21	36	37
Fertility rate (%)	64	90	88

[1]Not available for 1997 owing to high rate of induced heats (see text). [2]21 divided by average interval between heats x 100 [3]Excludes services on nine cows before arrival. [4]May be overestimate due to unrecorded services when bull running with cows.

Table 2-52: Control Farm fertility - 1996 to 1998

	1996	1997	1998
No. of calvings	41	43	18
Calving spread (months)	12	12	NA
Non-detected oestrus (%) (60-days)	NA[1]	NA[1]	ND[2]
Submission rate (%)	NA[1]	NA[1]	ND
Heat detection rate (%)[3]	NA[1]	NA[1]	ND
Calve to first service (days)	NA[1]	NA[1]	ND
Calve to conception (days)	NA[1]	NA[1]	ND
No. cows served	41	34	ND
Conception rate to all services (%)	49	54	ND
Services per conception	2	1.9	ND
No. conceived	37	32	ND
Fertility rate (%)	90	94	ND

[1] Not applicable owing to high rate of induced heats (see text). [2]Not done – project concluded March 1998. [3]21 divided by average interval between heats x 100.

Figure 2-1: Cow body condition score and weight for periparturient period 1997.
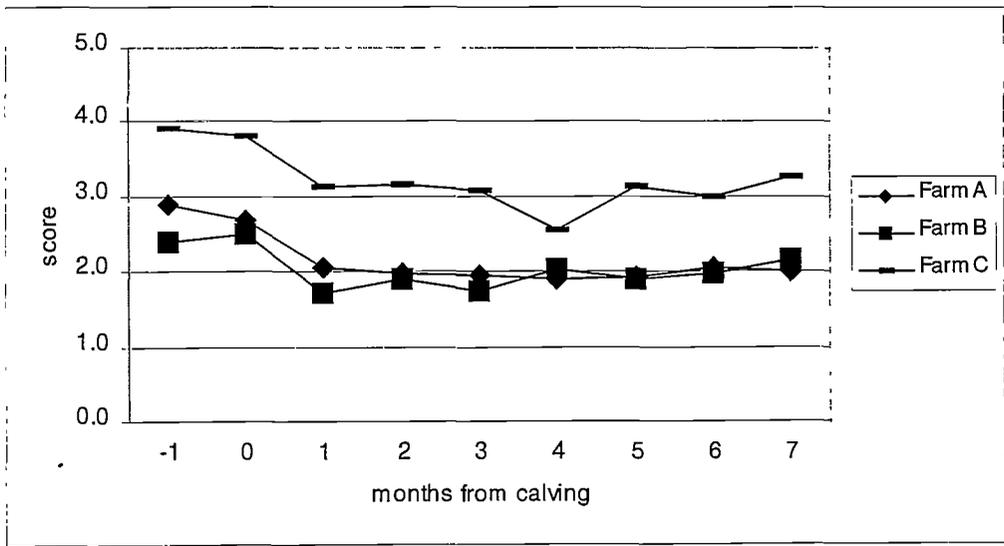
Farm A = Index Farm A, Farm B = Index Farm B, Farm C = Control Farm

Figure 2-2: Milk protein to fat ratios for periparturient period 1997.
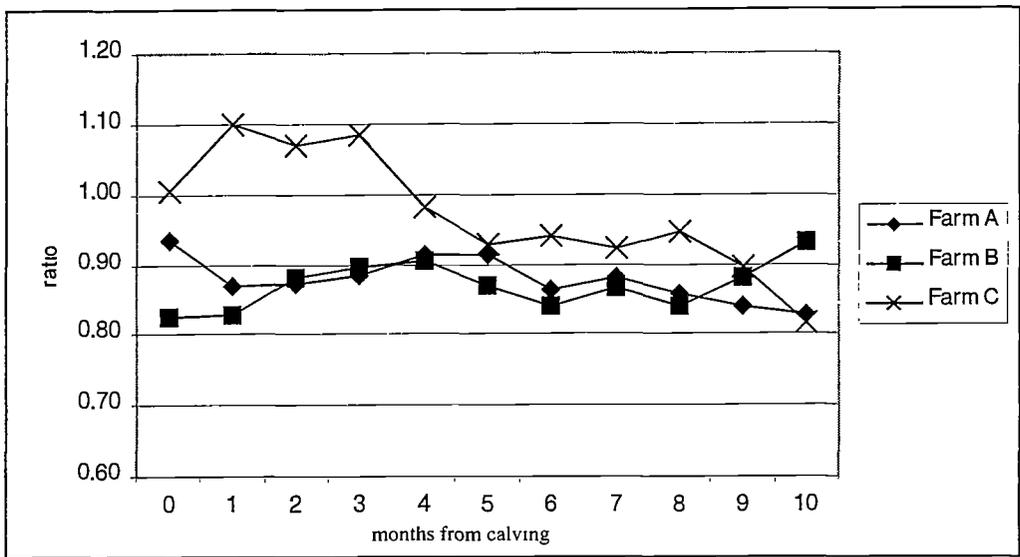
A = Index Farm A, B = Index Farm B, C = Control Farm

Figure 2-3: Mean monthly red blood cell counts for three monitor farms – April 1996 to March 1988
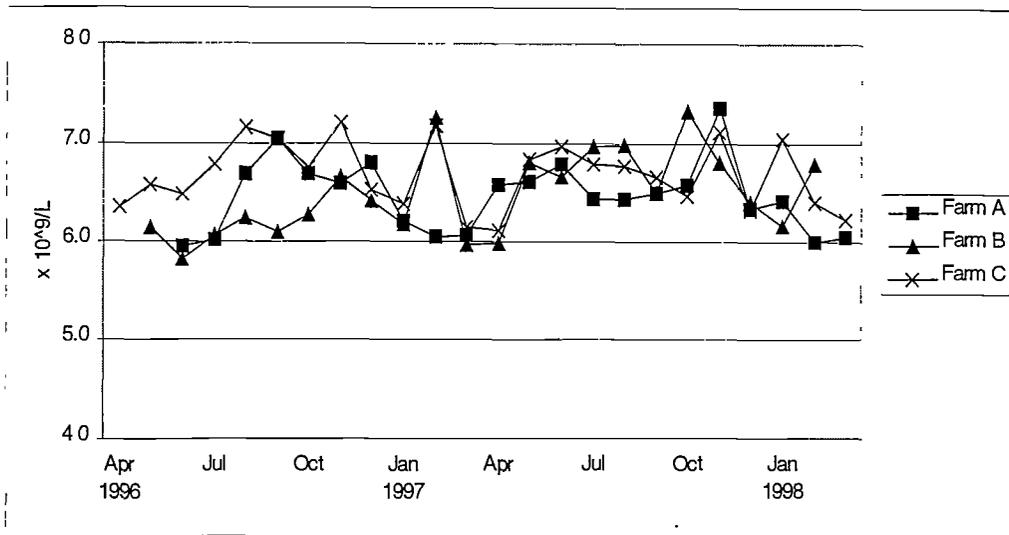
A = Index Farm A, B = Index Farm B, C = Control Farm

Figure 2-4: Mean monthly blood urea concentrations for three monitor farms – April 1996 to March 1988
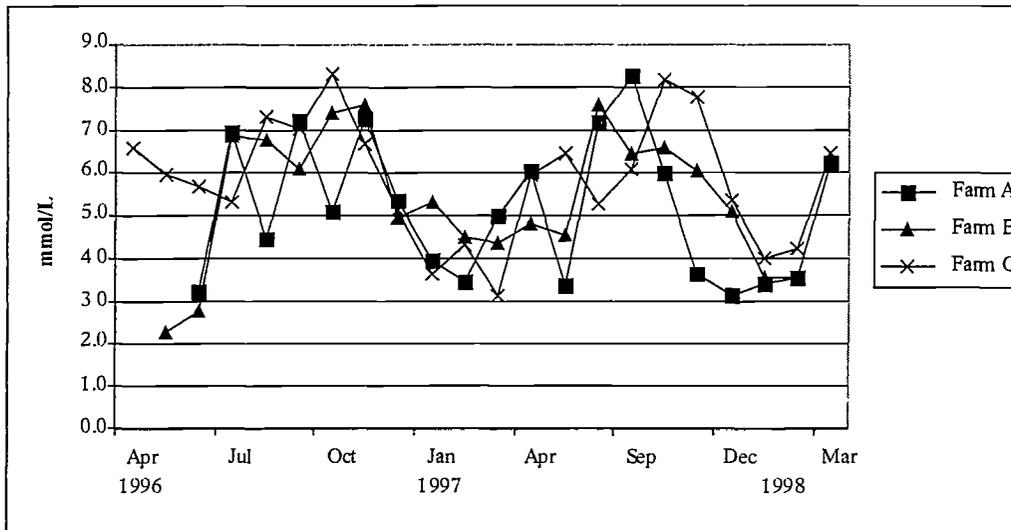
A = Index Farm A, B = Index Farm B, C = Control Farm.

Figure 2-5: Mean monthly blood βHB concentrations for Monitor Farms – April '96 to March '98
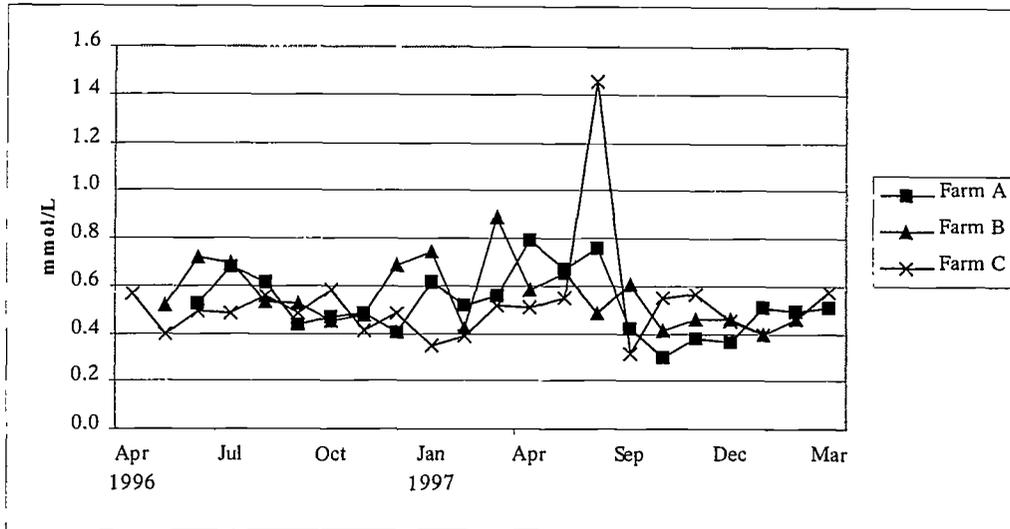
A = Index Farm A, B = Index Farm B, C = Control Farm.

Figure 2-6: Mean monthly blood GLDH concentrations for Control Farm – 1997
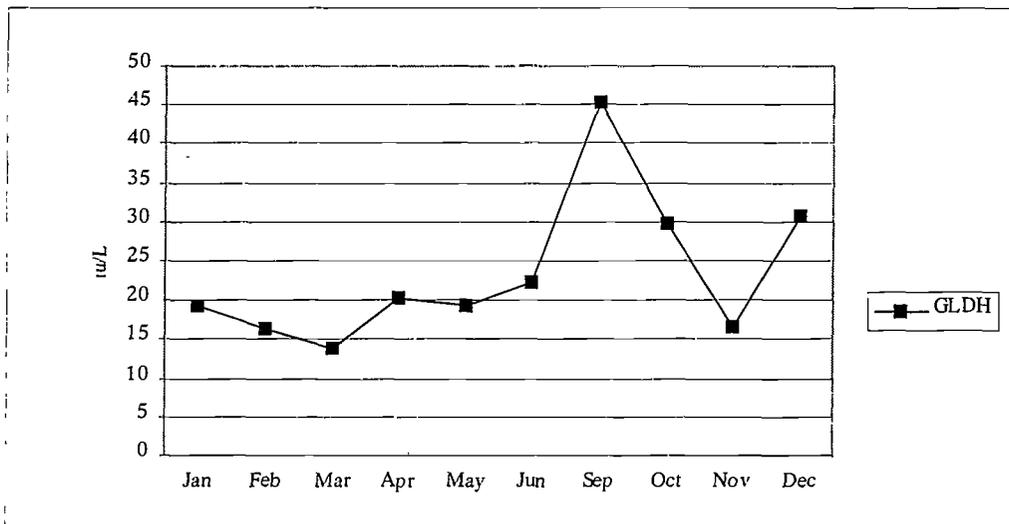
CHAPTER THREE

LONGITUDINAL STUDY ON FOUR FARMS

Following identification and investigation of the two Index Farms, a wider study was initiated to investigate claims that other farms in the Askeaton area had experienced an excess of animal disease. This comprised a Retrospective Survey, which is described in Chapter Five, and involved the identification and investigation of other farms whose herdowners considered they had an excess of animal health problems.

It was originally envisaged that a decision regarding further prospective longitudinal studies of animal health and production would be made in the light of the results of the Retrospective Survey (EPA, 1995). However, delays in the compilation of data collected in the Retrospective Survey meant that a decision had to be made regarding prospective studies on selected farms before any conclusions could be drawn from the results of the Retrospective Survey.

As originally outlined in the EPA Interim Report (EPA, 1995), three categories of farms were to be selected for a Longitudinal Study, i.e. · four 'problem' herds in the Askeaton area, four non-problem herds in the Askeaton area and four 'normal' herds from outside the area. However, for the following reasons this approach was not adopted:

1. Advice received at the time indicated that the numbers of farms involved in such a comparative study would have been insufficient to ensure statistical validity.

2. The study design might be too narrowly focused to deal with the relatively wide range of conditions which were identified on the 'problem' farms.

3. The resources required for such a study on 12 farms in two separate areas of the country (i.e. Askeaton and control areas) would be considerable.

4. If such a comparative study were undertaken, additional resources would not have been available for investigation of remaining 20 'problem' herds which were not selected for inclusion in a longitudinal study.

It was decided, therefore, to pursue an alternative approach which involved a Longitudinal Study of animal health and production on five of the problem herds in the Askeaton area, combined with an appropriate level of investigation of the disease problems on the remaining 20 farms. The results of investigations carried out on the farms which were not selected for inclusion in the Longitudinal Study are included with the individual farm analyses of the Retrospective Survey (Chapter Five). The present Chapter describes the design and outcome of the Longitudinal Study of animal health and production on the selected 'problem' farms. Analysis of the historical animal health problems on these farms is also presented in Chapter Five.

Five farms were selected according to the procedures and criteria outlined in the Longitudinal Study Protocol (EPA 1996, unpublished). In July 1996, arrangements for the implementation of the Longitudinal Study were drawn up and agreed in consultation with the veterinary advisor to the Askeaton and Ballysteen Animal Health Committee. The Veterinary Investigation Team also met the Animal Health Committee to present details of the proposed study. In August 1996, the Veterinary Investigation Team met the selected herdowners to discuss their participation in the study. One of the originally selected herdowners declined to participate in the study and a replacement was selected according to the procedures and criteria previously used. Following agreement of the herdowners regarding details of participation, farm visits commenced in October, 1996.

One of the herdowners (Farm T) voluntarily withdrew from the study in spring 1997. As insufficient data were collected to allow meaningful analysis, this herd is not included in the following report of the study results. However, a number of disease problems were investigated on this farm. An account of these is included in the analysis of this farm (Farm ID 08) for the Retrospective Survey (Chapter Five).

The objectives of the Longitudinal Study were to determine the nature and incidence of animal health and production problems experienced on the participating farms and to investigate the immediate and underlying causes. In addition, it

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was hoped that the availability of contemporaneous information on a range of production and animal health parameters would assist investigation and identification of underlying factors contributing to the occurrence of any unusual conditions encountered or to an unusually high incidence of other more common conditions.

Farms in the Longitudinal Study were visited at least monthly by the project clinician to carry out animal health inspections and to collect records. At the start of the study, a group of monitor animals was selected for quarterly blood-sampling and condition-scoring on each farm. These groups initially comprised six cows and six growing animals. Additions and withdrawls were made from the groups during the study to allow for sales and culling. Details of haematology and biochemistry components which were analysed in blood samples from these animals are given in Appendix 5 and Appendix 6. Other blood samplings and analyses were carried out as required for investigation of specific disease conditions.

In addition to veterinary investigations, analyses of soil, herbage and fodder on the four farms were carried out by Teagasc in 1997. Results of these analyses are reported elsewhere (Soil, Herbage, Feed and Water Volume). A farm management audit was also carried out by Teagasc staff on three of the four participating farms.

DESCRIPTION OF FARMS

In order to maintain anonymity, only summary descriptions are given for each of the four farms participating in the Longitudinal Study. Details of stock numbers or other data which could identify the farms in question are specifically omitted.

In the following analysis, an outline is given of the main animal health and production problems encountered on each of the farms, together with a discussion of the probable or possible causes and contributory factors. Sporadic or individual animal cases of disease are only referred to in so far as they constituted part of an extended problem or were of an unusual nature. Periodic reports on animal health and production were issued to the herdowners and their attending veterinary practitioners throughout the study. Recommendations were also made for control or prevention of specific health problems encountered.

Note re disease incidence results: Tables of disease incidence on the four farms are presented at the end of this chapter Results are based on data provided by the herdowners concerned Incidence rates are given for diseases and classes of cattle where a reasonably accurate estimate of the population at risk (PAR) can be made and where the total number of cases is known In most cases, this applied to peri-parturient and lactational diseases in cows and to diseases of calves up to

about a month old where cumulative incidence rates are given Miscellaneous diseases of cows, as well as diseases of animals in the 1 month to 2 5 year-old age group, are given as annual rates based on a PAR of estimated average animal numbers per month

In relation to outbreaks of disease where the immediate population at risk is only a sub-group of animals on the farm, e.g. outbreak of coughing in a group of calves at grass, and where the duration of the outbreak is not known with any certainty, the number of outbreaks is given in the table and details of numbers of animals affected — where available — are given in the text.

Description of Longitudinal Study Farms

Farm	Type	Size
LS1	Dairy, store	Medium
LS2	Dairy, suckling	Medium large
LS3	Dairy, beef, suckler	Medium
LS4	Dairy, beef, store	Medium
LS5	Dairy, beef, suckler	Large

FARM LS1

The main animal health problems recorded in the Retrospective Survey Report on this farm (Farm 05) were infertility in cows and illthrift in calves and weanlings. The first visit of the Longitudinal Study was at the end of October, 1996. The main problems reported at that time were poor condition in cows, extended calving season due to infertility problems in previous years and illthrift in calves.

Due to failure to reach agreement with the herdowner regarding implementation of the Teagasc farm management audit, it was not possible to encompass the full extent of the Longitudinal Study as envisaged in the Project design.

ANIMAL CONDITION AND NUTRITION

Calves and growing stock

According to the herdowner, illthrift had been a problem in calves and weanlings for some time. Newborn calves were said to be small and failed to thrive - either indoors or on pasture.

The report on the DAF investigation of ill-thrift and stunting on this farm in August 1996 (DAFRD unpublished) noted that while some calves were small, there was no evidence of stunting. At the time of the first visit of the Longitudinal Study in October 1996, no specific disease problems were

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reported in the calves and weanlings. Animals in the group comprised a wide range of sizes and ages due to the extended calving season. Calf and weanling thrive were variable during the 1997 grazing season. Again, the group had a wide age-spread - and included a few late 1996 calves which were small for their age. It was also a feature in that year that the later-born calves performed less well than their older comrades. In 1998, the majority of calves were sold before the summer. Thrive in the five calves reared on grass throughout the summer of 1998 was affected by an outbreak of respiratory disease in June (see below).

There is no doubt that clinical and sub-clinical disease contributed to poor thrive in some growing animals on this farm. An outbreak of respiratory disease was reported to have affected all of a group of 16 housed calves in April 1997 (see below). Cases of chronic respiratory tract inflammation in the calf weanling group, including calves born after the April incident, were investigated on a number of occasions throughout the summer and autumn of 1997. A case of tick-borne fever was diagnosed in the group in September 1997. An outbreak of respiratory disease in calves at grass in June 1998 had a significant impact on their subsequent performance. Further details of these disease incidents are given below.

Another factor which may have had a negative influence on calf health and thrive on this farm is the practice of adding later-born calves to groups of older calves at grass. In summer/autumn 1997, for example, animal ages in the calf/weaner group ranged from about four to over 20 months. Mixing of age-groups in this way is not generally recommended as older animals may act as reservoirs of infection for the younger animals. In addition, later-born calves may receive a high parasite challenge after joining older calves on contaminated pastures. Even with regular anthelminthic treatment, a transient high level of intestinal parasitism can have a negative impact on growth rates. It is significant in this regard that poor thrive on this farm was generally more of a problem in calves born later in the year.

Mixing calves of widely varying ages can also have a negative effect on feed intake. Due to unequal competition for trough space, younger animals may not always receive their intended share of concentrate ration. Low blood copper concentrations recorded in some animals in this group on a number of occasions (see below), together with low iodine concentrations (7.0 to 13.0 µg/l) detected in three yearlings which were with the calf group in June 1997, suggest that concentrate intake was uneven. Had they all

received the recommended allocation of approximately 1 kg per head per day this should have been sufficient to ensure adequate supply of both these elements.

Cows

According to the herdowner, poor body condition in cows had also been a problem for some time. At the initial study visit to the farm in October 1996, about a third of the cows (dry and milking) had body condition scores under 2.0. The remainder had scores of 2.0 - 3.0. While these scores represent a poor performance for the time of year, there was evidence that concentrate supplementation for some cows may have been inadequate to meet production demands. Owing to the fertility problems of previous years, over two-thirds of the cows were still in milk and about a third were in the first half of lactation. At the time, milking cows were on silage and 2.3 kg of concentrates per day. Pregnant dry cows were on hay only.

Following analysis of the 1996 silage crop, which indicated that it was of poor quality (Soil, Herbage, Feed and Water Volume), it was recommended that milking and dry cows should receive substantially increased concentrate rations. It was recommended that milking cows should receive a daily allowance of approximately 2.2 kg concentrates per 4.6 kg of milk produced and dry cows should receive an allowance of 2 to 3 kg depending on condition. These recommendations, which were substantially in excess of the then current and proposed over-winter feeding regimen, were only partially implemented by the herdowner.

A further factor which must be taken into account in relation to the historical problem of poor cow condition on this farm is the practice of out-wintering cows after calving. This is not a common practice in dairy herds in this country owing to variable weather conditions. It is well recognised that inclement weather imposes an additional nutritional stress on cows (Anon, 1989). Failure to meet this requirement can contribute to poor cow condition at calving and severe negative energy balance post-calving. It is significant in this regard that the winters of 1993-94 and 1994-95 - which preceded periods when post-calving anoestrus/sub-oestrus were said to be at their worst (see below) - were wet cold and windy. The breeding seasons of 1996 and 1997, on the other hand, when heat detection was not reported to have been a problem, were preceded by relatively mild winters.

Despite the fact that concentrate supplementation recommendations were not implemented in full by

the herdowner, and allowing for a few cows which remained in poor condition throughout the study, cow condition generally improved over the two years of the study. While individual cases of persistent poor condition may have been disease-related - either clinical or sub-clinical - there was no evidence throughout the study that cow thrive was affected at a herd level by underlying disease problems.

It is not unusual in any herd to have individual cows which may exhibit poor condition either intermittently or persistently. This may be due to nutritional, production, or disease factors which are peculiar to those animals. One cow in this herd, for example, which had a persistently low body condition score, also had raised concentrations of the liver enzyme GLDH throughout 1997. Although this was clearly evidence of liver dysfunction, no specific condition or cause was identified.

In relation to the incidents of respiratory disease reported in cows in 1997 and 1998, and which were said to have followed incidents of atmospheric pollution (see below), there was no evidence that they had a significant impact on cow condition. With the exception of one cow which developed signs of pneumonia, cows' appetites were not said to have been affected. Two of the reported incidents occurred in the summer months (1997 and 1998) when cows were in generally good condition. There was no evidence following these incidents that cows in the affected group were subject to any unusual changes in body condition.

Two other animal health events which the herdowner considered were related to atmospheric pollution were reported in January and February 1998. While it is likely that cow condition would, in any case, have been declining in most cows at this time as part of the normal productive cycle, there is no evidence cows were subject to a more severe decline than would have been expected over the spring of 1998.

ANIMAL HEALTH

Details of recorded disease incidents are given in Table 3-1 and Table 3-2.

Calves and growing stock

The main health problems recorded in calves and young stock were respiratory disease and poor thrive. Two outbreaks of respiratory disease were reported. The first was in April 1997. According to the herdowner, all 16 of a group of housed calves began coughing the day after 'caustic smells' were noticed in the air around the farm. There was no

veterinary or laboratory investigation of this incident and the animals were treated by the herdowner.

Some of these calves, along with older as well as later-born calves, were the subject of veterinary investigations for illthrift and intermittent coughing while at grass during the following summer and autumn. Three animals from the group which were born a month after the April incident, and which had been coughing and showing signs of poor thrive in autumn 1997, still had signs of chronic respiratory disease when examined in January, 1998.

While the only specific diagnosis made in relation to the calf/weaner group in 1997 was a single case of tick-borne fever (see below), haematology results consistent with the presence of inflammatory conditions were noted in blood samples collected on a number of occasions. Although individual animals had serum antibody titres to virus pathogens, there was no specific indication that virus infection contributed to the respiratory problems in 1997.

Marginal copper deficiency may have had a limiting effect on calf thrive at times. Almost a third of blood samples collected from the calf/weanling group during the summer and autumn of 1997 had copper concentrations below 9.0 μmol/l (normal range 9.4 - 24 μmol/l). Although it has been the practice on this farm to administer copper to calves and weanlings at intervals, the method used, i.e. oral drench, provides only short-term cover. While recommendations regarding the use of long-acting depot preparations were made to the herdowner during the Study, the practice of oral drenching was continued throughout.

A case of tick-borne fever (TBF) was also diagnosed in the calf/weanling group in September 1997. This is a chronic condition, the causative agent of which is spread by ticks. Animals affected for some time would be expected to show signs of illthrift. While the agent of TBF was only identified in one of six animals sampled, diagnosis is generally difficult, and demonstration of its presence in one animal in the group raises the distinct possibility that others were infected.

The only significant disease outbreak recorded in calves in 1998 was coughing in a group of five calves at grass in June. Clinical examination of the calves confirmed the presence of a moderately severe respiratory tract inflammation. Haematology findings were consistent with a chronic respiratory tract inflammation with infectious involvement.

The animals showed a good response to antibiotic therapy - though according to the herdowner subsequent growth rates were lower than expected. According to the herdowner, this outbreak was related to an incident two weeks previously which he considered was atmospheric pollution – but which had not been associated with any clinical signs of illness at the time.

The only deaths recorded in the first year of the Study (November 1996 to end 1997) were a stillborn calf and a one-and-a-half year-old heifer which had been severely growth-retarded from an early age. Three calves and a one-and-a-half year old heifer died in 1998. The first two calf deaths in 1998 occurred within a short time of birth - one following a difficult delivery from a cow with severe mastitis. The third calf death was due to a congenital cardiac anomaly. The death of the heifer was due to injuries she sustained after breaking into a neighboring field.

Cows

Significant disease incidents in the cows in 1997 and 1998 were also largely respiratory in nature. An outbreak of coughing involving most of the cows was investigated in June 1997. According to the herdowner, this was a direct result of their exposure to atmospheric pollution which he had noted two days earlier. At the time of the veterinary visit to the farm, most of the cows exhibited coughing. Evidence of upper respiratory tract inflammation was confirmed on clinical examination of a number of cows. However, they appeared otherwise healthy and haematological and biochemical analysis of blood samples collected showed no evidence of a significant systemic inflammatory response. With the exception of one cow which developed signs of pneumonia two weeks later, all of the cows recovered uneventfully and without treatment. No specific diagnosis was made regarding the cause of the outbreak. Serological investigations did not show any evidence of viral involvement.

A second outbreak of coughing in cows was reported to have followed a further alleged incident of atmospheric pollution in January 1998. However, clinical examination of a single cow presented for veterinary examination some days after the incident failed to show any signs of significant respiratory tract inflammation. According to the herdowner, an incident of irritability in cows at milking in February 1998 may also have been due to atmospheric pollution.

At the end of June 1998, the herdowner reported that some cows were showing respiratory signs as a result of the alleged incident of atmospheric pollution which had been reported at the beginning of that month (*see above re calves*). However, clinical examination of a number of cows showed no significant evidence of respiratory tract inflammation and there was no evidence of a systemic inflammatory response on subsequent haematology analysis of blood samples.

A number of cows were also said to have been coughing on the day after another alleged incident of atmospheric pollution in November, 1998. No veterinary investigation of this incident was carried out as notification was not received until some weeks after it occurred.

Other than the respiratory conditions described above, disease incidence in the cow group was generally low. One case each of mastitis and milk fever was recorded in 1997. Two cases of clinical mastitis and one of sub-clinical mastitis (persistently high milk cell count) were recorded in 1998. Lameness was not reported to have been a problem in either year.

The most significant feature of the animal health investigations on this farm was the repeated claims that disease outbreaks - mostly respiratory – had followed exposure to atmospheric pollution. However, it should be noted that none of the alleged pollution incidents were confirmed by laboratory analysis of air samples. Although the herdowner had been supplied with an instrument for collection of air samples, only one sample was submitted over the two-year period of the Study and the findings on this were not significant (Environmental Quality Volume). Neither were the claims of environmental pollution corroborated by concurrent reports from herdowners of neighboring farms or of other farms in the locality. In fact, throughout the Study, only two other reports of atmospheric pollution were made by other herdowners in the vicinity - one involved the confirmed emission of dust from a mud stack on the Aughinish industrial site and no source was identified for the second. Neither of these reports coincided with those made by the herdowner of Farm A(LS).

From a theoretical standpoint, had the animals on this farm been exposed to 'caustic' or other irritant fumes of a sufficiently high concentration then it is quite likely that they could have developed respiratory signs similar to those reported. The fact that clinical and laboratory investigation of the incident in the cows in June 1997 failed to identify a specific cause cannot be taken as evidence either for or against the possibility that it was due to atmospheric pollution. Outbreaks of respiratory

disease are common in cattle of all ages and the ability to make a diagnosis will depend on factors such as timing and extent of clinical sampling, availability of appropriate laboratory tests, and nature of causative agent. Most outbreaks are multifactorial in origin and the diagnostic rate can be low even following extensive laboratory investigation (Gibbs, 1997).

On the one hand, not all infectious agents capable of causing respiratory disease can be readily identified by laboratory tests. On the other hand, had the incidents been due to inhalation of irritant fumes, the subsequent respiratory signs (mainly coughing) would not have been sufficiently characteristic to distinguish between infectious and irritant causes. Other possible causes of the outbreak - and which probably could also not have been identified by clinical or laboratory investigation - include irritation or allergy due to inhaled dusts (e.g. feed dust) or pollens. The cows in question were housed twice daily for milking where they received dry feed.

Regarding the three reported instances of respiratory signs in cows following alleged exposure to atmospheric pollution in 1998, there is insufficient evidence to comment on possible causes. The presence of respiratory tract inflammation was not confirmed by veterinary examination of cows in January and June 1998. Clinical and laboratory findings in the outbreak of respiratory disease in calves in June 1998 were sufficiently characteristic to suggest that it was most likely to have been infectious in origin. No veterinary or laboratory investigation was requested of the reported incident affecting cows in November, 1998.

In the absence of evidence regarding the nature of any specific irritant substances to which the animals on this farm might have been exposed, it was not possible to identify appropriate analyses to be carried out on clinical pathology samples from affected animals. As no animal losses were associated with the incidents, no post mortem material was available for examination or analysis. The only evidence, therefore, to support an association between these outbreaks and alleged pollution relates to their reported concurrence in time.

Other individual animal problems identified by the herdowner as being possibly due to atmospheric pollution included abortion (two cases), abnormal oestrus behavior in a cow (one case), a stillborn calf and a calf with a jaw abscess. However, these are all common conditions which are far more likely to have been due to infectious, hormonal or traumatic causes than to environmental pollution. In relation to those cases which were specifically investigated, there was no evidence that they were due to unusual factors such as atmospheric pollution.

No cows were reported to have died throughout the period of the Study.

FERTILITY

Infertility was reported to have been a problem on this farm since about 1990. According to the herdowner, the main problem was of cows and heifers failing to show signs of heat. Analysis of breeding records provided by the herdowner for the Retrospective Survey indicated a high rate of non-detected oestrus over the six years for which records were available. The incidence of repeat-breeders (i.e. multiple returns to service) was also reported to have been a problem in later years (Chapter Five). Artificial insemination was used exclusively on the farm up to 1994. A combination of natural and artificial service was used in 1994 and 1995. However, despite this, problems with heat detection persisted until 1996. Heat detection was not reported to have been a problem in 1996 when a bull was used for virtually all services. No results of fertility examinations were available for any of the bulls used during this period.

Details of fertility performance during the Longitudinal Study (1997 and 1998) are given in Table 3-3. Fertility performance was good in 1997. About a third of recorded services were natural and two-thirds were by artificial insemination. The submission rate for the first three weeks of the breeding season was 85 per cent. The overall fertility rate was high in 1997. Only one cow served in 1997 failed to calve in 1998. She had been confirmed pregnant by rectal examination at 56 days after service but repeated six weeks later and was assumed to have suffered an early abortion.

A bull was used for all services in 1998. Detailed fertility records were only maintained for the first month of the breeding season. Despite the incomplete records, it was clear that performance was down on 1997. The overall fertility rate for the year was also down. Ultra-sonic scanning carried out in September 1998 indicated that between 75 and 80 per cent of the cows were pregnant. However, this may have been somewhat of an underestimate as the bull had been with the cows up to a week prior to the scanning and pregnancies less than 30 days duration would not have been detected.

In the absence of comprehensive records it is not possible to determine the reasons for the lower conception performance in 1998. Although natural service was used throughout the breeding season, no information was available regarding bull fertility. Early embryonic losses may have been a factor. According to the herdowner, hormonal induction of heat was used on a number of cows in summer 1998 as an aid to fertility performance - despite the fact that the bull was running with the cows throughout. This is not a common practice as induction of heat in cows which are in early pregnancy may lead to embryonic expulsion. Available records show that at least one pregnant cow received hormone treatment as she was subsequently shown to be pregnant by scanning only 15 days after an artificially induced heat.

MILK PRODUCTION

Although individual cow milk recording was not carried out on this farm, available records indicated that production was satisfactory in 1997 and 1998. Total sales were. close to quota in both years. Average yield per cow for 1997 was estimated at about 3,596 kg on approximately 391 kg concentrates per head. Final figures for milk production in 1998 were not available when monitoring of the herd ceased in September. However, it is likely that production was comparable to 1997. Estimated average yield per cow up to September was about 3,300 kg on about 489 kg concentrates. A reportedly more rapid decline in yields in the latter part of the year than in 1997 may have reflected the poor weather conditions in the summer and autumn of 1998.

Note: owing to the historical fertility problems discussed above, production figures for both years include extended lactations of cows which had failed to go in calf in the previous year. These will have inevitably reduced average herd yields

BLOOD ANALYSIS RESULTS

Blood samples were collected from groups of animals - young stock and cows - on 13 occasions throughout the two-year period.

Haematology

Summary haematology statistics for cows and dry stock are given in Table 3-4. Mean red cell parameters for all samplings from cows and growing stock were generally within normal ranges throughout the study. Individual-animal values also generally remained within normal ranges. No evidence of anaemia was detected at any time throughout the Study. Mean MCV values for growing stock were generally between 30 and 40 µL - which is below the reported reference range of 40 – 60 µL. However, MCV values vary

considerably depending on breed and age - with younger animals having lower values (Schalm *et al.*, 1988). As most of the animals in the group were under six months of age, and in the absence of any evidence of anaemia, it is unlikely that this finding was of any clinical significance.

White cell counts in blood samples from cows were also generally within normal ranges. However, raised counts – generally due to a neutrophilia – were detected at times in samples from young stock. These were collected during investigations of chronic illthrift and respiratory signs and are consistent with a systemic inflammatory response to infection.

Overall, the haematology findings do not indicate any evidence of anaemia or other abnormality of red or white cell production or homeostasis at herd level. Individual values outside reference ranges were consistent with nutritional or infectious influences which are observed on farms elsewhere.

Biochemistry

Blood biochemical parameters were mostly within normal ranges throughout the period of observation. However, raised concentrations of globulins, βHB and the liver enzyme GLDH were recorded in cows and young stock on a number of occasions. Some raised globulin concentrations (>51.0 g/l) were recorded on most sampling occasions. These were probably associated with mild intercurrent sub-clinical inflammatory conditions, e.g. abscess, mastitis, respiratory infection, and were not, on their own, regarded as being of particular significance. βHB concentrations above 0.9 mmol/l detected in cows on a number of occasions were indicative of periodic negative energy balance (Whitaker *et al.*, 1993).

GLDH activities above 25.0 iu/l were detected in cows and growing stock on several occasions. In most cases, values were between 25.0 and 50.0 iu/l and, in the absence of any other specific findings, were not considered to be of particular significance. However, one animal, an eight year old cow, had GLDH activities of between 50 and 90 iu/l on four of the five samplings in 1997. This cow, which had a history of illthrift, had delivered a premature deformed calf in early 1998. Blood samples collected on a number of occasions in 1997 also had raised AST and globulin concentrations. Although no specific diagnosis was made to account for the illthrift or abnormal blood picture, it is likely that they were inter-related and were associated with unidentified liver lesions, e.g. abscesses or fibrosis.

Blood copper concentrations below the normal range of 9.4 – 24.0 μmol/l were detected in about a quarter of all samples collected during the two-year period – mostly from young stock. Recommendations regarding depot copper supplementation were made on a number of occasions (*see above*).

CONCLUSION

While problems with illthrift and respiratory disease in young stock and cows were observed on this farm, performance overall was satisfactory during the almost two-year period of the Longitudinal Study. In relation to poor thrive, there is sufficient information to suggest that nutrition, management and infectious agents were important contributory factors. Animal health was generally good and no serious outbreaks of disease were encountered. Although a number of outbreaks of respiratory disease were investigated in cows and young stock these were relatively mild in nature and no losses occurred.

The fact that no specific determination could be made regarding the possible association between some of these outbreaks and alleged incidents of atmospheric pollution is due partly to the absence of material for analysis (i.e. suspect air samples) and partly to the relatively mild and non-specific nature of the clinical signs observed in reportedly affected animals. Although specific infectious causes of the outbreaks were not identified, this is as likely to have been a function of variables such as timing of sampling, stage and severity of illness and availability of suitable tests as of any indication that infection was not involved. As no losses occurred, no material was available for pathology investigations. Regardless of their nature or origin, the incidents were apparently restricted in their geographical distribution as no other concurrent reports of atmospheric pollution in the vicinity were brought to the attention of the investigating team.

In relation to fertility, while both heat detection and conception performance were good on this farm in 1997, conception was below target in 1998. However, owing to incomplete breeding records - and absence of information on bull fertility - no conclusions can be drawn regarding the causes of reduced performance in 1998. Although individual cow records were not available, overall milk production appeared to have been satisfactory.

The results of haematology and biochemical analyses carried out on blood samples collected during the study showed no evidence of unusual health problems. Changes consistent with inflammatory, nutritional and metabolic conditions were noted at times. The most consistent finding was of a mild copper deficiency in some young stock at grass - probably indicating inadequate supplementation. However, it is unlikely that this had more than a marginal impact on animal health.

FARM LS2

The main animal health problems recorded in the Retrospective Survey Report on this farm (Farm 06) were infertility, perinatal calf mortality and ill-health in calves. The first visit of the Longitudinal Study was in November, 1996. The only significant animal health problems reported at the time were a recent laboratory diagnosis of BVD virus infection in a calf born the previous April and illthrift in a yearling bull. The main problems reported to have occurred during 1996 were post-calving vaginal discharge and repeat-breeding in cows, perinatal calf losses and poor milk production. A case of BVD virus infection was confirmed in a yearling heifer in February 1996. Two incidents of irritability at milking were also reported to have occurred - one shortly after an emission from a local factory was reported to have blown over the farm.

The Longitudinal Study on this farm commenced in December 1996 and concluded in December 1997 at the request of the herdowner for management reasons. A number of farm visits were also made in 1998 to investigate specific disease problems.

ANIMAL CONDITION AND NUTRITION

Cow body condition was moderate at the first farm visit of the Longitudinal Study in November, 1996. About a third of the cows (all but one milking) had condition scores under 2.0 at the time. Based on analysis of samples from the 1996 silage crop, Teagasc recommendations for concentrate supplementation for dry and milking cows represented a moderate increase on existing practice. These recommendations were implemented in part in 1997. Other than expected variations associated with production status, and occasional cases of ill-health, cow condition was not a specific problem at herd-level during the Longitudinal Study.

ANIMAL HEALTH

Details of reported disease occurrence are given in Table 3-5 and Table 3-6.

Calves and growing stock

The main disease problem recorded in young stock in 1997 was a high incidence of stillborn calves. Six stillbirths were reported in total. While the incidence is above target, and indicates a significant problem, straightforward diagnoses could be made in most cases. Four had a history consistent with difficult calving or perinatal trauma and one had an enlarged thyroid and lesions of pneumonia. The sixth was not submitted for laboratory examination.

Post-perinatal calf health was good. No deaths were recorded and respiratory and enteric conditions were not reported to be a problem. In early summer 1997, some calves were reported to be showing poor thrive and discolouration of the coat. Blood samples collected in August had marginally low copper concentrations. The calves showed no other significant signs of ill health and, at the end of the year, they were in good to moderate condition.

No specific disease problems were reported in older growing stock.

Cows and adult stock

Cow health was generally good. Two adult deaths occurred during 1997. The first, a cow, was euthanased following development of nervous signs. The second, a heifer, was due to babesiasis (redwater). There is a history of babesiasis in certain areas of this farm where old, rough pastures provide suitable tick habitats (see Chapter Five). Two other cases of redwater were reported in 1997 - both recovered following treatment.

Mastitis and lameness were the main adult animal disease problems recorded on the farm. Eleven cases of clinical mastitis were recorded in 1997. Laboratory investigations indicated involvement of *Staphylococcus aureus* - a common cause of infectious mastitis. Nineteen cases of lameness were recorded. Although relatively high, at 1.6 cases per month this is not an exceptional incidence (Appendix 1). There was evidence of involvement of the usual risk factors associated with outbreaks of lameness, i.e. rough surfaces and wet conditions underfoot.

Three cases of metritis were reported. Other single-case conditions in cows comprised tail haemorrhage, vulval tear with haemorrhage, pneumonia, grass tetany, chronic nasal discharge, and abscessation of the ventral abdominal wall.

Following investigations in the spring of 1998, severe fluke infestation was identified in a number of adult animals. It is most likely that this infestation was due to infection acquired in the autumn of 1997.

MILK PRODUCTION

Milk production was poor on the farm in 1997 given the breed of cow and reported rates of concentrate supplementation. Average yield per cow was 4,028 kg on 634 kg concentrates. From Table 3-7 it can be seen that production was below expectations across lactation groups. Given reported feeding and genetic merit of the cows, results could have been significantly higher.

Analysis of monthly production records for 1997 indicated that many cows had exhibited a rapid decline from peak yields between April and June. Of 36 cows which had peak yields in early April, about a half showed reductions in yield of between 30 and 40 per cent over the following two months; the remainder showed reductions of between 20 and 30 per cent. These are well in excess of the 10 per cent per month normally expected at this stage of lactation and indicate that a significant part of the shortfall in milk yield occurred at this time.

The reason for the rapid decline over this period could not be determined retrospectively. A Teagasc farm audit carried out in June 1997 reported an adequate supply of grass at the time. Other than individual cases of mastitis and lameness (see below), there was no evidence of a specific health problem affecting cows at that time. The milking cows were in generally good health and analysis of blood samples collected showed no evidence of underlying illness. Had the Study continued into 1998, it had been proposed to carry out a comparative trial of milk production by selected animals located on the farm and on a control farm elsewhere. Other factors to have been investigated would have included grazing and milking management.

Specific disease problems which would have had a negative effect on the yields of individual cows at various times of the year include lameness and mastitis. Nineteen cases of lameness were reported in 1997. In addition to the eleven reported cases of clinical mastitis, relatively high herd somatic cell counts during the year also indicated a significant degree of subclinical mastitis.

It is known that mastitis can reduce the yield of an affected cow by between 15 and 40 per cent (Beck et al., 1992), while a case of lameness can reduce yields by between 5 and 10 per cent (Kossaibati and Esslemont, 1997). The severe fluke infestation detected in animals in the spring of 1998 probably

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originated in infection acquired during the latter part of the milking season in 1997. This may also have affected milk yields at the time. A further factor which may have had an adverse effect on milk yields was a suspected problem with stray voltage in the milking parlour. This was to have been investigated further in 1998 had the Study continued.

Lactation lengths in many cows were also short in 1997. Average lactation length for the herd was 237 days. Six cows had lactations of under 200 days and 14 had lactations of under 230 days. These are well short of the optimum 305-day lactation. While the management decisions to dry off cows early undoubtedly reflected the already lower than expected yields in many of the cows, they will, inevitably, have had a significant negative impact on total yields for the year.

In addition to the above factors contributing to the poor milk production performance on this herd in 1997, it should also be noted that the herdowner was concerned that animal health problems experienced by some of the animals born in 1994 and 1995, i.e. the first and second lactation cows in 1997, may have had a long-term effect on their milk production potential. He was also of the opinion that some of these problems may have been due to exposure to environmental pollution in 1994 or 1995. Although no specific evidence of underlying long-term health or developmental problems was observed in these animals in 1997, this was one of the issues to have been addressed in the proposed trial of milk production in 1998 (see above).

FERTILITY

Details of fertility performance in 1997 are given in Table 3-8. Artificial insemination was used exclusively for the first two weeks of the breeding season. A combination of artificial insemination and natural services was used for the remainder of the season. The submission rate for the first three weeks of the breeding season was only 46 per cent. This is well below target and indicates a problem of non-detected oestrus in the early part of the season. Up to five bulls were used for services during the remainder of the season. Indicators of conception performance cannot be calculated as routine recording of services ceased once natural mating had commenced. The percentage pregnant of served for the year was 83 per cent, which is just below target. Except for the very poor heat detection rate at the beginning of the season, this is not an unacceptable performance.

BLOOD ANALYSIS RESULTS

Six cows and six yearling heifers were initially selected as monitor animals on this farm. Blood samples were collected on six occasions between December 1996 and February 1997. Samples were collected from a selection of animals on a further three occasions in 1998.

Haematology

Summary haematology statistics for cows and dry stock are given in Table 3-9. Haematology values were generally within normal ranges for the duration of the Study. There was no evidence of anaemia in young or adult stock. Total white cell counts above the normal range were not detected in cows at any time. Occasional raised counts in young stock (maximum $14.2 \times 10^9/l$) were probably associated with intercurrent sub-clinical infections.

Biochemistry

The majority of biochemical parameters were also within normal ranges throughout the period of the Study. Variations in protein, phosphorus, βHB and urea were probably metabolic in origin and related to changes in nutrition and production status and season. Occasional low phosphorus concentrations were seen in older cows or around peak milk yield. These were normal physiological responses to production demands.

Raised activities of the liver enzymes γGT and GLDH were detected in samples from some adult animals in the spring of 1998. Subsequent investigations confirmed that they were due to fluke infestation (see above).

CONCLUSION

Animal health overall was good on this farm during the 12-month period of the Longitudinal Study. The main problem in calves was stillbirths. The majority of cases were associated with difficult calvings. Post-perinatal calf health was good. Cow health was also generally good. Mastitis, lameness and fluke infestation were the main adult disease problems encountered. There were no serious or unexplained disease outbreaks.

Milk production was poor in 1997. While a number of probable contributory factors were identified – viz mastitis, lameness, short lactations, stray voltage – a full investigation of the problem planned for 1998 could not be initiated owing to the unplanned cessation of the study at the end of 1997.

While fertility performance could not be accurately assessed owing to incomplete records and use of multiple bulls for service, the number of cows in calf for the year was close to target.

The results of haematology and biochemistry analyses carried out on blood samples collected from the monitor animals during 1997 were generally within normal ranges. Changes consistent with inflammatory, nutritional and metabolic conditions were noted at times. Raised concentrations of liver enzymes associated with fluke infestation were detected in blood samples from some adult stock in spring 1998.

FARM LS3

The main animal health problems recorded in the Retrospective Survey Report (see Chapter Five) on this farm (Farm 07) were perinatal calf mortality, infertility, and poor milk yield. The first visit of the Longitudinal Study was at the end of October, 1996. No specific disease problems were reported at that time. High milk cell counts had been a problem in 1996 and production was reported to have been below expectations.

ANIMAL CONDITION AND NUTRITION

Animal condition was generally good at the time of the initial visit. All but four of the cows had body condition scores over 2.0. Based on analysis of samples from the 1996 silage crop, Teagasc recommendations for concentrate supplementation for dry and milking cows represented a moderate increase on existing practice. These recommendations were largely implemented in 1997. With the exception of a small number of imported Holstein cows which tended to have only poor to moderate condition, animal condition was generally satisfactory throughout the two-year period of the study.

ANIMAL HEALTH

Details of recorded disease incidents are given in Table 3-10 and Table 3-11. Animal health was generally good throughout the Study.

Calves and growing stock

Two stillbirths occurred in 1997 and two in 1998. Three of the four were associated with calving difficulties. No post-perinatal mortalities were recorded in 1997. Two calves died of coli-septicaemia (a common peri-natal infection) in 1998. Laboratory investigations indicated the primary problem in these cases was probably impaired immune status due to inadequate colostrum intake. One case of severe diarrhoea was

reported in a seven-week old calf in 1997. No other significant problems were reported throughout.

Cows and adult stock

The main problem in cows was one of sub-clinical mastitis. Although only four cases of clinical mastitis were recorded in 1997, the results of laboratory investigations in that year indicated widespread infection of the milking herd with *Staph. aureus*. A comprehensive control program was implemented which included dry cow therapy, modifications to the milking routine and culling of persistently infected cows. While cell counts were lower throughout most of 1998, and only three cases of clinical mastitis were recorded, quarter-sampling at the end of the year indicated that infection with *Staph. aureus* remained widespread.

Other conditions recorded in 1997 and 1998 were metritis or vaginal discharge, retained placenta, lameness and milk fever. The incidences of these conditions were within acceptable limits.

Two cows died during 1997 - one was euthanased following development of milk fever and post-parturient recumbency. The second was a case of grass tetany. One cow died in 1998 as a result of abomasal ulceration and haemorrhage.

FERTILITY

Fertility was not reported to have been a problem on this farm in the years immediately preceding the Longitudinal Study. The results of available fertility indices for the two years of the Longitudinal Study in 1997 and 1998 are given in Table 3-12. The submission rate of 85 per cent for the first three weeks of the 1997 breeding season indicates heat detection was good at the time. Details of conception performance cannot be determined owing to incomplete service records. The percent pregnant of cows served in 1997, at 82 per cent, was just below target. Little information is available regarding fertility performance in 1998 as a bull was used for the majority of services and few records were kept. The reported overall fertility rate (percent pregnant of cows served) at 93 per cent was well within target.

MILK PRODUCTION

Milk production was satisfactory in 1997 and 1998. Average yield per cow in 1997 was 4,078 kg on 519 kg concentrates. The comparable figure in 1998 was 4,210 kg on about 700 kg concentrates. Yield per cow in 1998 was significantly reduced by the addition of a group of purchased in-calf heifers to the herd at the beginning of the year.

BLOOD ANALYSIS RESULTS

Blood samples were collected from the selected monitor animals – six each of young stock and cows - on eight occasions throughout the two-year period.

Haematology

Summary haematology statistics for cows and dry stock are given in Table 3-13. Group mean haematology parameters remained within normal ranges throughout. With the exception of a single case of anaemia in a cow in October 1998 - possibly due to internal bleeding from an abomasal ulcer - individual-animal erythrocyte parameters also generally remained within normal ranges. White cell counts in blood samples from cows and young stock were also generally within normal ranges. Occasional raised counts were consistent with intercurrent sub-clinical infections.

Biochemistry

Blood biochemical parameters were mostly within normal ranges throughout the period of observation. Raised globulin or GLDH (liver enzyme) activities were recorded in individual samples on a number of occasions. These were not considered to be of any clinical significance. Recommendations regarding supplementation were made at appropriate times to address seasonal findings of low blood magnesium in cows and marginal copper in young stock.

CONCLUSION

The main animal health problems on this farm prior to the commencement of the Longitudinal Study were perinatal calf mortality, infertility, and poor milk yield. In contrast, animal health was generally good throughout the two-year period of the Longitudinal Study. While losses due to stillbirths and neonatal infections were recorded, rates were well within normal ranges. Cow health was also good. The two deaths which occurred were due to production-related diseases.

Although inadequate records were available to make an accurate assessment, fertility performance appeared to have been good. Despite the problem of widespread subclinical mastitis – the extent of which was undoubtedly partly a function of its non-recognition in previous years - milk production was satisfactory in 1997 and 1998. Haematology and biochemistry results on blood samples collected from the monitor animals were generally within normal ranges. Occasional values outside reference ranges were associated with production status or individual cases of ill-health.

FARM LS5

This is a large dairy herd. The main problems reported in the Retrospective Survey Report (see Chapter Five) on this farm (Farm 09) were infertility, perinatal calf mortality and ill-health in calves. The first visit of the Longitudinal Study to this farm was in December 1996. No specific disease problems were reported at the time. An outbreak of diarrhoea in calves was reported to have occurred during the previous summer.

ANIMAL CONDITION AND NUTRITION

Excess condition (overfat) of some dry cows and heifers – which resulted in a high incidence of dystocia (see below) - was a problem during the 1997 calving season. Animal condition otherwise was generally satisfactory throughout 1997 and 1998. Analysis of silage samples from the 1996 and 1997 crops indicated that the quality was good. Concentrate supplementation during the 1997 and 1998 milking seasons was largely in line with Teagasc recommendations.

ANIMAL HEALTH

Details of recorded disease incidents are given in Table 3-14 and Table 3-15. Results for 1998 are incomplete due to the absence of comprehensive records. Animal health performance overall was satisfactory over the two years of the Study.

Calves and growing stock

The main problems in calves in 1997 were perinatal mortality and diarrhoea. The incidence of perinatal mortality (mainly stillbirths) was high owing to calving problems associated with the overfat condition of some cows and heifers (see below). Post-mortem findings in calves submitted for laboratory examination were consistent with deaths being secondary to difficult or prolonged calvings.

An outbreak of diarrhoea in calves, due to cryptosporidial infection, occurred in January 1997. Further sporadic cases occurred over the following three months. There were no losses and all cases responded to therapy. Several cases of diarrhoea and associated illthrift were recorded in weanlings at grass during the summer. An outbreak of respiratory disease, characterised by coughing and nasal discharge, occurred in September. The majority of calves were affected. No specific pathogen was identified by a subsequent laboratory investigation. Although growth rates of some calves were reduced, no losses occurred and the majority had improved significantly by mid-November.

There were seven perinatal calf losses in 1998 . Two cases were from sets of twins which is a known risk factor for perinatal mortality. Post-perinatal calf health was satisfactory in 1998. An outbreak of respiratory disease in the last batch of housed calves in the spring responded to antibiotic therapy. A further outbreak of respiratory disease in calves at grass following a spell of cold and wet weather also responded to antibiotic therapy. No losses were recorded in either outbreak.

Cows and adult stock

The main health problem in adult animals was excess condition of cows and heifers in 1997 leading to a high incidence of dystocia. Almost a half of the calvings in that year were recorded as being difficult. Associated stillbirths have been discussed above. Dietary control measures were introduced during the calving season to address the problem.

The incidences of retained foetal membranes and vaginal discharge/metritis were also above normal in 1997 as a direct result of the calving problems. The incidence of mastitis was within the normal range in 1997. The incidences of other conditions were within acceptable limits in 1997. Inadequate information is available to assess disease incidences in 1998. However, other than a serious mastitis problem in bought-in cows – and which had to be dealt with by radical culling - there were no reports of significant disease problems in adult stock in that year.

Two cows died in 1997. Both were calving-related. The first was from peritonitis secondary to a caesarian section; the second was euthanased in post-parturient recumbence. Three other cows were culled to the abattoir in 1997 due to periparturient conditions. Records of animal deaths are incomplete for 1998. However, based on available information, the death rate would appear to have been low and comparable to 1997.

FERTILITY

The results of available fertility indices for 1997 and 1998 are given in Table 3-17. Analysis of results for the 1997 breeding season indicated that heat detection performance was good overall. This reflected the attention to heat detection in that year and the use of teaser bulls. Accuracy of heat detection was also good as indicated by the distribution of inter-service intervals given in Table 3-16. Conception performance, however, was below target. This was reflected in a high incidence of repeat-breeder cows (i.e. cows with four or more services). As few pregnancy diagnoses were

carried out during 1997, the problem was only identified retrospectively on the basis of the 1998 calving results.

Teaser bulls were again used in 1998 to assist heat detection during the first six weeks of the breeding season. Calving to first service and submission rates at this time were close to target indicating an acceptable heat detection performance. However, the distribution of inter-service intervals in Table 3-16 indicates that accuracy of heat detection was not good. The ratio of 18-24 : 36-38 day intervals was 3.2 compared to a target of 7.0 (Esslemont, 1992). This may reflect a reduced intensity of heat detection following commencement of natural service in mid-July.

Although the repeat-breeder rate was well within target, there is evidence that conception rates may still have been below target. While no pregnancy diagnoses were carried out in 1998, data for non-return rates suggested that over 20 per cent of cows served in 1998 failed to conceive.

Fertility performance had been an area of concern on this farm for some years. Analysis of information in the Retrospective Survey Report (Chapter Five), as well as the results of the Longitudinal Study, indicate that the main problem has been one of poor conception rates with a consequent high incidence of repeat-breeders.

As discussed elsewhere (see page 6), the main causes of repeat-breeding are conception failure and early embryonic loss. Conception performance is a function of cow and bull fertility, accuracy of heat detection, semen quality and handling, timing of insemination, and artificial insemination technique. In relation to the 1997 performance on this farm, the most significant factor likely to have affected cow fertility is the very high rate of calving problems – almost a half of calvings were described as difficult. It is well recognised that uterine infections and inflammation secondary to dystocia are a common cause of infertility in the cow (Oltenacu et al., 1990). Fifty percent of the repeat-breeders on this farm in 1997 had a recorded history of severe calving problems. Given the high incidence of dystocia in the herd in that year, it is likely that others may have suffered sub-clinical infection.

Although calving and repeat-breeder problems were significantly reduced in 1998, there is evidence that conception performance remained below target. While in-depth investigation was precluded by the absence of pregnancy diagnosis results, analysis of available records suggests that reduced accuracy of heat detection was likely to

have been a contributory factor (*see comment re inter-service intervals above*). The possible contribution of bull fertility to the reduced conception performance in 1997 and 1998 cannot be estimated as no fertility examinations of bulls were carried out.

A further factor which must be taken into consideration when assessing overall fertility performance on this farm relates to milk yield. Average yields were relatively high with individual cows giving up to 7,274 kg per year. Recent studies by Teagasc have shown that conception performance can be significantly reduced in high yielding cows O'Farrell *et al.*, (1997). The results of blood (*see below*), soil, and herbage analyses carried out on this farm during the two-year Study provide little evidence to suggest that mineral deficiency had a significant impact on fertility performance. To the extent that copper deficiency may occur on this farm, it is likely to be secondary to raised molybdenum concentrations on certain pastures.

In conclusion, while conception performance on this farm was below target, it was not exceptionally so. Conception rates vary widely on dairy farms. In the UK DAISY survey, for example, conception rates ranged from 34 to over 60 *per cent*, with services per conception ranging from 1.5 to 2.8 (Esslemont and Spincer, 1993). There is, therefore, no reason to suggest that fertility performance on this farm was suppressed due to unusual factors. In relation to the repeat-breeder problem, it is likely that a significant proportion of these in 1997 were secondary to the earlier calving problems. Reduced accuracy of heat detection was probably a contributory factor in 1998. While the respective roles of the other factors most commonly associated with reduced conception, i.e. bull fertility, timing of insemination, semen handling and quality, and embryonic mortality, were not determined, they are far more likely candidates for involvement than external factors such as environmental pollution.

MILK PRODUCTION

Milk production was good on this farm in 1997 and 1998. Average yield per cow in 1997 was estimated to be 5,264 kg on 250 kg concentrates per head and an average lactation length of 277 days. The comparable figures for 1998 were 5,182 kg for 118 cows on 550 kg concentrates per head and an average lactation length of 287 days. The relatively low concentrate usage on this farm reflects the good quality of silage.

Milk quality was also good in both years with low to moderate cell counts (SCC) reflecting the generally low herd incidence of mastitis. A number of cows bought-in in early 1998 were culled shortly after calving because of mastitis problems.

BLOOD ANALYSIS RESULTS

Blood samples were collected from groups of animals - young stock and cows - on seven occasions throughout the two-year period.

Haematology

Summary haematology statistics for cows and dry stock are given in Table 3-18. Haematology values were generally within normal ranges throughout the period. Marginally raised white cell counts on occasions were consistent with systemic inflammatory responses.

Biochemistry

Biochemistry parameters were also generally within normal ranges throughout. Marginally low copper values (total five samples) were recorded on two occasions in 1997. However, these were not considered to be of clinical significance. Concentrations of other blood minerals were, with a few exceptions, within normal ranges throughout. Low globulin concentrations in cows in January 1997 were probably related to stage of pregnancy. Occasional raised GLDH activities were probably dietary in origin.

CONCLUSION

The main problems which had been reported on this farm in the years immediately preceding the commencement of the Longitudinal Study were infertility, perinatal calf mortality and ill-health in calves (Chapter Five). While animal health was generally good during the period of the Longitudinal Study, specific problems were recorded in relation to stillbirths, diarrhoea and respiratory disease in calves. However, the former (stillbirths) were clearly associated with calving problems while the latter were infectious in origin. The main problems encountered in cows were calving difficulties associated with excess body condition and a consequential increased incidence of retained foetal membranes and metritis.

While fertility performance overall was satisfactory, conception rates were below target. Although detailed analysis was precluded by the absence of comprehensive breeding records, a relatively low accuracy of heat detection and reduced fertility of cows following difficult calvings were identified as probable contributory

factors. Milk production was good for the two years of the study with average yields per cow of around 5,001 kg Blood haematology and biochemistry analysis results were generally within reference ranges throughout.

OVERALL CONCLUSION

The findings of this study must be interpreted in the light of obvious limitations regarding the intensity of monitoring which could be implemented consistent with the private and commercial nature of the farming operations concerned. While herdowner compliance with the requirements of the study was generally good, the level of record-keeping varied considerably between herds and even between years within herds. Fertility records were generally inadequate for a detailed assessment of performance and information on concentrate feeding was largely based on reported average daily rates. For practical reasons, no attempt was made to assess the accuracy of the latter estimates. While there is no reason to doubt these estimates, the experiences on the DAF-managed farm (Index Farm B) in the first year of the Monitor Study in relation to feed hoppers (Chapter Two) highlight the fact that the accuracy of these mechanisms cannot be taken for granted.

Only a limited amount of information was available in relation to the supply and quality of grass and stored fodder on the Longitudinal Study farms. Although silage analysis results were available for all four farms for the 1996 crop, grassland assessment was limited to a visual inspection of each farm in 1997 and no monitoring or assessment of grazing management was carried out for reasons of resource limitations.

Allowing for these constraints, the results of the Longitudinal Study showed no evidence that animal health or production on any of the four farms were subject to unusual adverse influences during the periods of observation. While there was a wide range in production performance between farms, results were, with the exception of milk production on one farm (Farm LS6), generally compatible with expectations for comparable operations.

The incidence of disease on each of the four farms was generally within acceptable limits and no serious outbreaks of disease were encountered. The conditions reported largely comprised those commonly-observed on commercial farms, *viz* stillbirths and neonatal diseases in calves, mastitis, lameness and infertility in cows. In most cases, straightforward diagnoses could be made and there was no evidence of a unusually high incidence of diseases for which no diagnosis could be made. While the causes of a number of outbreaks of respiratory disease in cows and calves on one farm (Farm LS5) were not identified, the cases were generally mild in nature and clinical and laboratory examinations did not reveal any unusual features.

Tables

Table 3-1: Recorded disease incidents in calves and growing stock - Farm LS1

	1997 (from 10/96)	1998 (to 9/98)
Calves (0–4 weeks):		
	Incidence[1]	Incidence[1]
Born dead/died at calving	0.057	0.080
Congenital deformity	0.029	0.040
Post-perinatal deaths	0.000	0.000
Diarrhoea[2]	0.058	0.000
Respiratory disease	1 outbreak[3]	0.000
Navel/Joint Ill	0.000	0.000
Other Diseases	0.000	0.000
One month - 2.5 yr:		
	Incidence[4]	Incidence[4]
Deaths	0.024	0.023
Diarrhoea	0.024	0.000
Respiratory disease	0.000	1 outbreak
Other Diseases	0.000	0.000

[1]Cumulative incidence; PAR = total calves born [2]Requiring treatment other than milk withdrawal [3]Housed calves April 1997 – see text. [4]Annual incidence, PAR = average number animals per month.

Table 3-2: Recorded disease incidents in Cows - Farm LS1

	1997 (from 10/96)	1998 (to 9/98)
Parturient/lactation Conditions	Incidence[1]	Incidence[1]
Abortion	0.058	0.000
Difficult calving	0.000	0.040
Milk Fever	0.029	0.000
Downer cow	0.000	0.000
Retained Placenta	0.029	0.000
Vulval discharge/metritis	0.000	0.000
Ketosis	0.000	0.000
Tetany	0.000	0.000
Mastitis	0.029	0.080
Other	0.000	0.000
Miscellaneous Conditions	Incidence[2]	Incidence[2]
Deaths	0.000	0.000
Disease Culls:	0.000	1
Haematomas	0.000	0.000
Respiratory disease	0.700[3]	2 outbreaks[4]
Pneumonia	0.037	0.000
Lameness	0.000	0.000
Skin Lesions	0.000	0.000
Redwater	0.000	0.000
Irritability at milking	0.000	1 outbreak
Cystic ovary	0.031	0.033
Abscesses	0.000	0.000

[1]Cumulative incidence, PAR = total calved. [2]Annual incidence; PAR = average monthly number of cows. [3]Majority of cows reported showing signs of upper respiratory tract condition in June – see text. [4]Incidents reported by herdowner, not confirmed by subsequent field/laboratory investigation – see text.

Table 3-3: Fertility Performance for Farm LS1 – 1997 and 1998

	1997	1998	Target
Calving season	All year	January – June.	4 months
Breeding season	1 May – 31 July.	1 May – end September.	4 months
Services:			
Artificial insemination	May to July	None	
Bulls (1)	May to end breeding season	May to end breeding season	
Calving to first service (days)	67	NA[2]	60 – 70
Submission rate (%)	85	65	80
Calving to conception	92	NA	
Conception to 1st service	58	NA	
Services per conception	1.4	NA	
Fertility rate (%)[1]	96	75 – 79	> 85

[1]Percent pregnant of served. [2]Not available.

Table 3-4: Haematology statistics for Longitudinal Study Farm LS1

		1996 Dec	1997 Mar	Jun	Jul	Sep	Oct	1998 Dec	Jan	Jun	Jul	Aug
Cows												
RBC	Avg.	6.2	6.0	6.9	6.9	6.7	6.5	6.0	6.1	6.7		6.9
	StDev.	0.4	0.7	0.5	0.5	0.8	0.5	0.7		0.7		1.0
	N.	7	10	16	8	7	2	7	1	7		7
MCV	Avg.	52.0	50.3	49.4	48.4	48.9	53.3	51.0	36.9	50.9		48.9
	StDev.	5.0	4.0	4.8	5.7	4.9	3.3	4.3		3.3		2.5
	N.	7	10	16	8	7	2	7	1	7		7
PCV	Avg	32.0	29.8	34.0	33.2	32.7	34.8	30.6	33.0	33.9		33.8
	StDev	3.8	2.8	3.1	5.4	2.9	0.4	1.7		3.0		3.7
	N	7	10	16	8	7	2	7	1	7		7
WBC	Avg	7.0	6.6	8.6	9.4	7.4	7.2	6.7	53.2	7.9		6.9
	StDev	1.9	1.6	1.7	1.1	1.4	0.7	1.9		3.1		2.2
	N	7	10	16	8	7	2	7	1	7		7
Dry Stock												
RBC	Avg	8.4	8.4	9.2		8.4	9.4	9.9	9.1	9.5	10.2	7.1
	StDev	0.8	1.3	1.9		0.8	1.2	0.9	0.4	0.5	0.4	
	N	6	7	4		6	7	6	3	13	6	1
MCV	Avg	39.5	43.9	38.9		36.4	34.3	34.3	33.2	38.9	36.5	44.4
	StDev	6.6	5.8	5.0		2.6	3.5	2.3	1.6	4.0	1.3	
	N	6	7	4		6	7	6	3	13	6	1
PCV	Avg	33.0	36.4	35.0		30.6	32.0	33.7	29.7	36.7	37.2	31.5
	StDev	5.0	2.5	3.5		3.1	3.3	2.5	0.6	3.0	2.0	
	N	6	7	4		6	7	6	3	13	6	1
WBC	Avg	11.5	8.3	8.9		9.9	10.1	9.5	32.9	9.3	12.7	7.5
	StDev	3.3	2.3	4.7		3.8	2.9	2.0	0.8	1.7	1.3	
	N	6	7	4		6	7	6	3	13	6	1

Table 3-5: Recorded disease incidents in calves and growing stock - Farm LS2

	1997
Calves (0–4 weeks):	*Incidence[1]*
Born dead/died at calving	0.082
Congenital deformity	0.000
Post-perinatal deaths	0.000
Diarrhoea[2]	0.000
Respiratory disease	0.000
Navel/Joint Ill	0.000
Other Diseases	0.000
One month - 2.5 yr:	*Incidence[3]*
Deaths	0.030
Diarrhoea	0.000
Illthrift	0.180 – 0.240
Redwater	0.090
Other Diseases	0.000

[1]Cumulative incidence; PAR = total calves born. [2]Requiring treatment other than milk withdrawal.
[3]Annual incidence, PAR = average number animals per month.

Table 3-6: Recorded disease incidents in Cows - Farm LS2

		1997
Parturient/lactation Conditions		*Incidence[1]*
Abortion		0.014
Difficult calving		0.085
Milk Fever		0.000
Downer cow		0.000
Retained Placenta		0.000
Vulval discharge/metritis		0.042
Ketosis		0.000
Tetany		0.014
Mastitis		0.155
Other		0.000
Miscellaneous Conditions		*Incidence[2]*
Deaths		0
Disease Culls:		
	Udder problems	0.033
	Lameness	0.033
	Infertility	0.033
	CNS	1
Haematomas		0.000
Respiratory disease		0.033
Lameness		0.317
Skin Lesions		0.000
Irritability at milking		0.000
Conjunctivitis		0.050
Abscesses		0.033

[1]Cumulative incidence, PAR = total calved. [2]Annual incidence; PAR = average monthly number of cows.

Table 3-7: Estimated average yields per cow (1997) grouped by lactation number for Farm LS2.

	Yield (gals)
All Cows	886
First Lactations	667
Second Lactations	824
Rest: (3+ lactations)	1000

Table 3-8: Fertility Performance for Farm LS2 – 1997

	1997	Target
Calving season	January – June[1]	4 months
Breeding season	1 May – 30 Sept.	4 months
Services:		
Artificial insemination	April – September	
Natural service (5 bulls)	Mid-May to September	
Calving to first service (days)	76	60 – 70
Submission rate (%)	46	80
Calving to conception	NA[2]	
Conception to 1st service	NA	
Services per conception	NA	
Fertility rate	83	> 85

[1]One cow calved October, 1997. [2]Not available

Table 3-9: Haematology statistics for Longitudinal Study Farm LS2

		1996 Dec	1997 Apr	Jul	Aug	Sep	1998 Feb	Mar	Aug
Cows									
RBC	*Avg.*	6.7	5.8	6.3	5.7	7.2	6.2	6.2	6.6
	StDev.	1.1	1.1	0.7	2.1	0.7	0.4		0.6
	N.	7	7	7	3	7	7	1	6
MCV	*Avg.*	51.9	52.1	51.2	48.2	50.5	50.6	51.8	55.6
	StDev.	7.6	5.9	3.4	6.9	4.6	3.2		5.4
	N.	7	7	7	3	7	7	1	6
PCV	*Avg*	34.1	30.0	32.2	26.7	36.4	31.4	32.3	36.4
	StDev	3.6	3.1	2.1	7.1	3.1	2.0		3.4
	N	7	7	7	3	7	7	1	6
WBC	*Avg*	7.7	6.4	7.6	8.6	7.7	7.4	12.8	6.9
	StDev	1.0	1.0	1.5	2.8	1.2	1.2		0.9
	N	7	7	7	3	7	7	1	6
Dry Stock									
RBC	*Avg*	9.3	8.4	8.3	8.5	8.9	7.3	6.6	7.5
	StDev	0.8	0.9	0.6	1.8	0.8	0.6	0.3	0.9
	N	6	6	6	9	6	6	5	5
MCV	*Avg*	37.8	41.0	43.1	40.5	43.4	45.0	47.8	50.4
	StDev	2.1	3.3	3.1	7.2	2.4	2.1	3.4	6.3
	N	6	6	6	9	6	6	5	5
PCV	*Avg*	35.0	34.5	35.8	33.4	38.8	32.9	31.7	37.3
	StDev	2.2	3.6	2.5	3.0	3.7	2.9	1.4	4.4
	N	6	6	6	9	6	6	5	5
WBC	*Avg*	11.5	10.4	10.9	9.9	11.8	9.2	8.0	8.7
	StDev	1.6	1.6	1.6	2.1	1.8	1.9	1.1	1.7
	N	6	6	6	9	6	6	5	5

Table 3-10: Recorded disease incidents in calves and growing stock - Farm LS3

	1997	1998
Calves (0–4 weeks):		
	Incidence[1]	*Incidence[1]*
Born dead/died at calving	0.045	0.048
Congenital deformity	0.000	0.025
Post-perinatal deaths	0.000	0.05
Coli-septicaemia	0.000	0.05
Diarrhoea[2]	0.000	0.000
Respiratory disease	0.000	0.000
Navel/Joint Ill	0.000	0.000
Other Diseases	0.000	0.000
One month - 2.5 yr:		
	Incidence[3]	*Incidence[3]*
Deaths	0.000	0.000
Diarrhoea	0.070	0.000
Illthrift	0.000	0.000
Other Diseases	0.000	0.000

[1]Cumulative incidence, PAR = total calves born. [2]Requiring treatment other than milk withdrawal.
[3]Annual incidence, PAR = average number animals per month.

Table 3-11: Recorded disease incidents in Cows - Farm LS3

	1997	1998
Parturient/lactation Conditions		
	Incidence[1]	*Incidence[1]*
Abortion	0.000	0.000
Difficult calving	0.033	0.048
Milk Fever	0.033	0.000
Downer cow	0.017	0.000
Retained Placenta	0.033	0.048
Vulval discharge/metritis	0.083	0.071
Uterine prolapse	0.000	0.024
Tetany	0.017	0.000
Mastitis	0.067	0.071
Other	0.000	0.024
Miscellaneous Conditions		
	Incidence[2]	*Incidence[2]*
Deaths	0.049	0.024
Disease Culls:	0.000	0.000
Mastitis	0.195	0.214
Lameness	0.000	0.024
Haematomas	0.000	0.000
Respiratory disease	0.000	0.000
Lameness	0.146	0.095
Skin Lesions	0.000	0.000
Irritability at milking	0.000	0.000
Conjunctivitis	0.000	0.000
Abscesses	0.000	0.000

[1]Cumulative incidence, PAR = total calved [2]Annual incidence, PAR = average monthly number of cows.

Table 3-12: Fertility Performance for Farm LS3 – 1997 and 1998

	1997	1998	Target
Calving season	January – June	January – May	4 months
Breeding season	April – July	May – August	4 months
Services:			
Artificial insemination	First 5 weeks of breeding season	First 2 weeks of breeding season	
Natural service (1 bull)	June/July (1 bull)	May – August (2 bulls)	
Calve to first service (days)	NA	NA	60 – 70
Submission rate (%)	85	NA	80
Calving to conception	NA	NA	
Conception to 1st service	NA	NA	
Services per conception	NA	NA	.
Fertility rate	82	93	> 85

Table 3-13: Haematology statistics for Longitudinal Study Farm LS3

		1996 Nov	1997 Feb	Jun	Sep	Dec	1998 Apr	Aug	Oct
Cows									
RBC	*Avg.*	6.4	6.0	6.6	6.4	6.2	6.2	6.4	6.0
	StDev.	0.7	0.7	1.2	0.5	0.8	0.9	0.9	1.8
	N.	9	7	7	8	8	7	7	6
MCV	*Avg.*	51.8	52.6	49.6	51.1	50.7	53.4	51.7	51.8
	StDev.	6.5	6.4	3.4	5.9	6.1	6.3	3.5	4.0
	N.	9	7	7	8	8	7	7	6
PCV	*Avg*	32.8	31.6	32.7	32.5	31.5	32.5	33.3	30.4
	StDev	4.2	4.7	6.0	4.0	4.0	3.6	5.2	8.1
	N	9	7	7	8	8	7	7	6
WBC	*Avg*	9.3	7.1	8.0	8.2	7.3	7.5	8.4	8.9
	StDev	2.7	1.6	1.4	1.3	1.1	2.1	1.1	1.3
	N	9	7	7	8	8	7	7	6
Dry Stock									
RBC	*Avg*	9.2	8.2	7.5	8.7	7.6	7.9	7.9	7.8
	StDev	0.4	0.4	1.1	0.9	1.5	0.7	0.6	0.5
	N	5	5	5	5	5	5	5	5
MCV	*Avg*	39.9	41.4	43.1	42.9	45.1	47.0	49.6	49.7
	StDev	2.2	2.0	2.6	3.0	1.7	2.7	2.3	2.7
	N	5	5	5	5	5	5	5	5
PCV	*Avg*	36.5	33.9	31.9	37.0	34.0	37.1	39.2	38.9
	StDev	1.0	1.2	3.3	3.0	6.0	2.2	3.5	2.2
	N	5	5	5	5	5	5	5	5
WBC	*Avg*	13.5	9.8	5.9	8.8	8.1	8.7	9.3	8.5
	StDev	2.0	0.7	1.8	2.3	1.2	1.8	2.1	1.6
	N	5	5	5	5	5	5	5	5

Table 3-14: Recorded disease incidents in calves and growing stock - Farm LS5

	1997	1998[1]
Calves (0–4 weeks):	*Incidence*[2]	*Incidence*[2]
Born dead/died at calving[3]	0.120	0.070
Congenital deformity	0.000	0.000
Post-perinatal deaths	0.000	0.000
Coli-septicaemia	0.000	NA
Diarrhoea[4]	0.230[5]	NA
Respiratory disease	0.010	NA
Navel/Joint Ill	0.000	NA
Other Diseases	0.000	NA
One month - 2.5 yr:	*Incidence*[6]	*Incidence*[6]
Deaths	0.004	0.004
Diarrhoea	0.050	NA
Respiratory disease	1 outbreak[7]	NA
Lameness	0.013	NA
Other Diseases	0	NA

[1]Only partial disease incidence data available for 1998 [2]Cumulative incidence; PAR = total calves born. [3]Includes calf that died from dystocia-associated lesions two weeks after delivery.[4]Requiring treatment other than milk withdrawal. [5]Outbreak of cryptosporidiosis [6]Annual incidence; PAR = average number animals per month [7]Approx. 80% incidence

Table 3-15: Recorded disease incidents in Cows - Farm LS5

	1997	1998[1]
Parturient/lactation Conditions	*Incidence*[2]	*Incidence*[2]
Abortion	0.000	NA
Difficult calving	0.436	NA
Milk Fever	0.011	NA
Downer cow	0.021	NA
Retained Placenta	0.128	NA
Vulval discharge/metritis	0.106	NA
Uterine prolapse	0.000	NA
Tetany	0.000	NA
Mastitis	0.160	NA
Cystic ovary	0.032	NA
Other	0.000	NA
Miscellaneous Conditions		
	Cases	Cases
Deaths	0.020	NA
Disease Culls:		
Peri-parturient	0.020	NA
Mastitis	0.020	NA
Lameness	0.020	NA
Respiratory disease	0.000	NA
Lameness	0.102	NA
Cystic ovary	0.031	NA
All other	0.000	NA

[1]Only partial disease incidence data available for 1998.[2]Cumulative incidence; PAR = total calved (dairy cows). [3]Annual incidence, PAR = average monthly number of cows (dairy cows).

Table 3-16: Accuracy of heat detection for Farm LS5 – percent distribution of inter-service intervals

Interval (days)	1997	1998	Target	Interference Level
< 18	4	14	< 10	15
18 – 24	66	49		
> 24	29	37	< 30	35
Ratio 18-24:36-48	6.8	3.2	7.0	

Table 3-17: Fertility Performance (cows) for Farm LS5 – 1997 and 1998

	1997	1998	Target
Calving season	January – July	February – June	4 months
Breeding season	April – September	May – August	4 months
Services:			
Artificial insemination	April – July	May – July	
Natural service (1bull)	July – September	July – August	
Calving to first service (days)	80	64	60 – 70
Submission rate (%)	79	74	80
Heat detection rate	57	61	80
Calving to conception	114[1]	NA	80 – 85
Conception to 1st service	44	NA	60
Conception to all services	35	NA	60
Services per conception	2.8	NA	1.65
% Repeat-breeders	19	4[2]	< 10
Culling for infertility	12	NA	< 10
Fertility rate	76	~79[3]	> 85

[1] High due to inclusion of five cows that did not conceive in 1996 [2] May be underestimate due to incomplete breeding records. [3]Based on approximate non-return rate

Table 3-18: Haematology statistics for Longitudinal Study Farm LS5

		1997				1998		
		Jan	Apr	Jul	Nov	Jan	Apr	Aug
Cows								
RBC	*Avg.*	5.6	6.0	6.3	6.5	6.3	5.6	6.3
	StDev.	0.4	0.5	0.4	0.8	0.9	0.8	0.2
	N.	5	5	5	5	4	4	3
MCV	*Avg.*	51.1	49.9	52.2	51.6	52.8	53.5	45.5
	StDev.	2.9	2.9	2.9	2.7	2.6	1.9	1.3
	N.	5	5	5	5	4	4	3
PCV	*Avg*	28.5	29.8	32.8	33.4	33.0	30.0	28.5
	StDev	2.3	2.1	2.0	3.5	3.5	4.4	0.5
	N	5	5	5	5	4	4	3
WBC	*Avg*	6.2	7.3	6.8	7.0	5.7	5.9	8.8
	StDev	1.0	2.0	1.0	1.6	0.2	1.7	1.1
	N	5	5	5	5	4	4	3
Dry Stock								
RBC	*Avg*	7.7	8.1	7.5	7.2	7.1	6.2	6.6
	StDev	1.1	1.2	0.9	0.5	1.0	0.4	0.8
	N	7	7	7	7	6	6	5
MCV	*Avg*	39.7	41.9	46.4	48.4	46.0	46.9	47.0
	StDev	5.2	5.0	4.0	3.4	4.9	4.0	3.0
	N	7	7	7	7	6	6	5
PCV	*Avg*	30.0	33.4	34.5	34.7	32.2	29.0	30.7
	StDev	2.7	2.5	3.9	2.0	2.8	2.5	3.6
	N	7	7	7	7	6	6	5
WBC	*Avg*	8.8	8.2	7.1	10.3	7.8	7.2	8.6
	StDev	2.8	2.0	1.4	2.7	2.3	1.7	2.5
	N	7	7	7	7	6	6	5

CHAPTER FOUR

RETROSPECTIVE STUDY ON TWO INDEX FARMS

This Chapter comprises an examination and analysis of the reported and observed animal health problems on the two Askeaton Index Farms in the years immediately preceding the commencement of the Monitor Studies – i.e. up and including 1995. The background to the problems on these farms has already been outlined in the EPA Interim Report (EPA, 1995). The following analysis is based on information contained in the Retrospective Survey Report for each farm. Details of the methodology and background to the Retrospective Survey are given in Chapter Five. As discussed therein, it must be emphasised that much of the information for the Retrospective Survey was based on individual recall (i.e. herdowner and veterinary practitioner) and periods of up to ten or more years may have elapsed since the occurrence of some of the reported events. A list of all disease incidents reported in the two individual Farm Reports, together with available incidence data, is given in Appendix 13.

INDEX FARM A (ID 01)

(Interview date: 12-3-1996)

This is a dairy farm of approximately 24 hectares. Cow numbers on the farm between 1990 and 1995 ranged from a minimum of 28 in 1993 to a maximum of 36 in 1995. All replacements were reared on the farm. A more complete description of this farm is provided in the EPA Interim Report (EPA, 1995).

The main source of information on the animal disease history for this farm comprises a diary of events kept by the herdowner as well as the results of the interview carried out for the Retrospective Survey. Information made available from the herdowner's diary during the course of the Retrospective Survey investigation is listed in Appendix 7.

While the records for this farm are extensive, it is important to note that they are by no means comprehensive and, in many cases lack essential descriptive details. In relation to perinatal calf mortality, for example, while many losses are recorded, little information is provided regarding the circumstances or history of the events, i.e. whether born alive or dead, history of dystocia,

dam age, etc. There are also significant inconsistencies in the records. Again in relation to calf deaths, for example, while the herdowner's diary records ten deaths in 1988 (Appendix 7), the Retrospective Survey Report, which is based on an interview with the herdowner, refers to calf mortality problems as having commenced in 1989.

Conflicting information is also given in relation to cow losses prior to 1990. A report of a consultant's investigation of the farm in 1993 (Dowding and Dowding, 1994) - which collected contemporary information on animal losses at the time - refers to eight cow and 14 calf deaths. However, these figures conflict with the data in Table 4-2 below which is based on information collected from the herdowner's diary. There are also significant inconsistencies between the herdowner's diary and the Retrospective Survey Report in relation to the time-scales of some of the reported problems.

Though obviously referring to significant events, some of the information in the Retrospective Survey Report is also of little value for analysis owing to its anecdotal nature. In one case, for example, the veterinary practitioner is reported as referring to "... *a ruptured liver abscess in a heifer which died after calving*". However, in the absence of other clinical details, it is not possible to determine whether the animal died as a result of the ruptured liver abscess or whether the latter was a complication of a calving-related death. Information regarding either of these diagnoses would be particularly relevant to the analysis below of the reported problems of abscessation and perinatal mortality. As no date was given, it is also not possible to correlate the incident with any of the entries in the herdowner's diary.

Again, while the herdowner's description of a bullock which "... *bled from both hind feet within two weeks of returning to the farm*" is unusual, it is of little value in terms of identifying a cause. The animal in question had recently returned from being examined in UCD Veterinary Faculty Large Animal Clinic where no significant health problems had been noted.

In addition to the Retrospective Survey Report, the reports of several investigations carried out on Index Farm A in 1993 have been used as sources of

information. The main findings and conclusions of these reports are discussed further below.

ANIMAL HEALTH AND PRODUCTION PROBLEMS

The main problems reported by the herdowner, and which are described in some detail in the Retrospective Survey Report, were infertility, pining and mortality in cows and growing stock, increased perinatal calf mortality, diarrhoea in calves, skin lesions in cows and growing cattle, and irritability in cows at milking. While the exact time at which animal health performance deteriorated to a point where it was a matter of concern is not known, available records indicate that mortality on the farm was above average since 1988 (Table 4-2).

The main problems reported by the herdowner's veterinary practitioners were mastitis, infertility, pining, perinatal mortality and skin lesions in cows. The veterinary practitioners considered that there was an excess of animal health problems on the farm. They suggested that there was generally a poor response to treatment in the herd due to unknown underlying factors.

Infertility

Note: No direct comparison can be made between fertility performance on either of the Index Farms pre- and post-commencement of the Monitor Projects in 1996 as the herds were divided and new stock introduced for the purposes of the Projects.

Infertility was reported to have been a problem in Index Farm A since 1990. A summary of fertility performance for the years 1992 to 1995 is given in Table 4-1. However, it should be noted that the results for 1992 and 1993 are only of limited value for analysis as they are only based on a proportion of the herd (about a third of cows in 1992 and a half in 1993).

Both artificial insemination and a bull were used to serve cows in most years. While the Retrospective Survey Farm Report states that 'The primary problem was of cows repeating irregularly, up to 15 weeks after service.', it is clear from Table 4-1 that the underlying problem was one of heat detection. Non-detected oestrus (i.e. missed heats) and submission rates were outside target in all years. Because of this, the calving to service and calving to conception intervals were also above target. This led to an extended calving season in succeeding years which, by 1995, was seven months duration.

According to the herdowner, repeat-breeding was also a problem – with cows repeating at irregular intervals. Other fertility parameters were generally within the expected range of performance for herds

of this size (Appendix 1). Although an infertile rate of 27 per cent was recorded for 1992, this represented three cows not in calf of a total herd size for analysis of only 11 cows (i.e. full breeding data was only available for 11 cows in that year).

Perinatal calf mortality

Perinatal mortality is reported to have been a problem since 1989 and to have continued into the 1995 calving season. However, little information is available regarding the circumstances or calving history of calf losses up to and including 1993. Although the Retrospective Survey Report refers to six cases of 'perinatal mortality' (Table 18 in Retrospective Survey Report), it is unlikely that this reflects total losses for the period. While total calf losses of 45 are recorded for the period 1988 to 1995 in Table 4-2, insufficient information is available from farm records to determine what proportion of these were attributable to the immediate perinatal period.

References in the herdowner's diary (Appendix 7) suggest that leptospirosis may have been responsible for some calf losses in 1989. According to the herdowner's veterinary practitioner, vaccination against leptospirosis commenced in the autumn of 1989 in response to a history of stillbirths.

It would also appear that calving problems were a significant factor in relation to perinatal calf losses - at least in the early years. Both the herdowner and the veterinary practitioner refer to foetal oversize as having been a factor in certain years - though the veterinary practitioner states that "... dystocia was not a problem at all stillbirths.". In 1988, the herdowner noted that "calves were larger than usual ... (and) ... there was an increased incidence of calving difficulty and veterinary assistance at calving".

A number of references in the Retrospective Survey Report are consistent with the effects of intrauterine hypoxia on calves during delivery. These include the veterinary practitioner's reference to "... fluid in the second water bag (being) abnormally viscous, brown or bloody ..." and the herdowner's to "... jelly like sticky dark yellow/brown mucous ...". These indicate the presence of meconium (foetal intestinal contents) which is a characteristic finding in calves which have experienced hypoxia prior to birth (Randall, 1978; Lopez and Bildfell, 1991). Hypoxia occurs during difficult or prolonged calvings and may result in the death of the calf or the birth of a weak calf which may die shortly after birth. According to the herdowner "... most affected calves were alive

at birth but died within minutes ...". The Dowding report (Dowding and Dowding, 1994) also states that "... (the) *newborn calf would take a few steps and die ... intensive nursing* (was) *required to keep them alive ...* (they were) *slow to suck ...".* These descriptions are consistent with the effects of hypoxia on the central nervous system during delivery.

All six of the calves which died in 1994 were submitted for laboratory post-mortem examination. Histories and findings are given in Table 4-3. Four of the six were full-term. Two of these had lesions consistent with BVD virus infection; there were no specific findings on the other two. Diagnoses of colisepticaemia and enteritis were made in the case of the remaining two calves which died in 1994. Two calves are reported to have died in 1995. A diagnosis of death consistent with intrauterine hypoxia was made in relation to one submitted for laboratory post mortem examination (Table 4-3).

Perinatal calf mortality was not a problem during the period of the Monitor Study from 1996 to 1998 (Chapter Two).

Diarrhoea in young calves

Diarrhoea is reported to have been a problem in young calves since 1990. According to the herdowner, calves were generally affected in the second week of life. Calves born indoors were most often affected. However, little information is available on the incidence of the problem. Table 18 in the Retrospective Survey Report - which lists clinical signs prior to death in cases where information was available - only refers to three cases of calf diarrhoea. Three of the four post-perinatal calves (i.e. over one day old) submitted to Limerick RVL between March 1991 and December 1995 had post-mortem findings consistent with diarrhoea. However, it is unlikely that either of these statistics are an accurate reflection of the problem. In the circumstances, it is not possible to draw any conclusions regarding the incidence, severity or cause of calf diarrhoea problems on this farm up to 1995.

Other than the single outbreak of relatively low severity which occurred in spring 1997 (Chapter Two), diarrhoea was not a problem in calves during the period of the Monitor Study from 1996 to 1998.

Pining and mortality in cows and growing stock

Although pining and mortality in cows and growing cattle is reported to have been a problem since 1991, examination of mortality data from the herdowner's diary (Appendix 7) indicates that there were significant adult losses from 1988. Three cow deaths are recorded for each of the years 1989, 1990 and 1991 (Table 4-2).

Incidence data for pining are only available for 1992. In that year, seven to nine of 33 cows and heifers were said to have been affected by pining and four died. The first reported case was a three-year-old heifer which lost body condition after calving in July/August 1992 and died in December of the same year. The animal was not submitted for laboratory examination and no specific clinical diagnosis was made. Although none of the other four deaths in that year were submitted for laboratory post-mortem examination, clinical diagnoses of mastitis (two cases), downer cow (one case) and leg injuries (one case) are given in the Retrospective Survey Report. According to the herdowner's account of the incidents:

"affected cows were generally in moderate body condition pre-calving. Pining occurred after calving. in the spring. Some went 'down' pre or post calving and others did not put on body condition over the summer and failed to milk well, especially in 1993, despite good herbage quality and quantity."

Few other specific clinical signs were noted – though some (number unspecified) were said to have skin 'lumps'. According to the herdowner's veterinary practitioner:

"pining in adult cattle was a definite herd problem. Internal abscessation was suspected as a cause in many cases. The response to treatment in pining animals was poor, often resulting in downer animals. Such cases were associated with bad winters, heavy rainfall and yarded animals. Illthrift, without any other specific signs, was also a problem in weanlings. They generally improved when moved to an out-farm."

Summary details of necropsy submissions to Limerick RVL up to the end of 1995 are given in Table 4-6. Prior to 1993, the only pathology submissions to Limerick RVL comprised a calf in 1991 and an aborted foetus in 1992. Findings consistent with diarrhoea were recorded in the former and no significant changes in the latter. Of the ten adult or growing animals which died in 1993, only three cows and one weanling were submitted for laboratory post-mortem. Diagnoses were made in all cases. Two of the cows had lesions of suppurative mastitis with secondary abscessation. *Actinomyces pyogenes* was isolated from the lesions. The third died as a result of hypomagnesaemic tetany – a common metabolic condition in grazing adult cattle. The weanling had lesions consistent with BVD virus infection.

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The single cow to die in 1994 had lesions of multiple internal abscessation secondary to severe septic foot and skin infection. *Dermatophilus congolensis*, a common cause of pustular dermatitis in cattle (Scott, 1988), was isolated from the skin lesions. A 10-month old weanling is also reported to have died from a 'rupture' (Appendix 7) in 1994. The carcase was not submitted for laboratory post-mortem examination.

Of the three terminally ill cows which were euthanased and submitted for laboratory examination in 1995, one submitted in March 1995 had lesions of parasite infestation and the other two were in very poor body condition and exhibited excessive tooth wear (incisors worn to gum level). The latter finding can be associated with cows feeding from a compacted silage face and, once tooth wear has developed beyond a certain point, silage intake can be reduced to the point where malnutrition results.

Although growing animals - weanlings and older dry stock - were also said to have been affected, little information is provided in the Retrospective Survey Report regarding signs or incidence. The only clinical description is that they " *were in poor body condition ... had poor coats, (and) ... no signs of respiratory disease or diarrhoea*". Other than the weanling with lesions of BVD in 1993 (*see above*), no animals in this age-group were submitted for laboratory post-mortem examination.

Skin lesions in cows and growing cattle

According to the herdowner, this problem began in 1990 and continued until 1994. The following are the herdowner and veterinary practitioner descriptions from the Retrospective Survey Report:

"All ages of cattle, including the bull, except calves, were affected, both indoors and at grass, particularly in the spring. Between 1992 and 1994 the farmer estimated that up to a third of the cows had lumps under the skin. This problem was most severe in 1992 and 1993. The lumps occurred all over the body, with up to 30 lumps/cow varying in size up to the size of a plate. In some cases the lumps fell off and the hair fell out resulting in the cows licking at the lesion resulting in bleeding. "

and

"... lumps all over the body of certain cows including in the legs and udder, varying in size from that of a sliotar to that of a football. Once the abscess fell off, the animal recovered. Up to 12 cows were affected in 1994. There was a poor response to antibiotic treatment of these cases. The private veterinary practitioner diagnosed this

problem as superficial abscessation in an unusually large number of animals in the same herd, without obvious cause. ... (The veterinary practitioner) suggested animals may have been immunodeficient, unable to prevent normal bacteria causing problems. He also noted that it was easy to rub the hair off the cows backs and the skin appeared reddened. ... At grass some cows, in both the black and white skin, had scabs and alopecia resembling rain-scald. As it was not rain-scald no treatment was given (in these cases). "

References to the problem are also made in reports of a number of farm investigations carried out in 1993. A report on a field visit by an RVL veterinary officer on 23 April 1993, the day after two cows had been received at the laboratory for post-mortem examination (Table 4-3) states that:
"... (although) most of the animals were in good condition ... three 1½ year old cattle were seen to have pyoderma at different body sites which were all discharging pus. ... A cow was observed with a severe mastitis displaying pus-discharging fistulae. " .

Only *Strep. faecalis* (an opportunist invader or secondary contaminant) was isolated from swabs of the skin lesions collected during this visit. The report concluded that the incidence of pyoderma was exceptional.

A report by a private laboratory (see below) of June 1993 refers to *"two or more cows (with) multiple subcutaneous abscesses ..."*. The Dowding report (Dowding and Dowding, 1994) noted one cow in August 1993 with evidence of healing subcutaneous lesions. A reference in the report to *"two cows sent to (the) factory in April 1993 ..."* suggests that they may have had suppurative mastitis and secondary abscessation.

No active cases of pyoderma were observed at a further farm visit by DAF laboratory staff in December 1993, but the report of the visit refers to " *... evidence of healed pyoderma lesions ..."* in some (number unspecified) animals in a mixed-age group.

Other than the field visit by an officer of Limerick RVL in April 1993, the only laboratory investigation of this problem involved the four cows submitted to Limerick RVL in 1993 and 1994 for post-mortem examination (see above) and one submitted to UCD Veterinary Faculty Large Animal Clinic in July 1993. A diagnosis of shoulder abscess (also with evidence of a pulmonary abscess) was made in relation to the latter. A *Streptococcus* species was isolated from the shoulder lesion.

Irritability in cows at milking

The following extract from the Retrospective Survey Report is based on the herdowner's account of the problem:

"This problem began in 1992 approximately and some 10 to 12 cows were affected annually. Affected cows were difficult to milk, kicking the milker, necessitating the use of 8 'kick bars'. This problem is ongoing and occurs after cows graze 'coloured grass'. While the milking machine has been serviced, the presence of stray electricity voltage has not been examined. The practitioners recalled individual excitable cows at TB tests and queried whether the skin abscesses could cause this problem of irritability. The farmer attributed this problem to airborne pollution."

There are a variety of possible explanations for this behavior ranging from cow temperament to management factors. It is obviously not possible at this stage to make a diagnosis. As the problem was only said to affect a proportion of cows it is unlikely to have been environmental in origin. In the circumstances, it is not possible to determine what, if any, significance this condition had in relation to the historical animal health problems on this farm. It was not reported to have been a problem during the period of the Monitor Study.

Mastitis

Although mastitis is not specifically referred to as a herd problem in the Retrospective Survey Report, there is ample evidence that it was a significant factor in relation to the serious animal health problems between 1992 and 1994. In addition to the four cases of suppurative mastitis in 1993 referred to above, the post-mortem report on the cow submitted for post-mortem examination in December 1995 refers to the presence of 'mammary fibrosis' - which is evidence of a history of chronic mastitis.

The veterinary practitioners also considered mastitis to have been a problem on the farm and their call log indicates that approximately one third of all calls to the farm in 1994 and 1995 were to mastitis cases. Bulk milk cell counts (SCC) were also above 400,000 for each of the years 1992 – 1994 (range 402,000 – 701,000) which is consistent with a significant degree of udder inflammation.

Reduced milk yield

Annual milk sales (allowing for estimated quantities fed to calves) for the period 1987 to 1995 are given in Table 4-4. Sales showed an overall decline from around 113,650 kg in 1987 to

63,644 kg in 1994. They reached their nadir in 1992 at around 59,098 kg with an estimated yield per cow of around 2,159 kg for the year. This compared to an estimated yield per cow of around 3,410 kg in 1987 before the problems began.

While highly unsatisfactory, this performance is hardly surprising given the animal losses and health problems encountered during the period. In addition to the direct effect of illness on cows' yields, replacement of losses with first-calvers will also have contributed to an overall reduction in herd yield. A further reduction will have resulted from the fertility problems which led to an extended calving season and consequent loss of synchrony between calving and periods of optimum grass growth. Mastitis is also likely to have had a negative effect on yields.

EARLY FARM INVESTIGATIONS

Several farm investigations were carried out in 1993 which identified nutritional factors as contributing to animal health problems. The first of these, by a veterinary officer of Limerick County Council in January 1993, concluded that there was evidence of iodine and selenium deficiency. The second, commissioned by Aughinish Alumina and carried out by a private laboratory in June 1993, concluded that the problems were due to 'impaired immune function' associated with inadequate availability of copper, iodine and selenium. The third investigation, commissioned by Limerick County Council, commenced in August 1993 and concluded that many of the reported animal health and production problems were consistent with iodine deficiency (Dowding and Dowding, 1994). A fourth study, also on commission from Aughinish Alumina, was carried out by a second private laboratory in September 1993. The report of this investigation referred to evidence of inadequate copper and iodine and high selenium.

Although all four of these investigations agreed on the finding of iodine deficiency on the farm in 1993, the possibility that inadequate iodine status was responsible for the reported animal health problems remains open to serious doubt. The conclusions of these investigations regarding the iodine status of the farm were based on the results of blood and herbage analysis results. However, the reported concentrations for herbage iodine of between 0.1 and 0.5 mg/kg in 1993, though low, are not untypical of Irish pastures (McGrath and Fleming, 1988). As animals receive most of their iodine from the soil, this is the preferred source for analysis.

Although no soil analysis results are available for 1993, extensive soil tests carried out by Teagasc in 1995 on the farm demonstrated concentrations of around 5.9 mg/kg iodine, which is an acceptable value. Given that the bulk of environmental iodine replenishment comes from the sea and lakes *via* atmospheric moisture and rain (not *via* animal manure as suggested in the Dowding report of the 1993 investigation - Dowding and Dowding, 1994) - and is unlikely to vary considerably from year to year - it is reasonable to assume that similar, and therefore adequate, values obtained in 1993.

The blood analyses on which the conclusions of the 1993 reports were based comprised thyroxine and plasma inorganic iodine (PII) estimations. However, due to its extreme variability, blood thyroxine is not regarded as a reliable estimator of iodine status (Convey *et al.*, 1977; Rogers *et al.*, 1993; Mee *et al.*, 1995). In addition, 14 of the 15 blood samples collected from adult animals in January 1993 had thyroxine concentrations which were within the reported normal range of 54 - 111nmol/l (radio immune-diffusion analysis - Kaneko, 1989). Only six of the 18 samples referred to in the June 1993 private laboratory report were significantly below the normal range and no information is given regarding the types of animal from which these samples originated. Given that blood thyroxine values are strongly influenced both by age and reproductive status, it is not possible, therefore, to determine the significance of the thyroxine analysis results.

Although low plasma inorganic iodine (PII) concentrations were detected in blood samples collected from animals on the farm in January 1993, this is also not regarded as a reliable indicator of clinical iodine deficiency (Whitaker, 1999). Blood PII is an indicator of current iodine intake. Animals on low iodine rations, e.g. unsupplemented grass or silage, are likely to have low values while animals on higher iodine diets, e.g. concentrates, will have correspondingly higher values. Low PII values are commonly found in blood samples from Irish herds. Between a third and two-thirds of all blood samples tested at Teagasc Grange Laboratory from 1991 to 1997, inclusive, had low PII concentrations (Rogers, 1997). Despite this, problems of the type or severity seen on the Askeaton farm are not similarly widespread.

The clinical signs of iodine deficiency are well described (Jubb *et al.*, 1991). In the pregnant cow, it may lead to aborted and stillborn calves – some of which are likely to have enlarged thyroids, i.e. goitre (Seimiya *et al.*, 1991). Goitre can also occur in affected growing animals and adults. However,

outbreaks of this type have generally been associated with severe deficiency in areas where human goitre has also been recognised as a problem (Hetzel *et al.*, 1990). Although stillbirths and perinatal calf mortality were a problem on Index Farm A, there is little experimental evidence in the literature to support the view that they can be caused by a marginal deficiency of the order observed on this farm.

In addition, no evidence of goitre or follicular hyperplasia was detected on examination of thyroids from calves submitted for pathology examination. The results of recent studies have raised significant doubts regarding the role of iodine deficiency in stillbirths and weak calf syndrome in cases where there is no gross or microscopic evidence of thyroid hyperplasia (McCoy *et al.*, 1995). McCoy *et al.*, (1997), failed to induce abortion or stillbirth in heifers on experimentally-induced iodine-deficient diets.

The presence of copper deficiency is mentioned in the reports of both of the private laboratory investigations in June and September 1993. However. little evidence is presented in either report to support this finding. None of the reported blood copper concentrations were below the normal range of 9.24 – 24 µmol/l (converted from Puls, 1994). Although milk copper was referred to in the September report as being low, with the exception of iodine, milk mineral concentrations are not generally regarded as reliable indicators of trace element status (Puls, 1994). Other than expected seasonal variations, extensive analysis of blood samples collected from animals on the farm during the period of the Monitor Study failed to reveal evidence of a significant herd problem with copper supply (Chapter Two).

Evidence for the presence of selenium deficiency is also equivocal. None of the reported glutathione peroxidase concentrations (a selenium-containing enzyme) were below the minimum recommended value of 40 units/ml PCV. Although the report of the January 1993 investigation commissioned by Limerick County Council referred to low herbage selenium concentrations, extensive analysis of soil and herbage carried out by Teagasc in 1995, 1996 and 1997 indicated that while some results were marginal others were high and overall, selenium supply on the farm was likely to be adequate. Blood selenium concentrations were also generally within acceptable limits throughout the period of the two-year Monitor Study on this farm (Chapter Two).

The suggestion in the June 1993 private laboratory report that the problems were due to *'impaired*

immune function' is also not supported by the laboratory evidence and appears to be solely based on the presence of a *'relative neutropenia'* and absence of band (immature) neutrophils in stained blood smears. Besides the fact that an assessment of immune function could not be made on the basis of a routine haematology analysis, the reported total neutrophil counts (1.16-$3.17 \times 10^9/l$) are within the normal range in all six samples and the absence of band neutrophils cannot be taken to be of any clinical significance. The normal range for bands in the bovine is from zero to 120 cells/µl (Schalm *et al.*, 1988).

The reference to raised IgG concentrations indicating *'an attempt to compensate for reduced phagocytic activity'* is also speculative. In the first case, no evidence of reduced (neutrophil) phagocytic activity is provided and in the second, there is little if any scientific basis for the suggested association between phagocytic activity of neutrophils and IgG production by lymphocytes. Although increased levels of serum IgG and IgM have been reported in cattle affected with BLAD - a genetic immune system defect (Nagahata *et al.*, 1994) - there is no evidence to suggest that the increased immunoglobulin concentrations are linked to reduced neutrophil phagocytic activity.

In conclusion, while marginal iodine, copper, and possibly also selenium status may, at times, have had a limiting effect on animal performance. it is highly unlikely that they could have been responsible for the severe disease problems reported on this farm between 1991 and 1995. As the EPA Interim Report (EPA 1995) stated "... *although herbage selenium, copper, iodine (and zinc) were lower than those considered adequate for animal nutrition, they were not abnormal in an Irish context".*

EPA-COORDINATED INVESTIGATIONS IN 1995

Although reported animal losses were at their height on this farm in 1992 and 1993, conditions on the farm were not good when the EPA-coordinated investigation got underway in early 1995. The Teagasc report of the farm inspection of 22 March 1995 (EPA, 1995) stated that it had been severely grazed in the previous months and there was heavy poaching of the pastures - particularly along boundaries and hedgerows. The latter were also said to have shown signs of grazing. The farm was also overstocked - largely owing to the inability of the herdowner to sell dry stock which were in poor condition. The report of a joint VLS/Teagasc visit to the farm on 23 March 1995 refers to the generally poor condition of about 30 weanlings present on the farm at that time. Cows were reported to range from poor to good body

condition. Other than patchy alopecia (hair loss) on some cows, which was probably due to lying in passageways, skin lesions were not a problem.

CONCLUSION

The severity of the animal health problems experienced on this farm is beyond question. The purpose of the present exercise has been to describe the nature and extent of the historical problems and, based on available information, to attempt to identify the causes and any underlying factors which may have contributed to the overall high incidence of disease. Unfortunately, as already noted in the EPA Interim Report (EPA,1995), this exercise is severely hampered by the almost complete absence of laboratory or other specialist involvement throughout most of the period during which above-normal losses occurred. Despite the fact that 13 cows and 33 calves were reported to have died in the five-year period from 1989 to 1992, the only pathology submissions to Limerick RVL in the same period comprised a calf and a foetus. Prior to 1993. no consultations took place with Limerick RVL regarding the severity of the developing problem. This is particularly surprising given the apparently chronic nature of the illnesses in many of the affected animals. Even following involvement of the RVL in 1993, only a minority of adult bovine losses was submitted for pathology examination in that year. Although almost all losses were submitted in 1994, all but one were new-born or young calves and therefore not representative of the severe adult losses seen in previous years.

For the purposes of the present analysis, the main problems of interest on the farm were infertility, calf deaths and illness (including pining and skin lesions) and deaths of adult animals. Although irritability of cows at milking was also reported to have been a problem this cannot be investigated further at this stage due to its transitory nature. Mastitis, though not specifically mentioned in the Retrospective Survey Report as a problem, was sufficiently widespread in some years to have had a significant negative impact on milk quality. Milk production also declined sharply throughout the period as a direct consequence of cow illness and deaths. These problems are discussed individually in the following paragraphs.

While fertility performance was reported to have been below target, the main problems - i.e. reduced heat detection and repeat-breeding - are not unique to the Askeaton area and are commonly seen on farms elsewhere. As discussed elsewhere (*see* page 5 *et seq.*). heat detection is largely a function of

management and there is no specific evidence to suggest that such was not the case on this farm. On the contrary, according to the Retrospective Survey Report, heat detection was carried out "... *in the yard at morning and evening milking and during the day, if cows were in paddocks close to the yard*". While this is probably a fairly typical regime for a small non-intensive dairy herd, it is substantially short of the generally accepted recommendation that, for the first four weeks of the breeding season at least, heat detection should comprise four 20-minute periods of observation daily (Sreenan and Diskin, 1984). The fact that heat detection aids such as tail paint (cows) and a chin-ball marker (bull) were not used, can only have further reduced performance.

Conception performance is also dependent on a range of cow, bull and management factors. While it is not possible at this remove to identify those responsible for reduced performance in the present case, the veterinary practitioner's comment that repeat-breeding "... *primarily affected cows following metritis with individual cows in poor body condition also affected*" is consistent with experience elsewhere (Oltenacu *et al.*, 1990; Ferguson, 1991). Although a bull was used in some years, the possible contribution of bull infertility to the problems cannot be determined as there is no record of fertility examinations having being carried out on bulls.

Overall, therefore, while fertility was well below target in certain respects, performance could not be described as exceptionally poor. The main problems - i.e. heat detection and repeat-breeding - are not uncommon and their consequence, an extended calving season, was an inevitable outcome. Despite the poor heat detection performance, the pregnancy rate remained over 80 *per cent* in most years. The use of a stock bull in some years probably helped to maintain conception rates at a reasonable rate.

While the specific causes of the reduced fertility performance on this farm cannot be definitively identified at this stage, they are far more likely to have been related to the more commonly-accepted animal and management risk factors than to environmental pollution. Although the possibility that mineral deficiencies contributed to infertility was considered during the early investigations, the results of detailed soil, herbage and blood analysis carried out between 1996 and 1998 indicate that mineral nutrition is unlikely to have had more than a marginal limiting effect on cow fertility.

Reported calf mortality rates were well above average and indicate a serious problem in some years. Forty five calf deaths are reported to have occurred during the period 1988 to 1995. While the history indicates that the majority were either calving-related or were due to diarrhoea, there is insufficient evidence to apportion losses between the two categories. However, the descriptions of the problems do not indicate any unusual features and there is sufficient evidence to suggest involvement of risk factors which are commonly associated with these problems elsewhere.

Both perinatal calf mortality and calf diarrhoea are common problems world-wide. The former, which may also be termed 'weak calf syndrome' is most frequently associated with difficult or prolonged calvings (Menzies *et al.*, 1996; Collery *et al.*, 1996). Based on available evidence, it is clear that dystocia was a factor in many of the perinatal calf losses on this farm. Some of the clinical descriptions regarding stillborn or weak-born calves in the Retrospective Survey Report are consistent with the effects of hypoxia. The latter is probably the most common cause of death in calving-related losses (Kasari, 1989).

Infectious agents and environment (i.e. housing, feeding and hygiene) are the usual risk factors for calf diarrhoea. Although the incidence of diarrhoea may have been high on this farm, there is insufficient information to confirm this. The fact that the problem was reported to have occurred primarily in calves born indoors is consistent with a build-up of infection during the calving season – a common finding in outbreaks elsewhere. This is supported by the veterinary practitioner's reference to a *"problem* (of) *deep bedding in the* (calf) *house"*. Coccidiosis, a protozoal infection, was also apparently a recurrent problem on the farm.

By far the most severe problem experienced on this farm prior to 1996 was that of illness and deaths of adult animals. Between 1988 and the end of 1995, approximately 29 cows are reported to have died or been culled due to illness. An unknown number of other cows were also reported to have been affected and recovered.

Although weanlings and other growing stock were said to have been affected, only five losses are recorded for this age-group in the Retrospective Survey Report and only one animal was submitted for laboratory post-mortem examination. Because of this, no specific conclusions can be drawn regarding possible causes of the problems in growing stock.

Even in relation to adult stock, diagnoses can only be made or suggested for a small proportion of cases owing to the very limited nature of

contemporary veterinary investigations. Calving problems - though primarily mentioned in relation to calf deaths - may have accounted for some of the cow losses in the early years. Clinical diagnoses of mastitis, downer-cow and trauma are recorded for four cows which died in 1992. Diagnoses of mastitis, grass tetany, parasitism and secondary malnutrition were made on six cows which were submitted for laboratory post-mortem examination between 1993 and 1995. The causes of illness and deaths in the other cases - by far the greater part of the total affected - cannot be determined at this stage owing to the absence of contemporary records.

The description of an outbreak (or outbreaks) of subcutaneous abscessation and skin lesions in cattle on this farm between 1990 and 1994 is unusual. While insufficient information is available to determine the full extent of the problem - either in terms of the number of animals affected or the exact period during which cases occurred - it is clear that it cannot be considered in isolation from the pining and mortality in cows discussed above. According to the herdowner, the problem was at its worst in 1992 and 1993. These were also the years when cow mortality peaked.

However, again owing to the limited availability of contemporary clinical and pathology data, the underlying causes of the majority of cases must remain a matter of speculation. According to Jubb *et al.* (1991) - a standard veterinary pathology text - virtually all bacterial pyodermas (abscesses) are secondary to exogenous or endogenous triggering factors.

The commonest exogenous causes are penetrative wounds to the skin (Radostits *et al.*, 1994). In a farm situation, these are generally associated with injection site reactions or with trauma against sharp objects such as cubicle rails, door-posts, gates and fencing. However, while these risk factors were probably responsible for occasional cases of subcutaneous abscesses on this farm - as they would have been on any farm - it is unlikely that they could have accounted for the unusually high incidence of the condition in 1992 and 1993. Although the problem had virtually disappeared by 1995, there is no evidence that this was associated with significant structural modifications to the housing or external environment.

Prolonged wetting of the skin can also predispose to infection. It is likely that a number of cases of skin lesions in 1994 - possibly the majority - were due to *Dermatophilus congolensis* infection. This organism, which is the causative agent of dermatophilosis, was isolated from the animal

submitted for post-mortem examination in 1994. Dermatophilosis occurs most frequently during periods of wet weather (Scott, 1988). The winter of 1993-94 and spring of 1994 were particularly wet. Rainfall was above normal in each of the five months from December 1993 to April 1994. In addition, the veterinary practitioner's descriptions of some cases as "*... easy to rub the hair off the cows backs ... skin appeared reddened ... hair loss and gray crusted skin ... around the eyes*" are consistent with dermatophilosis.

Endogenous causes of superficial abscesses mainly comprise secondary localisation of infection from other suppurative lesions in the body. Suppurative mastitis was undoubtedly responsible for a proportion of the cases on this farm. However, other than the two laboratory-confirmed cases of mastitis in 1993 (and possibly two others culled to the factory around the same time; *cf* Dowding report - Dowding and Dowding, 1994) - no estimate can be of the extent to which suppurative mastitis contributed to the problem.

The most significant feature of the outbreak of skin lesions, however, is that it was clearly part of a larger problem of animal disease at herd level. The overall picture on this farm was of adult animals under severe health stress over a relatively prolonged period of time. In the circumstances, therefore, it is likely that many cases of abscessation were a secondary consequence of reduced resistance to disease in animals of already poor health and condition.

This, of course, still leaves unanswered the larger question regarding the underlying reason for the high incidence of illness and death in adult cattle on this farm. Given the diversity of reported syndromes, it is unlikely that there was a single cause. Although diagnoses can only be made or inferred for a proportion of affected animals, there is little evidence of a common pattern among the cases where diagnoses were made - either the clinical diagnoses reported for 1992 or the few post-mortem diagnoses available from 1993 to 1995. In relation to the latter, three had lesions which were due to bacterial infections, one to parasite infestation, one to viral infection (weanling), and two were probably nutritionally compromised due to excessive tooth wear.

On the basis of the available evidence, therefore, the most reasonable conclusion which can be drawn would appear to be that while the majority of problems were probably of diverse and unrelated aetiology, there were also in existence, at times between 1988 and 1995, one or more underlying factors which had the effect of

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increasing the severity of some diseases and perhaps, even of reducing the overall level of herd resistance to infectious disease.

While the precise identity of these factors cannot be determined at this stage, a number of potential candidates can be ruled out. In the first case, it is clear that there is no evidence that a single infectious entity was responsible for all or .even the greater part of the losses. Though infection was identified as the cause of illness and death in several cases, the agents concerned were unrelated. The possible involvement of Bovine Immunedeficiency Virus was ruled out on the basis of negative blood test results in December 1994 (EPA, 1995). In the second, while the supply of specific mineral nutrients such as copper and selenium was probably marginal at times, there is no evidence that mineral deficiency was a significant contributory factor to the main problems reported.

There is a broad range of other animal, management (nutrition, housing, hygiene, health care) and environmental influences which may have had an overall negative effect on animal health. Animal age, breed and body condition, probably played a role in relation to the fertility and calving-associated problems - as they do on all farms. There is also little doubt that management was an important determinant of fertility performance - as is also the case elsewhere (Esslemont and Kossaibati, 1996). Management undoubtedly also played a significant role in relation to calf mortality (sire selection, calving management, calf housing and hygiene). Again, this statement can be applied to virtually all farms as management factors are probably the major determinants of calf health and survival (Lance *et al.*, 1992).

However, regarding the role played by animal and management factors in relation to the most serious problem - that of adult bovine illness and deaths – the situation is less clear. Although inadequate nutrition was undoubtedly the main reason for the poor condition of animals on the farm at the start of the EPA investigation in 1995 ("... *overstocking, severe poaching of land, grazing of hedgerows, poor quality silage* ..."; EPA 1995) the problem of mortality of adult stock was well past its peak at this stage. On the other hand, there is insufficient information to determine the nutritional state of stock on the farm in earlier years. While two of three silage samples analysed in 1993 were of poor quality (DMD 63.2% in April and 59.8% in October 1993), no information is available regarding concentrate supplementation which may have been provided to make up the deficit. The

only information in relation to cow body condition at this time, is the veterinary practitioner's reference to the poor condition of "*individual cows*" in relation to the fertility problems.

Potential environmental influences which may have affected animal health comprise weather and atmospheric pollution. While it cannot have been the primary cause, poor weather undoubtedly contributed to the severity of some of the problems described. As discussed elsewhere (*see above* and pages 101 and 141), weather conditions over the winter-spring periods of 1993-94 and 1994-95 were cold, wet and windy. According to the veterinary practitioner, the pining and mortality problems in cows were associated with "... *bad winters, heavy rainfall and yarded animals.*".

The suggestion that environmental pollution was responsible for the problems on this farm has been the primary driving force behind the entire investigation of animal health in the Askeaton area. However, almost the sole apparent link with pollution throughout has been one of location, i.e. proximity to sources of potentially noxious emissions. On the other hand, no evidence has been found to date either of the presence of potentially toxic concentrations of pollutants in the area or of animal disease problems which could readily be identified with a specific toxic cause. On the contrary, most of the disease problems reported comprised conditions which are commonly seen elsewhere. The unusual feature of their occurrence on this farm relates to their incidence and severity. The question then is, could environmental pollution have been responsible for, or contributed to, the clearly higher than average incidence of animal disease on this farm?

In relation to the reduced fertility performance and calf mortality secondary to calving difficulties, the answer is most probably 'no'. Besides the fact that potentially straightforward explanations exist for their occurrence, environmental pollution could have had little bearing on performance parameters such as heat detection, conception and ease of calving. On the other hand, while the severe health problems in cows, as well as any of the other problems due to infectious agents, may well have been associated with a degree of reduced resistance to disease, to attribute it to environmental pollution begs the question as to why the problems would appear to have been unique to this farm. Similar problems were not recorded elsewhere in the area between 1988 and 1993. Although a high incidence of adult losses also occurred on Index Farm B (*see below*), these were in 1994 and 1995 which was after the period when a high incidence of unexplained losses occurred on Farm A. None of

the other 25 farms in the Retrospective Survey reported problems of comparable type, severity and duration throughout this period (Chapter Five).

In conclusion, it is clear that there was a high incidence of animal health problems on this farm at times between 1988 and 1995. While many cases were unexplained, it is likely that this was due more to the failure to recruit the appropriate degree of specialist investigative assistance throughout most of the period when exceptional losses occurred than to any specific features of the conditions reported. While calf mortality was unacceptably high in some years, potentially straightforward explanations involving the commonly-accepted risk factors could have accounted for the bulk of cases. The same applies to infertility. Although performance was outside target in some years, it was not exceptionally so and there would not appear to be any justification to cite unusual causes.

One area where a major question mark must remain is in relation to the occurrence and causation of pining, abscessation and deaths in adult cattle. Both morbidity and mortality were unusually high. While diagnoses were made or have been suggested for a proportion of the cases, the cause or causes of the majority of cases must remain unknown. However, this again is largely due to the inadequacy of contemporary records and, on its own, is an insufficient basis to suggest that unusual factors such as environmental pollution were involved.

On the contrary, the weight of negative findings in relation to environmental pollution throughout the investigation, together with the absence of a clearly defined disease syndrome − or group of related syndromes - argue strongly against the presence of a single underlying cause such as environmental pollution. In the circumstances, it can only be concluded that a combination of factors, probably all of which - with the exception of weather- were unique to this farm, acted in concert over a period of years to contribute to an increased incidence of illness and death in adult cattle. There is no evidence to suggest that environmental pollution was involved.

INDEX FARM B (ID 02)

(Interview date:27/8/96)

This is a dairy farm of approximately 34 hectares. Cow numbers on the farm between 1990 and 1994 ranged from a minimum of 69 in 1990 to a maximum of 74 in 1991. Breeding replacements were generally bought-in annually. By 1995, there

were only 52 cows on the farm due to non-replacement of animal losses (*see below*). A more complete description of this farm is provided in the EPA Interim Report (EPA, 1995).

Animal health and production records for this farm are far less extensive than those for Index Farm A. Although reference is made to disease problems extending back to the late 1980s are given in the Retrospective Survey Report - and are summarised below - little detailed information is available on animal disease and mortality prior to 1994. Even for 1994, the first year in which substantial losses were reported to have occurred, clinical details of disease incidents are scanty.

ANIMAL HEALTH AND PRODUCTION PROBLEMS

The main problems reported by the herdowner, and which are described in the Retrospective Survey Report, were illthrift, illness and mortality in cows and growing cattle, abortion, calving difficulty and infertility in cows, and illness and deaths in calves. The main problems encountered by the veterinary practitioners were cow deaths, listeriosis in cows, calf diarrhoea, ketosis, salmonellosis, mastitis and lameness. They considered that there was an excess of animal health problems in the herd.

Details of animal mortality on the farm for the years 1990 to 1995, based on information supplied by the herdowner, are given in Table 4-5. Summary details of necropsy submissions to Limerick RVL up to the end of 1995 are given in Table 4-6.

Abortion

Abortion was reported to have been an annual problem in the herd − though exact figures for annual losses are not given. A brucellosis abortion 'storm' occurred in 1984 following which the farm was depopulated. Thereafter, abortions were reported to have occurred at the rate of one or two per year. Peak incidence was in 1994 when four were reported. *Listeria monocytogenes* was isolated from one of two aborted foetuses submitted to Limerick RVL in spring 1994 and again from a foetus submitted in January 1995. The veterinary practitioners also referred to abortions due to *Listeria* infection having occurred in previous years. Baled silage was considered the likely source. Serological evidence of active *Leptospira hardjo* infection was also demonstrated in the herd in 1989.

Calf diarrhoea and pneumonia

Calf diarrhoea and pneumonia were reported to have been an annual problem with losses at about six to seven calves per year. However, little

information is supplied regarding numbers of calves affected with either condition. It is also unclear as to when the conditions were first regarded as a problem. Although the data in Table 4-5 covers the years 1990 to 1995, reference is also made in the text of the Retrospective Survey Report to high mortality in 1987 associated with a severe outbreak of enterotoxigenic *E. coli* infection.

The veterinary practitioner is also quoted in the Retrospective Survey Report as referring to a serious problem with calf diarrhoea – though this comment may only refer to 1995 as, while four calls to pneumono-entritis in calves were listed in the practitioner's call log for that year, none were listed for any of the years 1990 to 1994, inclusive. Salmonellosis was diagnosed on a number of occasions in both calves and cows. Calf losses due to dystocia were not considered to be a significant problem.

Reference is made in the Retrospective Survey Report to an outbreak of respiratory disease in calves (no date given) which was considered to have been due to IBR virus infection. No carcases or other clinical pathology material from cases of diarrhoea or pneumonia were submitted to Limerick RVL up to and including 1995. The report of a VLS visit to the farm on 23 March 1995 noted that suckled calves were housed in loose boxes with inadequate ventilation.

Illthrift, illness and mortality in cows and growing cattle

During the early 1990's, one to three cows were reported to have died each year (1-4 *per cent*) - in most cases following milk fever or gangrenous mastitis. According to the herdowner, the first unexplained death occurred in 1993 when a cow was found dead in a field in December of that year. No post-mortem examination was carried out. Also in 1993, a problem of cows showing difficulty in rising in the cubicle sheds became apparent. It was more serious in 1994 and 1995 and, as a result, the majority of cows had to be outwintered in these years. It is reported that on one day five cows had to be assisted to rise in the cubicle houses. All ages of cows were affected. In relation to this problem, the veterinary practitioners refer to *"lameness ... in older cows with overgrown claws"*, and also to *"hock injuries associated with cubicle layout"*.

The herdowner's veterinary practitioner reported that while no obvious problems were noted in the cows at a herd TB test in October 1993, a dramatic deterioration in body condition score was noted at a visit in March 1994. The weather at the time was

said to have been particularly severe. However, although 14 cows were reported to have died in 1994 - nine between January and May – the veterinary practitioners' descriptions of cases in the Retrospective Survey Report only refer to one case of milk fever, three of pining and two of lameness. No post-mortem examinations were carried out on animals which died in 1994.

An extract from the veterinary practitioner log for calls to cows for the period in 1990 to 1995 is given in Table 4-7. From that, it is apparent that there was a significant increase in the number of calls to calving-related problems in 1994. Although the category of calls with the single biggest increase was milk fever, a proportion of these may, in fact, have been to other periparturient problems as calls are generally logged a veterinary practitioner's office before a farm visit has taken place.

Animal health problems were at their worst in the spring of 1995. The veterinary practitioners describe the following syndrome in cows at that time:

"Affected cows had rapid, shallow breathing ... nasal discharge, normal rectal temperature, lethargy and poor milk yield. Some cows also had alopecia and reddened skin and had a 'tender gait' (not laminitis). Such cows deteriorated and died within a month of showing such signs. Appetite appeared normal and there was no diarrhoea. ..."

The generally poor condition of stock recorded at inspections of the farm by VLS staff in March and May 1995 has also been described in the EPA Interim Report (EPA, 1995). Lactating cows were in poor body condition and weanlings and other stock were thin and undersized. Severe body fat depletion was the most consistent finding on the 11 adult bovine carcasses (nine cows, two bulls) examined in Limerick RVL in February and March 1995.

Seventeen cows, a bull and a weanling were said to have died in 1995 (six cases were euthanased *in-extremis*). Ten cows, the bull and the weanling were submitted to Limerick RVL over a four-week period in February-March 1995. Summary post-mortem findings for these are given in Table 4-6. In the case of all except two, findings were consistent with diagnoses of severe acute or chronic inflammatory conditions of infectious origin.

Seven cows had lesions of pneumonia. Lesions of listeriosis and chronic fluke infestation were found in the bull. Recognised bacterial pathogens isolated from submissions included *Listeria*

monocytogenes, Pasteurella, Salmonella typhimurium and *A. pyogenes*. The weanling submitted for post-mortem examination was one of a group of animals which had been in poor condition over the winter. A number had developed suppurative lesions around the lower limb joints and a suppurative arthritis was observed in the animal submitted for post-mortem examination. *A. pyogenes*, a common cause of suppurative infections, was isolated from the lesions. *Salmonella bredney* was also isolated from the intestines of this animal. According to the veterinary practitioners, affected animals did not respond to antibiotic therapy.

Skin conditions in cows and growing cattle

This problem was reported to have begun in the spring of 1994 and to have affected the majority of cows both then and again in 1995. Dry stock were also affected. According to the Retrospective Survey Report:

"Hair loss occurred over both the black and the white skin, primarily on the upper body. The farmer described the hair as 'burned off' the yearling cattle as if acid was poured on their backs. Both yarded and outwintered cattle were affected. Cattle scratched and in 1995 some bled markedly.... (the) cows would not lick their coat, an observation (the herdowner) attributed to airborne pollution contamination of the hair"

The veterinary practitioner reported that:

"... the hair detached easily when rubbed, the skin was painful to touch and the cows were scratching"

All calls in the veterinary practitioner log specifically referring to skin problems ('Cows itching') were in 1995 when a total of six was recorded.

A report of a visit by DAF laboratory staff in May 1995 noted:

"Areas of alopecia ... on the cows ... Extensive hair loss with pinkish discolouration and flaking of the skin"

Although both laboratory staff and the veterinary practitioner considered that the lesions were probably originally due to *Dermatophilus infection* ('rain scald'), there were no specific laboratory findings on skin or hair samples collected in May 1995. This may have been due to the fact that weather conditions had improved significantly by then and lesions were in the recovery stage. Although very wet and cold weather had been experienced up to and including March of that

year, rainfall and sunshine amounts had been around normal from early April onwards.

Lameness

Lameness was reported to have been a problem at times but detailed incidence rates are not available. According to the herdowner, 10-12 cows were lame in some years. The Simmental bull was reported to have been lame in both hind legs since late 1993. The veterinary practitioners recalled overgrown hooves and hock cellulitis in cows, particularly in older cows, but did not regard it as a major herd problem. Only two calls are recorded in the veterinary practitioner log from 1990 to 1995 under the heading 'lameness'. Lame cows - and cows with overgrown hooves - were noted by DAF laboratory staff during a farm visit on 23 March 1995. An outbreak of suppurative arthritis was also reported to have occurred in a group of four housed weanlings over the winter of 1994-95 (*see above*).

Calving problems

Prolonged calvings - necessitating caesarian delivery in some cases - were reported to have been a problem in 1995. According to the veterinary practitioner, a number of downer-cows had to be induced to calve in 1995 and caesarian sections were performed on three. Two calls are recorded in the veterinary practitioner log to calving events in 1990 and four in 1994 - none are recorded for the intervening years (Table 4-7).

Twinning

According to the herdowner there was a dramatic rise in the incidence of twinning from about one per year in the 1980s to four to six per year in the 1990s - the latter all sired by the Simmental bull. However, no calving records are available. The veterinary practitioners did not recall an increase in the incidence of twinning.

Infertility

Infertility is reported to have been a problem since 1990. However, no breeding records were available for analysis. With the exception of the period from 1985 to 1988, when artificial insemination was used on a proportion of cows, service was entirely by stock bulls.

According to the Retrospective Survey Report, the primary problem was of cows either not showing signs of oestrus or of an oestrus of short duration - as short as 30 minutes. The problem was largely dealt with by leaving the bull with the cows all summer. As a result, most of the cows eventually became pregnant and few were culled for

infertility. However, the inevitable result of this was a greatly extended calving season. There was little veterinary intervention in relation to infertility on the farm.

Mastitis

Mastitis was reported by the veterinary practitioners to have been an annual problem in the herd. Conditions in the cubicle houses were cited by them as possible contributory factors. *Staphylococcus aureus* and *Streptococcus uberis*, common bacterial pathogens, were isolated from milk samples submitted for laboratory analysis in 1991. Milk cell count results are not available prior to 1994. Bulk counts for 1994 and 1995 were above a half million cells/ml which is consistent with a herd mastitis problem.

Reduced milk yield

Annual milk sales for the period 1990 to 1995 (allowing for estimated quantities fed to calves) are given in Table 4-8. Milk production is said to have decreased in the late 1980s and through the 1990s. However, the only information available regarding production prior to 1990 is that 240,938 kg were sold in 1986. Yield per cow was around 3,182 kg from 1990 to 1993, Yields fell to 2,932 kg in 1994 and 1,041 kg in 1995. A large amount of the 1995 production was discarded owing to the *Salmonella typhimurium* outbreak (*see* page 96).

EPA-COORDINATED INVESTIGATIONS IN 1995

The State veterinary authorities first became aware of the serious animal health problems on this farm in March 1995. Prior to that, the only submissions for laboratory examination had been an aborted foetus in November 1994 and a cow at the end of February 1995. The farm was visited by veterinary personnel of the DAF Veterinary Laboratory Services (VLS) on 23 March 1995. Teagasc investigations also commenced at this time. A report was prepared by VLS staff which described the animal health problem as it existed at that time (23 March 1995) and recommendations were made regarding further investigations. The most significant findings were the generally poor condition and health of cows and other stock. Teagasc investigations also noted the very poor condition of pastures and evidence of overgrazing where stock had been outwintered. Further details of farm and animal health investigations in 1995 have already been reported in the EPA Interim Report (EPA, 1995).

CONCLUSION

The severity of the reported animal health problems on this farm in 1994 and 1995 is beyond question. The main problems reported were abortion, calf diarrhoea and pneumonia, illthrift, illness and deaths in cows and growing cattle, skin problems in cows and growing cattle, lameness, calving problems, twinning, infertility, mastitis and reduced milk yield. There is insufficient information in the Retrospective Survey Report to assess the significance or otherwise of the references to deaths and disappearances of wildlife on the farm.

To deal with the less severe problems first, while lameness was reported to have been a problem, there is insufficient information to assess its incidence. Neither is there any specific evidence to suggest that unusual factors were involved. On the contrary, references to overgrown hooves and trauma in the cubicle-house, together with evidence of poor drainage from the holding yard (*see below*), indicate the presence of risk factors commonly associated with this problem.

Although cow fertility was reported to have been poor, very little can be said regarding performance as no breeding records were kept. Fertility management appears to have been loose since the reconstitution of the herd in 1984. The main problem was reported to have been one of heat detection – said to have been due to cows with short .or no visible oestrus. According to the Retrospective Survey Report, there was little or no veterinary investigation of cows not seen on heat and, despite the fact that all services had been by natural mating since 1989, there is no reference to fertility examination of bulls having been carried out. On the other hand, it is reported that one of the two bulls, the Simmental, had been lame in both legs since late 1993. This would undoubtedly have had a significant negative effect on his fertility performance in 1994 and, while he was withdrawn from service, would have meant that only the nine year old Hereford bull was available. As the latter died in early 1995, it would be reasonable to expect that fertility performance in 1994 and 1995 would have been severely compromised.

Overall, while it is not possible to identify the specific causes of infertility owing to the absence of breeding records, the following points can be made on the basis of the Retrospective Survey Report:

- The practice of running the bull with the cows throughout the summer is not common in intensive dairy operations and would inevitably lead to an extended

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calving pattern owing to the failure to identify and remove cows of low fertility (the Retrospective Survey Report states that few cows were culled for infertility).

- Failure to identify and remove cows of low fertility would also have had a negative effect on herd fertility in succeeding years.

- As a bull was used throughout the season it is very unlikely that an intensive heat detection program was also in operation - therefore the actual degree and duration of oestrus expression by cows must be open to question.

- One of the bulls (the Simmental) was reported to have been lame in both hind legs since late 1993 and was likely to have had significantly reduced fertility performance. The second bull died as a result of listeriosis and chronic fluke infestation in spring 1995. All services in 1995, therefore, would have been by the lame bull.

- The reported or confirmed poor condition of many cows in 1994 and 1995 would undoubtedly have contributed to reduced fertility performance in these years.

While abortion was reported to have been a problem, neither its incidence nor occurrence were exceptional. The annual incidence of between 2 and 4 *per cent* is close to average rates reported elsewhere (Appendix 1). Although all four of a group of heifers which had been bought in March 1994 aborted (two in 1994 and two in 1995), *Listeria* infection, a common infectious cause of abortion, was confirmed in two of the cases. In relation to the herd *Listeria* problem, the most likely source of this was silage (Blood and Radostits, 1990). Although *Salmonella* was not isolated from any aborted foetuses, it was present on the farm and may also have caused abortion.

Although there was said to have been a high incidence of twinning in the 1990s (approximately 5.4 - 11.5 *per cent* per annum), records are not available to confirm this. The possibility of a genetic component would have to be considered as they were all said to have been sired by the same bull.

The precise extent of the calf diarrhoea and pneumonia problem cannot be determined. While losses were above average at around 10 *per cent* per year, they were not exceptionally high and no information is available regarding the actual number of animals affected. Unfortunately, no pathology specimens from affected calves were submitted for laboratory analysis between 1990 and 1995. While the exact causes of the problems cannot be identified at this stage, therefore, there is no specific evidence to suggest that factors other than those normally cited - i.e. infection, management and nutrition - were involved.

Although mastitis was said to have been an annual problem in the herd, little detailed information is available. Recognised bacterial pathogens were isolated from four of five milk samples submitted for laboratory analysis in 1991. High cell counts (SCC) in 1994 and 1995 would certainly confirm the presence of a herd problem. Although incidence data is not available, it is likely that infection was widespread by 1995 as many chronically infected cows had to be culled from the herd in 1996 after its take-over by the DAF (Chapter Two).

While the incidence of mastitis was probably above average, there is no information to suggest that it was exceptional. Rates of up to 30 cases per 100 cows per year are not uncommon on commercial dairy herds elsewhere (Appendix 1). According to the veterinary practitioners, housing conditions (poor hygiene) may have contributed to the problem. The high age structure of the herd (*see* page 20), combined with the fact that involuntary losses in 1994 would have prevented selective culling, probably also resulted in a higher proportion of chronically affected animals in the herd in 1995.

Milk yield is said to have decreased in the late 1980s and through the 1990s. However, there is no information on yield per cow prior to 1990. Although yields were at an only moderate 3,182 kg per cow from 1990 to 1993, inclusive, there is no evidence of a significant decline over the period. Factors which may have had a negative influence on yields include grass management (*cf* references to grass type and quality in the First EPA Interim Report, 1995), cow age structure, mastitis, and extended calving pattern due to poor fertility performance. Yield per cow dropped to 2,932 kg in 1994 and to 1,041 in 1995. However, given the severe disease problems and high mortality in these years, the results are not unexpected. Fourteen cows are reported to have died in 1994 out of a total milking herd of about 65 and a further 17 in 1995. Although little information is available on cow condition in 1994, it was poor throughout a large part of 1995 - and there is also evidence that grass supply was critically low at a crucial time for milk production in that year. In the circumstances,

it is not necessary to look further for an explanation for poor milk production in 1994 and 1995.

The illness and deaths of adult and growing cattle in 1994 and 1995, together with the skin problems and calving difficulties which occurred around the same time, were undoubtedly the most severe problems on this farm. However, owing to the total absence of pathology submissions, or other laboratory involvement, it almost impossible to determine the possible causes in 1994.

With regard to the adult animal losses in 1995, specific diagnoses were made in most cases where carcases were submitted for laboratory post-mortem examination. Post-mortem findings were generally consistent with acute or chronic inflammatory conditions from which a variety of common bacterial pathogens were isolated. Aside from severe body fat depletion which was present in all cases - pneumonia was the most common lesion, being detected in six cows. Three well-recognised infectious conditions played a significant role in relation to disease outbreaks, i.e. pasteurellosis, salmonellosis, and listeriosis. The bacterium *A. pyogenes*, which is a common cause of suppurative conditions, was also isolated from one case of chronic pneumonia.

Based on the clinical description and history, the skin lesions reported in 1994 and 1995 were most likely due to *Dermatophilus* infection. Although not confirmed by laboratory tests, the samples in 1995 were collected during a period of improving weather and the lesions were probably healing at this stage. Dermatophilosis is a common skin disease associated with prolonged exposure to wet weather. Conditions were ideal for the development of this infection in the winter/spring periods of 1994 and 1995.

The calving difficulties described were also clearly secondary to the intercurrent health and body condition problems. According to the veterinary practitioner, calving had to be induced in several downer cows in 1995.

While the diseases diagnosed in the animals submitted for post-mortem examination in March 1995 were not unusual, what was exceptional was their occurrence at an unusually high incidence in adult animals. Up to five of the 10 cows submitted had lesions consistent with pasteurellosis. *Pasteurella multocida*, the cause of pasteurellosis, was isolated from three. While pneumonic pasteurellosis is a common disease of cattle, it is most frequently seen in animals between six months and two years of age (Blood and Radostits, 1990). Outbreaks in cows are uncommon.

Predisposing factors for pasteurellosis can largely be summarised under the one heading, i.e. environmental stress. Specific risk factors include the stresses of moving and mixing animals, poor housing conditions, nutritional stress (i.e. food deprivation) and inclement weather (Blood and Radostits, 1990). There is evidence that all of these existed on the farm at the time of the most severe losses.

Salmonella typhimurium was also isolated from three cow carcasses in March 1995 indicating a significant intercurrent problem of salmonellosis. Again, while acute outbreaks do occur in adult cattle, salmonellosis is more often a disease of young stock (Blood and Radostits, 1990). Predisposing factors for outbreaks of salmonellosis are similar to those for pasteurellosis and are also stress inducers. The most important are nutritional stress, inclement weather and poor environmental hygiene (Blood and Radostits, 1990). An outbreak of this severity in adult cattle is consistent with a breakdown in farm hygiene.

Listeria was isolated from an aborted foetus and a bull in the spring of 1995. This is a common infectious cause of abortion and systemic disease in cattle and sheep. Accepted risk factors for listeriosis include cold and wet weather and poorly preserved silage (Blood and Radostits, 1990) - both of which were present on this farm over the winter-spring of 1994-95. As there was a history of listeriosis on the farm (sporadic abortions, deaths of adult animals) the appearance of clinical cases at this time would not be unexpected.

The overall picture, therefore, in relation to the adult animal problems, is of an unusually high proportion of animals succumbing to a variety of infectious conditions. However, while these are acute conditions, and much of the pathology findings in the animals examined post-mortem were of an acute nature, it would be incorrect to consider that their occurrence at this time represented an acute and unexpected outbreak of disease in an otherwise healthy herd. Given the contemporary descriptions of conditions on the farm at the time - animals pining and in very poor body condition, chronic skin infections, downer cows - as well as the emaciated condition of carcases submitted for post-mortem examination, it is clear that they were largely acute terminal conditions in animals which had been in declining health and condition for some time.

The reports of large numbers of cows exhibiting difficulty rising in the cubicle house in the winter-spring periods of 1994 and 1995 also suggest extreme weakness. While inadequate hoof care and

trauma related to deficiencies in housing design were probably contributory factors (cf Retrospective Survey Report), it is likely that in many cases the weakness was an expression of malaise in cows in general ill-health and condition.

Lack of ventilation in the cubicle houses was also referred to by the herdowner's veterinary practitioners as being a possible contributory factor in relation to the health problems experienced. Inadequate ventilation in the cubicle houses was also noted in a report of a visit to the farm by DAF laboratory staff in March 1995.

The most important question, therefore, relates to the underlying reasons for the poor condition and severe losses of cows in the springs of 1994 and 1995. While it is not possible at this stage to draw up a comprehensive list, it is important to consider any identifiable factors which may have been involved. Poor weather was undoubtedly a contributory factor. The bulk of losses occurred during or following prolonged periods of bad weather in the winters and springs of 1994 and 1995. By March 1994, when the veterinary practitioners first noted a severe deterioration in cow body condition, the majority of the cows had been outwintered, in varying stages of pregnancy, over a period of particularly inclement weather. The months of December 1993 to April 1994, inclusive, were wet and windy. Similar weather, though colder, was prevalent in the winter-spring of 1994-95. The paddocks on which cows were outwintered in 1994-95 (and possibly 1993-94) were exposed to the prevailing westerly winds and had little shelter. In the Teagasc soil survey, one was classified as largely rocky (EPA, 1995).

Under such conditions, it is inevitable that animals would have suffered a significant degree of stress. It is also of interest to note that the skin condition, which occurred around the same time, was consistent with rain scald. This, as the name implies, is associated with wet weather (Scott, 1988). Similar skin lesions were reported on cows on Index Farm A in the spring of 1994 and on one of the farms in the Retrospective Survey (Farm ID 06), in the spring of 1995 (Chapter Three). Skin lesions and partial hair loss (alopecia) are also a non-specific accompaniment of debilitation (Blood and Radostits, 1990) which was common to cattle on both of the Index Farms.

The fact that most of the cow losses in 1994 - and possibly also in 1995 (though cf Table 4-5) - were said to have been young animals could also be construed as a lack of acclimatisation to conditions on the farm. The majority of replacement breeding stock were bought-in at the beginning of each year

and therefore had to adjust either to less than ideal conditions in the cubicle house or to outwintering during cold wet weather in a state of advanced pregnancy.

Nutrition is also likely to have played a role. While little information is available on the nutritive status of the herd in the winter-spring of 1993-94 - other than the veterinary practitioner's reference to animals being in poor body condition - inadequate nutrition combined with relative overstocking was undoubtedly a factor in the winter-spring of 1994-95. The nutrient requirements of animals are known to be increased under circumstances of inclement weather (Anon., 1989; Fox and Tylutki 1998). However, on this farm all of the cows and approximately 20 dry stock aged 10 months to two years were outwintered on poor quality silage alone during the winter of 1994-95 (EPA, 1995). The Teagasc report on the farm inspection in March 1995 (EPA, 1995) also noted that in addition to severe poaching of the fields on which the cows had been outwintered, the pastures were bare and the hedgerows showed signs of grazing. Even by May, there was little sign of grass growth.

The generally poor condition of all ages of stock on the farm in the spring of 1995 has already been recorded and severe body fat depletion was the most consistent finding in animals submitted for laboratory post-mortem examination in spring 1995 (EPA, 1995). Although it is not possible at this remove to determine to what extent poor condition was secondary to inappetance as a result of intercurrent illness, and to what extent primarily due to inadequate supply, there is evidence that the latter played a role. According to the Retrospective Survey Report, it was not the practice to feed concentrates to dry cows over winter. However, the quality of silage available to animals over the winter of 1994-95 was poor (EPA, 1995) and inadequate to maintain condition in dry cows without additional concentrate supplementation. The fact that no preparations were made throughout 1995 for winter fodder production must also raise questions regarding feeding management on the farm.

Taking these two factors together, i.e. out-wintering in poor weather conditions and inadequate nutrition for dry cows, it would be inevitable that a substantial proportion would have calved down in very poor condition. It is highly significant, therefore, that virtually all of the cows submitted for post-mortem examination in the spring of 1995 had died close to or shortly after calving.

General farm hygiene at the time may also have been a significant contributory factor to the development of infectious disease problems - in particular those due to *Salmonella*. Despite a dry summer, the open slurry pit was reported by VLS staff to have overflown in the autumn of 1995. If this had occurred during the wet winters of 1993-94 and 1994-95 – and it is difficult to see how it wouldn't - then it would have greatly increased the risks of outbreaks of salmonellosis. Marks of dried slurry observed on the walls of the holding yard in autumn 1995 also suggested that cows had been standing knee-deep in slurry at times during wet weather. Besides contributing to the incidence of foot problems, this would also substantially increase the rate of exposure to faecal bacteria.

While deficiencies of specific mineral nutrients may have contributed to the problems, it is unlikely that they were the primary causes. The potential for selenium deficiency certainly existed on the farm. Teagasc analysis of soil and herbage carried out between 1995 and 1997 (EPA, 1995) indicated that concentrations were below the recommended range for animal nutrition - though not low enough to inevitably lead to deficiency. Marginally low concentrations were also recorded in blood samples collected from 10 animals in May 1995.

Analyses carried out as part of the immunology studies in 1998 indicated that blood selenium concentrations would be likely to fall well below the normal range in unsupplemented animals on silage or grass. However, these animals remained healthy throughout and there was no evidence of a significant impairment of their immune functions (*see* Chapter Seven). In addition, dry cows on the farm were reported to have had access to a pre-calver mineral supplement in the spring of 1995, and tissue concentrations of animals examined post-mortem were within normal ranges (EPA, 1995).

Inadequate copper supply is also unlikely to have been of more than marginal significance. Although herbage concentrations were below the recommended range, they were comparable to values on many other Irish farms where problems with clinical copper deficiency do not occur. There is no evidence from the results of soil and herbage analyses carried out on this farm to suggest that inadequate supply of minerals other than copper and selenium could have been responsible for the animal health problems encountered.

The other main factors to consider in relation to the overall poor health and condition of the stock at this time are specific infections and environmental pollution. While a number of bacterial pathogens were directly responsible for the terminal acute conditions in many of the animals which were submitted for post-mortem examination in March 1995, almost no pathology or clinical pathology material was submitted before that time. It is not possible, therefore, to determine the possible role played by infectious agents in relation to the more chronic problem of pining and poor condition. No evidence of active or recent infection with Bovine Immunodeficiency Virus, or any of the more common bovine viruses such as BVD, IBR, or PI_3, was detected in paired blood samples collected from five cows in April and May 1995. Chronic mastitis may have been involved in some cases - though probably only a minority. Lesions of mastitis were only reported in one of the cows submitted for post-mortem examination.

Fluke infestation may also have played a role. Lesions of chronic fluke infestation were detected in a number of the adult animals examined post-mortem in spring 1995 and no regular fluke control program was used on the farm (Retrospective Survey Report: "*After wet summers only (not in 1994/95), the cows were treated for fluke with Trodax in November*"). The years 1994 and 1995 are known to have been periods of high fluke infestation nationally.

Although the herdowner considered that environmental pollution was responsible for the animal health problems on this farm, there is no direct evidence to support this contention. While disease incidence was certainly above normal, this obviously does not suggest a specific cause - either pollution or otherwise. None of the main disease syndromes reported had features which would suggest a specific toxic aetiology. To the extent that there was evidence of a common underlying mechanism - i.e. increased susceptibility to infectious disease - the history and clinical and post-mortem findings suggest that this was most likely secondary to chronic debilitation and environmental stress.

In conclusion, while there was undoubtedly a severe animal health problem on this farm in 1995 and probably also in 1994, it is likely that the diseases were multifactorial in origin. As with many outbreaks of severe disease - particularly those characterised by the presence of multiple syndromes - it is not possible to definitively identify all of the underlying influences that may have acted to tip the balance from health to disease for so many animals on this farm. The combined effects of out-wintering pregnant cows during periods of particularly inclement weather in 1994 and 1995, together with inadequate pre-calving nutrition, will undoubtedly have increased the

numbers of animals with a heightened susceptibility to disease around calving time. There is no evidence to suggest that the problems were due to environmental pollution.

OVERALL CONCLUSION

The animal health and production problems on these two farms were undoubtedly severe, both in incidence and expression, at times over the eight-year period from about 1988 to 1995. However, there is little or no evidence to suggest that they had a common cause such as environmental pollution. The most severe problems on each farm occurred at different time periods and there was little overlap in terms of the clinical syndromes described.

Because of the lack of contemporary case-history records, as well as the very limited use of laboratory facilities by the herdowners concerned, many of the diagnoses discussed above can only be viewed as opinions regarding the most likely causes based on the available evidence. It must be accepted that alternative explanations, some involving environmental factors such as pollution, could be adduced. However, in contrast to the confirmed or suggested diagnoses outlined above, these would have to be considered as speculative and not based on the presence of any specific supporting evidence.

Were the problems on the two farms to have been due to environmental pollution then it would be expected that (a) the same or similar disease syndromes would have been reported on other affected farms in the area and (b) there would have been a fair degree of overlap in terms of the time periods during which problems were experienced on affected farms. While these issues are dealt with elsewhere in relation to the larger population of 'problem' farms in the Askeaton area (Chapter Five), comparison even between the two Index Farms - which were only about a kilometre apart by line of sight - gives little grounds for suggesting a common cause.

In the first case, the most prominent problem reported on Index Farm A comprised chronic suppurative lesions in cows (suppurative mastitis, subcutaneous abscesses, etc.). While cows were also affected on Index Farm B, the majority of animals examined post-mortem had acute or sub-acute systemic lesions which were probably of relatively short duration. Although both farms reported pining and death of affected animals, these are not uncommon consequences of many chronic conditions. In fact, the only major syndrome which was common to both farms was

that of dermatophilosis (skin infection) – and was most likely associated with an environmental factor common to both farms, i.e. wet weather.

In relation to the temporal aspects of disease occurrence, problems on the two farms were at their worst at different times. While cow losses peaked on Index Farm A in 1992 and 1993, losses substantially above normal were not reported to have commenced until the spring of 1994 on Index Farm B.

If environmental pollution were to have been responsible for the severe animal health problems on these farms then the question must also be asked as to what pollutants were likely to have been involved. While a list of pollutants for which there are potential sources in the area has already been published (EPA, 1995), most attention in the Askeaton area has been focused on the elements aluminium, fluorine and sulphur - the first two because of the proximity of a bauxite refinery and the latter because the refinery – as well as a number of other, more distant, industries - generate sulphur dioxide emissions from the combustion of hydrocarbon fuels.

However, besides the fact that environmental and tissue analyses and monitoring have shown no evidence that the farms have been subject to excess deposition by any of these three substances - and in relation to fluorine the only identified source is natural geochemical deposits (EPA, 1995) - there is little or no clinical evidence that the disease outbreaks which have been reported could be reasonably attributed to these compounds. Both aluminium and fluorine produce specific clinical syndromes. The former, while rare and difficult to reproduce, is mediated *via* an induced phosphorus deficiency and the signs include joint stiffness and pica (Crowe *et al.*, 1990). Fluorine toxicity, or fluorosis, is a well-recognised syndrome which gives rise to bone and teeth abnormalities (Krook and Maylin, 1979). While not all of the characteristic signs may be seen on every occasion, a wide range of other possible causes must be considered in the differential diagnosis when, as in the case of the Askeaton investigation, the major presenting signs comprise common conditions such as illthrift, lameness and hoof overgrowth.

The potential effects of excess sulphur deposition, while less easy to predict, are also mediated *via* interference with availability of other essential elements - in this case copper and possibly selenium (Suttle, 1974; Suttle, 1975; Poole and Rogers, 1989). Again, while some of the clinical signs reported on the two farms could have been due to inadequate copper or selenium supply, and

as discussed above, natural shortages of these elements may have played a secondary role in relation to some of the problems, it is extremely unlikely that the main problems on the two farms, i.e. acute and chronic suppurative conditions in cows, pining and high mortality, could have been due primarily to sulphur-induced deficiencies of copper or selenium. In the first case, neither the monitored nor likely sulphur emissions in the area would have been high enough to have had any significant effect in relation to uptake of these minerals (Appendix 15). In the second case, neither contemporary nor subsequent animal, plant, and soil analyses showed evidence that the farms were likely to have suffered severe deficiency of either element. Even had there been marginal reduction of copper and selenium availability, the reported concentrate and mineral supplementation rates used on the two farms at various times would have been adequate to prevent severe deficiency. In the third case, even if deficiency had occurred, it is highly unlikely that it could have led to such high mortality. Given severe deficiency of either or both elements, one or more of the classical signs of deficiency would also have been apparent.

In conclusion, therefore, while it is clear that the two farms suffered an excess of animal disease, straightforward explanations involving the commonly-accepted infectious, nutritional and management risk factors can be adduced in the majority of cases. The fact that definitive diagnoses could not be made in many cases relates more to the paucity of contemporary pathology and analytical data than to the unusual nature of the conditions. Although the severity of some of the conditions suggest that exceptional circumstances obtained at times on each of the farms, there is no clinical, pathological or analytical evidence to suggest that environmental pollution was involved.

Tables

Table 4-1: Fertility records summary analysis 1992 – 1995 for Index Farm A. (extract from Retrospective Survey Report)

YEAR	1992	1993	1994	1995	1996	Average
Herd size for Analysis	11	19	27	26	18	20
Served % end period	92	89	93	85	94	91
Fertility index	45	55	56	32	36	45
Pregnancy Rate 1st serv.	54.6	82.4	68	68	24	59
Services per conception	1.7	1.3	1.6	1.4	2.5	1.68
Calving to 1st Serv	80.1	71.1	67.7	133	73	85
Calving to conception	91.1	75.4	76.6	186	96	105
Submission Rate (1st 3 weeks)	36	21	58	25	29	34
Non-detected Oestrus %	42.9	57.1	22.2	69.2	54	49
Percentage returns 18-24 days	50	50	44.4	NA[1]	20	33
Infertile Rate	27.3	5.9	12	18	12	15
Heat Detection Rate %	57	80	90	86	69	76
Heat Detection Efficiency %	40	60	42	10	21	35
Services – cows failed	0.7	2	2	0.5	2	1.44
Calving spread (no. months)	6	5	6	7	5	5.8

[1] Not available.

Table 4-2: Animal losses on Index Farm A – 1985 to 1995[1]

	Cows				H&B[2]		Foetuses	Calves
	PAR[3]	Deaths	Culls	PAR	Deaths	PAR	Aborted	Deaths
1985	(33)[4]	0	0	(24)	0	21	0	1
1986	(33)	1	1	(24)	0	35	1	4
1987	34	1	1	7	0	36	1	2
1988	32	0	0	16	0	34	0	10
1989	34	3	0	19	0	33	1	6
1990	34	3	0	28	0	36	0	5
1991	(33)	3	0	(24)	0	33	2	3
1992	33	4	0	24	0	23	0	9
1993	29	6	1	41	4	22	1	5
1994	35	1	1	30	1	34	1	5
1995	36	3[5]	4	30	0	31	0	2

[1] Based on Table 16 in the Retrospective Survey Report – which was based on data collected from the herdowner's diary. [2]Heifers and bullocks. [3]Population At Risk. [4]Numbers in brackets estimated. [5]Euthanased and submitted for PM examination.

Table 4-3: History and necropsy findings for carcases from Index Farm A submitted to Limerick RVL between 1991 and 1995[1].

Date	RVLRef	Specimen	Age	History	PM Findings
21/03/91	1134	Calf	1 week	Slight scour, died within few hrs	Enteritis, dehydration
14/04/92	1377	Foetus	8 month gest	·Heifer's calf	No significant findings
22/04/93	1572	Cows x 2	Adult	Down 2-3 weeks, pining	Both suppurative mastitis - *A. pyogenes*
06/05/93	1738	Foetus	9 month gest	Natural service sire	No significant findings
19/05/93	1859	Cow	4yro	Found dead in field	Hypomagnesaemia
21/05/93	1884	Calf	3 week	Scouring over 2 weeks	Enteritis, dehydration, intususception
21/05/93	1885	Hen	Adult	Few dead recently	Salpingitis/peritonitis
22/11/93	3523	Weanling	6 month	Illthrift, No treatment. At grass	Diarrhoea, dehydration, BVD-type lesions
07/02/94	562	Calf	5 day	No signs of illness	Colisepticaemia
06/04/94	1552	Calf	8 hour	Calf was 'moaning'	Cerebellar hypoplasia, BVD
19/05/94	2109	Calf	Full-term	Natural service sire	Cerebellar hypoplasia, BVD
15/06/94	2327	Calf	Full-term	AI sire, stillbirth	Atelectasis
20/06/94	2375	Calf	10 day old	Mouth inflamed	Enteritis, dehydration, emaciation
27/06/94	2426	Calf	Full-term	Born dead, AI, Heifer's calf	No significant findings
05/09/94	2885	Cow	7 yro	No treatment, at grass	Cardiac & pulmonary thromboembolism, etc.
07/03/95	1172	Cow	3 yro	Pining, down. No treatment	Ostertagiasis Type II
01/06/95	2388	Calf	Full-term	Stillbirth, AI	Intrauterine anoxia
06/12/95	4110	Cow	Adult	Pining (Euthanized)	Chronic mastitis, deformed hooves, incisors worn to gum
06/12/95	4111	Cow	8 yro	Pining (Euthanized)	Incisors worn to gum

[1] Modified from Table 13 in Retrospective Survey Report, see also Appendix A1-3 in EPA Interim Report (1995).

Table 4-4: Annual milk sales Index Farm A

1987	1988	1989	1990	1991	1992	1993	1994	(1995)[1]
24,890	22,697	21,714	17,521	18,878	13,719	14,320	17,689	(12,589)

[1]nine-month lactation.

Table 4-5: Animal losses on Index Farm B – 1990 to 1995[1]

	Cow		Bull		H&B		Calf		Foetus	
	PAR[2]	Died	PAR	Died	PAR	Died	PAR	Died	PAR	Died
1990	69	1-2	1	0	21	0	68	6-7	69	1-2
1991	70-74	3	2	0	26	0	69-73	7	70-74	1-2
1992	71	2-3	1	0	20	0	70	6-7	71	1-2
1993	72	3	2	0	21	0	71	9	72	1-2
1994	69	14	2	0	17	0	64	NA[3]	69	4
1995	60-70	17	1	1	20	1	58-68	NA	60-70	2

[1] Based on Table 6 in the Retrospective Survey Report. [2] Population At Risk [3]Not available

Table 4-6: History and necropsy findings for carcases from Index Farm B submitted to Limerick RVL in 1994 and 1995[1].

RVLRef	Date	Specimen	Age	History	PM Findings
3701	18/11/94	3 mo (?)	Foetus	2nd calver, bought March/April	NSF
168	11/01/95	6/7 mo	Foetus	2nd calver, bought March/April	*Listeria monocytogenes* isolated
1025	27/02/95	3 yr	Cow	Aborted in Nov. Pining since.	Chronic suppurative pneumonia. *A. pyogenes* isolated
1114	06/03/95	5/6 yr	Cow	Pining. In pain Down precalving.	Acute serofibrinous pneumonia/pleurisy *P. multocida* isolated.
1200	08/03/95	7 yr	Cow	Down precalving. Down for 2 weeks	Necrotic pneumonia/pleurisy. Chronic interstitial nephritis Severe metritis *S typhimurim* isolated.
1201	08/03/95	10 yr	Bull	Down, in pain Treated by PVP	Listeriosis and chronic fluke
1202	08/03/95	6 yr	Cow	Pining for weeks Pregnant, close to calving	Advanced autolysis. No significant findings
1203	08/03/95	5/6 yr	Cow	Pining, recently calved.	Severe necrotising pneumonia and pleurisy
1231	09/03/95	3 yr	Cow	Went down after induced calving. Euthanased.	Poor body condition. No specific lesions.
1232	09/03/95	11 yr	Cow	Pining since calving 3 weeks prev	Serofibrinous exudative pneumonia pleurisy, Interstitial nephritis *P. haemolytica* isolated
1303	13/03/95	3 yr	Cow	Bought one year. Calved Pining for few weeks.	Diffuse exudative pneumonia *P. haemolytica* isolated.
1446	21/03/95		Cow	Pining. Recently calved.	Poor body condition Suppurative metritis - *S. dysgalactiae* isolated. Mastitis - *Serratia marescens* isolated. *S. typhimurim* isolated from internal organs
1447	21/03/95		Cow	Emergency caesarian	Severe diffuse serofibrinous pneumonia *S typhimurium* isolated.
1638	30/03/95	8 mo	Weanling bull	Pining 2 – 3 weeks. Suppurative arthritis phalangeal joint.	Euthanised. Suppurative arthritis - *A. pyogenes* isolated *S bredney* isolated from intestines Focal protozoal-type encephalitis

[1] Modified from Table 7 in Retrospective Survey Report, see also Appendix A1-4 in EPA Interim Report (1995).

Table 4-7:Extract from veterinary practitioner log for Index Farm B: cow calls 1990 – 1995

	1990	1991	1992	1993	1994	1995
Visit cow	2	3	3	2	8	0
Wash out	2	6	1	3	5	1
Milk fever	2	1	4	1	7	1
Mastitis	0	0	3	3	4	3
Calving	2	0	0	0	4	1
Cow down	0	0	0	1	2	4
Cows itching	0	0	0	0	0	6
Caesarian	0	0	0	0	0	3
Grass tetany	1	0	0	0	0	0
Lame cow	0	1	0	0	1	0
Pneumonia	0	0	1	0	0	0
Cow staggering	0	0	0	0	1	0
Cow scouring	0	0	0	0	1	0
Total	9	11	12	10	33	19

Table 4-8: Annual milk sales and yield per cow for the period 1990 to 1995

	1990	1991	1992	1993	1994	(1995)[1]
Milk Sold	42061	42267	44732	44167	39648	13645
Yield Per Cow[2]	717	720	709	690	645	229

[1]A large proportion of milk was discarded in 1995 due to identification of *Salmonella typhimurium* infection in cows [2]Total annual sales per farm divided by reported number of cows.

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CHAPTER FIVE

RETROSPECTIVE STUDY ON 25 FARMS

Introduction and Background

A retrospective investigation of animal disease and production problems was carried out on 27 farms in the Askeaton area (including the two 'index' farms) commencing in December 1995. The overall purpose of the Retrospective Survey was to investigate claims that there had been an abnormal or unusual incidence of animal health problems in the wider Askeaton area, and if so, to attempt to identify the underlying causes.

The objectives of the Retrospective Survey have been described elsewhere (EPA, 1995). Briefly, they comprised:

- Identification by self-disclosure all farms reported to have an excess of animal disease and to define their geographical distribution.

- To describe the clinical manifestations of the disease problems reported on each farm

- To determine whether or not the farms had a common disease syndrome and to describe - as far as possible - the clinical, biochemical and pathological characteristics associated with each syndrome.

- To describe the temporal distributions of the disease syndromes, i.e. times of onset and duration.

- To compare levels of particular animal diseases on affected farms with an appropriate reference population.

- To identify possible risk factors.

An interim report on the Retrospective Survey has already been published (EPA, 1997a). This report gave details of methodology relating to farm identification and data collection. It also gave summary descriptive statistics of the raw data on farm size, production and reported animal health problems. The present analysis comprises a detailed examination of the reported problems together with an assessment of their incidence, severity and possible causation.

Some differences will be noted between the two reports regarding the tabulation and categorisation of data on disease occurrence. These are due to differences in the scope and objectives of the respective reports. The 1997 interim report was largely descriptive and comprised a summary of all reported disease incidents regardless of severity, duration or type. The present report comprises a detailed analysis of the individual Farm Reports (*see below*) and concentrates on disease incidents that are considered to be of epidemiological significance in the context of the overall investigation of animal health and production in the Askeaton area.

In the following assessment and analysis, only the main problems on each farm, i.e. those which were of economic or animal health or production significance in terms of numbers of animals affected or duration of effect, are considered in detail. The summary descriptions provided are based on the herdowners' accounts contained in the individual Farm Reports and are intended to provide sufficient information to support the analysis of each case. Complete descriptions of each reported problem are contained in the individual Farm Reports (DAF 1996, unpublished).

For each farm, an outline is given of each reported problem followed by consideration of its incidence, severity and presentation (i.e. type of disease). Case descriptions are summarised from information provided in the Retrospective Survey Report. Comments by the herdowners' PVP regarding cause and diagnosis are included where available. An assessment is made in the case of each farm of the severity or otherwise of the problems reported and, where possible, an opinion is given regarding factors which may have caused or contributed to the problems. These opinions are based on the histories supplied and comprise the risk factors which are most commonly associated with the conditions identified. They are not intended to be comprehensive or to exclude the possibility that other unidentified factors were also involved. In the case of each farm, opinions are also offered (i.e. by reference to what could be expected on farms elsewhere) as to whether the problems occurred at an unusually high incidence, or were of an unusual nature. All available information on the incidence of the main disease

problems reported on each of the 27 surveyed farms is also tabulated in Appendix 13.

IDENTIFICATION OF AFFECTED FARMS AND DEFINITION OF STUDY AREA

Ideally, investigation of an epidemiological problem of this nature would involve comparison of disease incidences in cross-sectional samples of farms from the affected and appropriate control areas. However, this was not possible in the Askeaton investigation owing to time and resource limitations. In the first case, owing to widespread local concerns regarding human and animal health, the investigating team was under considerable pressure to follow-up on all reports of animal disease problems. In the second case, owing to the relatively scattered distribution of 'problem' farms – ultimately 27 in a population of around 1,000 farms over an area of approximately 400 square kilometres – a large number of farms would have had to be surveyed in order to ensure inclusion of a significant number of 'problem' farms. This would have had to be matched by a similar number from the control area. The resources for such an exercise would have been substantial.

In the circumstances, and in consultation with the local Askeaton and Ballysteen Animal Health Committee, a decision was made to include farms in the survey on the basis that their herdowners considered they had an excessive incidence of animal disease problems. Farmers in the area were notified of the survey requirements by public meeting and *via* the Askeaton and Ballysteen Animal Health Group.

While this obviously represented a highly biased sample, it was hoped that for the purposes of the investigation the bias would have been sufficiently marked to allow the selected farms to be taken as a reasonable approximation of the total number of farms in the area which had - or whose herdowners considered they had - an excessive incidence of animal disease problems. Although this expectation cannot be confirmed, given the method of selection - together with the heightened level of public awareness - it was probably the most representative sample of farms which could have been gathered short of sampling the entire farm population in the area.

Twenty-five herds were identified by this means. With the inclusion of the two index farms, the total number of farms included in the Retrospective Survey was 27. One other herd, which was not reported to have had an excess of animal disease, was initially included as a control at the request of the Askeaton and Ballysteen Animal Health Group.

However, as there would be no sound statistical basis for its comparison against the other 27 herds, this farm is not considered further in the following analysis. The results of the Retrospective Survey for the two index farms were presented in Chapter Four. The present chapter presents the results for the remaining 25 herds.

The study area was defined as the area comprising the District Electoral Divisions (DEDs) in which herds with a reported excess of animal disease were identified[1]. A map of the study area, together with the approximate location of the 27 farms, is shown in Figure 5-1.

DATA COLLECTION

The function of the Retrospective Survey was to collect information on farm management, nutrition. and animal health and production, on the farms over the period during which problems were reported to have occurred at an excess rate. The information collected is contained in individual Farm Reports that were based on herdowner interviews carried out in late 1995 and early 1996. Copies of these Farm Reports were subsequently sent to each of the herdowners concerned for comment and correction in 1996 (EPA, 1997a). Together, these reports comprise the Retrospective Survey Report.

Information on management and nutrition was collected on each of the 27 farms under the headings feeding, animal condition, fertilizer and slurry usage, housing, breeding, calving, milk production, mineral supplementation and disease control measures. However, because of its retrospective and largely undocumented nature, the value of this information related mainly to its role in describing the nature of the farm operation. Comments in the present report, therefore, in relation to management and nutrition are of a general nature and refer only to the potential impact of stated practices on reported animal health and production problems.

DISEASE INCIDENCE AND OCCURRENCE

The design of the Retrospective Survey intended that morbidity and mortality rates of reported disease conditions on the surveyed farms would be compared with those of an appropriate reference

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1   Ballynacarriga, Kildimo, Dromard, Ballyallinan, Rathkeale rural, Rathkeale urban. Croagh, Kilcornan, Pallaskenry, Castletown, Iveruss, Askeaton West, Nantinan, Kilscannell, Riddlestown, Askeaton East, Lismakeery, Craggs, Aughinish, Shanagolden. Shanid, Loghill, Mohernangh, Dunmoylan west.

population. However, a number of reservations must be highlighted at this stage both in relation to the nature of the data collected and to the availability of comparative reference populations.

While the information in the Retrospective Survey Report is extensive, it is neither comprehensive nor balanced. Much of the data collected was based on individual recall as few herdowners maintained permanent records of animal health and production. The inherent biases and unreliability of this type of information in epidemiological research has been highlighted by other workers (Martin *et al.*, 1975; Sackett, 1979). While corroboration was sought from other sources in the present study - such as the herdowner's private veterinary practitioner (PVP) and other specialist farm advisors – this also generally amounted to individual recall. Inevitably, sources often differed in recollection of specific events.

There is also a strong element of recall bias in that much emphasis is placed in each Farm Report on the main problems experienced - while little information is provided in areas where performance was considered satisfactory. For example, one Farm Report might contain a detailed history of problems relating to outbreaks of respiratory disease in calves but might contain no information regarding stillbirths, perinatal mortality or other forms of calf ill health. This cannot be taken to imply that no losses occurred in these areas – only that performance was regarded as satisfactory by the herdowner concerned. Because of this, it was generally not possible to determine actual morbidity or mortality rates for the main categories of disease or production problems reported. This problem is illustrated by the substantial gaps in the table of disease incidence in Appendix 13.

This deficit of information is particularly important in relation to cow fertility where it cannot be assumed that performance was below target only on those farms where it was highlighted as a problem – and for which breeding records were available for analysis. It is far more likely that even within the group of farms where it was not recognised as a problem, a range of performance from within to well below target is masked by the absence of comprehensive breeding records.

Regarding the normality or otherwise of disease incidence on the surveyed farms, there is at present no comprehensive source of reference data either on the occurrence of disease on Irish farms or on the expected distribution of 'problem' farms within a given area. Although a baseline postal survey of certain animal health and production indices was

carried out in the Aughinish area in 1979 - 81 (Rogers and Poole, 1984), most of the information collected on disease occurrence related to the presence or absence of specific diseases on a farm rather than to disease incidence at farm level. While this deficit has been addressed to some extent by the animal health questionnaire survey which was carried out in the Askeaton and other areas in 1996 (*see* Chapter Six), this only gives indirect information on disease incidence *via* data collected on rates of treatment for disease.

In the absence of comprehensive data from Irish sources, therefore, internationally published data on animal disease incidence (Appendix 1) are used as the main basis for comparison with reports of disease incidence on the 27 surveyed farms. Where necessary, account is also taken of specific conditions applying under Irish farming conditions.

ONSET AND DURATION OF PROBLEMS

Reservations also apply in relation to the quality of data collected regarding times of onset and duration of reported problems. While precise dates are available in some cases, the accuracy of information is generally limited to season and year. In many cases, it is also not possible to determine when a particular problem began or ended or whether problems reported in one year recurred in following years.

ADDITIONAL SOURCES OF INFORMATION

In addition to information contained in the Retrospective Survey farm reports, the following analysis of animal disease history on the 27 surveyed farms also draws on the results of extensive VLS and Teagasc investigations which took place in the area from 1995 onwards. In 1996, a stock inspection was carried out by an officer of the DAF on 10 of the 27 farms that had reported problems with illthrift or stunting in cattle (DAFRD unpublished). This report is referred to, where appropriate, in the individual-farm analysis below. The two Index Farms (IDs 01 and 02) were also the subjects of a detailed two-year Monitor Study (Chapter Two) between 1996 and 1998. Five further farms were included in a Longitudinal Study of animal health and production (Chapter Three) during 1997 and 1998 (one withdrew from the study in early 1997).

Seventeen of the remaining 19 farms were re-visited by VLS and Teagasc staff in 1997. (One herdowner declined to participate in the 1997 investigations and one farm had no stock). Stock were inspected and a detailed history of animal health and production was collected from the

herdowners. Blood samples were collected for analysis from a selection of cows and growing stock. Other clinical pathology material was also collected as indicated. Full reports on the visits, together with the results of all laboratory analyses were sent to the herdowners and their veterinary practitioners. A summary of the main disease and production problems reported at the 1997 re-visits is included in the following analysis.

Note: Pathology and clinical pathology specimens submitted to the Limerick Regional Veterinary Laboratory from the Askeaton area from September 1995 until mid-1997 are listed in Appendix 14. These are amended versions of Tables 3.8-3 and 3.8-4 in the Second Interim Report (EPA, 1998) which contained some errors.

FARM STATISTICS

In order to maintain anonymity, only summary herd descriptions are given for each of the farms participating in the Retrospective Survey. Details of stock numbers or other data which could identify the farms are specifically omitted.

Farms were classified according to the following size ranges:

Small	10 – 30 hectares
Medium	30 – 50 hectares
Medium large	50 – 120 hectares
Large	over 120 hectares

FARM DETAILS

Distribution of surveyed farms according to enterprise type, size, livestock units and stocking rate:

Enterprise Type:

	Number Farms
Dairy (sole)	6
Dairy (mixed)	12
Beef/suckler	9
Other	1

Farm Size(hectares):

	Number Farms
< 40	9
40 – 80	12
>80	6

Livestock Units:

	Number Farms
< 50	4
50 – 100	14
100 - 200	7
> 200	2

Stocking Rate (LU/acre):

	Number Farms
< 0.5	5
0.5 - 0.75	11
0.75 - 1	7
1.0 - 2.0	4

MILK YIELDS

It has been suggested that milk production can provide a useful indicator of herd health (Stein, 1986). A high incidence of conditions such as lameness, mastitis and illthrift (regardless of cause), for example, could be expected to depress herd yield. Acute outbreaks of infectious conditions, e.g. leptospirosis (milk drop syndrome) can have a dramatic, though shorter-term, effect on yields. Other factors which could negatively affect herd yields include infertility (i.e. due to late calving and/or a reduction in the total number of cows milking), poor weather (wet, cold, drought) and a number of management decisions such as withholding of milk for calf-feeding.

Information on total milk sales and quality (protein, fat, cell and bacterial counts) was available for the 18 dairy herds in the sample of 25 farms. This was based on annual creamery returns for one or more of the years 1990 to 1995, inclusive. For the purposes of investigating evidence of a trend in milk production on individual farms, annual yield per cow was estimated as the total annual sales per farm divided by the reported number of cows. However, it should be stressed that this method only provides a crude estimate of yield per cow as it makes no allowance for non-milking (e.g. barren) cows, nor does it allow for milk withheld from sale due to quota restrictions or requirements for calf-feeding.

In the context of claims of widespread animal health problems in the Askeaton area, available milk production data for the 18 dairy herds in the survey were examined to determine if there was any evidence of a coordinated pattern of reduced yields over the period of interest. If such a pattern was detected, then it could provide evidence of a negative influence on milk production in the area as a whole.

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Individual Farm Assessments

The following sections comprise summaries, interpretations, and analyses of the individual Farm Reports for 25 of the 27 self-identified problem farms visited in the Retrospective Survey. The historical problems on the two Index Farms have already been described in Chapter Four.

FARM RS01

Main Enterprises: Dairy
Herd size   Small

This was the original Index Farm (A) in relation to reports of animal health problems in the Askeaton area. A full analysis and assessment of animal disease history is presented in Chapter Four. For the purposes of the present analysis, a summary of the animal health and production problems is given in Table 5-1. The opinion of the herdowner's PVPs regarding the main problems encountered is given in Table 5-2.

FARM RS02

Main Enterprises: Dairy, store
Herd size   Medium

This was the second Index Farm (B) in relation to reports of animal health problems in the Askeaton area. A full analysis of animal disease history is presented in Chapter Four. A summary of the animal health and production problems is given in Table 5-3. The opinion of the herdowner's PVPs regarding the main problems encountered is given in Table 5-4.

FARM RS05

Main Enterprises: Dairy, store
Herd size   Medium

ASSESSMENT

This was Farm LS1 in the Longitudinal Study – the results of which are presented in Chapter Three. A summary of the historical animal health and production problems on this farm are given in Table 5-5. The opinion of the herdowner's PVPs regarding the main problems encountered is given in Table 5-6. The main animal health problems recorded in the Retrospective Survey Report on this farm were infertility in cows and ill thrift in calves and weanlings. The reported incidences of other conditions, i.e. downer cows, abortions and stillbirths - were not unusually high.

From the information supplied in the Retrospective Survey Report, it is clear that the fertility problems largely related to heat expression and detection. Non-detected oestrus rates (and consequently submission rates) were well outside target in all years (1989 - 1995). There was also a relatively high infertile rate in 1991 and 1992 (19 and 35 *per cent*, respectively). Artificial insemination was used exclusively up to 1994. Although a bull as well as AI was used for services in 1994 and 1995, the problem appears to have persisted. Non-detected oestrus was not a problem in 1996 when a bull was used for all services. According to the herdowner, the main problem was one of cows not showing signs of heat. The incidence of repeat breeders (i.e. low conception rate) was also reported to have been a problem in later years.

As discussed elsewhere (*see* page 6), non-detected oestrus is a function both of the heat detection regimen in operation on a farm and the level of heat expression by cows. However, the former, i.e. heat detection procedures, are generally regarded as the most important factors influencing the overall rate of heat detection (Esslemont and Kossabiti, 1996). While it is not possible at this remove to retrospectively assess heat detection practices, aspects of the breeding management noted on this farm during the period of the Longitudinal Study which may have contributed to reduced performance included absence of a fixed-period heat observation routine, absence of artificial heat detection aids (i.e. tail-painting of cows or chin-ball markers on bulls), and incomplete breeding records.

The extent to which reduced expression of heat may have contributed to the fertility problems prior to 1996 cannot be determined. However, post-calving negative energy balance may have contributed to the heat detection problem at times. It is well recognised that severe or prolonged negative energy balance can delay the onset of visible heats post-calving (Butler and Smith, 1989; Ferguson, 1991). Investigations during the period of the Longitudinal Study regarding silage quality and rates of concentrate supplementation, indicated that post-calving energy balance could have been a critical factor in some years for subsequent breeding performance (*see* Chapter Three).

The practice of out-wintering cows before and after calving on this farm could also have put added stress on the cows' energy reserves during periods of bad weather. It would generally be regarded as normal management practice to increase supplementation to out-wintered animals at these times – a practice which does not appear to have been followed on this farm based on information supplied in the Retrospective Survey Report.

The winters which preceded the years of poorest performance on this farm, i.e. 1993/94 and 1994/95, were particularly inclement. In the Retrospective Survey Report, the herdowner's PVPs refer to cows in "poor body condition with inactive ovaries" in 1995. In contrast, the winters which preceded years of improved fertility performance, i.e. 1995/96 and 1996/97, were mild.

It is obviously not possible at this stage to determine the factors responsible for the reportedly low conception rates. Given the extensive use of natural service from 1994 onwards, bull fertility is likely to have had a significant impact on performance. However, no information is available regarding fertility assessments of bulls throughout the period.

Illthrift in calves and weanlings is reported to have been a problem since 1989. However, little information is available regarding numbers affected or severity of effect. The primary problem was reported to have been poor body condition at grass in summer and poor price at sale. Of 20 – 25 calves on the farm in 1995, most were said to have been affected. No deaths were reported over the period.

While it is not possible at this stage to determine the actual causes of the problem, the contributory factors cited in the Retrospective Survey Farm Report - coccidiosis, respiratory conditions, viral pneumonia and hooze - are all common causes of ill thrift in young stock. The DAF 1996 investigation into reports of stunting and ill-thrift of cattle reported that "some of the calves (on this farm) were small but none of them were stunted" at the time of the inspection in August 1996.

A further factor which may have contributed to poor calf thrive on this farm is the practice of mixing calf age-groups while at grass. The possible significance of this practice in relation to both nutrition and parasite infestation is discussed further in Chapter Three.

While calf performance continued to be only moderate during the period of the Longitudinal Study - 1997 and 1998 - no severe or unusual outbreaks of disease occurred and no post-perinatal calf deaths were recorded throughout the period. Factors identified which may have contributed to poor thrive included marginal dietary copper status, chronic respiratory disease and tick-borne fever (Chapter Three).

Overall, while the level of calf performance achieved on this farm was far from optimal, it would not give specific grounds for citing evidence of unusual causes such as environmental pollution.

The management factors outlined above, as well as the occurrence of a variety of common calf diseases, could account for the uneven growth rates seen in calves during their first year at grass. No significant problems of thrive were reported in older cattle grazing the same land during the period of the Longitudinal Study.

The remaining animal health problems highlighted in the Retrospective Survey Report - downer cows and abortions/stillbirths - did not occur at an unusually high incidence (Appendix 1). Only two cows died over the five-year period from 1990 to 1995 – which represents an annual incidence of around one per cent. Both cases (1995) went down before calving and were reported to have been in poor body condition. It is quite possible that these deaths were related to pre-calving nutrition. Poor weather in the spring of 1995 may also have been a contributory stress factor. Other possible causes include milk fever and the spinal injury suggested by the PVP in relation to one cow.

The reported incidences of abortion/stillbirth in 1994 and 1995 were not unusually high. Nor was this a significant problem in following years. A single stillbirth occurred in each of the years 1996 and 1997 and two in 1998.

Though not referred to as having been a problem, there is evidence of a high incidence of mastitis on the farm – either clinical or subclinical – in 1992 and 1993. Bulk milk cell counts (SCC) of over two million were recorded for each of these years. Total bacterial counts (TBC) of 168,000 and 365,000 indicate milking hygiene may also have been a problem   .

In conclusion, while this farm experienced significant animal health and production problems - mainly in relation to infertility and calf thrive - no evidence has been found to specifically indicate that they are associated with unusual causes such as environmental pollution. In relation to infertility, which was the main problem from an economic point of view, there is no reason to suggest that factors other than management and nutrition need be cited to account for its occurrence.

FARM RS06

Main Enterprises: Dairy, suckling
Herd size: Medium large

ASSESSMENT

This was Farm LS2 in the Longitudinal Study – the results of which are presented in Chapter Three. A summary of the historical animal health and

production problems on this farm is given in Table 5-7. The opinion of the herdowner's PVPs regarding the main problems encountered is given in Table 5-8. This farm reportedly experienced a high incidence of animal health and production problems since the early '90s. The most significant problems between 1992 and 1996 were deaths, pining, mastitis, infertility, lameness, skin lesions and poor milk yield in cows; perinatal calf mortality; an outbreak of a locomotor disorder in calves and the birth of deformed calves.

The health problems in cows were at their worst in 1994 and 1995. The herdowner considered that they were related and may have been associated with environmental pollution - citing as evidence white dust which 'burnt' grass in March, 1994 and damage to trees (reported by herdowner to veterinary officer from Limerick RVL during field visit in 1995). Four cows were reported to have died in 1994. Two died in January - one a sudden death a week before calving which was suspected as having been due to milk fever. The second cow died after a difficult calving. Two more died during the summer within 12 hours of developing toxic mastitis on the day after calving. A post mortem finding of fatty liver syndrome was also reported by the PVP on the last of these. While the incidence of mortality at six *per cent* is high, it is not exceptional. Straightforward explanations are available for all four deaths, i.e. milk fever, dystocia (difficult calving), toxic mastitis with or without fatty liver.

Pining was first reported to have been a problem in a cow after calving in July 1994. Approximately 10 – 12 other cows were reported to have shown signs of pining after calving and produced excess, but not diarrhoeic faeces, while yarded. However, no further information is available regarding the time of year when these cases occurred or of the results of any veterinary investigations. According to the herdowner, the generally poor body condition of cows, together with a stiff gait in some, was again noted in May 1995 and was recorded on video.

There is insufficient information to make any comments regarding the possible cause or causes of this problem. Laboratory investigation appears to have been limited to analysis of blood samples collected from nine cows in June 1995. The only specific findings from these were mild anaemia in two samples and two had low phosphorus concentrations. However, neither of these findings are of particular clinical significance in lactating cows – both can be production-related and the latter may also be age-related (*see* page 35). The DAF investigation into reports of ill-thrift and stunting of cattle reported that animals on this farm

were in good condition and there was no evidence of stunting at the time of the inspection in August 1996[1] (DAFRD unpublished).

Mastitis was reported to have been a particular problem in 1994 and 1995 (three and nine cases, respectively). Some were of severe toxic mastitis and two affected cows died in 1994 (*see above*). In February 1994 two cows developed *E. coli* mastitis and did not respond to intravenous antibiotics. In August 1994 a cow developed mastitis a week before her expected calving date. The problem continued in 1995 despite an upgrading of the milking machinery. Cases (type unspecified except one referred to as *E. coli*) occurred in April, May, June and July – the majority around calving. According to the Retrospective Survey Report, the private veterinary practitioners recalled attending a relatively large number of acute or gangrenous cases of mastitis in 1994 and 1995 in lactating cows after calving.

From the history, the majority of cases of mastitis would appear to have been what are termed environmental mastitis. The commonest cause is the bacterium *E coli* and outbreaks are most likely to occur after calving. Known risk factors are poor hygiene and inclement weather. Much of the housing on this farm was old and it was probably difficult to maintain adequate hygiene at all times – particularly during the type of wet weather which occurred during the springs of 1994 and 1995.

According to the herdowner (Longitudinal Study Interim Report January 1997) mastitis was not a problem in 1996. Although it was a significant problem in 1997 - the first year of the Longitudinal Study – it was of the clinically milder infectious form (*Staph. aureus*) and no cases of *E. coli* or toxic mastitis occurred (Chapter Three).

Cow fertility was also reported to have been a problem since 1994. According to the herdowner, the main problems were a high rate of irregular returns to service (six weeks to three months) and silent heats leading to an extended calving season. The reported infertile rates for dairy cows in 1994 and 1995 were 8 and 16 *per cent*, respectively. Analysis of available breeding records for the years 1993 to 1996 indicated that the main problem was

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1 The herdowner requested that a second inspection be carried out as he was not present at the time of the official inspection. However, owing to DAFRD staff changes, a second inspection did not take place. The officer who carried out the initial inspection was satisfied that a thorough inspection of the animals had been performed and the report was a fair representation of animal condition.

a high rate of non-detected oestrus. The latter was above target in 1993 and 1995. Although indices relating to the number of services per cow were generally within target, these were of doubtful accuracy due to the number of unrecorded services.

The extent to which cow (i.e. anoestrus and suboestrus) and management (efficiency of heat detection) factors contributed to the problem of non-detected oestrus cannot be determined at this stage. Although a high rate of non-detected oestrus was also recorded in 1997, the first year of the Longitudinal Study, breeding records were not sufficiently comprehensive to allow a full analysis of performance. As discussed elsewhere (see page 6), the rate of non-detected oestrus in commercial dairy farming is largely a function of heat detection. However, it is likely that anoestrus in the form of delayed resumption of heats due to excess negative energy balance post-calving may also have contributed to the problem in some years. The poor condition of some cows in the springs of 1994 and 1995 was noted by the PVPs who refer to attending "old cows in moderate to poor body condition". Post-calving anoestrus due to excessive negative energy balance is a well-recognised phenomenon (Butler et al., 1996). Poor weather conditions in these years will also have contributed to nutritional stresses on animals at grass. Published reports by Teagasc on controlled fertility trials at Moorepark in the 1994 breeding season have referred to the effect of the poor weather on fertility performance in that year (Dillon and Crosse, 1997; O'Farrell et al., 1997).

Another factor which may have contributed to fertility problems in 1994 is the relatively high incidence of post-calving metritis in that year. Ten or 11 cases of metritis, some requiring repeated washouts, were reported to have occurred in April. Metritis is a recognised risk-factor for reduced conception performance (Barr et al., 1993a). It is likely that the incidence of these cases was associated, to some extent, with the relatively high degree of intervention in calf delivery (see below).

Despite the fact that there was a high degree of bull usage on this farm - five were used in 1997 – there is no record of their having been assessed for fertility. In the circumstances, therefore, it is not possible to determine the contribution of bull infertility to conception problems.

Overall, while fertility performance was below target on this farm, it is not possible to define the exact nature or extent of the problem due to inadequate records. However, based on available evidence, there is no reason to suggest that the

causes were other than those commonly associated with infertility problems in dairy herds elsewhere.

Milk production was reported to have been poor on this farm from about 1993. However, there is conflicting evidence regarding actual production for the period 1990 to 1996. Based on the data in the Retrospective Survey Report, there was a dramatic reduction in estimated yield per cow from 5,805 kg in 1992 to 3,950 kg in 1993 (Table 5-63). However, a verbal account received from the herdowner in January 1998 indicated a more gradual decline from 5,228 kg in 1993 to around 4,091 kg in 1995 and 1996. Analysis of the latter records indicated that the bulk of the decline involved cows of 3rd and higher lactation. Their average yield per cow for this group fell from 5,828 kg in 1993 to 4,478 kg in 1996. Yields of first and second calvers showed little change during this period.

The possible causes of decreased yields in this herd are largely a matter of speculation at this stage. They include infertility, herd age structure, possible fluke infestation, and stray voltage in the milking parlour. Based on fertility data in the Retrospective Survey Farm Report (Table 18 in Retrospective Survey Report), there is some evidence that a significant number of cows milking in 1994 had not conceived in 1993 - and were, therefore, at the end of long lactations. This would have had a significant depressing effect on herd yield. The average age of cows milked in the herd is also likely to have declined significantly over the period 1993 to 1995 due to the rapid increase in herd size - from 23 cows in 1992 to 50 in 1995. In 1995, about 30 per cent of cows milked were first-calvers.

A further factor which may have had a negative effect on milk production is liver fluke infestation. While the Retrospective Survey Farm Report describes an adequate fluke dosing regime, this failed to a significant degree over the winter of 1997-98 (see page 67). If similar failures occurred in the winters of 1992/93 or 1993/94, when national warnings of severe fluke infestation were in force, then liver fluke could have had significant impact on milk production. A problem of stray voltage in the milking parlour, identified in 1995, may also have had an effect on milk production. However, according to the herdowner, there was no obvious response to some remedial work which was carried out. Factors identified during the Longitudinal Study in 1997 which will have had a negative effect on milk production included mastitis, problems with milking machine operation, and short lactations, (average 237 days in 1997).

Perinatal calf mortality was reported to have been a problem on this farm since 1994. The incidences for the three years 1994 to 1996 were 7, 13, and 10 per cent, respectively. In 1994 and 1995, affected calves were full-term and were from heifers and cows. Calves were either born dead or died within a short time of calving. Affected calves which were born alive had 'gelatinous mucous in the mouth and nose, laboured breathing and heart beat' and died within 15 minutes of birth. According to the herdowner, cases were not specifically associated with difficult calvings or malpresentations. In 1996, the majority of affected calves were said to have been from heifers (EPA Interim Report; Longitudinal Study, January 1997). Two were twins and two others had breathing difficulties associated with difficult or prolonged calvings. The incidence of stillbirths in 1997 was just under 9 per cent - the majority of which were related to calving difficulties (see Chapter Three).

While above normal, the reported incidence of perinatal mortality on this farm is not exceptionally high. Stillbirth and perinatal mortality is a common problem world-wide with reported incidence rates ranging from three to over six per cent (Appendix 1). The main risk factors comprise dystocia due to absolute or relative foetal oversize and prolonged or unattended calvings. There is evidence on this farm of a high degree of intervention in calvings (according to the herdowner, "approximately half of all calvings are assisted, with a calving jack"). Based on PVP comments regarding calls to heifers being 'not fully dilated', some of the interventions may have been premature. The reference above to 'gelatinous mucous ...' in relation to newborn calves is consistent with the effects of hypoxia during delivery.

The relatively high proportion of heifers in the herd may also have contributed to the incidence of stillbirths. About 30 per cent of cows milked in 1995 were first-calvers and the PVPs also refer to the small size of some heifers at calving. The incidence of dystocia is known to be significantly higher in heifers than in cows (Busato et al., 1997). Mineral imbalance may also have had an influence on calving difficulties. According to the Retrospective Survey Farm Report, seven animals were treated with calcium before calving in 1995. Depending on clinical history, it is possible that uterine inertia as a result of hypocalcaemia may have resulted in an increased incidence of prolonged calvings in that year. In conclusion, while perinatal mortality has obviously been a problem on this farm, the incidence has not been exceptionally high and the cases can largely be attributed to the usual periparturient risk factors.

Other problems reported in cows on this farm were skin lesions, lameness and irritability at milking in cows. The report of skin lesions in eight cows in the spring of 1995 is not unusual - particularly in the context of the poor weather conditions during this period. The PVP's diagnoses of 'rain scald' (with or without Dermatophilus infection) and trauma (i.e. abrasions), are reasonable given the clinical history. The same condition was reported in cows on Index Farm B at around the same time (see page 100).

Similarly, the reports of lameness in cows are neither unusual nor of a particularly high incidence. The PVP's comments regarding old cows and poor roadways are relevant. The reported intermittent problem of irritability of cows in the milking parlour may have been due to stray voltage. Some corrective work was done in 1996 and more was to be done in 1997.

The description in the Retrospective Survey Report of an outbreak of a locomotor disorder in calves in the spring of 1995 — which the herdowner considered was related to in-utero exposure of calves to the effects of environmental pollution - presents an unusual clinical picture. New cases appeared over a period of about one month and signs, which were sudden in onset, included stiff gait, shuffling hind limb gait, walking on the tips of their hooves, arched back, exaggerated forelimb gait, licking the limewashed walls and spasm of the back muscles. Calves were bright and had normal rectal temperatures. No diarrhoea or joint or navel involvement was observed. The only significant findings on blood samples collected from four calves at the time were mild anaemia in two and raised white cell count and neutrophilia in one. Blood phosphorus concentrations were normal. Evidence is conflicting regarding the number of animals affected. According to the herdowner, eight of 20 calves were affected — the PVP reported that three were affected. Three affected calves were sent to UCD Veterinary Faculty Large Animal Clinic where the problem was intensively investigated.

There are many possible causes of this outbreak. Spinal abscesses were among the diagnoses considered by the attending PVPs. A comparable condition - osteomyelitis and fracture of a cervical vertebra - was subsequently diagnosed in one of the calves sent to the Veterinary Clinic and was suspected in a second. Salmonella infection was suggested as a possible cause of the osteomyelitis. This would not be inconsistent with the history. Both calves were reported to have suffered diarrhoea some weeks prior to the onset of locomotor signs and, while not a problem, cases of

salmonellosis had occurred on this farm in previous years.

On the basis of clinical signs, a congenital cerebellar disorder was suggested in the case of the third calf submitted to the Veterinary Clinic. While blood from this calf was BVD antibody and virus negative, the possibility of BVD virus infection having played a role in the outbreak cannot be completely ruled out. Detailed investigations of the herd in 1996 by a VLS virology specialist indicated that BVD virus had probably been circulating among the dams of these calves in the previous autumn. BVD virus was subsequently isolated (RVL ref. 914/1996) from a calf which was born in April 1995 and had probably been infected (in-utero) at that time. BVD virus was also isolated from a weanling born in April 1996 (31M, RVL Ref. 3313L).

Another factor which could have contributed to the locomotor signs is hypomagnesaemia. The Veterinary Clinic report refers to low blood magnesium in two of the three calves submitted. Locomotor disorder due to hypomagnesaemia in milk-fed calves is a recognised syndrome (Doxey, D.L., 1983).

The possible involvement of lead, aluminium and fluoride toxicity can effectively be ruled out on the basis of analytical results on blood and tissues from calves submitted to the Veterinary Clinic. Blood lead concentrations were not indicative of toxicity. While one of two blood samples collected had an aluminium concentration of 74 µg/l which is above the normal range of 9 – 20 µg/l reported by Puls (1994), according to Valdiva et al., (1978) this value would not be indicative of toxicity. The single bone sample analysed had an aluminium concentration of 43.2 mg/kg which is within the normal range. In addition, blood phosphorus concentrations in samples collected from these and other calves on the farm in April and June 1995 were within normal ranges. As aluminium toxicity is mediated via induced phosphorus deficiency (Crowe, et al., 1990), aluminium intake sufficient to cause clinical signs would have been accompanied by hypophosphataemia.

Blood and bone fluorine concentrations were within the normal ranges and this, combined with the absence of clinical signs of fluorosis, indicates that fluorine toxicity was not responsible for the outbreak. In addition, the results of environmental monitoring and analyses have shown that the only known potential source of excess fluorine in the area is natural soil deposits (EPA, 1995). As these calves had been housed since birth, it is unlikely that they would have had access to fluorine by this

means. Lead, aluminium and fluorine concentrations were also within normal ranges in blood samples collected from nine cows at pasture in June, 1995.

An incidence of 2.5 per cent deformed calves was reported over the period 1994-95. Deformities included incomplete closure of the linea alba, a neck deformity, hydrocephalus, and a case of growth retardation. The latter was subsequently diagnosed as suffering from congenital BVD infection. BVD virus is a recognised cause of intra-uterine growth abnormalities and could also have been responsible for the case of hydrocephalus. Given one, and possibly a second, diagnosis of infectious causation, the incidence of unexplained cases deformities is not unusually high (Appendix 1).

A severe outbreak of redwater (babesiasis) was reported to have occurred on this farm in May 1995, affecting all six of a group of purchased heifers. Response to treatment was reportedly poor and a number relapsed. While the incidence is high over a short period, the history is consistent with the cases having occurred in previously unexposed animals, i.e. purchased heifers - as opposed to the existing indigenous cows. The field on which the heifers were kept had many areas which would provide an ideal tick habitat and had been associated with redwater cases in the past. A case of tick-borne fever in a bullock grazing the field had also been diagnosed some years previously. Three further cases of redwater occurred in heifers in this field in 1997.

In conclusion, while this farm definitely had a higher than normal incidence of animal health and production problems at times during the period 1992 to 1996, the problems were not, in themselves, unusual and neither was there any evidence of a single underlying cause such as environmental pollution. On the contrary, reasonable explanations are available for most of the problems. These include weather, housing conditions, post-calving sepsis (metritis), spinal abscesses, salmonellosis, BVD virus infection, and, possibly, chronic fluke infestation.

FARM RS07

Main Enterprises: Dairy, calves
Herd size: Small

ASSESSMENT

This was Farm LS3 in the Longitudinal Study – the results of which are presented in Chapter Three. A

summary of the animal health and production problems on this farm are given in Table 5-9. The opinion of the herdowner's PVPs regarding the main problems encountered is given in Table 5-10. The main problems on this farm were perinatal calf mortality, infertility, and poor milk yield. Information provided on the other problems mentioned – pining in cows and a deformed calf – does not suggest an unusually high incidence.

A significant perinatal mortality problem was reported to have existed from 1992 to 1995. In 1993 and 1994 about two-thirds of the calves were either born dead or were weak at birth and required resuscitation. The incidence of cases in 1992, 1995 and 1996, at between about 9 and 11 per cent, was still above the accepted average range of about 6 to 8 per cent (Appendix 1). Stillbirth and perinatal mortality was not a problem during the period of the Longitudinal Study in 1997 and 1998.

Weak calf syndrome is not an uncommon problem in dairy herds (Rice et al., 1986). Incidences of over six per cent have been recorded in affected herds (Appendix 1). The main risk factors for weak calf syndrome have been discussed elsewhere and relate to parturition (foetal presentation, size), calving management and possibly also mineral status of the dam. In relation to the present case, insufficient information is available to determine the exact causes of the problem at this stage. According to the Retrospective Survey Report, some of the losses were associated with difficult calvings. The PVPs also refer to relative foetal oversize in heifers as being a contributory factor. There is also evidence of premature intervention in calvings (Retrospective Survey Report; Longitudinal Study EPA Interim Report). Unattended calvings in the cubicle house (up to five per year according to the herdowner) may also have been a contributory factor (see EPA Interim Report of Longitudinal Study).

Fertility performance was also reported to have been a problem on this farm. However, no breeding records were maintained. According to the herdowner, the main problem was of cows repeating regularly. The average infertile rate over the three years 1993 to 1995 was reported to have been 27 per cent. The problem was more severe in heifers which had an average infertile rate of 62 per cent over the period.

In the absence of breeding records, it is obviously not possible to determine the nature or extent of the problem. Factors which may have contributed to poor performance include herd age structure, bull fertility and inadequate record-keeping. It is likely that the original repopulation of the herd with 'cull'

cows (see below) would also have had a significant negative effect on herd fertility. The PVP refers to the high age structure of the repopulated herd. While a bull was used for part of the breeding season each year, there is no record of fertility assessments having been carried out. Because of this, and as no breeding records were kept once the bull has been put in with the cows, the contribution of the bull to conception performance cannot be estimated. Fertility performance was good to moderate during the Longitudinal Study in 1997 and 1998. Heat detection was good at the start of the seasons and a pregnancy rate of 81 per cent was achieved in 1997 and between 80 and 90 per cent in 1998. As the bull ran with the cows for most of the breeding season in 1998, no detailed information on fertility performance is available for that year.

Overall, while fertility has been variable on this farm - and even allowing for absence of detailed records - it can probably be classified as falling within the range of performance for farms of this size and type. There is no evidence to suggest that unusual factors such as environmental pollution contributed to shortfalls in performance.

Reduced milk yield was reported to have been a problem since 1992. According to the herdowner, prior to 1990 the average milk yield per cow was 4,546 kg. In 1995 it was between 3,182 and 3,637 kg per cow. Factors which may have contributed to this problem include cow type, age distribution and mastitis. The herd was repopulated in 1991 with 'cull' cows following an outbreak of tuberculosis. Given the variable quality of this type of cow, a reduction in milk yield in the following years would not have been unexpected. An inevitably high rate of cow replacement with first calvers in following years would also have had a negative impact on production. While mastitis was not stated to have been a problem in the Retrospective Survey Report, it probably also had a negative effect on yields. Milk somatic cell counts during 1997, the first year of the Longitudinal Study, were sufficiently high to lead to payment penalties. Investigations during that year indicated that almost the entire herd was affected by clinical or subclinical staphylococcal mastitis.

Overall, while this herd has had significant animal health and production problems, most notably in relation to perinatal calf mortality and mastitis in cows, these problems are not unique to the Askeaton area and there is no evidence to suggest that factors other than those normally considered were involved.

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FARM RS08

Main Enterprises: Dairy, beef, store
Herd size: Medium

ASSESSMENT

This farm was originally selected to participate in the Longitudinal Study (Farm LS4) but withdrew voluntarily some months after its commencement (Chapter Three). A summary of the animal health and production problems on this farm are given in Table 5-11. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-12. This herd reported problems with lameness, downer cows, infertility, calf diarrhoea and pneumonia. Although congenital calf deformities are also mentioned as a recurring problem, there is insufficient information to comment on the incidence of specific deformities. At 1.5 *per cent*, the overall incidence was not unusually high (Appendix 1). It should also be noted that the PVP did not consider that there was an excess number of deformed calves in the herd.

The lameness, downer cow and infertility problems were severe and were undoubtedly interlinked. Lameness was reported to have been a problem in the herd since 1989/90. While the number of cases per year was not recorded, in the autumn of 1995, 10 to 12 of about 40 cows at grass were reported to have become lame. Response to treatment was poor and animals lost condition. Contributory factors suggested by the PVP and chiropodist included infection (e.g. Mortellaro's disease), poor condition of roadways, and advanced state of lesions on presentation for treatment.

This problem was fully investigated by staff of Limerick RVL during field visits in September and November, 1996. A severe lameness problem was confirmed. Some animals had been affected for a number of years. The overall picture was of chronic hoof problems with little recourse to veterinary assistance. The main contributory factors were identified as poor condition of housing and roadway surfaces, absence of a suitable foot care program, and inadequate or inappropriate treatment of affected cows.

Approximately five or six cows were reported to have gone down and died each year from 1989 to 1996. Most cases occurred during the housing period - generally in older cows or cows in poor body condition. Cases occurred both before and after calving. While the PVP was apparently not called to cases in later years, he recalled attending cows with milk fever and chronically lame cows which went down. In the absence of further information, it is not possible to determine the

individual causes of these deaths. However, based on herd history and the results of the laboratory investigation in 1996, lameness was certainly responsible for a proportion of the deaths. Most of the losses in 1996 were directly attributable to lameness. A relatively high incidence of dystocia ("*10 to 12 difficult calvings annually, particularly following use of the Charolais bull ...*") could, where followed by post-parturient paralysis, have accounted for some of the cases.

Fertility performance in this herd was also poor. Although no records are available, the herdowner estimated that 20 *per cent* of cows were infertile each year. The infertile rate in 1996 was 30 *per cent*. While a detailed assessment of the problem is not possible in the absence of records, lameness was undoubtedly a major contributory factor. It is well recognised that lameness has a significant negative effect on fertility performance (Lucey *et al.*, 1986b; Collick *et al.*, 1989) – mainly *via* delayed or reduced expression of heat post-calving.

Other factors which may have had a negative effect on fertility performance include absence of a set heat detection regime, bull fertility (although bulls were used for all services since 1993 there were no records of bulls having been assessed for fertility), and an apparently high rate of post-calving uterine infection. The PVP log lists six calls to cases of retained placenta in each of the years 1993 and 1994 - as well as to three cases of uterine infection in 1993. As it is unlikely that the PVP was called to attend every case of each condition, the total number of cases is probably somewhat higher.

A severe annual problem of calf diarrhoea and pneumonia was also reported to have existed on this farm. However, there appears to have been minimal veterinary or laboratory involvement in its investigation. Although most calves were said to have been affected each year since 1989/90, it appears that only one calf, and no clinical material, was submitted for laboratory examination throughout the period. A full investigation of this problem would have addressed issues such as colostrum status of calves, housing environment, hygiene and general calf management. The results of field investigations by laboratory staff in 1997 indicated that housing is likely to have played a significant role. The apparently high incidence of dystocia was also probably a contributory factor in that many calves would have been severely stressed after difficult births. In the circumstances, there is no reason to suggest that causes other than the usual risk factors for calf diarrhoea, i.e. infectious agents, management and housing, need be cited to account for this problem.

Milk production performance was poor on this farm. Total sales fell by 40 *per cent* in 1994 and a further 29 *per cent* in 1995. The herdowner considered that late calving and infertility were contributory factors. Other factors which are likely to have had a negative effect on yields include lameness and widespread sub-clinical mastitis. Although not mentioned in the Retrospective Survey Report, mastitis was obviously a significant problem on this farm. High bulk milk cell counts were recorded in all years from 1994 to 1996.

FARM RS09

Main Enterprises: Dairy, beef, suckler
Herd size: Large

ASSESSMENT

This was Farm LS5 in the Longitudinal Study – the results of which are presented in Chapter Three. A summary of the animal health and production problems are given in Table 5-13. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-14. The main problems reported in the Retrospective Survey Report were infertility, perinatal calf mortality and ill-health in calves.

The fertility problem between 1994 and 1996. the years for which fertility data was available, appears to have been largely characterised by reduced conception performance (2.1 services per conception in 1994 compared to a target of 1.65). Performance in all other respects, i.e. heat detection and overall fertility rate, appear to have been well within acceptable ranges. While it is not possible to identify the precise causes of the reduced conception performance at this stage, the risk factors usually considered comprise cow and bull fertility status, accuracy of heat detection, semen quality and handling, timing of insemination, and AI technique. Although outside target, conception performance was not exceptionally so. The intervention level for herds of this size is 1.96 services per conception (Appendix 1). Conception continued to be an area of under-performance during the Longitudinal Study in 1997 and 1998. Possible contributory factors, and which may equally have applied to the problem in earlier years, are discussed in more detail in Chapter Three.

Perinatal calf mortality was reported to have been a problem on this farm since the early 1980s. The incidence in the mid-eighties was said to have been around 10 *per cent* which is higher than rates reported from surveys of commercial farms elsewhere (Appendix 1). In 1994 the incidence was 35 *per cent*. It was 17 *per cent* in 1995. Despite the

duration, and latterly the severity of the problem, there appears to have been little laboratory involvement prior to 1996. The only recorded full-term laboratory submissions up to this time were two calves in 1994 and one in 1995. While only four calves were lost at birth in 1996 – three of which were submitted for laboratory examination - there were 10 stillbirths in 1997, the first year of the Longitudinal Study (Chapter Three). Of the three calves examined in 1996, one was from a set of twins, one was from a heifer, and *Campylobacter foetus* – a recognised cause of early abortion – was isolated from the third.

It is obviously not possible at this remove to identify the specific causes of the problem prior to 1996. A full laboratory investigation would have had to consider the usual risk factors for perinatal calf mortality (weak calf syndrome). These include dystocia, uterine inertia (e.g. due to hypocalcaemia), and factors associated with calving management (e.g. premature intervention).

Dystocia is by far the most common cause of perinatal mortality (McDermott *et al.*, 1992). In this regard, it is important to note that the PVPs referred to dystocia as being the commonest reason for 23 calls they made to attend calvings on. this farm between 1994 and 1996. As the incidence of dystocia is significantly higher in heifers than cows, the high proportion of heifers in the herd in 1995 (stated to be 45 *per cent* in the Retrospective Survey Report) will also have increased the risk of calf mortality due to dystocia at that time. Factors contributing to the incidence of stillbirths in 1997, the first year of the Longitudinal Study, are discussed in Chapter Three.

Although deficiencies of the minerals selenium and iodine have been associated with perinatal mortality, it is unlikely that they were significant contributory factors in relation to the problem on this farm. While low blood glutathione peroxidase concentrations were recorded in samples collected between 1993 and 1995, the lack of response to selenium supplementation in 1995 would not support its involvement. There was also no evidence to suggest that iodine deficiency was involved. The results of all analyses for the iodine-containing thyroid hormone thyroxine carried out between 1993 and 1995 were within the normal range. Plasma inorganic iodine concentrations of blood samples collected from milking cows during the Longitudinal Study in 1997 were also within acceptable limits. There was also no pathological evidence of thyroid follicular hyperplasia in calves submitted for laboratory post-mortem examination.

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Calf diarrhoea was also reported to have been a problem on this farm since 1991. Calves were initially affected at approximately 14 to 21 days of age - then progressively at younger ages and usually in the latter part of the calving season. Affected calves had a watery diarrhoea. However, in the absence of specific information regarding incidence rates, it is not possible to comment on the severity or otherwise of the problem.

Calf diarrhoea is a common problem on farms elsewhere with incidence rates of up to 100 *per cent* being reported (Appendix 1). Problems can be particularly severe in large spring-calving dairy herds due to the build-up of pathogens during the calving season – which is consistent with the history given above. Recognised infectious pathogens – viruses and protozoa – were identified on several occasions by laboratory examination of clinical specimens from calves on this farm. Evidence of widespread protozoal (cryptosporidia) infection was again detected in diarrhoeic calves in 1997. Two of four calves blood-sampled at this time also had low blood immunoglobulin concentrations indicating inadequate supply or absorption of colostrum. In the circumstances, there is no indication to suggest that factors other than those normally cited, i.e. infectious agents, diet, and build-up of environmental infection, were the main factors responsible for the incidence of calf diarrhoea on this farm.

An outbreak of pneumonia - coughing and raised temperature - was reported to have occurred in housed calves in 1995. Signs persisted while the calves were at grass. Although response to treatment was slow, no deaths occurred. Tests for virus on nasal swabs were negative. However, despite these negative results, there is nothing in the description of the outbreak to suggest that the cause was other than infectious. A positive identification of virus in nasal swabs is highly dependent on factors such as stage of infection and method of collection and delivery - a negative result does not necessarily rule out the possibility of viral involvement. Outbreaks of respiratory infection of this type are common in housed calves with residual coughing and lung damage often persisting later on grass.

The incidence of mastitis cases described in the Retrospective Survey Report – 23 to 34 *per cent* annually from 1989 to 1992 – although moderately high, is within the reported range for a dairy herd of this size (Appendix 1). Other problems reported on this farm included pneumonia in a bull, sudden deaths in young cattle and ill thrift in cows. However, the incidence of these problems was low

and well within reported normal ranges (Appendix 1).

Overall, while this farm has experienced problems with fertility, calf diarrhoea and, most notably dystocia and perinatal calf mortality, there is no evidence to suggest abnormal or unusual factors were involved in their occurrence. The infertility was similar in type and degree to that observed on many other dairy farms of this size. The calf disease problems, while severe, are comparable to outbreaks seen on farms in other areas and their occurrence can largely be attributed to infectious causes.

FARM RS12

Main Enterprises: Dairy, store
Herd size: Small

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-15. The opinion of the herdowner's PVPs regarding the main problems encountered is given in Table 5-16. The main problem reported was infertility – primarily of cows repeating to service at regular and irregular intervals. Analysis of fertility data for the years 1994 to 1996 indicated a high rate of non-detected oestrus (56 – 67 *per cent*). As discussed elsewhere, the rate of non-detected oestrus is largely a function of breeding management. This applies to an even greater degree in small herds where heat expression may be reduced owing to reduced opportunities for interaction between cows on heat (*see* page 6). Although insufficient information is available to determine the causes of reduced fertility performance on this farm, there is no specific reason to suggest that factors other than those commonly involved on farms elsewhere were involved.

Though not specifically referred to as problems, data in the Retrospective Survey Report suggest that the incidences of lameness and mastitis were also relatively high. However, insufficient information is available to discuss aetiology.

UPDATE 1997

This farm was not re-visited in 1997 at the request of the herdowner.

FARM RS13

Main Enterprises: Beef, store, dairying,
                            sheep, goats
Herd size: Small

A summary of the animal health and production problems on this farm is given in Table 5-17. The opinion of the herdowner's private veterinary practitioners regarding the main problems encountered are given in Table 5-18.

The main disease problem reported - which occurred exclusively on the outfarm - was an outbreak of illness in dry stock (bullocks and heifers) characterised by diarrhoea, ocular and nasal discharge, salivation, and recumbency. Subcutaneous abscesses were reported in some animals several months after recovery. All of 27 cattle at risk were affected and 16 died. A similar condition was reported in adult sheep and goats. All were affected and mortality was reported to be 100 per cent (25 sheep, 20 goats). Deaths were also reported to have occurred in wildlife.

Despite the severity and duration of this outbreak (December 1991 to April 1992), there was minimal involvement of the herdowner's PVP and no consultation with the local Regional Veterinary Laboratory. No clinical or morbid pathology material was submitted for laboratory examination. In the circumstances, therefore, it is not possible to make more than general comments on the outbreak.

Management on the farm was extensive. There was no cattle housing on the outfarm for overwinter accommodation and no fertilizer was applied to pastures. Cattle on the outfarm were fed hay alone from December onwards. In 1991, hay feeding commenced at the end of December – shortly before the first cases occurred.

Based on the clinical signs, infectious conditions which could have been considered include fluke infestation, salmonellosis, *Campylobacter* infection (winter dysentery), malignant catarrhal fever (cattle), and BVD virus infection (cattle) to name a few. The potential role of nutrition in relation to the development of the problem must also be considered. The generally rough quality of pasture on the outfarm, combined with stocking density (47 cattle, 41 sheep and 20 goats on 20 hectares in summer 1991; 20 two-year old bullocks were sold in September/October; disease outbreak occurred in December, 1991), and absence of any supplemental fodder up to the end of December, raise the possibility that many animals on the outfarm may have been in poor state of nutrition at the time of the disease outbreak.

Cobalt deficiency may also have been a contributory factor in relation to the problem in sheep. Manganese concentrations (761 – 1,322

mg/kg) high enough to inhibit plant uptake of cobalt were detected in soil samples collected on the outfarm in 1997.

The possibility of a toxic cause of the outbreak obviously cannot be ruled out. However, owing to the complete absence of material for analysis, its possible identity can only be a matter of speculation. There is nothing in the reported clinical history which would suggest involvement of a particular toxic agent. Neither is there anything in the history which could be reasonably associated with atmospheric sulphur dioxide emissions - the potential pollutant most often considered in the context of the Askeaton investigation. Even if exposure to a relatively high concentration of SO_2 was to have occurred, it would probably have resulted in an acute and relatively short-lived outbreak of upper respiratory signs in affected animals – not the prolonged and predominantly enteric condition reported here. In the context of local geography, it would also be extremely unlikely that such severe exposure would be confined to a single premises. No similar outbreaks were reported on other farms in the area either at that time or at any time since then.

Overall, therefore, while no specific diagnosis can be made for the severe outbreak of disease on this farm, this is largely due to the fact that expert assistance was confined to three farm visits by the veterinary practitioner and no laboratory investigation of any kind.

UPDATE 1997

No stock were present on the outfarm in 1991/92

FARM RS14

| Main Enterprises: | Single-suckling, store |
| Herd size: | Small |

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-19. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-20. The main problems reported were respiratory disease and diarrhoea in cattle.

In the autumn of 1993, one yearling of approximately 15 was affected by respiratory disease and illthrift. A four to five month-old homebred heifer, in a group of approximately 11, died in autumn 1994 following illthrift and respiratory disease. A two week old calf and a six to eight month old purchased calf developed respiratory disease and illthrift in September 1994. The PVP reported a good response to treatment. Of

approximately 15 year-and-a-half old cattle on the farm in the autumn of 1995, three had respiratory disease and diarrhoea and lost body condition.

The outbreaks of respiratory disease described above would not appear to have been unusual either in type or severity. Their occurrence was consistent with an infectious aetiology. The presence of BVD virus infection was also subsequently confirmed in two animals in 1996. The animals in question were described as stunted in the report of the DAF stunting/ill-thrift investigation of August 1996 (DAFRD unpublished).

Two cow deaths were also reported – one in August/September 1994 and the other in October 1995. One was an old cow with purulent mastitis and the other was consistent with BVD virus infection. In conclusion, there is no evidence either of an unusually high incidence of disease on this farm or of a problem of unusual or undiagnosed diseases.

UPDATE 1997

This farm was re-visited in September 1996 to investigate reports of ill-thrift in growing animals. BVD virus infection was confirmed in two affected animals. Some animals also had low blood copper concentrations.

FARM RS15

Main Enterprises: Dairy, store
Herd size: Medium

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-21. The opinions of the herdowner's PVPs regarding the main problems encountered are given in Table 5-22. The main problems comprised infertility and an outbreak of ocular discharge in cows. However, insufficient breeding records were available to analyse fertility performance in any detail. From the history, the main problem appeared to have been non-detected oestrus – though overall pregnancy rates, at between 80 and 90 per cent, were within the normal range for herds of this size. As discussed elsewhere, heat detection is a function of fertility management and is extremely unlikely to be caused by atmospheric pollution.

Copper deficiency, as a result of a low pasture copper to molybdenum ratio, may have had a negative effect on fertility in heifers not receiving concentrate supplementation. Analysis of partial breeding data for 1997 indicated that the only significant problem with fertility performance at

that time was a low submission rate at the start of the season due to missed heats.

An outbreak of conjunctivitis was reported in four of about 40 cows in summer, 1995. According to the herdowner, the PVP diagnosed 'pinkeye' which is an infectious condition. The herdowner also reported that in the same month a 'red dust' was deposited on the cows backs and on the pastures. Investigations by Teagasc and DAF staff at the time revealed crown rust in the pastures. This is a fungal infection which gives rise to a reddish discoloration of affected grasses. It is not known if there was any connection between the occurrence of crown rust in the pastures and the presence of a red dust on the cows.

UPDATE 1997

Infertility continued to be a problem in 1997. A complete analysis of the problem was not possible owing to inadequate breeding records.

FARM RS16

Main Enterprises: Dairy.
Herd size: Medium large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-23. The opinions of the herdowner's PVPs regarding the main problems encountered are given in Table 5-24. The problems comprised a series of probably interrelated periparturient conditions, i.e. slow calving, sub-clinical milk fever and attendant complications, e.g. stillborn calves, downer cows, pining cows. Infertility was also a problem.

Delayed calvings have been a problem on this farm since 1992. A report of VLS investigations on the farm in 1997 concluded that it was associated with uterine inertia due to sub-clinical milk fever. Recommendations were made regarding control. According to the PVPs, there were also problems of dystocia in heifers due to relative foetal oversize and premature assistance at calving.

Reference is made in the Retrospective Survey Report to the deaths of four cows in 1992 and two in 1995. However, none were submitted for laboratory post-mortem examination and, at this remove, it is not possible to identify the likely causes. The histories suggest they occurred around calving and may have been secondary to dystocia or other peri-parturient complications.

The incidence of perinatal calf mortality was around 10 per cent over the four-year period from 1992 to 1996. However, given the history of

calving difficulties, and the fact that half of the calf losses were twins, this figure is not exceptional. The incidence of twins itself, at 4.0 *per cent* was not abnormal (Appendix 1). Only one calf was submitted for laboratory examination over the period 1992 to 1995.

A serious outbreak of virus-type pneumonia was reported to have occurred in 1997. However, although between 12 and 15 calves were lost, there were no submissions to the laboratory. A farm investigation by laboratory staff in 1997 indicated that calf housing may have been a contributory factor.

Infertility was reported to have been a problem since at least 1993. The limited records available for analysis indicate that the problem was predominantly one of non-detected oestrus. However, the infertile rate in 1995 at 7.3 *per cent*, the only year for which it could be computed, was well within the normal range. Services on the farm were by DIY artificial insemination and natural mating. However, no information was available regarding the fertility status of bulls or details of the extent to which they were used during the breeding season. Reduced copper availability, as a result of high herbage molybdenum, may have had a negative effect on fertility at times.

A number of problems affecting individual animals at the time of the Retrospective Survey farm visit in 1995 were also described. These comprised spinal deviation in a cow and yearling, skin lesions in a cow and illthrift in a cow. With the exception of a genetic relationship between the deformed cow and yearling, there was no evidence that the problems were connected or formed part of a disease pattern. Their incidence was not abnormal.

UPDATE 1997

This herd was reported to have experienced a serious problem with respiratory disease in calves in the spring of 1997. Although there were 12–15 deaths, none were submitted for laboratory post mortem examination. At the 1997 summer re-visit, problems with calf housing were identified and recommendations for improvement were made. Several cases of coliform mastitis were also reported to have occurred during the calving season. These may also have been housing-related.

FARM RS17

Main Enterprises: Beef, suckling.
Herd size: Medium large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-25. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-26. The main problems related to the calving period. Other problems of lesser severity were diarrhoea and illthrift in yearlings, reduced milk yield and respiratory disease in weanlings.

The perinatal problems, which dated from about 1990-91, comprised difficult calvings, calf mortality, and downer cows and were undoubtedly inter-related. From the history, relative foetal oversize was obviously a major factor in the incidence of dystocia. Both the PVP and the Teagasc advisor are reported as referring to the size of calves sired by the Charolais bull. The PVP also refers to 'prolonged assistance' as being a contributory factor and there is also evidence of both premature and excessive assistance in calvings. Calf losses were reported to have been between 16 and 20 *per cent* per year.

Much of the calf mortality would appear to have been secondary to dystocia based on the history. Some followed posterior presentation deliveries which are typically difficult deliveries. The yellow 'slime' reported on newborn calves was probably meconium and is consistent with hypoxia during a prolonged or difficult calving. The relatively high incidence of 'downer' cows was probably also dystocia-related. Four to five cows are reported to have gone down each year after calving.

The reported incidence of calf deformities on this farm was not abnormal.

A number of cases of diarrhoea and ill thrift in yearling cattle between 1991 and 1995 were described. Neither the incidences nor descriptions of the cases were unusual. Possible diagnoses included salmonellosis, coccidiosis and bracken poisoning. A condition resembling redwater (babesiasis) was also described in a heifer.

Outbreaks of pneumonia in calves at grass were reported to have occurred between 1991 and 1993. The histories are consistent with viral infection - with or without an underlying lungworm infestation. Outbreaks of this type are common in late summer or autumn. Neither the reported morbidity nor mortality were unusual.

A low incidence of sudden deaths (about 1 *per cent*) was reported in recently-weaned calves after de-horning between 1990 and 1994. There are many possible explanations for these deaths -

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including complications associated with dehorning - and the incidence is not abnormal.

Milk yields were said to have been low between 1991 and 1993. However, no data was provided and, in the circumstances, no particular significance can be attributed to the claim. From information provided in the Retrospective Survey Report, factors which may have contributed to a reduction in yields include the relatively high rate of replacement heifers in the herd each year, a mastitis problem, and *Leptospira hardjo* infection (milk drop syndrome). The presence òf the latter was identified in the herd in 1995 and 1997.

Overall, while this farm had a significant number of animal health problems, they largely related to the perinatal period and can generally be attributed to the usual risk factors associated with difficult calvings. Other problems were probably largely infectious in origin and, while economically significant, the outbreaks are consistent with normal farming experience.

UPDATE 1997

Although no significant animal health problems were reported at the 1997 re-visit, serology tests identified recent active *Leptospira hardjo* infection in the herd.

FARM RS18

Main Enterprises: Dairy, store
Herd size: Small

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-27. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-28. Infertility was the only significant problem reported. According to the herdowner, it had been a problem since at least 1989. Analysis of records for the years 1993 to 1996 indicated that the main problem was one of non-detected oestrus in two out of the four years with a consequent extended calving pattern (Table 4 in Retrospective Survey Report and Table 11 {farm code 127} in Interim Report {EPA, 1997}). In addition to the usual factors affecting heat detection, the small size of the herd must be taken into account in assessing performance. As discussed elsewhere (*see* page 6), it is well recognised that heat detection is more difficult in small herds due to reduced opportunities for interaction between cows on heat.

Although some low blood coppers were reported from cows, it is not possible to state what significance this may have had on fertility.

Three other isolated and apparently unrelated cases of illness were reported in cattle. They were not of any specific significance in the context of the present investigation.

UPDATE 1997

Infertility continued to be a problem in 1997. However, no breeding records were available for analysis.

FARM RS19

Main Enterprises: Dairy, store
Herd size: Medium large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-29. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-30. Infertility was the only significant problem reported. Analysis of partial breeding records from 1990 to 1995 indicated high rates of non-detected oestrus in the start of the season leading to low submission rates and an extended calving pattern (four months in 1992 increased to eight months in 1995). The factors which are generally associated with reduced heat detection efficiency have been discussed elsewhere.

An improved submission rate in 1995 may have been due to the introduction of tail-painting to assist heat detection before the bull was put with the cows. However, a poor conception rate later in the season may also have been associated with use of a new bull. As no information is available regarding bull fertility, their contribution to fertility performance cannot be determined.

The herdowner's reference to a history of 'regular repeats' is strongly suggestive of a bull fertility problem. However, an analysis which was carried out on available breeding data for 1994 and 1995 indicated that the percentage of regular (i.e. 18 - 24 day) returns to service was below target — indicating an increased incidence of irregular repeats. This is more likely to have been due to factors such as inaccurate heat detection (before introduction of the bull) or increased embryonic mortality.

Reduced copper availability, due to high molybdenum concentrations in some grazing areas, could also have been a limiting factor in relation to fertility performance at times. However, the extent of this cannot be determined. Recommendations for twice annual copper depot administration were made in 1997 on the basis of low blood copper findings at the time.

Overall, while fertility performance on this farm was well below target in some years, the problems appear to have been similar to those experienced in many other herds and there is no evidence that unusual factors were involved.

UPDATE 1997

Infertility continued to be a problem when the farm was revisited in 1997 at which time a bull was being used to serve all cows. However, no breeding records were available for analysis.

FARM RS20

Main Enterprises: Suckling, store
Herd size: Medium large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-31. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-32. The main problem was respiratory disease in weanlings in 1995 and in calves in 1996.

In November 1995 an outbreak of respiratory disease occurred in about 20 of a group of 77 outwintered weanlings. Infectious pneumonia was diagnosed by the PVP. No material was submitted for laboratory investigation. In the spring of 1996, between 25 and 30 calves died out of over 100 home-bred and purchased calves. The main causes of death were reported to have been calving-associated (perinatal mortality), diarrhoea and virus pneumonia. The PVP referred to treating five cases of diarrhoea or pneumonia. A single calf was submitted for laboratory examination at the end of May, 1996. Post-mortem findings were consistent with Respiratory Syncytial Virus (RSV) infection.

Overall, while the history indicates severe outbreaks of disease in calves, the majority of cases were probably infectious in origin. Others were said to have been calving-associated. The fact that home-bred and bought-in calves were involved in the 1996 outbreak could have contributed to the severity of the outbreak due to the combined effects of the stresses associated with mixing, together with variations in their immune statuses.

According to the herdowner, the calf problems were largely solved by an intensive program of mineral supplementation instituted in 1996. This was based on the results of milk analyses carried out by a commercial laboratory. However, the relationship of milk mineral concentrations to nutrient requirements is open to question. Puls (1994) suggests that iodine is the only mineral which can be usefully measured in milk. The

results of analyses of herbage samples collected on this farm in 1979/81 and 1994 indicated that, with the exception of selenium which was adequate in 1979/81 and marginal in 1994 (the difference possibly reflecting sampling locations), mineral concentrations were within acceptable ranges. Evidence of marginal selenium status was the only significant finding following analyses of blood samples also collected from cattle at these times.

A number of other isolated cases of disease in cows in 1994 and 1995 were reported. These comprised diarrhoea (two cases), eye lesion (one cow) and facial paralysis (two cows). The incidence of these conditions is not unusual and straightforward explanations are available. The two cases of facial paralysis, for example, were consistent with Listeria monocytogenes infection. The reported incidence of deformed calves in 1994 and 1995 (two cases in total) was not unusual. A number of other isolated problems was reported in cows and a yearling in 1996 which were well with the range of normal farming experience.

UPDATE 1997

No specific health problems were reported at the re-visit in 1997.

FARM RS21

Main Enterprises: Suckling, store & beef
                     cattle, sheep
Herd size: Medium-large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-33. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-34. The main problems reported were retained foetal membranes, infertility and grass tetany (hypomagnesaemia) in cows, sore eyes in cattle, subcutaneous lumps in cows, illthrift in adult cattle, deformed calves and illthrift in lambs.

About 12 of 41 cattle, mainly heifers, were reported to have retained placentas post-calving in spring 1995. The PVP who attended these cases suggested that the use of a continental (i.e. large) sire on the heifers may have been a contributory factor. Low to marginal herbage and blood selenium concentrations have also been detected on this farm. However, the extent to which selenium deficiency may have been a contributory factor cannot be determined. While selenium has been reported to be a risk factor for retained placenta (Julien et al., 1976) recent work has cast doubt on this conclusion (Wichtel, 1998).

Three cows of a group of 20 - 30 were reported to have died as a result of grass tetany in the spring of 1993. However, while the losses were significant this is not an unusually high incidence in an outbreak. Outbreaks of grass tetany occur when blood magnesium concentration is below normal. Blood magnesium concentration, in turn, is a function of dry matter intake. There is no apparent basis to associate the occurrence of grass tetany with atmospheric pollution in the context of the Askeaton investigation.

Infertility in cows and heifers was reported to have been a problem since 1993. All matings were by natural service. As no breeding records were kept, and no fertility examination results of bulls were available, it is not possible to determine the nature or causes of the problem. However, the reference to 13 out of 23 heifers served by a single bull in 1994 subsequently being found barren is strongly suggestive of low bull fertility.

Conjunctivitis ('pinkeye') was reported to have been a problem since 1991. Up to 90 *per cent* of cattle on the home farm were affected in 1995 and cases lasted from one day to a week. The causes of conjunctivitis include infectious, mechanical (trauma, foreign bodies), and chemical agents. Infectious conjunctivitis, which may be caused by bacteria, mycoplasmas or viruses, is common in livestock. Bacterial conjunctivitis is particularly common in summer and autumn (Blood and Radostits, 1990). The agents may be spread by flies. The clinical descriptions of these cases are consistent with infectious conjunctivitis. Irritation due to grass seeds and pollens can also cause conjunctivitis. The possible involvement of environmental pollution could only have been investigated by analysis of air samples collected in the immediate vicinity at the time of the outbreaks.

About 10 of 30 Friesian cows purchased to fatten were reported to have developed subcutaneous lumps in January 1995. Although these animals received no veterinary attention, the descriptions are consistent with subcutaneous abscesses. The causes obviously cannot be determined at this stage. Factors to be considered would have included abrasions and injection site reactions.

Illthrift is said to have been a problem in adult cattle (two to two-and-a-half year old) since 1991. However, information is only provided relating to 15 animals bought in 1991 and there appears to have been little if any veterinary involvement in the problem. The PVP recalled attending suckler calves with diarrhoea and pneumonia in 1995 and 1996. The DAF report on an investigation into stunting of cattle on farms in the Askeaton area in

1996, found no evidence of stunting of cattle on this farm (DAFRD unpublished). Overall, insufficient evidence is provided in the Retrospective Survey Report to assess the severity, duration or causes of this problem.

Deformed calves were reported to have been born in 1994 and 1995. However, the incidence at one per year was well within the normal expected range.

Illthrift in lambs is reported to have been a problem since 1989. In 1995, there was diarrhoea and pining in lambs. There was no veterinary involvement in investigation of the problem. At this stage, it is obviously not possible to determine the causes. Problems of this type are common in sheep flocks - especially in the latter part of the grazing season. The causes are many and include parasitism, mineral deficiency, bacterial infection and grass quality.

In summary, while this farm reported a wide range of problems, some at an apparently high incidence, there is no specific evidence to associate any of them with unusual causes such as environmental pollution. In most cases the problems appear to have been of a type seen on farms elsewhere and straightforward explanations can be adduced to account for their occurrence. Due to the nature of the enterprise, there was a high rate of movement of adult cattle through this farm. This is a specific risk factor for an increased incidence of a variety of diseases - both due to mixing of cattle from various sources and to the often unknown health history of purchased adult animals. Bruning-Fann and Kaneene (1992) reported that disease rates were several times higher on farms which bought in calves than on those which did not.

UPDATE 1997

No specific health problems were reported at the re-visit in 1997.

FARM RS22

Main Enterprises: Dairy, pig fattening
Herd size: Medium large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-35. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-36. Infertility was said to have been a problem since 1991. Low conception rates and a prolonged breeding season were reported to have been the main problems. Detailed breeding records are not available. According to the herdowner, signs

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included cows not holding to first service and repeating regularly – with a few returning to service at longer intervals.

Because of the absence of breeding records, it is not possible to make an assessment of fertility performance on this herd. However, the reported infertile rate of around 5 per cent per year is well within acceptable limits for a herd of this size (Appendix 1). Although it was suggested that low trace element status may have had a limiting effect on fertility performance, the analytical evidence in this respect is either inadequate or conflicting. Grass samples collected in July 1995 and analysed by a commercial laboratory are said to have had a high sulphur and low selenium and iodine content. However, the actual results are not available. While soil samples collected by Teagasc in 1994 had low selenium concentrations, analysis of blood samples (glutathione peroxidase) collected between 1992 and 1996 indicated only marginal selenium deficiency and selenium concentrations of samples collected in 1997 were normal.

An outbreak of respiratory disease in cows associated with a drop in milk yield occurred in June 1995. Clinical and laboratory findings were consistent with a viral infection. Almost all cows were affected.

UPDATE 1997

Fertility continued to be a problem in 1997 - though not of a serious nature. According to the herdowner, genetic merit of cows and high milk production may have been contributory factors. No breeding records were available for analysis. Serology results indicated active *Leptospira hardjo* infection in herd. This may also have contributed to infertility.

FARM RS23

Main Enterprises: Multiple-suckling
Herd size: Medium

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-37. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-38. The main problems reported were respiratory disease in calves and weanlings and an outbreak of locomotor disease in calves.

Respiratory disease was reported to have been a problem in housed calves and weanlings since 1991. While severe, the history and clinical descriptions are consistent with outbreaks seen elsewhere and which are usually associated with a

combination of infectious and environmental (housing and weather) factors. Known bacterial, mycoplasmal and viral pathogens were isolated from outbreaks on this farm. Housing and mixing of calves of different ages and sources (home-reared and bought-in) were also cited as contributory factors by the PVP. Ventilation and hygiene deficiencies of housing were also referred to in Teagasc and VLS reports of the 1997 farm visits. The report of the DAF investigation into claims of stunting and ill-thrift, referred to the poor condition of some animals due to overstocking at the time of the farm visit in July 1996 (DAFRD unpublished).

Between November 1993 and March 1994, six of ten suckler calves were reported to have died within two weeks of birth after showing signs of collapse, lethargy, inability to rise and splayed legs. No carcases were submitted for laboratory pathology examination during the outbreak. While a diagnosis of white muscle disease (selenium/vitamin E deficiency) was made by the PVP at the time, the possibility of the involvement of secondary copper deficiency in the outbreak should also be considered. Although selenium deficiency (glutathione peroxidase analysis) was not detected in blood samples from dams of five calves submitted for laboratory examination in October 1994, the mean copper concentration was at the lower end of the normal range. Herbage samples collected from this farm on 1997 had a relatively low herbage copper:molybdenum ratio indicating the potential for induced copper deficiency.

In conclusion, while the above disease incidents were undoubtedly of a severe nature, the most likely causes are infectious, management and nutritional factors. There is no specific evidence to suggest involvement of environmental pollution.

1997 UPDATE

Respiratory disease in bought-in calves was the main problem reported at the 1997 re-visit. Recommendations were made regarding essential improvements to ventilation of cow and calf houses and calving pens.

FARM RS24

Main Enterprises: Suckling, store cattle, sheep
Herd size: Medium-large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-39. The

opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-40. The main problems reported were respiratory disease and diarrhoea in cattle and the death of two cows.

An outbreak of respiratory disease was reported to have occurred in all ten of a group of two-year old cattle at grass in summer, 1994. According to the herdowner, it was characterised by coughing and ocular discharge. No deaths occurred and no material was submitted for laboratory examination. Coughing persisted for about eight months and animals failed to thrive. Diarrhoea with fresh blood in the faeces was also seen after the cattle had been moved to aftergrass in autumn, 1994. The PVP recalled attending a severe outbreak of pneumonia in purchased weanlings.

While the herdowner considered that atmospheric pollution was responsible for this outbreak of disease, there is no specific evidence to support this suggestion. The history is consistent with an infectious cause, e.g. parasites, bacteria and/or viruses. Possible explanations for the appearance of blood in the faeces include the effects of eating lush grass (i.e. diarrhoea and rectal bleeding), coccidiosis, and salmonellosis. Wet windy weather conditions from December 1994 to February 1995 may have contributed to the prolonged nature of the outbreak.

One cow died in November 1994 and one in May 1995. The PVP's diagnosis of grass tetany in the first case is consistent with the reported history and clinical signs. The cow which died in May 1995 was recumbent for several days before death. Laboratory post mortem findings indicated the animal was emaciated. Although this case was said to have been one of four affected cows, the only information available in relation to the other animals is that they were in poor body condition. While no conclusions can be drawn regarding the cause of illness in these four cows, the fact that they were outwintered and had received no concentrate supplementation indicates that nutrition must be considered as a potential contributory factor. Poor weather in the spring of 1995 would also have resulted in an increased energy requirement for outwintered animals.

Overall, the incidence and severity of animal health problems reported on this farm were not unusual in terms of normal farming experience.

UPDATE 1997

A serious problem of stillbirths and dystocia was investigated at the herdowner's request in May

1997. Relative foetal oversize, and possibly also high calcium intake on lush grass — leading to uterine inertia, were regarded as the main contributory factors. No further significant animal health problems were reported during a re-visit later in the summer of 1997.

FARM RS25

Main Enterprises:	Dairy, store and beef cattle
Herd size:	Medium

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-41. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-42. The main problems reported on this farm were perinatal calf mortality, disease and illthrift in weanlings, mastitis, lameness, infertility and unexplained deaths of animals of all ages.

Perinatal calf mortality was reported to have been a problem in 1989/90. Between 10 and 12 of 50 calves born from November 1989 to February 1990 were reported to have died within an hour of birth. While this is a high incidence, the history is consistent with losses due to prolonged or difficult calvings. Most calvings were said to have been assisted and some were large, overterm calves. The herdowner's description of brown discoloration of calves is also consistent with meconium-staining associated with hypoxia during calving.

Diarrhoea and pneumonia, followed by chronic illthrift, were also reported to have been an annual problem in weanlings at grass. Signs were said to have persisted when animals were housed over winter and it was at this time that most losses occurred.

Based on the history and reported clinical signs, these outbreaks are consistent with infectious causes, i.e. parasites, bacteria and viruses. Parasitic pneumonia (hooze) and gastro-enteritis were diagnosed in autumn 1994 and again in autumn 1997. Pasteurellosis and salmonellosis were also diagnosed during laboratory investigation of the 1997 outbreak. Housing on the farm is old, poorly ventilated and difficult to clean. This undoubtedly contributed to the persistence of disease outbreaks over-winter.

Poor thrive in weanlings has been a problem for some years. In addition to the after-effects of infectious disease as outlined above, it is likely that nutrition also played a role. According to the Retrospective Survey Report, calves received no

compound ration in their first year. The report of the DAF investigation of ill-thrift and stunting on this farm, carried out in June 1996, stated that "*most of the thirty-four calves ... appeared to be on the verge of stunting and were small for their age*". They were being fed little if any meals and very poor quality hay. The report concluded that the condition of the animals was solely due to nutritional factors (DAFRD unpublished).

A serious lameness problem existed on this farm for some years. Investigations carried out in the latter half of 1997 indicated that the most important contributory factors were traumatic foot damage as a result of the poor condition of roadway surfaces compounded by softening of hooves due to poor drainage of yard and roads. These were compounded by an inadequate program of foot care together with lack of facilities for examination and treatment of feet.

Mastitis was also reported to have been a severe problem. The history in the Retrospective Survey Report, as well as the results of farm investigations carried out in 1997 and 1998, indicated the involvement of both infectious and environmental (i.e. milking machine, hygiene and housing) factors. The milking machinery, in particular, was old and was not subject to regular maintenance. High milk somatic cell counts were recorded on a number of occasions and a variety of infectious agents were isolated from milk samples.

Although not specifically referred to as a problem, milk yields were low on this farm throughout the period for which records are available (1991 - 1997). Average yields per cow fell from 2,832 kg in 1991 to 2,518 in 1994 and were only around 2,728 kg in 1997. The major factors contributing to this included the restricted use of concentrates in early lactation, late calving due to infertility, a high proportion of heifers in some years, mastitis, lameness and pasture management (inadequate fertilizer usage and intermittent overstocking). Inadequate water supply for cows at grass was also identified as a probable contributory factor in 1998.

Infertility was reported to have been a problem since 1993. However, detailed records were not kept and very little information was available on fertility performance. Due to the combined effects of an extended breeding season and low conception rate, calvings took place throughout the year. It is likely that this also had a negative impact on heat detection results. Inadequate concentrate supplementation and poor cow condition post-calving are also likely to have negatively affected overall herd fertility performance. Although a bull was used for all services, there was no record of

any fertility assessments of bulls having been carried out. Their possible contribution to fertility performance, therefore, cannot be estimated. However, it is most likely that the severe lameness experienced by the stock bull in 1997 had a significant negative impact on libido and performance.

The reported problem in relation to unexplained animal deaths largely related to the period from 1996 onwards and was not described in the Retrospective Survey Report. According to information provided by the herdowner in July 1997, about 30 cattle died in 1996. These comprised about 10 cows and the remainder were calves and yearlings. Most of the deaths occurred in the spring and autumn. Some cows died around calving.

A further seven animals, including three cows, were reported to have died in the first six months of 1997. None of the losses in 1996 or the first half of 1997 were submitted for laboratory examination and there was little veterinary involvement in their investigation. Because of the lack of information on the circumstances surrounding these losses, it is not possible to determine their exact causes. Based on history supplied by the herdowner, it would appear that specific conditions included toxic mastitis, bloat, perinatal downer cow syndrome, pneumonia, salmonellosis and other infectious conditions. Given the severity of the lameness problem, it is likely that this may also have contributed to losses.

Inadequate nutrition, especially in the immediate pre- and post-calving period, is also likely to have been a contributory factor. Silage quality in 1997 was poor (DMD 55 *per* cent - first cut). Given the restricted use of concentrate supplementation – both pre- and post-calving - it is likely that many cows would have calved down in poor condition and, as a result, had a reduced resistance to infection and an overall lowered health status. Cows were reported to have been in poor or moderate body condition at the time of the Retrospective Survey farm visit in December 1995 and many cows were observed to be in poor condition at farm visits in the second half of 1997.

Intermittent copper deficiency may also have contributed to the severity of some disease outbreaks *via* a reduced immune response. Low copper and high molybdenum concentrations were recorded in some herbage samples collected on the farm in 1997.

Ringworm was reported to have been a problem in calves for some years. According to the herdowner

it was less severe in 1997 due to copper supplementation. Other problems reported on this farm included neonatal calf mortality and an outbreak of abortion in 1989. The latter was attributed to salmonellosis. The reported incidence of neonatal calf mortality was well within acceptable limits.

Overall while this herd suffered severe animal health and production problems, they can largely be attributed to a combination of on-farm factors, i.e. infectious agents, management and animal nutrition.

UPDATE 1997

This farm was the subject of an on-going laboratory investigation during 1997 and 1998. The problems encountered have been discussed above.

FARM RS26

Main Enterprises: Suckling, sheep
Herd size: Medium

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-43. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-44. The main problems reported were virus infection in calves (BVD) and calving problems in cows. Three of 30 calves developed diarrhoea and mouth ulcers in 1994. Laboratory tests indicated the problem was due to BVD virus infection. This is a straightforward diagnosis and there is no indication to suggest that other factors were involved.

Dystocia (difficult calving) was reported to have been a problem in 1995. All of 30 calvings required assistance. However, no cows and only one calf died as a result. According the Retrospective Survey Report *"the farmer and the private veterinary practitioners attributed this problem to overcondition of the dry cows and use of an overly muscular bull"*.

Except for the dystocia problem in 1995, and for which there is a relatively straightforward diagnosis, this farm does not appear to have had a particularly high incidence of animal disease.

UPDATE 1997

Dystocia was again reported to have been a problem in 1997. Large calves sired by the bull, and overcondition of cows, were probably the main contributory factors.

FARM RS27

Main Enterprises: Dairying, calf rearing.
Herd size: Medium large

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-45. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-46. The main problems reported were infertility, illthrift in growing cattle and abortion.

Infertility was reported to have been a problem in 1994. However, detailed breeding records are not available. The main problem was reported to have been repeat breeders and an extended calving season. The infertile rate for 1994 was reported to have been 27 *per cent*. Repeat breeders and an extended calving season were also noted at the 1997 veterinary farm visit.

In the absence of detailed records it is not possible to determine the nature or extent of the fertility problem on this farm. The main factors associated with repeat breeding have been discussed elsewhere. As bulls were used extensively for services on this farm, any investigation of breeding performance would have required detailed information regarding their fertility status. Post-calving energy status may also have had an effect on fertility as the report on the veterinary visit to this farm in May 1997 noted that cows were *"in moderate to poor condition and some were losing weight."*

Although herbage samples collected in 1994 were reported to have had a relatively high sulphur content (0.59 *per cent*), there is no evidence that this had a significant affect on copper availability for stock. Herbage copper:molybdenum ratios were high in 1994 and 1997 and blood copper concentrations of cattle sampled in 1993, 1995 and 1997 were generally within the normal range. Selenium availability, on the other hand, could have had an influence on fertility performance. Herbage selenium concentrations were marginal ($\cong$ 0.05 mg/kg) in 1994 and 1997 and blood glutathione peroxidase concentrations (an indirect estimator of selenium status) of cattle sampled in 1993 and 1995 were also low to marginal. However, blood selenium concentrations were within the normal range in animals sampled in 1997. While hypomagnesaemia (subclinical) was identified as a problem in 1997, this is not likely to have had a direct effect on fertility.

Illthrift was reported to have been a problem in growing animals in 1995. However, little information was available regarding the age or type

of animals affected. The DAF investigation into reports of stunting and ill-thrift in cattle, found no evidence of either on this farm at the time of the inspection in August 1996 (DAFRD unpublished).

Reference is also made in the Retrospective Survey Report to 'viral pneumonia' having been a problem. This could certainly have reduced subsequent thrive of affected animals. A significant calf diarrhoea problem was reported to have occurred on this farm in the spring of 1997. Similar outbreaks in the past could have affected subsequent animal performance. The PVP log lists five calls for calf diarrhoea on this farm in 1995. Reference is also made in the Retrospective Survey Report to the likely effects of animals grazing on 'light land' during drought in the summer of 1995 (see below). Poor grass growth at this time could also have had a negative effect on growth rates.

Five of 60 to 65 pregnant cows and heifers were reported to have aborted between October 1994 and January 1995. Two were submitted for laboratory examination but no diagnosis was made. At 8 per cent, this does not represent an unusually high incidence of abortion and would be typical of an outbreak of infectious origin. The fact that no diagnosis was made would not be unusual. The diagnostic rate for abortion worldwide is under 30 per cent (Barr and Anderson, 1993b).

Overall, the problems reported on this farm could not be considered unusual either in type or severity and there is no evidence to suggest that causes other than those intrinsic to a normal farm environment need be cited to account for their occurrence.

UPDATE 1997

Many of the cows were observed to be in only moderate to poor condition at the re-visit in May 1997. This was probably due to excessive negative energy balance post-calving. Recommendations were made regarding concentrate supplementation. Recommendations for mineral supplementation were also made to control a significant problem of subclinical hypomagnesaemia which was detected by blood analysis.

Although fertility continued to be a problem, breeding records were not available for analysis. A severe outbreak of diarrhoea in newborn calves was reported to have occurred in the spring of 1997 with up to 20 calves affected. However, no clinical pathology or carcass material was submitted for laboratory analysis.

FARM RS29

Main Enterprises: Dairying, store cattle, single suckling
Herd size: Small

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-47. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-48. The main problem reported was illthrift and deaths in cows.

According to the herdowner, six cows out of a total of 36 died in the winter-spring period of 1990. Two or three cows were reported to have died each year since then. However, it is not clear from the Retrospective Survey Report whether the reported mortality represented the full extent of the problem or if other animals were affected by illthrift but recovered. Presenting signs reported by the PVP were diarrhoea, haematuria and pining; diagnoses considered included salmonellosis and bladder neoplasia.

However, no animals were submitted for laboratory post-mortem examination prior to mid-1995. Of the two submitted in that year, a specific diagnosis of ragwort toxicity was made in one. The other was emaciated and had lesions secondary to prolonged recumbence. A third cow submitted in 1996 was emaciated and had lesions of mastitis with abscessation.

An obviously serious outbreak of disease occurred on the farm in 1990. However, no information is available regarding its appearance or duration. Little further can be gleaned form the practitioner record of calls. According to the PVP log, there were 11 calls to the farm in that year. Three were related to calves or fertility treatments. Of the remaining six, three were to cases of redwater, one for milk fever, one for 'oedema', and one for a calving.

The reported annual cow mortality rate of around 6 to 10 per cent (two or three cows per year) in the following years is relatively high but few conclusions can be drawn on the basis of the information supplied. The only clinical information supplied by the herdowner is that "Affected cows had a normal appetite but were in poor body condition all year round.". The PVP reported having made about 15 calls to the farm over the five-year period 1991-95. Cases encountered included diarrhoea (suspected salmonellosis), haematuria (suspected bladder neoplasia) and pining with or without pyrexia.

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It is likely that a number of the cases were due to salmonellosis as its presence was confirmed by laboratory isolation on a number of occasions. Given that ragwort toxicity was diagnosed in one cow in 1995, and ragwort infestation of some grazing areas was observed during DAF veterinary farm visits in 1995 and 1997, it is quite probable that other deaths could also have been due to ragwort toxicity.

Even if only four or five of the deaths between 1991 and 1995 were due to these causes, it would bring the incidence of unexplained cow deaths down to between one and two per year. Surveys of animal deaths in Northern Ireland and Denmark (Menzies *et al.*, 1994; Agger and Willeberg, 1991) have reported annual cow losses of between 2.5 and 4.2 *per cent*. Menzies (1994) reported that mortality rates were highest on small farms (less than 70 dairy cows).

In relation to reported ill-thrift, mineral deficiency may have contributed to reduced animal performance - though to what extent is not known. Copper and selenium concentrations in blood samples collected from suckler cows and weanling in summer, 1997 were near or below the lower end of the normal ranges. All herbage samples collected in 1997 had selenium concentrations near or below the minimum requirement for animal nutrition of 0.05 mg/kg. Copper concentrations ranged from 6.7 to 10.2 mg/kg which, though common on Irish farms, could be associated with deficiency.

Other problems reported on this farm at a low incidence were abortion in cows (probably due to *Leptospira hardjo* infection based on serology results), a skin growth on a cow and dermatitis in two dogs. These cannot be considered unusual either in appearance or incidence.

In conclusion, while cow mortality was exceptionally high in 1990, insufficient information is available to suggest possible or probable causes. Although cow deaths continued to occur at a lower but still unacceptable rate between 1991 and 1995, and the causes can only be a matter of speculation at this stage, the actual losses only equated to one or two cows more per year than average rates reported elsewhere. If they represented the full extent of the reported cow problem, then they could not be taken to indicate an exceptional level of disease on the farm. Although clearly unacceptable from an animal health point of view, it is probable that many of the deaths could have been explained had full post mortem examinations been carried out. Other

reported cases of disease were well within normal ranges.

UPDATE 1997

The dairy herd had been disposed of in 1996 due to the reported severity of the animal health problems. At the 1997 summer re-visit some of the yearlings were in only moderate condition and had not fully shed their winter coats. Some blood samples had low copper and selenium concentrations (*see above*). No other significant disease problems were reported.

FARM RS30

| Main Enterprises: | Pedigree beef |
| Herd size: | Medium large |

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-49. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-50. The main problem reported on this farm was infertility. According to the Retrospective Survey Report, the incidence of repeat breeders was about 10-12 *per cent* in 1994-95 spring breeding season. Affected cows did not go in calf. Repeat breeding was again reported to be a problem at the re-visit to the farm in 1997 - though no details were available.

While a detailed analysis of the extent or nature of the problem on this herd cannot be made in the absence of breeding records, there is no evidence that a severe infertility problem existed. For an extensively managed suckler herd, and using a bull for all services, a 10-12 *per cent* infertile rate is not unacceptable. As outlined above, the problem of repeat breeding can be due to a variety of cow, bull and management factors. Four of the cows affected in 1994/95 had histories of conditions which could have affected their breeding performance, i.e. dystocia (two cows), uterine discharge, and evidence of hormonal dysfunction (signs of oestrus during pregnancy).

Other problems reported in the Retrospective Survey Report - sudden death in a calf and diarrhoea and respiratory disease in weanlings - were unusual neither in nature nor occurrence.

UPDATE 1997

The main disease problems reported at the revisit in 1997 were lameness in cows – possibly 'foul in the foot' and infertility (repeat breeders). An outbreak of diarrhoea in weanlings in the autumn of 1996

had been attributed by the veterinary practitioner to BVD virus infection.

FARM RS31

Main Enterprises: Dairying, store cattle
Herd size: Small

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-51. The opinion of the herdowner's PVPs regarding the main problems encountered are given in Table 5-52. The main problems reported were respiratory disease in weanlings and cows, redwater in cows and heifers, downer cattle, infertility, abortion and perinatal calf mortality.

An outbreak of respiratory disease in cows and weanlings was reported to have commenced in April 1994 and ended in September. Over half of the 33 cows and all of 31 weanlings were treated by the PVP at intervals throughout the period. The main presenting signs were a drop in milk yield (cows), dullness, cough, nasal discharge and marked pyrexia. The PVP's diagnosis was that the condition was infectious in origin and most animals responded well to treatment. While the herdowner considered that the severity and extent of the condition was due to immune suppression in the affected animals, there is no specific evidence to support this suggestion. Although the morbidity was high in cows and weanlings, this is not unusual for outbreaks of disease due to highly infectious agents. As no material was submitted for laboratory examination, the identity of the infecting agent cannot be determined.

An outbreak of redwater (babesiasis) was reported to have affected eight of 44 adult animals on the farm between June and September, 1995. Two cows died. While the incidence in this outbreak was high, it was not unusual and there is no reason to suspect that factors other than those normally associated with redwater outbreaks were involved. The incidence of redwater in any area is a function of tick population and activity (ticks are vectors of the agent of redwater). Some of the land on this farm is of a type which would provide an ideal tick habitat. In addition, the warm and dry weather in the summer of 1995 would have led to increased tick activity.

A problem of downer cattle was also reported to have occurred in 1995. A cow and a heifer were affected. However, the diagnoses reported by the PVP - milk fever in the cow and a broken leg in the heifer - are consistent with the histories and the

circumstances and are unusual neither in incidence nor appearance.

Infertility was reported to have been a problem in 1994. However, the only information available was that four of 40 cows did not go in calf that year. This is not a high infertile rate and is well within the normal range (Appendix 1). It is also possible that part of the problem may have been due to early undetected abortions as three other abortions were recorded in late 1994.

Abortion was reported to have been an annual problem since 1994. However, while three cases were reported in 1994, the reported incidence for 1994 and 1995. at under six *per cent* per year, is close to internationally reported normal incidence rates (Appendix 1). Despite the fact that there were no specific laboratory findings on samples from two aborted foetuses in 1995, the most likely causes are infectious. Infectious agents are said to be responsible for over 80 *per cent* of abortions worldwide (Barr and Anderson, 1993b). One of the abortions in 1994 was a set of twins. Twinning is also an accepted risk factor for abortion (Beerepoot *et al.*, 1992). No abortions were recorded for 1996.

Two calves died close to calving in 1995. However, this is not a high incidence given herd size and, based on the history, they were probably secondary to calving difficulties.

Overall, there is no evidence that animal disease occurrence on this farm was in any way exceptional. While the reported incidences of respiratory disease and redwater are high, they are consistent with what could be expected in outbreaks of infectious disease. The other conditions reported (milk fever, abortions/ infertility, perinatal mortality) were of a type and incidence common to many dairy farms elsewhere. Although there is no evidence that it could have been a significant factor in any of the disease problems discussed here, it is worth noting that analyses of soils collected on this farm in 1997 showed lead concentrations ranging from 176 to 1653 mg/kg. Values at the higher range could be toxic to livestock by direct ingestion.

UPDATE 1997

An outbreak of mastitis was reported to have occurred during the calving season. Possible contributory factors included housing and early dry-off of cows in the previous lactation. No samples were submitted for laboratory examination. Two stillbirths occurred in 1997 which were associated with difficult calvings.

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FARM RS32

Main Enterprises: Horse breeding, sheep rearing, suckling (cows), store and beef cattle.

Herd size: Large .

ASSESSMENT

A summary of the animal health and production problems on this farm is given in Table 5-53. The opinions of the herdowner's PVPs regarding the main problems encountered are given in Table 5-54.

The farm was originally selected to participate in the Longitudinal Study but withdrew voluntarily before its commencement. The visit to the farm on which the Retrospective Survey Report was based took place in December, 1995. The main problems reported were disease and deaths of calves, growing cattle and cows; skin lesions in cows, growing cattle, and sheep; illthrift and deaths of lambs and infertility in sheep; disease and deaths of horses. Other sporadic disease problems, affecting individual animals, were also reported and are not discussed here.

According to the herdowners, calf disease and mortality had been a problem for about 10 – 12 years. Written records had been kept since 1994 but were not presented for examination upon request[1]. Eighteen of 20 calves born to homebred cows were said to have died within minutes to weeks of birth in 1995. Many calves were said to have developed diarrhoea and pneumonia and responded poorly to treatment. None were submitted for laboratory post mortem examination and the private veterinary practitioner log in the Retrospective Survey Report, although incomplete for 1995, lists no record of calls to calves.

Given the limited information provided, as well as the absence of veterinary practitioner or laboratory diagnoses, it is not possible at this stage to determine the causes of these reported losses. However, the reference in the Retrospective Survey Report to purchased calves suckling the same cows having performed better, together with the herdowners' veterinary practitioner reference to cows in poor body condition at calving with problems of fetal oversize in heifers, is consistent with some of the losses having been calving-

related, i.e. hypoxia, weakness at birth due to dystocia, inadequate colostrum intake.

Illness and deaths of growing cattle and cows were also reported to have been problems during the same period. Affected cattle were said to have had a good appetite but lost body condition, had diarrhoea or eye discharges, become stunted and gone down. However, while around 30 cows were said to have died or been culled in the two-year period from November 1993, there appears to have been little veterinary involvement and none of the losses were submitted for laboratory examination. In the circumstances, it is not possible to make any suggestions regarding likely causes.

The herdowners considered that the problems in calves and cows were due to 'acid rain' reducing immunity in outwintered cattle.

Skin lesions in cows and growing cattle were reported to have been a problem since about 1993. Cows which pined were also described as losing their hair. As no samples were submitted for laboratory examination, and no veterinary diagnoses are available, it is not possible at this stage to suggest likely causes.

A high percentage of ewes and lambs were reported to have died in 1994 but no details were available. Infertility was also reported to have been a problem since 1993. However, there was no veterinary involvement in the problem and insufficient information is available to comment on possible causes.

Skin problems in sheep were reported to have been a problem since 1993. Wool and skin scraping samples collected by the private veterinary practitioner in 1993 were sent to Limerick RVL where *Dermatophilus congolensis* was identified. This is a common cause of skin problems in sheep and cattle and has been discussed elsewhere in this report (*see* Chapter Four). Portions of the same samples were also sent to a private analytical laboratory[2] which reported a wool pH of 1.45 (1:125 dilution), iron 0.3 g/kg, aluminium 0.15 g/kg, and attributed these results to an acid-rain problem. The basis on which this opinion was given is not known.

The most serious problems on this farm involved horses. According to the herdowners, they commenced in 1991 and were continuing at the time of the Retrospective Survey farm visit in December 1995. Signs reportedly associated with these problems included acute and chronic

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[1] Owing to the incomplete nature of the information relating to the animal health problems on this farm, as well as the unique occurrence (in the Askeaton area) of the undoubtedly severe equine problems, the data has not been included in some of the inter-farm assessments at the conclusion of this Chapter.

[2] Mercury Analytical Laboratory, Limerick.

diarrhoea, leg rashes, mouth ulcers, illthrift, lymphangitis, abortions, and foaling-associated conditions in adult horses; weakness, joint-ill, and pneumonia in foals (high incidence). The Retrospective Survey Report lists brief details on the deaths of horses over the period 1991 to 1995. These comprised 15 horses over two years old, 14 foals or yearlings, and 12 – 14 abortions. However, the report also notes that the information may be " ... *either incomplete or erroneous*" as the herdowners referred to other more complete records which were not presented despite repeated requests.

None of the losses were submitted for laboratory post mortem examination over the period 1991 to 1994 and few veterinary diagnoses were available. In the circumstances, and given the anecdotal and incomplete nature of the records, it is not possible at this stage to determine likely causes. The reports do, however, indicate serious health problems in horses over an extended period.

The farm was visited by a veterinary equine specialist from CVL Abbotstown and staff of Limerick RVL in May and June 1995. The report of the latter referred to a respiratory condition which had been recurring in adult horses in 1994 and up to the time of the visit. It was characterised by depression, nasal discharge, submaxillary lymph node enlargement, and abscessation. The report noted that these signs were consistent with a diagnosis of strangles. This is an infectious condition of horses caused by *Streptococcus equi*. In addition to the above signs, it can also cause lymphangitis and oedema of affected limbs – both of which were described at various times in horses on this farm. This diagnosis was later confirmed by the laboratory isolation of *Strep. equi* from a naso-pharyngeal swab collected from a horse at the June 1995 visit.

The Retrospective Survey Report also refers to a private veterinary practitioner recalling that cases of strangles occurred on the farm in growing and adult horses at grass in 1994 and 1995.

The report of the CVL equine specialist also noted that there was historical clinical evidence for the presence of larval cyathostomiasis in young horses. This is an intestinal parasitic condition of horses which is refractory to anthelminthic treatment and can cause chronic weight loss, diarrhoea of sudden onset, colic, and death in severe cases (Murphy and Love, 1997). The diagnosis was supported by biochemical analysis of blood samples from eight horses during the June 1995 farm visit. All had raised alkaline phosphatase concentrations and four had hypoalbuminaemia - findings which are

consistent with larval cyathostomiasis (Murphy and Love. 1997). In addition, haematology analysis indicated that four had eosinophilia, in one case marked, ·a finding which is consistent with parasitism. The offer of more intensive farm studies to investigate the parasite and other problems was not taken up by the herdowners.

On clinical grounds, the CVL specialist suggested that remnant lesions of dermatitis along the backs of young horses were consistent with 'rain scald', i.e. *Dermatophilus* infection. This suggestion would be supported by the record of cold wet weather over the winter of 1994/95 - and which has been discussed elsewhere in relation to the occurrence of the same condition in cattle in the area. Laboratory diagnoses of sarcoptic mite infestation (one sample) and ringworm (one sample) were also made on skin or hair samples from two horses during 1995.

Lesions of eosinophilic granulomatosis were reported by the Irish Equine Centre in a skin biopsy from a horse with dermatitis in 1995. Eosinophilic granulomatosis is a pathological description of a chronic inflammation and does not imply a specific aetiology. It would, however, be generally assumed to include an allergic component, e.g. reaction to parasitic larvae (Lindberg, 1985). A private veterinary practitioner considered that two other horses on the farm had signs of a clinically similar skin condition in 1995. Eosinophilia in a blood sample submitted to Limerick RVL from one of these is also consistent with parasitism or an other allergic-type reaction.

Despite the laboratory investigations on the farm in 1995, no entire carcasses of the horses which died (at least three mares and three younger animals) were submitted to Limerick RVL for post mortem examination. Pathology findings on viscera submitted from two foals were consistent with peritonitis and systemic bacterial infection. The findings of post mortem examinations on horses sent by the herdowners to the Irish Equine Centre in late 1995 to mid-1996 are the subject of a published report by Fogarty *et al.*, (1998) and are discussed further below.

At the time of the Retrospective Survey farm visit in December 1995, the following were reported by the herdowners as being then current problems:

- Illthrift in a Bay 2-year-old horse. This horse had a history of loss of body condition, skin 'rash' and diarrhoea. A few days after the visit, it was sent by the herdowners to the Irish Equine Centre for euthanasia and post mortem

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examination. The results of this are included in the report published by Fogarty *et al.*, (1998).

- Lymphangitis and swollen legs (oedema) in two horses.

- Overgrown hooves in a horse.

- Skin lesion in a chestnut horse.

- Stunting in yearling horses. According to the herdowners, foals throve during their first summer but did not grow to a normal size as yearlings. A DAF inspection of horses on the farm in May 1996, showed that over half were in poor condition.

Other problems reported in cattle at that time were: diarrhoea in suckling cows, lameness in a cow, eye loss in a yearling, and stunting in growing cattle. A DAF report on an inspection of cattle on the farm in May 1996 concluded that the majority were in 'fit condition' at that time – though some were small for their age.

This farm undoubtedly had severe animal health problems over an extended period. However, owing to the very limited use of veterinary or laboratory facilities, as well as the incomplete nature of records which were provided, it is neither possible nor appropriate to attempt to determine the likely causes of problems prior to 1995. While some contemporary diagnoses were made or suggested for cases occurring in 1995, it must be emphasised that these were based on two farm visits by laboratory staff and the submission of a limited amount of clinical or tissue samples on other occasions. No entire carcasses were submitted for laboratory pathology examination and requests by DAF veterinary staff to undertake more intensive farm investigations were not acceded to by the herdowners.

The 1995 diagnoses of strangles (confirmed) and larval cyathostomiasis (suspected), are highly significant in the context of horse health on this farm and together, could account for many of the problems described. Strangles is an infectious debilitating condition of horses which can persist on a farm for long periods with clinical outbreaks recurring after periods of stress, e.g. poor weather, mixing, transport (Blood and Radostits, 1990). The historical descriptions of respiratory disease with lymphangitis and swollen legs (oedema) in horses prior to 1995 are also consistent with a diagnosis of strangles.

Larval cyathostomiasis is a common cause of diarrhoea and colic in horses (Murphy and Love, 1997). Both diagnosis and treatment are difficult

because the condition is associated with the maturation of non-egg-laying larval stages in the wall of the intestine. While its presence on the farm could only have been confirmed by more extensive contemporary investigations - which were proposed but did not take place - the clinical history of chronic ill-thrift and diarrhoea in some horses, as well as the 1995 biochemistry findings, are consistent with its occurrence.

Evidence of parasitism was also reported by Fogarty *et al.*, (1998) in all six horses submitted by the herdowners to the Irish Equine Centre for post mortem examination from late 1995 to mid-1996. Although no quantitative data was reported by the authors, the descriptions of the lesions in some cases were suggestive of severe infestation. While the paper also claimed an association between lesions in these horses and uptake of environmental aluminium, a response to this has been published by Collery *et al.*, (1999). The latter pointed to the lack of evidence presented by Fogarty *et al.*, in support of their claim regarding aluminium and questioned the lack of emphasis in their paper on the possible role of parasitism in the development of the lesions in all six horses.

In conclusion, while this farm suffered a high incidence of disease in a number of animal species, most significantly in horses, there is good evidence that infectious conditions played a major role in their occurrence. The fact that few contemporary diagnoses were made up to 1995 is directly attributable to the very low degree of laboratory or other veterinary assistance in their investigation. Although DAF laboratory and field investigations were initiated in the first half of 1995, offers of more intensive investigations in 1995 and 1996 were rejected by the herdowners.

ANIMAL HEALTH UPDATE ON RETROSPECTIVE SURVEY FARMS – 1997

Seventeen farms were re-visited in 1997. No serious problems were reported on the majority of farms and herdowners were generally either satisfied with performance or considered that under-performance, where it occurred, was due to on-farm factors. Six farms reported that fertility performance continued to be below target. However, breeding records were generally inadequate for a detailed analysis. Two farms reported recent serious outbreaks of calf disease. However, no samples had been submitted for laboratory examination. Based on histories, housing was identified as a probable contributory factor in both cases. Dystocia and stillbirths – both involving relative foetal oversize - were reported to

have been a problem on two farms. BVD virus infection was confirmed as the cause of illthrift in two adult cattle on one farm.

One farm continued to suffer a wide range of serious animal health and production problems. This farm was the subject of an extended investigation involving VLS and Teagasc staff throughout 1997 and 1998. A variety of management, nutritional and infectious factors were identified as being responsible for the problems.

Haematology parameters in blood samples collected during the 1997 re-visits were generally within reference ranges. The main finding from biochemistry analysis of samples was evidence of marginal to inadequate copper and selenium supply on a small proportion of the farms. While this is unlikely to give rise to serious health problems in animals on adequate diets, it could have a limiting effect on performance at certain stages of the production cycle. While a high proportion of blood samples had iodine (PII) concentrations below the reference range, this was not an unexpected finding given that the majority were taken from unsupplemented grazing animals during summer. There was no evidence to suggest that the results were indicative of a problem with clinical iodine deficiency in the area. All seventeen farms had mean blood phosphorus concentrations within the normal range. This is a significant finding given the concerns regarding potential environmental pollution by aluminium. Had the latter occurred then it might have been expected to have led to an induced phosphorus deficiency.

BLOOD ANALYSIS RESULTS (1997 FARM RE-VISITS)

Blood samples were collected from a selection of animals (approximately 10 cows and growing animals on each farm) on each of the surveyed farms which were re-visited during the summer of 1997. Mean haematology and biochemistry results are given in Appendix 9. As samples were collected during the summer months, it should be noted that the results largely reflect values in unsupplemented grazing animals.

Haematology

Mean haematology results for blood samples collected during the re-visits to these farms in summer 1997 are given in Appendix 9. Mean red cell parameters for all farms were within normal ranges. Packed cell volume (PCV) values at the lower end of the normal range were observed in samples from some lactating cows on a few farms.

This is a production-related response which has been reported elsewhere (Kappel et al., 1984). Mean total white cell counts (WCC) were also generally within normal ranges. Only one farm (No. 22) had a mean WCC above the reference rage of 5-10.5 x 10^9/l. This was due to two cows with high counts which were probably associated with intercurrent sub-clinical infections. Occasional other individual-animal total or differential white cell counts outside the reference ranges were generally associated with intercurrent infections - either clinical or sub-clinical. On Farm 25, for example, neutrophilias in a number of cows were associated with chronic foot lesions and mastitis (see page 130 et seq.).

Biochemistry

Mean biochemistry results for blood samples collected during the re-visits to these farms in summer 1997 are given in Appendix 9. Mean values for the majority of parameters were within reference ranges. Only four parameters were outside reference ranges on more than two farms. These were the minerals copper and selenium, the metabolite urea and the tissue enzyme CPK. As the latter largely originates from muscle and rises rapidly in response to mild trauma, it is likely that raised values were largely associated with the stresses of gathering and handling.

Twenty five per cent of all blood samples had low copper concentrations. Three farms had mean values below the reference range. On a further four farms, a half or more of the animals sampled had low copper values. Twenty per cent of blood samples were low in selenium. Mean values were low on three farms - one of these also had a low mean copper value. On a fourth farm, low selenium concentrations were recorded in a half of the samples - although the mean value was within reference range.

Overall, therefore, it is likely that over half of the farms surveyed may have experienced clinical or subclinical effects of inadequate copper or selenium supply at times in unsupplemented animals. These findings agree with those of Rogers and Poole (1984) based on blood samples collected in the area in 1982-83, and reflect the marginal soil and herbage availability of copper and selenium on farms in the area (Moneypoint, 1984).

In addition to inadequate supply, secondary copper deficiency may also be induced by the presence of excess molybdenum and sulphur in the diet. Pasture molybdenum concentrations above 5.0 mg/kg, for example, are likely to lead to problems with copper availability. Values between 2.0 and

5.0 mg/kg, where the copper-molybdenum ratio is less than 2.0, may also be significant. Based on the Teagasc analysis results of herbage samples from the surveyed farms, it is possible that up to six of the surveyed farms could experience molybdenum-induced copper deficiency under summer grazing conditions. Up to ten farms could be affected under winter grazing conditions. However, as only certain pastures are involved on each of the farms, this assessment assumes that the pastures in question are grazed or used for fodder conservation.

The results of sulphur analysis of herbage samples collected on these in 1997 are given elsewhere (Soil, Herbage, Feed and Water Volume). Values were within the reported range for farms elsewhere in Ireland. Based on these results, there is no evidence that herbage sulphur concentrations - regardless of source - would have been likely to have had a significant effect on copper availability at the time of sampling.

Three farms had one or more samples with selenium concentrations above the reference range and on one farm all samples had high values. This finding reflects the presence of a previously identified region of high soil selenium in the area which is geochemical in origin (Fleming, 1962).

Plasma inorganic iodine (PII) was measured as an indicator of recent inorganic iodine intake. Depending on the published reference range used (Mee et al., 1995; McCoy et al., 1997), either all or 85 per cent of samples had low PII concentrations. However, no evidence of clinical iodine deficiency was detected during the survey. According to McCoy et al., (1997), PII concentrations are unsuitable as the sole indicator of iodine status. This view is supported by the finding of PII concentrations below 20 µg/l in grazing heifers at Abbotstown where no clinical signs of iodine deficiency were detected at any stage of the study.

Overall, there was no evidence that phosphorus deficiency was a problem in the surveyed area. Mean phosphorus concentrations were within the reference range on all 17 farms (Appendix 9). Only 13 per cent of the total samples collected had phosphorus concentrations below the reference range of 1.4 – 2.5 mmol/l. On two farms, about a half of the samples had concentrations below the reference range. In addition to the effects of milk production and age, it is possible that supply was inadequate on one of the farms at the time as the herbage phosphorus was below the recommended minimum concentration.

Six farms had mean blood urea concentrations above the reference range. This finding is related to production characteristics of the farms in question and reflects factors such as grass quality and quantity, as well as rates of fertilizer application. Although raised urea concentrations have been reported to depress fertility (Butler et al., 1997) it is unlikely that this finding is of particular significance in relation to the present investigations.

Raised mean activities of one or both of the liver enzymes GLDH and AST on three farms were probably also production-related. Samples from one farm had raised mean GGT activities. This is also a liver enzyme and is released in response to cell damage. Animals on this farm had experienced fodder shortage and lesions consistent with fluke infestation were detected on post-mortem examinations.

Individual values of other blood parameters outside reference ranges were consistent with the occurrence of commonly-observed infectious or metabolic conditions.

INFLUENCE OF MANAGEMENT AND OTHER FACTORS ON ANIMAL HEALTH IN THE ASKEATON AREA

MANAGEMENT AND NUTRITION

Management and nutrition undoubtedly played a role in relation to the occurrence of disease and production problems in the Askeaton area – as they do on all farming operations. However, while the nature of the selection process, i.e. 'problem' farms, inevitably ensured that the sample contained a proportion of farms whose management regimes were clearly inappropriate to maintain an adequate level of animal health and performance, there is no evidence to suggest that management practices were significantly different from what would be expected in a cross-section of comparable herds elsewhere.

Problems identified where management and nutrition are likely to have been important include infertility, dystocia, lameness, respiratory disease in housed animals and inadequate condition in cows pre- and post-calving. This assessment is not unique to farms in Askeaton and would apply to any farms which experienced an above-normal incidence of these problems.

One aspect of nutrition which needs to be highlighted, and which, again, is probably not unique to Askeaton, relates to the possible impact of silage quality on animal health and performance

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over the winter/spring period of 1993-94 – a time when many of the farms reported problems were at their worst. Analyses of the 1997 silage crops on 21 of the surveyed farms, which were produced during a period of good growing conditions, indicated that quality was less than adequate on many of the farms (Soil, Herbage, Feed and Water Volume). Samples from 12 farms had DMD values below the recommended minimum of 65.0 *per cent* and five were below 60.0 *per cent*. Given the difficult growing conditions of 1993 (Keating and O'Kiely, 1997), it is likely that crops harvested in that year were, on average, of poorer quality than in 1997. As concentrate supplementation to dry cows was not widely practiced in the area, it is likely that many cows calved down in relatively poor condition in the spring of 1994. Owing to the inclement weather of the winter/spring of 1993-94, this effect would have been particularly significant in relation to cows which were outwintered.

MILK PRODUCTION IN THE ASKEATON AREA

Estimated average annual yield per cow (total milk sales divided by reported number of dairy cows – *see* page 112 for explanation) for each the 18 dairy herds, together with percentage annual changes, are given in Table 5-63. Taking the arbitrary figure of 10 *per cent* as being evidence of a notable reduction in yield per cow from one year to the next, the results show no evidence of an overall downward trend affecting the majority of herds at any time over the period 1990 – 1995. Five herds showed a reduction of over 10 *per cent* in 1993, three in 1994, and seven in 1995. Throughout the period, only two herds (farms ID07 and ID08 in 1992-93 and 1994-95, respectively) were affected in two consecutive years.

Of the seven herds affected in 1995, two were the original Index Farms and the significance of reductions in milk production in relation to the severe animal health problems which they experienced has already been discussed elsewhere (Chapter Four). To the extent that there was any evidence of a common underlying factor in relation to the other five farms in that year, late calving as a result of fertility problems was likely to have significantly affected yields, i.e. due to loss of synchrony between peak yield and period of maximum grass growth. This effect may have been further intensified due to poor grass growth in the summer of 1995 due to relative drought. Teagasc workers also reported that poor grass growth in the August/September of that year had a significant impact on milk production in a controlled trial at Moorepark (Dillon, Buckley, and Cliffe, 1998).

Other identifiable factors which may have contributed to reduced yields on these five farms included lameness (farm ID08), post-calving cow condition (farms ID05 and ID06), fluke infestation (farm ID06), and milk-drop associated with an outbreak of virus infection (farm ID022).

In 1992, the year in which yields were most severely affected on Index Farm A, only one other herd was affected. If the reduction in yields on Index Farm A in that year had been due to animal health problems associated with environmental pollution, then it is surprising that more of the 'problem' dairy herds were not similarly affected.

INFLUENCE OF WEATHER ON DISEASE INCIDENCE

Weather can have a significant influence on the occurrence of disease in animals - either housed or in the open (Webster, 1981). The incidence of respiratory disease, for example, is strongly influenced by temperature, humidity and air-flow in both housed and grazing animals (Bruning-Fann and Kaneene, 1992). As would be expected, a significant positive association has also been demonstrated between cold, wet, windy weather and increased calf mortality (Martin *et al.,* 1975).

The spread of conditions such as conjunctivitis in grazing animals, may be facilitated both by dust blowing during dry weather and by increased fly activity. This may account for some of the reports of outbreaks of conjunctivitis in the summer of 1995 which was dry and warm. A similar explanation may apply to some of the reported outbreaks or redwater which are also insect-borne.

For cows, winter and spring are the most critical periods as the stresses of advanced pregnancy or recent calving may be compounded by poor weather conditions. This is undoubtedly one of the most important reasons for the finding that death rates in cows are highest in the spring (Menzies *et al.,* 1995). In this regard, it is relevant to note that the results of the Animal Health Survey (Chapter Six) indicated that a substantially higher proportion of cows were outwintered in the immediate Askeaton area than in any of the other areas.

Analysis of weather patterns was carried out for the years 1990 to 1996 in order to identify any possible climatic factors which could have contributed to an increased disease incidence in the Askeaton area. A summary of weather patterns for the years 1991 to 1996 in the Limerick-Clare area is given in Appendix 8. The most significant feature of the weather over the period, and the only one which shows an obvious association with some of the reported disease problems in the Askeaton area, is

the very poor conditions over the winter-springs of 1993-94 and 1994-95. Wind and rainfall were above normal in the five months from December to April in both periods. In addition, March-April 1995 was unseasonably cold with snow and sleet.

There is also corroboration from other sources of the effects of poor weather conditions in Ireland in 1993 and 1994 on animal health and farm production. O'Farrell *et al.,* (1997) suggested that calving rates in DairyMIS herds may have been adversely affected by climatic and nutritional factors in 1994. Dillon, Buckley and Cliffe (1998) reported reduced fertility performance in unsupplemented cows at grass in the springs of 1993 and 1994 owing to poor grass growth and difficult grazing conditions at the time. Teagasc workers also noted the poor ensiling weather conditions in 1993 which resulted in poor silage quality (Keating and O'Kiely, 1997).

Poor weather during these periods undoubtedly contributed to the severity of disease on some of the most-severely affected farms in the Askeaton study. Outwintering of heavily pregnant or recently-calved cows on Index Farm B, as well as on two other 'problem' farms, in these years must have constituted a significant degree of environmental stress. In addition, some of the cases of skin lesions reported on the two Index Farms and on another 'problem' farm in the springs of 1994 and 1995 were probably also associated with wet weather (i.e. dermatophilosis or 'rain scald').

VETERINARY PRACTITIONER CALLS

Thirteen out of fourteen PVP's who attended livestock on the 27 study farms participated in a questionnaire-interview and supplied 'call registers' for livestock from affected farms. Details of calls to each of the 27 farms are included in the individual Farm Reports. Total call numbers per farm per year, as well as for each of the four most frequently-recorded identified categories of call, for the period 1991 to 1995, are given in Table 5-55.

Neither total nor category-specific calls showed evidence of a significant overall trend throughout the period. Total calls per farm per year ranged from a peak of 2.22 in 1990 down to 1.83 in 1993. These results do not provide any evidence of a significant increase in the level of practitioner activity on the 27 'problem' farms, as a group, over the period of observation.

CONCLUSION

The purpose of the Retrospective Survey was to determine if the undoubtedly severe animal health and production problems experienced on the two Index Farms were a reflection of a significant problem affecting the Askeaton area as a whole and, if so, to determine if there was any evidence of a common underlying aetiology. However, as outlined in the introduction to this chapter, circumstances dictated that compromises had to be made in relation to the design of this survey. Specifically, shortcomings existed both in relation to case definition and sample selection. In practice, both of these were left undefined and any herdowners in the area who considered they had an excess of animal health or production problems were invited to participate. Ultimately, 25 herds, in addition to the two Index Farms, were included in the survey. However, owing to the lack of case definition and the fact that the farms were self-selected, this does not necessarily imply either that all of these farms had severe disease problems or that they were fully representative of 'problem' farms in the area.

In the context of the overall Askeaton investigation, therefore, the main questions to be answered from the foregoing analysis of animal disease and production problems on the 27 surveyed farms are:

A. How many of the farms in the group could be said to have suffered an unusually high incidence of animal disease or production problems?

B. Is there any evidence that surveyed farms suffered a significant incidence of unusual or unexplained diseases?

C. Is there any evidence for the presence of a common underlying factor contributing to disease incidence on the surveyed farms?

D. Do the results of the assessment of disease incidence on the surveyed farms indicate evidence of an unusually high incidence of animal disease or production problems in the Askeaton area as a whole?

A – EVIDENCE OF AN UNUSUALLY HIGH INCIDENCE DISEASE ON SURVEYED FARMS

Despite the fact that the surveyed farms were selected on the basis that the herdowners concerned considered that they had suffered an excess of animal disease problems, it is clear from the above analysis that, on many farms, the problems were of a relatively mild degree. Disease problems which

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were classified as severe or moderately severe, together with the farms on which they occurred, are listed in Table 5-56. Only five farms can be said to have had a severe or moderately severe problem with adult bovine deaths, four with respiratory-enteric disease and four with perinatal calf mortality.

Adult bovine mortality

The high incidence of reported or confirmed adult bovine deaths on some of the surveyed farms was undoubtedly one of the most significant features of this investigation. Five farms reported a significant problem of deaths of cows or adult cattle (Table 5-57). However, death can be the end result of any disease process. In the case of these five farms, the causes of deaths comprised a broad spectrum of commonly-seen conditions with little or no evidence of an underlying pattern. This is not to minimize the severity of the problems experienced by the herdowners concerned – only to illustrate the point that it is not possible to group or compare farms on the basis of mortality without reference to the underlying causes. However, when broken down under the respective causes, e.g. dystocia, hypomagnesaemia, salmonellosis, mastitis, trauma, and parasitism, the incidence rates in most cases are much lower. The only adult categories where the incidences could be said to have been high were deaths associated with suppurative or systemic inflammatory conditions and those where the cause of death was unknown. The latter category existed largely due to the surprisingly small percentage of losses which were submitted for laboratory examination on most of the farms involved and the former comprised the two Index Farms – where the severity of the problems was beyond question.

Infertility

Summary details of fertility performance on the problem farms have already been reported (EPA, 1997). As outlined in that report, infertility was reported to have been a problem in cattle on 18 farms. However, adequate data for analysis was only available from eight farms (35 herd years) – all of which were dairy herds. Even within this group the results must be interpreted with caution owing to the incompleteness of the raw data and the small size of some of the farms (four of the eight farms had data for less than 25 cows). Besides the fact that little reference data were available for small herds, it is well recognised that fertility performance tends to be poorer where cows are kept in small groups (Allrich, 1993).

In relation to these eight herds for which records were available, the results of the analyses indicated that fertility performance was within normal ranges for about two-thirds of the 35 herd years examined (EPA 1997). Bearing in mind the reservations outlined above regarding data quality and scope, these results do not suggest that the problems were of unusual severity.

The problems identified by analysis of fertility records mainly comprised submission rates and non-detected oestrus. However, both of these parameters measure the same component, i.e. efficiency of heat detection. Non-detected oestrus (missed heats), is the most common type of infertility reported on dairy farms (Esslemont and Kossaibati, 1996). As discussed elsewhere, missed heats may be due to reduced or non-expression of heat by the cow or to inadequate observation or recording of heats by the herdowner. It is widely accepted that the latter, i.e. fertility management, is the most important cause of cows not being observed in heat (Esslemont and Kossabiti, 1996). Except in cases of severe debilitation or malnutrition, sub-oestrus and anoestrus are generally problems of individual cows in the immediate post-calving period.

Based on the above analysis of data from these eight farms, there is no evidence to suggest that unusual or abnormal factors were responsible for the fertility problems reported at herd level or that the group as a whole suffered a severe incidence of fertility problems. In relation to the remaining farms where insufficient data was available for analysis, little comment can be made regarding the nature or incidence of the problems. However, from available information, they would appear to have been similar to those commonly observed on farms elsewhere.

Bovine respiratory-enteric disease

Of the six farms which reported severe respiratory disease (Table 5-57), in one case there were no laboratory submissions and insufficient information is available to comment on the nature or severity of the problem. In the other cases, both clinical and laboratory findings were consistent with infectious causes. Housing was considered to have been a contributory factor in relation to at least three of the farms concerned. High morbidity in outbreaks of infectious respiratory disease is not unusual – especially where inadequate housing is a contributory factor (Roy, 1990). Bruning-Fann and Kaneene (1992) reported mortality rates for respiratory disease as ranging from 0 – 60 per cent on an individual farm basis while on a calf basis (i.e. area-wide) it ranged from 2 - 20 per cent. In

the circumstances, while the incidence of respiratory disease was high on some of the farms in the present investigation, there is no evidence to suggest that exceptional factors were involved.

Perinatal calf mortality

Perinatal calf mortality was reported to have been a severe problem on four farms (Table 5-57) – one of which was Index Farm A. However, while there were few laboratory submissions from any of the farms, and most of the information was based on individual recall, there is insufficient historical evidence to indicate either that the incidence was exceptionally high on an area basis or that risk factors other than those usually cited (i.e. prolonged or difficult calvings, unattended calvings and premature intervention or assistance) were present in most cases.

In relation to incidence of disease, therefore, there is no evidence that the majority of the surveyed farms in the Askeaton area were subject either to a high incidence of one or more unusual animal diseases or that morbidity or mortality rates for the diseases which did occur were excessive for the group as a whole. While losses on individual farms were undoubtedly highly significant for the herdowners concerned - particularly in the case of small herds - they could not, in epidemiological terms, be classified as evidence that the area as a whole had an unusual pattern of disease occurrence.

B – EVIDENCE FOR INCREASED INCIDENCE OF UNUSUAL OR UNDIAGNOSED DISEASES

Based on the foregoing descriptions and analysis of problems on the individual survey farms it is clear that there is no evidence that there was a high incidence of *unusual* animal diseases in the Askeaton area over the period of the Retrospective Survey. The diseases reported largely comprised those commonly seen on farms elsewhere, i.e. infertility, peri-parturient problems, infectious disease, etc. (Table 5-51). Neither is there any evidence of an abnormally high incidence of *undiagnosed* diseases. In most cases, straightforward explanations exist for the disease outbreaks. These largely comprise environmental, infectious, nutritional and management factors which are commonly associated with outbreaks of animal disease elsewhere. While in many cases no specific diagnosis was made at the time of the outbreak, and causes can only be suggested at this remove, this is probably due more to the limited nature of the investigatory process at the time, or the absence of historical information, than to the unusual nature of the outbreaks.

It should also be noted that the diagnostic rates for certain conditions such as abortion, perinatal calf mortality and illthrift are universally low. The diagnostic rate for abortion, for example, is only around 30 *per cent* - and this refers to cases which have been submitted for a full laboratory investigation (Barr *et al.*, 1993a). One of the outstanding features of the information collected in the Retrospective Survey, on the other hand, has been the evidence of relatively limited use of laboratory facilities or expertise - even in cases of prolonged or serious disease outbreaks.

C - EVIDENCE OF COMMON AETIOLOGY

The possibility that there had been a common underlying factor contributing to disease incidence in the Askeaton area could, in one sense, be said to be of significance. only *if* an excess of animal disease had been identified. However, given the accepted limitations regarding the foregoing assessments of disease incidence, and allowing for the possibility that an underlying factor such as environmental pollution could contribute to a small though not insignificant increase in disease incidence, examination of evidence for the existence of a common aetiology is still relevant to this investigation.

In the absence of evidence regarding the identity of any specific underlying factor, this question must be addressed indirectly, i.e. *via* expectations of what might have occurred were the problems to have been linked on most or all of the 27 farms. Assuming the presence of a common risk factor - more specifically environmental pollution originating from one or more defined locations - then the following would be expected:

i. Evidence of a temporal relationship in disease occurrence

ii. Evidence of a spatial relationship between affected farms in terms of disease severity and incidence.

iii. Evidence of a common syndrome - or group of related syndromes - on the affected farms.

i Evidence of a temporal relationship

Given that cause must precede effect - and that continued exposure to a putative risk factor should be accompanied by continued effect - it would be expected that there should be a strong degree of synchrony in terms of onset and duration of the reported problems on the affected farms. To investigate this possibility, the numbers of farms

reporting new cases of each of the listed problems per year are given in Table 5-58.

Allowing for an inevitable degree of clustering inherent in the study design – i.e. its focus on a specific time-period and the effects of herdowner recall bias - there is little evidence from these tables of any marked degree of synchrony in terms of onset and duration of reported problems. While there was a peak of 20 in the number of farms reporting new problems in 1994, there is also a wide spread of reports over the 10-year period from 1988 to 1997. Eight or more farms reported commencement of new problems in each of the seven years from 1989 to 1995, inclusive. Although the peak year for the reported commencement of fertility problems was 1994 at five new cases, there were four new cases in 1988 and three each in 1990 and 1993. It should also be noted that the peak in herdowner-reported fertility problems was not reflected in a similar increase in fertility-related private veterinary practitioner visits (see above). The peak year for onset of problems with respiratory-enteric disease was 1995 while for illthrift it was 1991 with four new cases. An interesting feature of the data in Table 5-58 is the peak in the number of farms reporting the onset of mortality problems (all ages of cattle) in 1994. However, as discussed above, the underlying causes of mortality on some of the farms were clearly unrelated.

Durations of the three most commonly-reported problems are given in Table 5-59, Table 5-60, and Table 5-62. Again, while there was inevitable overlap in terms of occurrence due to the relatively short period of interest (< 10 years), there is little evidence of a tendency towards synchronisation. Durations of all three conditions ranged from one to over 10 years.

In relation to infertility in particular, it is surprising that there was not more evidence of concurrence. Given the widespread presence both of the condition, and of the commonly accepted management and nutritional risk factors, it is likely that this partly reflects the absence of detailed records and consequent variations in individual herdowners' perception of the problem.

ii Evidence of a spatial relationship

The geographical distribution of the 27 surveyed farms, and their relationship to the largest and most immediate source of industrial emissions, i.e. a bauxite processing plant, are shown in Figure 5-1. Farms which had moderate-to-severe or severe problems ('problem' farms) are indicated by solid-outline rectangles. The remainder are indicated by dotted-outline rectangles. The distribution of farms shows no obvious pattern in relation to their classification as severe vs non-severe viz-a-viz their distance or direction from Aughinish. Such a relationship might have been expected if a significant proportion of the disease problems were causally-related to emissions from this site, i.e. severity of problems should reduce as distance from the source of assumed pollutant increases. The more remote sites of industrial emissions, i.e. Moneypoint and Tarbert generating stations, are not considered in this exercise as it would be expected that any concentration gradient of presumed pollutant would be significantly reduced and of little significance given their distance from Askeaton.

iii Evidence a common or related syndromes

A list of the main problems reported on the 27 farms, together with the numbers of farms reporting each problem is given in Table 5-62. It is clear from this that there was no evidence that a common syndrome occurred on all 27 farms. The four most commonly-reported problems were infertility in cows (18 farms), bovine respiratory-enteric diseases (13 farms), perinatal calf mortality (7 farms) and mortality in cows (6 farms). However, due to the almost universal presence of the infectious, nutritional and management risk factors with which they are generally associated, these are also common problems elsewhere. In the circumstances, it would be necessary to demonstrate a marked increase in their occurrence in a specified area to suggest that unusual underlying factors were contributing to their occurrence. As discussed above, infertility was only a severe problem on four farms, respiratory-enteric disease on four, and perinatal calf mortality on three. Although cow mortality was a moderately severe to severe problem on six farms, as discussed above the causes were multiple.

Given the positive effects of recall bias, the converse of the reported incidence figures for some of the common diseases is also highly significant in the context of the search for evidence of a common underlying cause. Twenty three farms, for example, did not appear to have had severe fertility problems; 22 farms did not have severe problems with respiratory-enteric disease of calves. Although lameness was reported on five of the farms, the 22 farms on which it was not reported to have been a particular problem included the original Index Farm.

Analysis of PVP calls to the 27 farms gives a similar picture of diversity in terms of conditions reported (see above).

D - DISEASE OCCURRENCE IN THE ASKEATON AREA AS A WHOLE

As discussed in the introduction to this Chapter, it is not possible to make a definitive assessment of the normality or otherwise of disease incidence in the group of 27 surveyed farms as a whole owing to the absence of comparative national baseline data. However, in so far as the group of farms may be said to represent an estimate of the total number of farms in the surveyed area with an excess of disease problems (*see* page 110 and Figure 1-1), it is clear that it is an overestimate. Based on the foregoing analysis of the individual Farm Reports, only just over half (16/27) of the surveyed farms were considered to have suffered moderately severe or severe problems (Table 5-56).

Assuming that this reduced total is a reasonable estimate of the number of farms in the Askeaton area with a history of an excess of animal disease problems over the period of interest, and given a total farm population of around 1,000 herds in the study area (*source* Central Statistics Office, 1991 census), then this amounts to an estimated proportion of about 1.5 *per cent* 'problem farms' in the study area.

The question then arises, '*could a similar proportion of problem farms be identified in other areas of the country?*'. While the information is not available to give a definitive answer to that question, neither the total number of problem farms (16 out of around 1,000), nor their relatively uniform distribution within the area, would provide strong evidence in support of the notion that the Askeaton area and surrounds was subject to specific environmental influences which were detrimental to animal health.

General Conclusion

In conclusion, and in answer to the questions posed on page 142:

- There is no evidence that there was an unusually high incidence of animal health and production problems in the Askeaton area as a whole over the period of interest, i.e. approximately 1985 to 1995. While individual farms had significant animal disease problems – in particular the two Index Farms - the total amounted to substantially less than the originally-identified 27 farms. Only 16 of the 27 self-identified 'problem' farms could, in fact, be said to have had severe or moderately severe animal health or production problems. Of these, only nine had more than one problem and only five had a multiplicity of problems (i.e. three or more). In so far as these 15 farms can be said to have been representative of the incidence of farms with an excess of animal disease in the area - and given a population of over 1,000 farms in the study area - then it does not appear to be exceptionally high.

- There is no evidence that there was a high incidence of unusual or undiagnosed diseases in the Askeaton area. The problems reported were of a type commonly seen elsewhere. To the extent that no diagnoses were made or could be suggested in many cases, this was probably due more to the very limited contemporary involvement of laboratory or other expert resources in their investigation than to any inherently. unusual features of the reported incidents.

- Other than infectious, management, and environmental factors which are common to most farms, there is no evidence of a common underlying factor having contributed to an increased incidence of animal disease in the Askeaton area. This assessment is based on:

  1. The absence of evidence of a significant degree of temporal or spatial clustering – other than that inherent in the study design - in relation to onset, duration and incidence of the most frequently-reported problems.

  2. The absence of evidence of a common syndrome on most or all of the 27 farms. Although infertility and respiratory-enteric disease were each reported on over half of the farms, these are common problems world-wide.

The overall conclusion of this section of the investigation is, therefore, that while there was undoubtedly an unacceptably high incidence of animal health and production problems on many of the surveyed farms there is no specific evidence to indicate either that this was part of an area-wide phenomenon or that herds in the Askeaton area had been subject to unusual environmental influences which had a negative effect on animal health.

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Tables and Figures

Figure 5-1: Location of major industrial sources of atmospheric emissions (blue arrows) relative to the 27 'problem' farms in the Askeaton area.
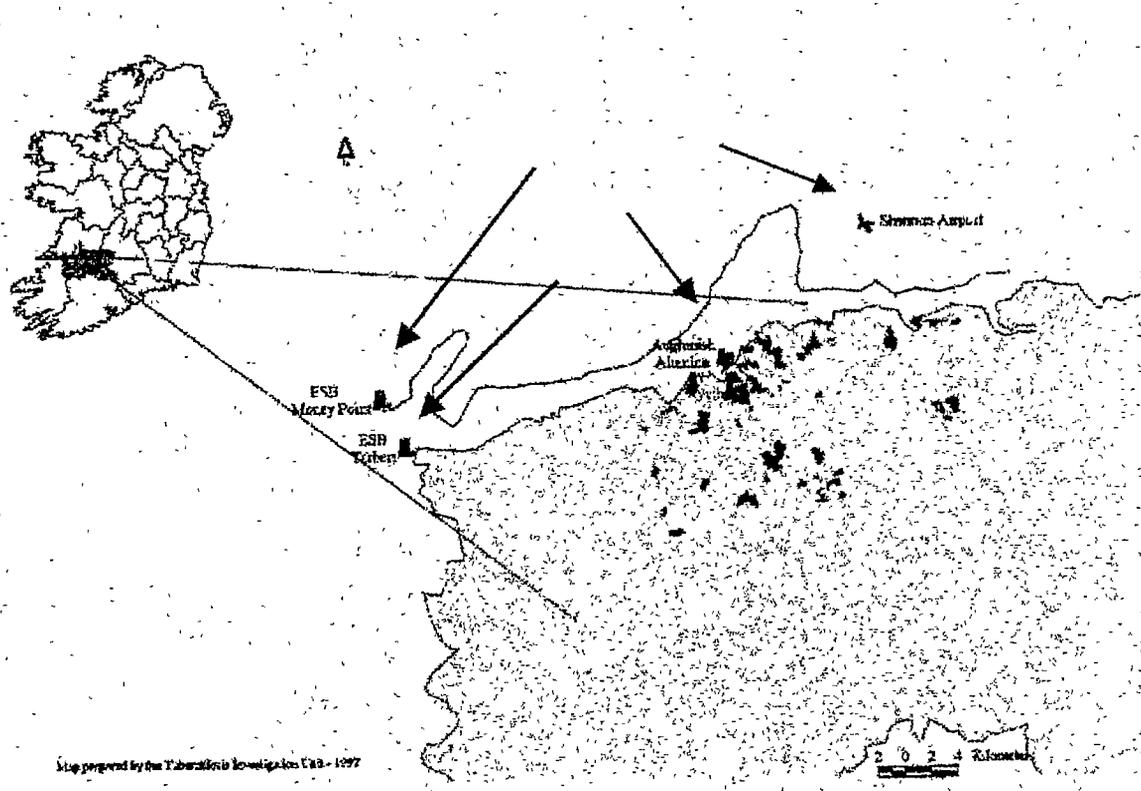
Table 5-1: Farm RS01 - Animal Health and Production Problems[1]

Description	Duration[2]	Severity[3]	Contributory factors[4]
Infertility	1990 – 1995	Moderate to severe	Heat detection, nutrition, bull (?)
Perinatal calf mortality	1989 – 1995	Severe	Dystocia, diarrhoea, housing
Pining and deaths in cows and growing stock	1988 (?) – 1993	Severe	Dystocia, hypomagnesaemia, mastitis, trauma, parasitism, unknown
Subcutaneous abscesses and dermatitis in cows	1990 – 1994	Severe	Infection, trauma, (wet) weather, debilitation, unknown
Mastitis	1992 – 1994.	Moderate to severe	Infection
Irritability in cows at milking	1992 – 1995 (?)	Mild	Stray voltage, cow temperament, management.
Reduced milk yield	1990 – 1995	Severe	Cow losses, age, debilitation, mastitis

[1] These footnotes apply to all following tables (NA = Not Available):

[2] Duration is inclusive of start and ending years.

[3] Severity takes into account factors such as proportion affected (incidence), duration of problem and description of cases. Even where losses occur, the condition may not be classified as 'severe' if the incidence was low.

[4] Contributory factors are those which, based on farm history and knowledge of disease/production problems, may have had a contributory effect. In some cases, specific diagnoses can be made.

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Table 5-2: Farm RS01 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors[1]
Mastitis, infertility, pining, perinatal mortality and skin lesions in cows	Yes	poor response to treatment in the herd due to unknown underlying factors

Table 5-3: Farm RS02 - Animal Health and Production Problems

Description	Duration	Severity	Contributory factors
Abortion	1994	Mild	Infection
Calf diarrhoea and pneumonia	NA	? severe in 1995	Insufficient information
Illthrift, illness and mortality in cows and growing cattle	1994 – 1995	Severe	No information for 1994. In 1995, infection, environmental stress (weather), malnutrition, dystocia.
Skin problems in cows and growing cattle	1994 – 1995	Severe	Infection, wet weather.
Lameness	NA	NA	Hoof overgrowth, trauma, wet conditions, other unknown.
Calving problems	1995	Severe	Dystocia secondary to debilitation.
Twining	? 1990s	NA	Insufficient information
Infertility	1990 - ?	NA	Insufficient information - no breeding records. Bull.
Mastitis	Annual	NA	Infection.
Reduced Milk Yield	1994 – 1995	Severe	Secondary to disease problems and cow losses.

Table 5-4: Farm RS02 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Mortality in cows, listeriosis in cows, calf diarrhoea, ketosis, salmonellosis, mastitis and lameness	Yes	NA

Table 5-5: Farm RS05 - Animal Health and Production Problems

Description	Duration	Severity	Contributory factors
Infertility in cows	1989 – 1996, 1998	Severe in some years	Fertility management, nutrition, bull fertility.
Ill thrift in calves and weanlings	1989 – 1998	Mild to moderate	Infection, mineral supply, grazing management.

Table 5-6: Farm RS05 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Infertility, illthrift and calf diarrhoea	Yes	Trace element deficiency, effects of drought, cow body condition

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[1] Response to question *'Please rank in order of importance the underlying factors which you consider contributed to the animal health problems on this farm'*.

Table 5-7: Farm RS06 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Perinatal calf mortality	1994 – 1997	Moderate	Dystocia, herd age structure, calving management, hypocalcaemia.
Locomotor disorder in calves	1995	Moderate to severe	Osteomyelitis, bone fracture, bacterial/viral infection, hypomagnesaemia.
Cow deaths	1994	Moderate to severe	Milk fever, dystocia, toxic mastitis, fatty liver.
Pining in cows	1994, 1995	Moderate to severe	Miscellaneous health problems in 1994/95, weather, metritis, fluke.
Mastitis	1994, 1995, 1997	Moderate	Infection, housing.
Poor milk production	1993/94 – 1997	Moderate	Miscellaneous health problems in 1994/95, infertility, mastitis, liver fluke, stray voltage in milking parlour.
Infertility in cows	1994 – 1997	Moderate	Fertility management, cow condition, bull fertility and management, metritis.
Skin lesions in cows	1995	Moderate to severe	Infection, rain.
Lameness in cows	1994, 1995	Moderate	Road surfaces, cow age.
Irritability at milking	1996	Mild intermittent	Stray voltage in milking parlour.

Table 5-8: Farm RS06 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Infertility, dystocia, illthrift, lameness in calves, mastitis and redwater	Yes	Cow condition, quality of some pastures.

Table 5-9: Farm RS07 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Perinatal calf mortality	1992 – 1995	Moderate to severe	Dystocia, herd age structure, calving management, mineral imbalance.
Infertility in cows	1994 – 1997	Moderate	Fertility management, herd age structure, bull fertility.
Poor milk production	1993/94 – 1997	Moderate	Herd age structure, mastitis.

Table 5-10: Farm RS07 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Mastitis, high SCC, downer cows, lameness.	Yes	Herd age profile (old), trace element imbalance.

Table 5-11: Farm RS08 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Cow lameness	1989 – 1997	Severe	Housing and roadway surfaces, foot-care management.
Infertility in cows	1989 – 1997	Moderate to severe	Lameness, fertility management, bull fertility
Downer cows	1989 – 1997	Moderate to severe	Lameness, milk fever, difficult calvings.
Calf diarrhoea and pneumonia	1989 – 1997	Moderate to severe	Insufficient history provided.
(Poor milk yield)*	? – 1996	?	Insufficient history provided.
(Mastitis)*	? – 1996	?	Insufficient history provided.

* Not specifically referred to in Retrospective Survey Report, but apparent from analysis of production data

Table 5-12: Farm RS08 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Lameness, dystocia, retained foetal membranes, leptospirosis and mastitis	Yes	Severity of cases on presentation.

Table 5-13: Farm RS09 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility	1983 – 1997	Moderate	Conception performance
Perinatal calf mortality	1984 – 1996, 1998	Moderate to severe	Dystocia, calving management, over-condition of cows.
Diarrhoea in calves	1991 – 1997	Moderate	Infectious agents, diet, housing.
Pneumonia in calves	1995	Moderate	Infectious agents.
Mastitis	1985 (?)	Mild to moderate	Infectious agents.

Table 5-14: Farm RS09 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Perinatal calf mortality, infertility, virus pneumonia and diarrhoea in calves.	Yes	Trace element imbalance.

Table 5-15: Farm RS12 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility	1989 – 1996	Moderate	Heat detection.
(Lameness)*	1991 – 1994 (?)	?	Insufficient history provided.
(Mastitis)*	1993 - 1994 (?)	?	Insufficient history provided.

*Not specifically described in Retrospective Survey Report, but apparent from analysis of production data and PVP comments.

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Table 5-16: Farm RS12 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Infertility, lameness, mastitis, dystocia and calf diarrhoea	Yes	Condition of roadways, cow condition, sire selection.

Table 5-17: Farm RS13 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Deaths in growing cattle, sheep and goats	Dec. 1991 – April 1992	Severe	Insufficient information. See text.

Table 5-18: Farm RS13 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Mortality in bullocks and heifers, mastitis, diarrhoea and salmonellosis in calves.	Yes	Unknown underlying factors.

Table 5-19: Farm RS14 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Respiratory disease and diarrhoea in cattle.	1993 – 1995	Mild to moderate	Infection.
Cow deaths (2)	1994 – 1995		Mastitis, virus infection.

Table 5-20: Farm RS14 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Diarrhoea in weanlings, pneumonia in weanlings, fluke in cows.	No	Poor response to antibiotic therapy, moderate cattle body condition.

Table 5-21: Farm RS15 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility	1990 – 1997	Mild	Fertility management, copper deficiency.
Conjunctivitis in cows	1995 (Aug)	Mild	Infection, mechanical irritation (dust, grass, etc.).

Table 5-22: Farm RS15 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Virus pneumonia in calves, infertility, retained foetal membranes, dystocia and redwater.	No	Iodine deficiency (infertility).

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Table 5-23: Farm RS16 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Delayed calvings	1992 – 1997	Moderate	Uterine inertia.
Deaths of cows	1992, 1995	NA	Calving-related.
Perinatal calf mortality	1992 – 1995	Moderate	Calving-related
Infertility	1993 – 1997	Moderate	
Calf pneumonia	1997	Severe	Infection, housing.

Table 5-24: Farm RS16 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Dystocia, retained foetal membranes, mastitis, downer cows, milk fever and grass tetany	No	None suggested.

Table 5-25: Farm RS17 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Dystocia	1990 – 1994*	Moderate to severe	Relative foetal oversize, calving management.
Perinatal calf mortality	1990 – 1994*	Moderate to severe	Relative foetal oversize, hypoxia, calving management.
Downer cows	1994*	Moderate to severe	Dystocia-related.
Diarrhoea and ill thrift in yearlings	1991 – 1995	Moderate	Infectious causes.
Pneumonia in calves	1991 – 1993	Moderate	Infectious causes.
Sudden deaths (calves)	1990 –1994	Mild	Multiple
Low milk yield	1991 - 1993	Unknown	Herd age structure, mastitis, milk drop syndrome.

*Herd depopulated in 1994 following outbreak of brucellosis.

Table 5-26: Farm RS17 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Mastitis in heifers, dystocia, redwater, lameness, diarrhoea (Salmonellosis and colibacillosis) in calves.	Yes	'acid rain' contributed to weak calves at birth..

Table 5-27: Farm RS18 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility	1989 – 1997	Moderate	Heat detection, possible mild copper deficiency.

Table 5-28: Farm RS18 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Routine clinical cases.	No	Not stated.

Table 5-29: Farm RS19 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility	1992 – 1997	Moderate	Heat detection, bull fertility, copper availability.

Table 5-30: Farm RS19 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Lameness, pneumonia, dystocia, infertility.	None	Not stated.

Table 5-31: Farm RS20 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Respiratory disease in weanlings	1995/96	Severe	Infection, mixing bought-in and home-reared calves.

Table 5-32: Farm RS20 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Retained foetal membranes, mastitis, pneumonia in calves.	Yes	None

Table 5-33: Farm RS21 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Retained foetal membranes (cows)	1995	Severe	Foetal size, selenium deficiency (?).
Grass tetany (cows)	1993	Moderate	
Infertility	1993 - ? 1997	Moderate to severe	Insufficient information. Bull fertility (?).
Conjunctivitis	1991 - ? 1995	Moderate to severe	Infection, abrasion, irritant.
Illthrift in adult cattle	1991 - ?	Insufficient information	Insufficient information
Deformed calves	1994, 1995	Mild	Genetic, unknown.
Illthrift in lambs	1989 - 1995	Insufficient information	Parasitism, mineral deficiency, bacterial infection and grass quality.

Table 5-34: Farm RS21 -PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Redwater, pneumonia and diphtheria in calves, retained foetal membranes (RFM).	Yes	Use of a continental cross sire on the heifers, frequent purchase of cattle.

Table 5-35: Farm RS22 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility	1991 – 1996	Mild	Breeding management, high RBI cows.
Respiratory disease and milk drop in cows	June 1995	Moderate	Virus infection

Table 5-36: Farm RS22 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Infertility, leptospirosis, bacilliary haemoglobinuria.	No	Trace element imbalance.

Table 5-37: Farm RS23 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Respiratory disease in calves and weanlings	1991 – 1996	Severe	Infection, housing, mixing.
Locomotor disorder in calves.	1993 – 1994	Severe	Unknown, mineral imbalance.

Table 5-38: Farm RS23 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Calf pneumonia and diarrhoea.	Yes	Housing, calf purchasing policy, infectious challenge.

Table 5-39: Farm RS24 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Respiratory disease and diarrhoea in cattle	1994 – 1995	Moderate	Infectious agents, weather.
Cow deaths	1994 – 1995	Moderate	Hypomagnesaemia, over-winter feeding and weather, other unknown factors.

Table 5-40: Farm RS24 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Pneumonia and diarrhoea in weanlings.	No	Not stated.

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Table 5-41: Farm RS25 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Perinatal calf mortality	1989 - 1990	Moderate	Dystocia.
Mastitis	1989 - 1997	Moderate to severe	Infection, milking machine and hygiene.
Diarrhoea, pneumonia, illthrift in weanlings	1991 - 1998	Moderate to severe	Infection, nutrition.
Lameness	1994 - 1998	Severe	Poor roadways, inadequate foot care program.
Infertility	1993 - 1998	Insufficient information	General cow health, nutrition, bull fertility.
High incidence of animal deaths	1996 - 1997	Severe	Multiple factors including infection, management and nutrition. ·

Table 5-42: Farm RS25 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Infertility, lameness, pneumonia in weanlings, salmonellosis in cows, dystocia.	Yes	Cow herd (old) age profile, recent intensification, inadequate calf housing.

Table 5-43: Farm RS26 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
BVD infection in calves	1994	Mild	Virus infection
Dystocia in cows	1995, 1997	Moderate	Bull size and overcondition of cows.

Table 5-44: Farm RS26 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Calf pneumonia, BVD, coccidiosis in sheep.	No	Not stated.

Table 5-45: Farm RS27 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility	1994, 1997	Insufficient information	Insufficient information
Illthrift in growing cattle	1995	Insufficient information	Secondary to infectious disease.
Abortion	Oct. 1994 – Jan 1995	Moderate	Unknown but probably infectious.
Hypomagnesaemia	May, 1997	Subclinical	Raised herbage potassium content.

Table 5-46: Farm RS27 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Infertility, leptospirosis, lameness, salmonellosis, mastitis.	No	Trace element imbalance.

Table 5-47: Farm RS29 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Mortality with illthrift in cows	1988 - 1996	Severe	Salmonellosis, ragwort toxicity and other unknown factors.
Salmonellosis in cows and weanlings	1988 - 1996	Moderate	Salmonella infection

Table 5-48: Farm RS29 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Diarrhoea and pining in cows, mastitis, diarrhoea and pneumonia in calves.	Yes	Virus infection.

Table 5-49: Farm RS30 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Infertility in cows	1994/95, 1997	Mild	Specific reproductive tract problems in 1994/95. Unidentified management and bull factors.

Table 5-50: Farm RS30 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Infertility, pneumonia, coccidiosis, milk fever, grass tetany and summer mastitis.	No	Not stated.

Table 5-51: Farm RS31 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Respiratory disease in weanlings and cows	April - September, 1994	Moderate	Infectious agent(s).
Redwater in cows and heifers	June - September, 1995	Moderate to severe	Infectious agent.
Downer cattle	1995	Low	Milk fever, broken leg.
Infertility	1994	Low	
Abortion	1994/95	Low	

Table 5-52: Farm RS31 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Pneumonia in all ages of cattle, calf diarrhoea, redwater, milk fever and mastitis.	Not stated	None.

Table 5-53: Farm RS32 - Summary of main animal health and production problems

Description	Duration	Severity	Contributory factors
Cattle:			
Perinatal calf mortality	1995	Severe	Calving-related
Calf diarrhoea and pneumonia	? 1995	Severe	Insufficient information
Morbidity and mortality in cows and growing cattle	1994-95	Severe	Insufficient information
Skin lesions in cows and growing cattle	1993 - ?	Unknown	Insufficient information
Ewe and lamb mortality	1994 - ?	Unknown	Insufficient information
Infertility in sheep	1993 - ?	Unknown	Insufficient information
Skin lesions in sheep	1993 - ?	Unknown	Insufficient information; one isolate of *Dermatophilus*
Miscellaneous diseases in horses	1991 - ?	Severe	Infectious agents – *Strep. equi*, parasites, *Dermatophilus*; others unknown

Table 5-54: Farm RS32 - PVP commentary regarding animal health and production problems

Main problems encountered	Severe (Yes/No)	Contributory factors
Cattle:		
Listeriosis	No	
Horses:		
Swollen limbs, respiratory disease in foals, illthrift and skin rashes.	Yes	

Table 5-55: PVP calls per farm to 'problem farms'.

	1990	1991	1992	1993	1994	1995	1996
Calving	2.0	2.7	2.5	2.2	2.3	2.1	2.1
Diarrhoea	2.2	2.5	1.8	1.8	1.8	2.3	2.1
Locomotor	2.5	2.0	2.1	1.8	2.6	1.5	2.9
Reproductive	2.4	2.6	2.1	2.6	2.6	2.5	2.1
Mastitis		2.4	2.3	2.6	2.9	2.5	2.4
All Calls	**2.2**	**2.0**	**1.8**	**1.8**	**2.1**	**1.9**	**2.0**

Table 5-56: Farms with problems classified as moderately severe or severe[1].

ID	Disease	Animal	Duration	Severity	Known or Suggested Contributory Factors
1	Infertility	Cow	1990 – 1995	2.5	Heat detection, nutrition, bull (?).
	PNM[2]	Calf	1989 – 1995	2.5	Dystocia, infection (diarrhoea), housing.
	Pining & death	Cow	1988 – 1993	3	Dystocia, hypomagnesaemia, mastitis, trauma, parasitism.
	Skin lesions	Cow	1990 – 1994	3	Infection, trauma, (wet) weather, debilitation.
	Mastitis	Cow	1992 – 1994	2.5	Infection
	Milk yield	Cow	1990 – 1995	3	Cow losses, age, debilitation, mastitis.
2	Resp-enteric	Calf	NA[3]	NA	Insufficient information
	Illthrift & death	Misc.	1994 – 1995	3	Infection, weather, malnutrition, dystocia.
	Skin lesions	Cow	1994 – 1995	3	Infection, wet weather.
	Lameness	Cow	NA	NA	Hoof overgrowth, trauma, wet conditions,
	Dystocia	Cow	1995	3	Secondary to debilitation and/or recumbency.
	Milk Yield	Cow	1994 – 1995	3	Secondary to disease problems and cow losses.
5	Infertility	Cow	1989 – 1996	3	Fertility management, nutrition, bull fertility.
6	Locomotor	Calves	1995	2.5	Osteomyelitis, bone fracture, bacterial/viral infection.
	Mortality	Cow	1994	2.5	Milk fever, dystocia, toxic mastitis, fatty liver.
	Pining	Cow	1994	2.5	Misc. health problems, weather, metritis, fluke.
	Skin lesions	Cow	1995	2.5	Infection, rain.
7	PNM	Calf	1992 – 1995	2.5	Dystocia, herd age, calving management, mineral nutrition.
8	Lameness	Cow	1989 – 1997	3	Housing and roadway surfaces, foot-care management.
	Infertility	Cow	1989 – 1997	2.5	Lameness, fertility management, bull fertility.
	Downer	Cow	1989 – 1997	2.5	Lameness, milk fever, difficult calvings.
	Resp-enteric	Calf	1989 – 1997	2.5	Insufficient history provided.
9	PNM	Calf	1984 – 1996	2.5	Dystocia, calving management, over-condition of cows.
13	Mortality	Misc	1991 – 1992	3	Insufficient information.
17	Dystocia, PNM, Downer cow	Cow	1990 – 1994	2.5	Relative foetal oversize, calving management.
20	Resp-enteric	Calf	1995 –1996	3	Infection, mixing bought-in and home-reared calves.
21	Infertility	Cow	1993 - ?1997	2.5	Insufficient information.
21	RFM[4]				
	Conjunctivitis	Cow	1991 - ?1995	2.5	Infection, abrasion, irritant.
23	Resp-enteric	Calf	1991 – 1996	3	Infection, housing, mixing.
	Locomotor	Calf	1993 –1994	3	Unknown, mineral imbalance.
25	Mastitis	Cow	1989 – 1997	2.5	Infection, milking machine and hygiene.
	Resp-enteric	Calf	1991 – 1998	2.5	Infection, nutrition.
	Lameness	Cow	1994 – 1998	3	Poor roadways, inadequate foot care program.
	Mortality	Misc	1996 – 1997	3	Infection, management, nutrition.
29	Mortality	Cow	1988 – 1996	3	Salmonellosis, ragwort toxicity.
31	Redwater	Cow	1995	2.5	Infection with *Babesia*.
32	Miscellaneous	Horse & cattle	1993 - ?	3	Insufficient information

[1] Severity classified as 1 = mild, 1.5 = mild to moderate, 2 = moderate, 2.5 = moderately severe, 3 = severe.
[2] Perinatal calf mortality. [3] Not available. [4]Retained foetal membrane

Table 5-57: Occurrence of disease problems classified as moderately severe or severe on survey farms.

Disease	Farm ID	Animal
Resp-enteric disease	2, 8, 16, 20, 23,25	Calves
Illthrift, pining	1, 2, 6	Cows/growing stock
Infertility	1, 5, 8, 21	Cow
Locomotor disorders	6, 23	Calves
Perinatal calf mortality	1, 7, 9, 17	Calf
Downer	1, 2, 8, 17	Cow
Mortality	2, 25	Growing stock (bovine)
Mortality	1, 2, 6, 13, 29	Cow, adult bovine
Mortality	13	Sheep, goats
Mortality	32	Horse
Lameness	2, 8, 25	Cow
Dystocia	1, 2, 17	Cow
Skin lesions	1, 2, 6	Cow
Skin lesions	32	Horse
Mastitis	1, 25	Cow
Redwater	31	Cows, heifers
RFM[1]	21	Cow
Reduced milk yield	1, 2	Cow
Conjunctivitis	21	Cow

[1] Retained foetal membrane

Table 5-58: Number of new cases per year of main animal health problems reported on the survey farms

Year of Onset	<88	88	89	90	91	92	93	94	95	96	97	98
Abortion	1							2				
Behavioural						1				1		
BVD								1				
Conjunctivitis					1				1			
Deformity								1				
Delayed calving						1						
Downer			1	1					1			
Dystocia			1		1				1			
Hypomagnesaemia							1				1	
Illthrift			2	1	1	1		1	2			
Infertility	1		4	3	1	1	3	5				
Lameness			1		1	1		2				
Locomotor							2		1			
Low milk yield	1				1		2			1		
Mastitis	1		1				1	1		1		
Mortality		1		1	1	1		4		1		
Perinatal calf mortality	1		2	1		2	1					
Redwater									1			
Resp-enteric disease	2		1	1	5		1	2	3		1	
Retained foetal membrane								1				
Salmonella		1										
Skin				1				1	1			
Twinning				1								
Total per Year	7	2	12	11	12	8	11	20	12	4	2	0

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Table 5-59: Infertility (cows): Onset and Duration on survey farms

Farm	<88	88	89	90	91	92	93	94	95	96	97	98
1				*	*	*	*	*	*			
2				*	*	*	*	*	*			
5			*	*	*	*	*	*	*	*		*
6								*	*	*	*	
7								*	*	*	*	
8			*	*	*	*	*	*	*	*	*	
9	*	*	*	*	*	*	*	*	*	*	*	
12			*	*	*	*	*	*	*	*		
15				*	*	*	*	*	*	*	*	
16							*	*	*	*	*	
18			*	*	*	*	*	*	*	*	*	
19						*	*	*	*	*	*	
21							*	*	*	*	*	
22					*	*	*	*	*	*		
25							*	*	*	*	*	*
27								*			*	
30								*	*		*	
31								*				

* Problem reported.

Table 5-60: Respiratory-enteric disease: Onset and Duration on survey farms.

Farm	Animal Type	<88	88	89	90	91	92	93	94	95	96	97	98
1	Calf	*	*	*	*	*	*	*	*	*			
2	Calf					*	*	*	*	*	*		
8	Calf				*	*	*	*	*	*	*	*	
9	Calf					*	*	*	*	*	*	*	
14	Growing cattle								*	*	*		
16	Calf											*	
17	Calf, Growing cattle					*	*	*					
20	Calf									*	*		
22	Cows									*			
23	Calf					*	*	*	*	*	*		
25	Calf					*	*	*	*	*	*	*	*
24	Adult								*	*			
31	Cows, weanlings								*				

* Problem reported.

Table 5-61: Perinatal Calf Mortality: Onset and duration survey farms

Farm	<88	88	89	90	91	92	93	94	95	96	97	98
2			*	*	*	*	*	*	*			
6								*	*	*	*	
7						*	*	*	*			
9	*	*	*	*	*	*	*	*	*	*		*
16							*	*	*	*		
17				*	*	*	*	*				
25			*	*								

* Problem reported.

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Table 5-62: Occurrence of disease problems on the surveyed farms

Disease	Animal	Farms
Infertility	Cow	1, 2, 5, 6, 7, 8, 9, 12, 15, 16, 18, 19, 21, 22, 25, 27, 30, 31
Resp-enteric disease	All	1, 2, 8, 9, 14, 16, 17, 20, 22, 23, 24, 25, 31
	Calf	1, 2, 8, 9, 16, 17, 20, 23, 25
	Growing cattle	14, 17, 31
	Cow	22, 31
	Adult cattle	24
Perinatal calf mortality	Calf	2, 6, 7, 9, 16, 17, 25
Mortality	All	1, 6, 13, 14, 16, 17, 24, 25, 29
	Cow	1, 6, 14, 16, 24, 29
	Bovine all ages	25
	Calf	17
	Misc species	13, 32
Illthrift	All	1, 2, 5, 6, 21, 27
	Adult cattle	21
	Cow	1, 2, 6
	Growing cattle	1, 5, 27
	Lamb	21
	Horse	32
Mastitis	Cow	6, 8, 9, 12, 25
Lameness	Cow	1, 6, 8, 12, 25
Low milk yield	Cow	1, 6, 7, 8, 17
Abortion	Cow	1, 27, 31
Downer	Cow	8, 17, 31
Dystocia	Cow	1, 17, 26
Locomotor	Calf	1, 6, 23
Skin lesions	Cow	1, 2, 6
Hypomagnesaemia	Cow	21, 27
Conjunctivitis	Cow	15, 21
Behavioural	Cow	2, 6
BVD virus	Calf	26
Deformity	Calf	21
Delayed calving	Cow	16
Redwater	Cow	31
Retained foetal membranes	Cow	21
Salmonella	Cow	29
Twinning	Cow	1

Table 5-63: Estimated annual milk yield per cow (YPC)[1] and annual percentage change for survey farms.

Farm		1990	1991	1992	1993	1994	1995
1	*YPC (gals)*	639	642	630	613	575	198
	% change		0.5	-1.9	-2.6	-6.3	-65.6
2		515	562	416	477	505	350
			9.0	-26.0	14.8	5.9	-30.8
5				576	574	657	481
					-0.5	14.6	-26.7
6			1185	1277	869	908	806
				7.8	-32.0	4.5	-11.3
7		474	486	405	270	574	556
			2.6	-16.7	-33.5	112.9	-3.1
8					687	400	327
						-41.7	-18.2
9			759	1119	1003	982	991
				47.5	-10.3	-2.2	1.0
12		629	597	566	596	695	637
			-5.0	-5.2	5.2	16.6	-8.3
13			432	486			
				12.5			
15			747	790	723	724	736
				5.7	-8.4	0.1	1.7
16			891	873	785	548	906
				-2.0	-10.2	-30.2	65.4
18		815	691	745	633	694	564
			-15.2	7.9	-15.1	9.7	-18.7
19			681	687	793	838	851
				0.9	15.4	5.7	1.5
22		703	660	752	931	882	725
			-6.2	14.0	23.8	-5.3	-17.8
25			558	576	529	496	501
				3.2	-8.1	-6.3	1.1
27		591	608	672	681	744	697
			2.8	10.6	1.4	9.2	-6.4
29			848	814	777	663	625
				-4.0	-4.5	-14.6	-5.7
31		685	628	736	820	898	821
			-8.3	17.2	11.4	9.5	-8.5

1 *See* page 112 for explanation

CHAPTER SIX

ANIMAL HEALTH SURVEY

Introduction

A survey of human health was commissioned by the Mid-Western Health Board as part of its investigations into claims of the presence of an environmental hazard in the Askeaton area (Human Health· Volume). The survey was administered to approximately 2500 individuals both within and outside the Askeaton area. In cases where the respondent lived on a farm containing cattle, a second questionnaire on animal health and production was also administered. The purpose of the latter questionnaire was to collect information on a range of animal health and production variables and to compare responses in the Askeaton area to those in other areas.

Materials and Methods

SURVEY DESIGN

Full details of the survey design are reported elsewhere (Human Health Volume). Briefly, a cross-sectional survey was administered to a total of approximately 2500 respondents in six areas in the Mid-Western Health Board area (Table 6-1 and Figure 6-1). Information for the animal health survey was collected on farm size, type, production and animal disease and deaths.

The survey involved two-stage cluster sampling with the designated districts comprising the primary units and the households comprising the secondary units. Four hundred and fifty individuals, each from a separate household, were interviewed per area. In cases where the respondent lived on a farm, and was in a position to supply the relevant information, a questionnaire on animal health and production was also administered. For the purposes of the survey, areas A and B comprised the 'exposed' region and areas C, D and E the 'non-exposed' region. As area F, Clarecastle/Ennis rural, was originally included in the human health survey owing to local concerns regarding environmental pollution in the immediate locality, it cannot be regarded as a 'control' area and has been excluded from the comparative section (i.e. exposed *vs* non-exposed regions) of the present analysis.

DATA HANDLING AND ANALYSIS

Questionnaires were visually checked for inconsistencies, recording errors and missing data. Obvious errors and omissions were corrected where corroborative data in related fields appeared reliable, e.g. the stated total number of cows could be cross-checked with the number of cows served and calved. Data were entered into a computer text file by a commercial data entry agency and subsequently imported into a Microsoft Access® database. Computerised validity checks were carried out on database entries.

Farms were classified as **dairy, suckler** or **mixed** based on cow numbers. Dairy farms were those with greater than 80 *per cent* dairy cows, suckler farms with greater than 80 *per cent* suckler cows, and the remainder were classified as mixed. Data on pining and ill-thrift in cattle, as well as for a range of production indices, were collected as categorical variables, e.g. for illthrift 0 = no problem, 1 = minor problem, 2 = major problem. In the following analysis, results on these variables are presented as frequency distributions of farms per area or region.

Descriptive results are presented by area (i.e. areas A to E) and comparative results by exposure status (i.e. exposed *vs* non-exposed). Details of stock numbers and type are largely confined to 1995 as this data was based on each respondent's answer regarding average numbers during that year. Data for 1996 were less representative as they related to the number of animals on the farm at the time the questionnaire was administered (mid-1996).

Disease incidence and mortality rates were calculated according to accepted formulae (Thrusfield, 1995). Mortality rates for each class of animal in 1995 were calculated as the reported number of deaths for the entire year as a proportion of the reported average number of animals present during the year. Mortality rates for 1996 were for the first six months of the year and were calculated as the reported number of deaths during that period as a proportion of the reported number of animals (in each class) present on the farm at the time the questionnaire was completed. No attempt was made to extrapolate the latter to a full year as it is likely that the majority of deaths would have

occurred in the first half of the year in line with the general calving pattern in the area (Menzies *et al.*, 1995). Comparisons of mortality rates, therefore, cannot be made between 1995 and 1996. It should also be noted that the method of calculation for 1995 which used the average number of animals for the year as denominator may overstate the actual mortality rate.

Mean farm-level incidence rates (morbidity or mortality) suffer a significant draw-back in comparative animal health studies in that they are generally skewed to the right, i.e. while a small number of farms may record a high incidence, the majority will have zero incidence. According to Waltner-Toews *et al.*, (1986a), the median is a better method to separate 'problem' from 'non-problem' farms. In the following analysis, median farm-level mortality rates were also used to compare the exposed and non-exposed regions. This was done by classifying farms on the basis of mortality status relative to the overall animal category median for each year. Using the categorical variables HIMORT95 and HIMORT96, herds were classified as having above normal mortality for a particular animal type (i.e. suckler cow, dairy cow, etc.) if their respective rates were above the overall median for the two groups (exposed and non-exposed) combined for each year.

Data on calving and fertility were collected for the 1995/96 breeding period by reference to the results of the 1996 calving season. The denominator for calving and lactational conditions (milk fever, retained placenta and mastitis) was the reported number of cows calved up to the time the questionnaire was completed. The denominator for peri-natal calf mortality in 1996 was the total number of calves born in 1996 up to the time the questionnaire was completed.

The incidence of other selected cattle diseases was estimated by requesting information on the number of animals treated (excluding prophylactic treatments – see Appendix 10, record 14) for each condition for the period 1 January to 31 May 1996. As treatments applied by herdowners and veterinarians have been combined in the present analysis to ensure sufficient numbers in each category, there may be some overstatement of incidence where animals received both veterinary and non-veterinary treatments. Farm incidence rates above one, i.e. due to repeat treatments of the same animal or animals, have been corrected to one for computational purposes.

For calving and lactational-related conditions in cows, the denominators for disease incidence

(treatment) in 1996 were the reported numbers of cows calved. For all other disease conditions and animal types, the denominators were the reported average numbers of these animals on the farm in 1995.

In the following analysis, farms with less than four animals in a category have been excluded from the comparisons of mortality rates in the exposed *vs* the non-exposed regions. The purpose of this was to reduce the disproportionate influence of small category size on rates (i.e. one death in a one or two-animal group represents a mortality rate of 50 or 100 *per cent*). In a herd with 20 dairy cows and two suckler cows, for example, the herd would be included in the dairy cow analysis but excluded from the suckler cow analysis. The main outcome of this was to reduce rates slightly in the non-exposed regions owing to the higher proportion of small herds. In area C, for example, when all herds were included, average cow mortality was 4.3 *per cent*. Exclusion of two single-cow herds, each with a 100 *per cent* mortality rate (i.e. one cow death per herd), reduced the average rate to 1.6 *per cent*.

Graphical and statistical analysis and reporting was performed using a Microsoft Excel® spreadsheet and the statistical analysis program Statistix®. The analysis unit of primary concern was the herd.

Results

FARM CHARACTERISTICS AND LOCATION

FARM SIZE AND STOCK NUMBERS

From a total of 2,479 completed human health questionnaires in the six areas, 680 answered 'yes' to the question *'Do you live on a farm that contains cattle?'*. Following validation of forms and data cleaning, a total of 590 were suitable for further analysis. Five hundred and fifty six (556) of these were in areas A to E and are included in the following comparative analyses.

Results on farm size and stock numbers are given in Table 6-1 to Table 6-5. Details of the total number of survey respondents per area, together with the numbers and percentages of valid responses from those living on cattle-farms, are given in Table 6-1. Areas C and D, i.e. Killadysart and Ennistimon, had the highest proportion of respondents on farms. Together, these two areas contributed about half of the total valid farm responses of the survey.

Area A accounted for over a third of all sheep in the six areas and almost a half of all horses.

Areas C and D were noticeably different from the other three areas in terms of farm size and type. Average farm size was significantly smaller (p < 0.05), and a higher proportion were described as suckler, in areas C and D than in areas A, B and E. Areas A and B, on the other hand, accounted for approximately 43 *per cent* of dairy cows but only 17 *per cent* of suckler cows. Average dairy cow numbers per farm in areas C and D (Table 6-5) were less than a third of numbers in areas A, B and E.

Similar differences, indicating more extensive farming in areas C and D, were also apparent in relation to milk production. Less than a quarter of herds in these areas reported average annual yields per cow of over 4,546 kg compared to almost a half of the herds in areas A, B, and E (Table 6-6). Concentrate feeding (Table 6-7) also tended to be lower in the former two areas. Stocking rate was also lower in areas C and D (Table 6-4).

Table 6-8 shows the proportion of cattle outwintered by area. The main points to note from this are the higher proportions of cattle outwintered on dairy and suckler farms in area A (Askeaton) than any of the other areas. Twenty seven *per cent* of respondents on dairy farms in area A reported that over half of the herd was outwintered compared to less than 10 *per cent* in areas B, C, D, and E. The difference was even more noticeable for suckler farms with 67 *per cent* of respondents in area A reporting that over half of the herd was outwintered.

From Table 6-9 it can be seen that the proportion of farms which had silage analysis carried out on a regular basis was low. Over a half of farms in the five surveyed areas had no silage analyses carried out in the previous three years – the figure for areas C and D was over 80 *per cent*. Even in the relatively more intensive areas A and B, only a third of farms reported that silage analysis was performed on an annual basis.

The distribution of farms by calving pattern is given in Table 6-10. Over three-quarters of farms in the five areas reported spring calving with most of the remainder classified as mixed. Only 12 farms reported autumn calving.

ANIMAL HEALTH DATA

MORTALITY

Mean farm-level mortality rates by area and animal category for 1995 are given in Table 6-11. No significant differences (p < 0.05; one-way ANOVA) were noted by area between dairy cow

and dry stock mortality rates. Suckler cow mortality was significantly higher in area A than in areas C and D and in area B than in areas C, D and F. Calf mortality in area C was significantly lower (p < 0.05) than in areas A, B, D, and E. The numbers of farms with sheep and horses were too small to permit statistical comparisons between areas.

Comparisons of results by exposure status (areas A and B = 'exposed', areas C, D, and E = 'non-exposed') are presented in Table 6-12 to Table 6-18.

Farm-level mortality rates by exposure status are given in Table 6-12. Overall mortality rates (i.e. total deaths in each category as a proportion of reported animal number in that category) are also given in Table 6-13 to allow comparison with other sources in the literature which use this method of analysis (e.g. Menzies *et al.*, 1995). Rates for cows (dairy and suckler) and calves are significantly higher in the exposed than the non-exposed group in 1995. The difference is most marked for suckler cow mortality where the mortality rate in the exposed group at 4.2 *per cent* is over three times that in the non-exposed group. Rates for most categories in 1996 were lower which reflects the fact that they were for six months only. Dairy cow, dry stock and calf mortality rates were significantly higher in the exposed than non-exposed region.

Median mortality rates for farms in the exposed and non-exposed regions for 1995 and 1996 are shown in Table 6-14. With the exception of calves, which had median mortality rates of 4.17 *per cent* in 1995 and 2.5 *per cent* in 1996, median mortality rates for all other categories of livestock were zero. This meant that herds with *any* deaths in a category were classified as having above-median mortality (i.e. HIMORTxx) for that category. The distributions of above-median (HIMORTxx) farms by exposure status are given in Table 6-15.

The odds of a herd having above-normal mortality by exposure status for 1995 and 1996 are given in Table 6-16. These were significantly greater for the exposed herds for dairy and suckler cows, calves and sheep in 1995 (e.g. farms in the exposed region were over three times more likely to have above-median suckler cow mortality than those in the non-exposed region). In 1996, they were significantly higher in the exposed herds for dairy cows, dry stock, calves and sheep. The effects of controlling for group size, i.e. number of animals per category per farm, are also illustrated in these tables. In 1995, only the odds ratios for cow mortality (dairy and suckler) remained significant following this adjustment. In 1996, on the other

hand, a highly significant odds ratio in relation to dairy cow mortality (p <0.0001), became non-significant by inclusion of group size in the regression equation.

The higher cow mortality rates on the exposed than the non-exposed farms in 1995 is further illustrated in Table 6-17 which shows the distribution of farms by mortality rates. The proportion of farms with zero mortality for both dairy and suckler cows is significantly lower (p < 0.05) in the exposed than the non-exposed group – 50 vs 75 and 68 vs 86 per cent, respectively. The proportion of farms with greater than 8 per cent suckler cow mortality is also significantly higher (p < 0.05) in the exposed group than in the non-exposed – 14 vs five per cent - though the number of farms in each category was small (nine and 16, respectively). The comparable figures for dairy cows are reversed, i.e. a higher proportion of farms in the non-exposed region had greater than 8 per cent suckler cow mortality - though the difference between the two regions is not significant (p > 0.05).

In order to try and identify characteristics of farms with higher cow mortality rates, the distributions of rates by cow numbers (dairy or suckler) per farm are given in Table 6-18. From these it is apparent that there is a 'bulge' in suckler cow rates for exposed farms in the 15 – 29 cow size range in both 1995 and 1996. In 1995, 10 of the 16 farms with above-median suckler cow mortality in the exposed region were in farms with 15 – 29 cows – and five of them reported three or more deaths. The reason for the higher mortality rate in this size-range of farms is not known.

ILL THRIFT

The distribution of farms by exposure status and presence or absence of a reported problem of ill-thrift is given in Table 6-19. Although the questionnaire allowed for a graded response in terms of the perceived severity of the problem, the 'moderate' and 'severe' categories have been combined owing to the very small numbers reporting severe problems (four farms in the case of cows, 14 for dry stock).

The proportion of farms reporting some degree of ill-thrift was significantly higher (odds ratio analysis) in the exposed than the non-exposed region for both cows and dry stock – 12 vs 5 and 24 vs 6 per cent, respectively. Although the total numbers reporting any degree of ill thrift were small, there was also a clear association between ill thrift and mortality in suckler cows in the exposed area but not in the non-exposed. Eight of 10 farms reporting ill thrift in suckler cows in the exposed

area also reported above-median suckler cow mortality (i.e. one or more cow deaths), compared to only 4 of 12 in the non-exposed. A similar association was not found between mortality and ill thrift in either dairy cows or dry stock.

DISEASE

The incidence of animal treatment for disease by exposure status is given in Table 6-21. However, the results should be viewed with caution as this part of the questionnaire had clearly given rise to confusion and was, in general, poorly completed. Of almost 60,000 potential data entries (see Appendix 10, record 14), only just over 10,000 were useable. Owing to the poor response rate, statistical analysis was not considered appropriate for this section of the survey.

The results indicate considerable variation between the exposed and non-exposed regions in terms of treatment for disease with little evidence of an overall pattern. The main points to note are the higher incidences of the calving and lactation-related conditions, i.e. milk fever, retained placenta and mastitis, as well as the generally higher rate of treatment for mineral deficiency, in the exposed than in the non-exposed group. Higher rates of treatment for liver fluke and skin disorders in suckler cows in the exposed group were, in both cases, largely due to single farms where all animals in a group were treated (i.e. incidence = 1.00).

Treatment rates for lameness were similar in the exposed and non-exposed regions for all classes of animal.

FERTILITY AND PERIPARTURIENT HEALTH

The reported incidences of fertility and periparturient animal health problems in 1996 are given in Table 6-20. The percentage of cows open was significantly higher (p<0.05) in the exposed than the non-exposed region. The percentage of assisted calvings, cows down or died within two days of calving, as well as the percentage of calves which were born dead or died within the first 24 hours of life, were also significantly higher in the exposed region. Differences between regions for other parameters, including abortion, twinning and congenital deformities were not significant.

Discussion and Analysis

The purpose of this survey was to compare the farm-level incidence of selected animal health problems on farms in the Askeaton area and surrounds ('exposed region') with farms elsewhere ('non-exposed' area). In the context of the wider

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Askeaton investigations, the objective was to determine if there was any evidence to support concerns that there had been an unusually high incidence of animal disease or deaths in the area.

It is necessary at this stage to highlight a number of characteristics of the survey design and implementation which are likely to have had a significant impact on the results generated. In the first case, this was an observational study and, like all studies of this kind, it may demonstrate an association but cannot provide direct evidence of the causes of any differences observed (Thrusfield, 1995). In the second case, the animal health section of the survey was an addition to a larger study of human health in the area. This may have had important implications both in relation to sample selection and to data collection. The fact that the study population was originally selected with human rather than animal health in mind may have contributed to the significant differences in enterprise type identified between the exposed region and two of the three areas in the non-exposed region, i.e. areas C and D. Post-survey reports from health-care workers who had been involved in administering the questionnaire also indicated that significantly more problems had been experienced in relation to collection of information on animal health and production than on human health. This latter observation was also supported by the relatively high number of returned animal health questionnaire forms (90 of 680) which were unsuitable for analysis given that the survey was based on personal interview.

Findings during the data validation phase of the exercise also indicated that significant problems had been encountered in relation to completion of certain sections of the questionnaire -- in particular the table of farm animal deaths (Record 11-10; Appendix 10) and the section on cattle diseases (Record 14-34; Appendix 10). However, despite these reservations, the results of the survey represent the largest and most comprehensive body of combined animal health and production data which have been collected on Irish farms in recent years.

PRODUCTION AND FARM MANAGEMENT

The results of the survey have highlighted a number of important differences between herds in the exposed and non-exposed regions in terms of size and enterprise type. Farms in areas C and D, which accounted for over three-quarters of the non-exposed survey population, were, on average, significantly smaller and a higher proportion were suckler than those in the exposed region. Other differences noted which support the picture of

farms in areas C and D being less intensive, include lower milk yield, lower concentrate usage, and lower frequency of silage analysis. These indicate significant differences in farm management between the exposed and non-exposed regions and, given the importance of farm management in relation to animal health and performance (Bruning-Fann and Kaneene, 1992), could have had an influence on some of the results of the survey.

Despite the apparently more intensive nature of farms in the exposed region (areas A and B), it is interesting to note that a substantially higher proportion of cattle were outwintered on farms in area A (Askeaton area) than on any of the other areas. This applied to both dairy and suckler farms -- 67 *per cent* of suckler farms in area A outwintered over half of the cattle compared to an average of 29 *per cent* in areas C, D and E combined. For dairy farms, the corresponding figures were 28 and 10 *per cent*, respectively. If these are an accurate representation of the overall position in the region, then it could be a significant finding in relation to animal health and performance in 1994 and 1995 given the poor weather conditions over the winters of 1993-94 and 1994-95.

FERTILITY

Indices for fertility and periparturient animal health performance for the two regions combined were generally comparable to results reported elsewhere (Appendix 1). Reported rates for abortion, congenital deformities, and calf deaths within the first month of calving were low compared to other reported values (Appendix 1). Although there were significant differences between the two regions in relation to the proportion of cows open (i.e. infertile rate), as well as to the incidence assisted calvings, cows down or dying, and perinatal calf mortality, values were, in any case, well within the range of rates reported from other studies (Appendix 1). It is also likely that a proportion of these differences could be accounted for by the observed differences in the degree of intensification between the two regions (i.e. dairy *vs* suckler -- *see* above).

ILL-THRIFT

Although the proportion of farms in the exposed region reporting some degree of ill thrift in cows or dry stock was significantly higher than in the non-exposed region, in the great majority of cases the problem was said to have been of only moderate severity. Only two farms in each area

(exposed and non-exposed) reported a severe problem with ill thrift in cows.

The reasons for the higher incidence of ill-thrift in the exposed region cannot be determined on the basis of the available data. As discussed elsewhere (see page 7), ill thrift is a non-specific expression of any factors (infectious, environmental or other) which result in an animal's energy intake being insufficient to meet requirements. While ill thrift has been reported as a finding in a wide variety of animal diseases (Blood and Radostits, 1990), there is little information in the literature on the occurrence of ill thrift per se.

ANIMAL MORTALITY

The farm-level mortality rates for dairy and suckler cows recorded in this survey (1.9 and 1.6 per cent, respectively in 1995 for all areas combined) are comparable to reports from elsewhere. Poole and Rogers (1984) reported mean annual rates of from 1.0 to 1.6 per cent mortality in the baseline survey of 25 farms (23 dairy, 2 suckler) carried out in the Aughinish/Askeaton area in 1979 – 1981. Menzies et al., (1995) reported an overall (i.e. as opposed to farm-level) dairy cow mortality value of 1.6 per cent for Northern Ireland and quoted a range of about one to 4.6 per cent from other surveys reported in the literature. They reported an overall mortality rate of 2.4 per cent for suckler cows in their own survey and quoted rates in the literature for 'range' (i.e. outdoor suckler) cows as from about 3.0 to 9.0 per cent.

In comparative terms, the most important finding was the higher suckler cow mortality rate in the exposed region than the non-exposed in 1995. At 4.2 per cent, it was over three times the rate in the non-exposed region.

The difference between the two regions related more to the number of farms which experienced any losses rather than to the numbers of farms with high mortality. While almost a third of farms in the exposed region experienced at least one suckler cow death compared to only 14 per cent of farms in the non-exposed region, overall mortality rates for farms which suffered any losses in the two regions (i.e. HIMORT95 farms) were similar – 7.4 per cent in the exposed compared to 5.9 per cent in the non-exposed region. The actual number of farms with a high rate of mortality (three or more suckler cows deaths) in the exposed region amounted to only five out of 142 valid responses.

The reason for the higher number of farms reporting at least one cow death in the exposed region in 1995 cannot be determined on the basis

of data collected in the survey. Although the larger average farm size in the exposed region might have been expected to have had an effect (i.e. the probability of at least one death occurring increases with increasing cow numbers), the odds ratios remained significant even after accounting for cow numbers per farm (Table 6-16). Differences in farm type between the two regions (i.e. suckler cow losses on 'suckler' farms as opposed to 'dairy' farms) do not appear to have been a significant factor either. The great majority of farms in both the exposed and non-exposed regions with above-median suckler cow mortality (HIMORT) were classified as suckler (i.e. over 80 per cent suckler cows) and therefore of similar type. It is also unlikely that recall bias was a significant factor given the importance which would be attached to the loss of a cow – in particular in the non-exposed region given the smaller average farm size and the greater significance attached to such loss.

The finding of an association between ill thrift and mortality in suckler cows (see page 166) is also interesting – though not surprising. Menzies et al (1995, 1996) reported that ill thrift was a common ante-mortem finding in a survey of bovine mortality in Northern Ireland.

The results of the present survey also indicated that a higher proportion of animals in the exposed region (Area A) were out-wintered. The potential influence of this on ill thrift and mortality must also be considered. Inadequate nutritional compensation for the effects of inclement weather would lead to an energy deficit which, if prolonged, would be expressed as ill thrift and an increased susceptibility to disease. However, the numbers of farms involved at these stages of the data analysis were small and so the findings must be interpreted with caution.

Although the odds of above-median mortality for some other animal classes in 1995 and 1996 were also higher in the exposed region, mortality rates were generally well within normal ranges reported elsewhere (Appendix 1) and therefore no particular importance can be attached to the findings. In several cases, differences between the exposed and non-exposed regions were no longer statistically significant once animal numbers (i.e. number of cows, heifers, etc. per farm) were taken into account, e.g. dairy cows in 1996 (Table 6-16). Calf mortality rates, at between six and 12.6 per cent in the exposed and non-exposed areas for 1995 and 1996 were comparable to those reported by Poole and Rogers (1984) for the Aughinish baseline survey of 25 farms. They were also comparable to rates reported from studies elsewhere (Appendix 1).

Morbidity

Collection of statistics on disease incidence invariably presents problems in relation to the definition of disease. A herdowner's definition of pneumonia, for example, may differ from a veterinarian's – and neither would be confirmed in every case by a pathological examination. In the present study, treatment rates were used as an indicator of morbidity. This is a reasonable approach as, while it does not necessarily ensure accuracy in terms of diagnosis, it at least measures events which the herdowner considered sufficiently important to warrant action. A similar approach was used by Waltner-Toews *et al.*, (1986) who regarded it as a useful proxy for disease incidence. However, Emanuelson and Oltencau (1998), suggested that the results should be interpreted with caution when used for inter-herd comparisons as management may be a confounding factor, i.e. better-managed farms may resort to treatment more often.

The results of this aspect of the present survey must also be viewed with caution owing to the high rate of invalid or non-responses (less than one-in-six of the responses for this section provided data suitable for analysis). Besides the fact that a poor response rate brings into question the representativeness of the collected data (Thrusfield, 1995), categories with a small number of responses tend to be unduly influenced by one or two respondents with extreme values. The higher treatment rate for skin conditions in dairy cows in the exposed compared to the non-exposed group (1.5 *vs* 0.2 *per cent*), for example, was entirely due to a 50 *per cent* treatment rate (six of 12 animals) on one farm.

Bearing in mind the foregoing reservations, and when taken as an indicator of disease incidence, treatment rates for the two groups combined were generally comparable to rates reported elsewhere. While the results show a considerable degree of variation between animal categories and exposure status, there is no evidence of an overall pattern of higher treatment for disease in the exposed than the non-exposed region. Although the incidences of milk fever, retained placenta and mastitis were higher in the exposed than non-exposed region, they were well within the range of rates reported from elsewhere (Appendix 1) – and undoubtedly, to some extent at least, reflected differences in enterprise type between the two regions (i.e. dairy *vs* suckler). The reported rates for treatment for lameness in cows in both regions were at the lower end of the scale when compared to other reported rates (Appendix 1).

Although treatment rates for mineral deficiency were higher for all classes of animal in the exposed than non-exposed region, the numbers of farms using treatment were small in both regions and it is unlikely that any special significance can be attached to the finding. In terms of estimating morbidity, treatment for mineral deficiency is the category least likely to reflect a specific diagnosis. In the Retrospective Survey, for example, infertility, rather than clinical deficiency, was the most frequently cited veterinary reason for mineral administration.

In conclusion, the most significant finding from this survey has been the relatively high rate of suckler cow mortality in the exposed region in 1995 when compared both to the non-exposed region and to rates reported elsewhere. The reasons for this difference cannot be determined from the results of the survey. Its significance must be viewed with caution in the light of the relatively small number of farms on which it was based as well as clear differences between the exposed and non-exposed regions in terms of the predominant farm types. Although attempts were made to control for differences in farm size and enterprise (dairy *vs* suckler), it is likely that a wide range of other important variables were unrecognised and therefore uncontrolled. While differences in morbidity and mortality between the two regions were noted for other animal types, rates were generally comparable to those reported on farms elsewhere.

Tables

Figure 6-1: Six survey areas in Askeaton Animal Health Investigation (Map MWHB).

Risk and Comparison Areas in the Askeaton Investigation

Area 1 - Askeaton
Area 2 - Rathkeale
Area 3 - Killadysert
Area 4 - Ennistimon
Area 5 - Moyne & Littleton District
Area 6 (Urban) - Clarecastle
Area 6 (Rural) - Clarecastle
Limerick City
Limerick
Tipperary North
Clare
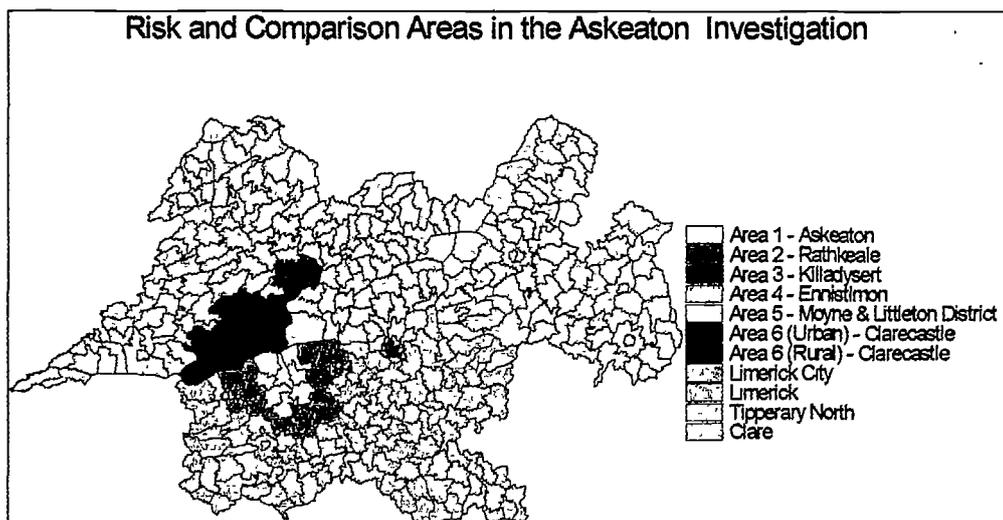
Table 6-1. Survey areas – response details.

Area Code	Area	Responses	Valid Responses with Stock
A	Askeaton	415	87
B	Rathkeale/Newcastlewest	446	55
C	Killadysart rural	393	169
D	Ennistimon rural	397	154
E	Littleton/Moyne rural	357	90
F	Clarecastle/Ennis rural	471	35
Total		*2479*	*590*

Table 6-2: Distribution of farms by size in the six surveyed areas.

Farm size (hectares)	Number (%) farms per area					
	A	B	C	D	E	F
< 20	18 (20.7%)	10 (18.2%)	70 (41.4%)	62 (40.3%)	13 (14.4%)	14 (40.0%)
20 – 40	32 (36 8%)	22 (40.0%)	76 (45.0%)	61 (39.6%)	34 (37.8%)	9 (25.7%)
40 – 60	20 (23 0%)	12 (21.8%)	15 (8.9%)	20 (13.0%)	20 (22.2.%)	5 (14 3%)
> 60	17 (19.5%)	11 (20.0%)	8 (4 7%)	11 (7.1%)	23 (25.6%)	7 (20 0%)

Table 6-3: Distribution of farms by enterprise type[1] in six surveyed areas (1995).

Category	Number (%) farms per area					
	A	B	C	D	E	F
Dairy	44 (51.1%)	37 (67.3%)	48 (28 4%)	32 (20.8%)	46 51.1%)	4 (11.4%)
Suckler	31 (35 2%)	11 (20 0%)	85 (50 3%)	93 (60 4%)	24 (26 7%)	16 45.7%)
Mixed	3 (3.4%)	3 (5.5%)	23 (13.6%)	24 (15 6%)	13 (14.4%)	4 (11 4%)
No cattle	9 (10.2%)	4 (7.3%)	13 (7.7%)	5 (3.2%)	7 (7.8%)	11 (31.4%)

[1] dairy = 80 *per cent* or more dairy cows, suckler = 80 *per cent* or more suckler cows, mixed = remainder of herds

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Table 6-4: Average farm size and adjusted stocking rate[1] per district (1995).

		A	B	C	D	E	F
Farm Size (hectares)		42.5 (3.0)[2]	44.3 (3.6)	27.3 (1.2)*	31.4 (1.9)**	48.9 (4.0)	44.4 (8.5)
Stocking Rate		0.76	0.77	0.54	0.51	0.77	0.62

[1]Animals per acre: *Cow = 1 unit, 1 – 2 yro bovine = 0.75 unit, < 1 yro bovine = 0 33 unit, horse = 1 unit, sheep = 0 5 unit)*, excludes farms with no stock in 1995. [2] *Standard error of mean*
* significantly lower (P <= 0.01, ANOVA Newman-Keuls post-hoc test) than areas A, B, and C.
** significantly lower (P <= 0 01) than area A

Table 6-5: Average number stock per farm[1] per district (1995).

	Number animals					
Area	Dairy cows	Suckler cows	Dry Stock	Calves	Sheep	Horses
A	21.1	8.5	33.3	29.7	21.6	2.5
B	33.1	5.5	43.6	40.1	7.4	0.1
C	8.3	10.0	14.3	16.7	2.1	0.5
D	6.3	12.7	13.3	18.7	5.3	0.4
E	24.0	10.7	47.1	35.0	12.4	0.7
F	10.2	18.0	27.5	20.1	24.8	1.8
All Areas	14.6	10.6	25.5	24.5	9.2	0.8

[1]Includes farms with no stock in 1995.

Table 6-6: Distribution of farms by annual milk yield (gals) per cow per district

	Number (%) farms					
Yield (gals)	A	B	C	D	E	F
<1000	27 (61.4%)	20 (57.1%)	52 (81.3%)	41 (78.8%)	28 (47.5%)	5 (50.0%)
> 1000	17 (38.6%)	15 (42.9%)	12 (18.8%)	11 (21.2%)	31 (52.5%)	5 (50.0%)

Table 6-7: Distribution of farms by annual concentrate supplementation rates per cow per area.

	Number (%) farms per Area					
Concentrates (kg)	A	B	C	D	E	F
<500	26 (49.1%)	10 (37.0%)	85 (79.4%)	88 (76.5%)	35 (46.1%)	10 (45.5%)
> 500	27 (50.9%)	17 (63.0%)	22 (20.6%)	27 (23.5%)	34 (53.9%)	12 (54.5%)

Table 6-8: Distribution of farms by proportion of cattle outwintered (1995-96) by farm type.

% stock out-wintered	% farms per area					
Dairy Farms	A	B	C	D	E	F
None	42%	56%	69%	61%	73%	75%
1 – 50%	31%	36%	29%	32%	18%	25%
> 50%	27%	8%	2%	6%	9%	0%
Suckler/Mixed Farms						
None	10%	45%	50%	46%	45%	29%
1 – 50%	23%	27%	21%	18%	32%	21%
> 50%	67%	27%	29%	36%	23%	50%

Table 6-9: Distribution of farms by number of silage analyses in three-year period 1993 – 1995.

	Number (%) farms per area					
No. Analyses	A	B	C	D	E	F
0	46 (62.2%)	29 (60.4%)	133 (91.1%)	114 (81.4%)	41 (49.4%)	23 (74%)
3	27 (36.5%)	18 (37.5%)	13 (8.9%)	25 (17.9%)	42 (50.6%)	8 (25.8%)
> 3	1 (1.4%)	1 (2.1%)	0 (0.0%)	1 (0.7%)	0 (0.0%)	0 (0.0%)

Table 6-10: Classification of farms by calving season per area.

	Number (%) farms per area					
Season	A	B	C	D	E	F
Spring	63 (81.8%)	43 (84.3%)	148 (94.9%)	135 (90.6%)	71 (86.6%)	18 (78.3%)
Autumn	1 (1.3%)	0 (0.0%)	3 (1.9%)	4 (2.7%)	1 (1.2%)	3 (13.0%)
Both	13 (16.9%)	8 (15.7%)	5 (3.2%)	10 (6.7%)	10 (12.2%)	2 (8.7%)

Table 6-11: Mean farm-level mortality by area – 1995[1].

	% Mortality (standard deviation)						
	A	B	C	D	E	F	All
Dairy Cows	2.3 (3.3)	2.6 (3.5)	1.6 (4.0)	2.2 (5.2)	1.1 (2.1)	1.1 (2.0)	1.9 (3.8)
Suckler Cows	3.8 (7.0)	5.0 (11.1)	1.1 (3.2)	1.0 (3.0)	1.6 (3.0)	0.8 (1.6)	1.6 (4.5)
Dry Stock[2]	1.3 (4.1)	0.7 (1.7)	1.6 (5.6)	0.6 (2.6)	0.7 (1.7)	2.1 (4.2)	1.1 (3.6)
Calves	8.7 (10.5)	7.7 (7.6)	4.9 (8.7)	6.0 (8.3)	6.4 (6.0)	3.6 (4.5)	6.2 (8.4)
Sheep	11.2 (18.9)	5.9 (3.7)	10.0 (22.5)	5.6 (13.8)	8.3 (8.3)	12.6 (25.8)	9 (18.2)
Horses	5.4 (12.4)	NA[3]	0.0	25 (50)	1.3 (3.1)	0.0	5.8 (20.0)

[1] Farms with less than four animals in a category are omitted from calculation for that category (see text). [2] Heifers & bullocks over six months. [3] No horses

Table 6-12: Farm-level mortality rates[1] by exposure status.

	% Mortality Rates (standard deviation)					
	1995			1996		
	Exp	Non-exp	p[2]	Exp	Non-exp	p
Dairy Cows	2.40 (3.37)	1.63 (3.91)	0.002	1.31 (2.32)	1.13 (3.01)	0.029
Suckler Cows	4.19 (8.43)	1.11 (2.99)	0.01	2.23 (6.02)	0.73 (2.41)	0.313
Dry Stock	1.06 (3.38)	1.05 (3.75)	0.335	1.05 (3.98)	0.64 (2.64)	0.051
Calves	8.28 (9.46)	5.55 (7.95)	0.002	12.66 (21.44)	6.65 (11.97)	<0.001
Sheep	9.96 (16.6)	8.79 (18.65)	0.324	9.66 (20.81)	5.57 (8.89)	0.168
Horses	5.43 (12.38)	5.98 (23.53)	0.745	3.38 (7.15)	0.65 (2.69)	0.870

[1] Numerator in 1995 = reported number of deaths per animal class for entire year. Numerator in 1996 = reported number of deaths in first six months of year. Denominator in 1995 = reported average number of animals in each class per farm for the year. Denominator for calf mortality in 1996 = number cows calved + twins. Denominator for other classes in 1996 = reported number of animals on farm at the time when the questionnaire was completed. Results exclude cases where denominator for a class is less than four animals (see text).
[2] Rank Sum Two-Sample (Mann-Whitney)

Table 6-13: Overall annual mortality[1] by exposure status.

	% Mortality			
	1995		**1996**	
	Exp	**Non-exp**	**Exp**	**Non-exp**
Dairy Cows	2.4	2.6	1.3	1.8
Suckler Cows	3.7	1.4	2.0	0.6
Dry Stock	1.0	1.3	1.0	0.7
Calves	8.9	7.1	12.8	11.1
Sheep	9.5	10.4	9.2	5.2
Horses	3.7	4.7	0.9	0.7

[1]Numerator in 1995 and 1996 = reported number of deaths per animal class (first six months of 1996). Denominator in 1995 = reported average number of animals in each class per farm for the year. Denominator for calf mortality in 1996 = number cows calved + twins. Denominator for other classes in 1996 = reported number of animals on farm at the time when the questionnaire was completed. Results exclude cases where denominator for a class is less than four animals (see text).

Table 6-14: Median farm-level mortality rates by exposure status (1995).

	Median Mortality Rate (%)					
	1995			**1996**		
	Exp	**Non-exp**	**All**	**Exp**	**Non-exp**	**All**
All Cows	1.14%	0.00%	0.00%	0.00%	0.00%	0.00%
Dairy Cows	1.25%	0.00%	0.00%	0.00%	0.00%	0.00%
Suckler Cows	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
Dry Stock	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
Calves	6.16%	2.25%	4.17%	2.87%	0.00%	2.50%
Sheep	5.44%	0.00%	0.00%	0.00%	0.00%	0.00%
Horses	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%

Note: includes all responses, i.e. group sizes < 4 are not excluded.

Table 6-15: Distribution of above-median (HIMORTxx[1]) farms by exposure status[2]

	Percent (number) farms					
	Dcow	**Scow**	**Dry**	**Calves**	**Sheep**	**Horses**
1995						
Exp.	51.1 (45)	31.8 (21)	21.6 (29)	63.1 (82)	77.3 (17)	14.8 (4)
Non-exp.	24.4 (47)	14.1 (43)	14.0 (49)	44.8 (175)	36.2 (25)	8.2 (4)
1996						
Exp.	38.6 (34)	16.2 (11)	20.4 (28)	61.6 (69)	69.6 (16)	6.9 (2)
Non-exp.	19.4 (36)	9.7 (30)	7.9 (29)	46.2 (172)	39.7 (27)	3.8 (2)

[1] HIMORT = mortality > median rate (xx = '95 or '96) [2]includes all responses, i e. group sizes < 4 are not excluded.

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Table 6-16: Odds ratios of above-median farm-level mortality (HIMORTxx[1]) for exposed *vs* non-exposed regions in each animal class in 1995 and 1996[2]

Model	Dairy Cows	Suckler Cows	Dry Stock	Calves	Sheep	Horses
1995						
Exposure alone						
Odd Ratios	3.36	3.05	1.46	2 05	5 07	3.22
95% Conf Intervals	1.96 – 5 57	1.63 – 5 71	0.88 – 2 42	1.36 – 3.10	1.07 – 24.1	0.80 – 12.99
P	<0 0001	0 0004	0.1442	0.0711	0.0405	0.1009
Exposure + group size[3]						
Odd Ratios	2.13	3.34	1.23	1.48	4.15	1 57
Conf. Intervals	1.18 – 3 86	1 70 – 6.55	0.73 – 2.1	0.95 – 2.31	0.85 – 20.33	0.28 – 8 94
P	0.0123	0.0004	0.4373	0.0828	0.0788	0.6092
1996						
Exposure alone						
Odd Ratios	3.29	1 81	2.61	2.16	3.72	3.19
95% Conf. Intervals	1.97 – 5.51	0.85 – 3 85	1.51 – 4.53	1.37 – 3 40	1.32 - 10.5	0.45 - 22.75
P	<0 0001	0.1232	0.0006	0.0009	0.0132	0.2485
Exposure + group size[3]						
Odd Ratios	1 35	1.66	2.11	1.52	2 92	1.17
Conf. Intervals	0.70 – 2.60	0.75 – 3.69	1.19 – 3.75	1.03 – 1.07	0.97 - 8.79	0.09 - 15.79
P	0.5457	0.2118	0.0104	0.0402	0.0563	0.9058

[1] HIMORT = mortality > median rate (xx = '95 or '96). [2] Analysis <u>excludes</u> herds of < 4 animals per class except horses.
[3] Number animals per class per farm

Table 6-17: Distribution of exposed and non-exposed farms by cow mortality rates (1995).

	Number (percent) Farms[1]			
Mortality Rate (%)	Dairy Cows		Suckler cows	
	Exp	Non-exp	Exp	Non-exp
0	43 (48.9%)	152 (75.2%)	44 (67.7%)	278 (85.5%)
0 – 4	23 (26.1%)	16 (7.9%)	6 (9.2%)	10 (3.1%)
4 – 8	18 (20.5%)	21 (10.4%)	6 (9.2%)	21 (6.5%)
> 8	4 (4.5%)	13 (6.5%)	9 (13.8%)	16 (4.9%)

[1] Farms with dairy and suckler cows are counted twice (i.e. as dairy and suckler).

Table 6-18: Farm-level mortality rates by cow numbers per farm.

	Mortality Rate (*per cent*)							
	1995				1996			
	Dairy		Suckler		Dairy		Suckler	
No. Cows	Exp.	Non-exp.	Exp.	Non-exp.	Exp.	Non-exp.	Exp.	Non-exp.
4 –14	1.28	1.48	2 56	1 03	0.00	1.71	1.85	0.51
15 – 29	3 32	1.93	8.79	1.06	1.61	1 18	4 08	0 81
> 29	2.19	1.20	2.04	1.50	1.33	1.24	0 44	1.32

Table 6-19: Distribution of farms by exposure status and presence or absence of ill-thrift.

	Number (%) farms			
Ill-thrift	Cows		Dry Stock[1]	
	Exp.	Non-exp.	Exp.	Non-exp.
Present	16 (12.3%)	21 (5.4%)	32 (23.5%)	22 (6.0%)
Absent	114 (87.7%)	371 (94.6%)	104 (76.5%)	347 (94.0%)
Odds ratio[2]	2.48		4.85	
95% confidence interval	1.25-4.90		2.71-8.71	

[1] Heifers, bullocks and calves. [2] Odds of occurrence in exposed *vs* non-exposed.

Table 6-20: Farm-level fertility and periparturient animal health data for exposed and non-exposed regions in 1996

	Exposed			Non-exposed			All[2]		
Condition/parameter	Mean	SD[3]	Median	Mean	SD	Median	Mean	SD	Median
Open (12.14)[4]	8.06*	12.2	5.36	4.31*	9.63	0.00	5.52	11.27	0.00
Late (12.16)	11.50	13.32	8.45	11.40	13.24	9.09	11.41	13 22	8.70
Abortions (12.12)	0.76	2.23	0.00	0.72	2.87	0.00	0.71	2 68	0.00
Assisted calvings (12.23)	20.97*	23.17	14.29	17.53*	23.37	0.10	18 38	23.27	11.11
Cows down[5] (12.25)	1.14*	3.14	0.00	0.73*	5.53	0 00	0.87	5.00	0.00
Cow dead (12.27)	0.92*	2.97	0.00	0.52*	5.36	0.00	0.63	4.7	0.00
Twin births (12 21)	3.08	7.17	0.00	2.29	4.16	0 00	2.50	5 10	0.00
Deformed calves (12.29)	0 37	1 15	0.00	0 44	2.33	0.00	0.42	2.11	0.00
Perinatal calf deaths[6] (12.31)	6.33*	10.53	4.08	2 69*	5 35	0.00	3 57	7.07	0.00
Calves dead[7] (12.33)	3.78	7.28	0.00	3 02	7.42	0.00	3.14	7.25	0.00

Incidence Rate (per cent) [1]

[1] Farms with < 4 cows served in 1995 omitted. [2] Six areas (A – F). [3] Standard deviation. [4] Record and field of questionnaire (see Appendix 10). [5] Within one month of calving/delivery. [6] Within two days of birth. [7] Three days to three months.

* Difference significant at p < 0.05 - exposed vs non-exposed (Mann-Whitney rank sum test).

Table 6-21: Farm-level incidence (per cent) of animal treatment[1] by exposure status – Jan 1 to May 31, 1996.

Treatment	Area	Bullock	Calf	Dcow[2]	Heifer	Scow[3]
Diarrhoea	Exp	0.8	20.7	2.1	2 2	5.1
	Non-exp	1.5	16.1	2.5	0.7	1.1
	Total	1.2	17.5	2 3	1.3	2.1
Eye disorder	Exp	2.8	5.7	2.2	5.6	8.3
	Non-exp	2.3	2.5	4 1	1.3	4.6
	Total	2.5	3.6	3.3	2 8	5.5
Fluke	Exp	1.3	0	0.2	1	5.1
	Non-exp	1.8	0.1	1.5	0.9	1.1
	Total	1.6	0	0.9	0.9	2
Lameness	Exp	1.7	0 3	9.7	3.5	5
	Non-exp	2.2	0.6	9 8	3	3.9
	Total	2	0.5	9.8	3.2	4.2
Mastitis	Exp			15.2		5 1
	Non-exp			9.5		3 4
	Total			11.5		3.9
Milk fever	Exp			4.1		0 3
	Non-exp			2.2		0.3
	Total			2.9		0 3
Minerals	Exp	4.4	4	10.3	5.9	6.2
	Non-exp	0.4	1.2	8 2	0.2	1.5
	Total	1.8	2.1	9	2.1	2.5
Pneumonia	Exp	3.1	10.2	0.2	3.2	0
	Non-exp	1.8	5 9	0.9	2.5	0.4
	Total	2 3	7.3	0.6	2.7	0.3
RFM	Exp			6.8		1.5
	Non-exp			4.4		2.1
	Total			5.4		1.9
Skin disorder	Exp	0 6	2.6	1.5	3	2.6
	Non-exp	0.7	0.8	0.2	2	0 1
	Total	0.7	1.4	0.8	2 3	0.7

[1] Prophylactic or therapeutic. [2] Dairy cow. [3] Suckler cow.

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CHAPTER SEVEN

IMMUNOLOGY STUDIES

An investigation into the immune status of cattle from the Askeaton area of Co. Limerick.

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INTRODUCTION

The existence of animal health and productivity problems on farms in the vicinity of Askeaton led to speculation that the animals may have had altered immune function. The Environmental Protection Agency (EPA) was advised that specific tests for immune system function be included in the continuous environmental monitoring program (EPA report, 1995). Studies of immune function were initiated in accordance with the protocol drawn up by the Immunology Advisory Committee (EPA report, 1995). The aim of this study was to assess the immune function of cattle on the farms under investigation. The study was not concerned with determining factors that may have led to animal health problems.

Increased susceptibility to infectious disease may reflect altered immune competence. Clinically, alterations in the immune system manifest as either chronic or recurring infections. Opportunistic infections in animals are also consistent with depressed immunity. An immunodeficient state may also predispose to certain autoimmune diseases or neoplasia. Several immunodeficiency diseases are genetically determined.. In cattle, genetic defects in immune function include the A45 trait, a lethal condition that occurs in Black Pied Danish cattle. Another inherited immunodeficiency is Bovine Leukocyte Adhesion Deficiency (BLAD), a condition characterised by persistent neutrophilia and absence of pus formation at sites of infection. Such inherited immunodeficiency diseases are rare. Unless the majority of animals on the farms under investigation were derived from a limited genetic pool, inherited immunodeficiencies are unlikely to be a cause of altered immunity.

The more likely causes of immunodeficiencies include infectious disease, stress, poor nutrition, malignancies, environmental or other toxic factors, immunosuppresive drugs or hormonal fluctuations. Immunodeficiency arising from these factors may be temporary or permanent. Viruses induce clinically apparent immunodeficient states in some animal species. Bovine immunodeficiency virus is associated with encephalitis and infections in cattle although it is disputed as to whether it leads to a clinically apparent immunodeficient state (Snider et al., 1996). Bovine herpesvirus-1 interferes with the host's antigen presentation machinery to evade the host's immune response (Hinkley et al., 1998). Other viruses such as bovine viral diarrhoea virus, exert a generalised immunosuppresive effect.

Temporary immunodeficient states have been reported in animals under stress. Calves transported by road develop both humoral and cellular immune disorders, which are thought to increase susceptibility to microbial infections (Murata et al., 1987; Murata and Hirose, 1991). Deficiency in a single nutrient or generalised malnutrition has long been known to have adverse effects on the immune system Dairy cows in early lactation may suffer from hepatic lipidosis when their energy demands outstrip energy intake from feed such that their body reserves are used to meet the energy deficit. In cows, hepatic lipidosis has been linked with impaired humoral and cellular responses (Wentink et al., 1997). Age is another factor that affects immune competence. In early life, the immune system is not fully functional and thus the new-born animal is particularly susceptible to infections. Nutritional status at age of weaning has been shown to affect immune responses in calves (Pollock et al., 1994). In older animals, the decline in numbers of T cells and their function is associated with increased susceptibility to infections.

Evaluation of immune competence relies on enumeration of the various elements of the immune system and establishing that these elements are functional. The objectives of the study were

- to compare baseline indicators of immune function in cattle from the Askeaton area with control groups and

- to compare antigenic immunological responses of cattle from the Askeaton area with control animals.

The investigation involved two studies, one with cows and a second with steers. Cellular and humoral components were quantitatively analysed.

Immune cells were assessed for their functional capabilities following non-specific and specific stimulation. The protocol adopted (Immunology Appendix A) analysed immune factors in the peripheral blood of animals within the study.

For longitudinal studies of immune function, the inability to obtain sequential samples limits such studies to sampling peripheral blood. The immune system comprises many tissues and organs within the body and the peripheral blood represents just one compartment within this system. Thus, results must be interpreted with this in mind. While peripheral blood levels of freely diffusable humoral components reflect tissue levels, immune cells may be sequestered extravascularly.

One of the most important aspects of immune monitoring is documenting changes that occur over time. This requires an accurate assessment of baseline data. In this study, no baseline data for cows prior to the reporting of the various animal health problems was available. Thus, immune profiles of animals before and after the 'perceived insult' that lead to the animal health problems could not be assessed. Consequently, a study was designed to compare the immune function of cows from the farm under investigation with healthy animals on the Veterinary Research Laboratory farm, Abbotstown. There were differences in breed between the cows based in Askeaton and Abbotstown. The Askeaton cows were predominantly British Friesian while the Abbotstown cows had a high Holstein Friesian component. To control for variability in immune responses between herds due to differences in breed, cows were assigned to groups based on location and origin (see below).

Group	Origin	Location
A	Abbotstown	Abbotstown
B	Askeaton	Abbotstown
C	Abbotstown	Askeaton
D	Askeaton	Askeaton

This study with cows was designed to test whether location alone rather than origin had an affect on immune status. Implementation of this protocol required the movement of animals between farms. Before the study commenced a system of controlled farm management practices were established on both farms.

In addition to breed, differences in age, pregnancy and lactation may also affect immune responses. It was not possible to match cows for these variables and this must be borne in mind when interpreting the results. However, age and pregnancy status was

included in the statistical analysis of the data. An additional study was undertaken using closely matched steers. Using steers matched according to size it was possible to remove constraining variables encountered in the design of the study with cows.

Selenium levels in the diet are known to influence immune function (Finch and Turner, 1996; McKenzie et al., 1998) and levels of blood selenium differed between farms. While efforts to control for selenium variability were not initiated, a study was conducted to assess whether blood selenium levels influenced immune responsiveness to a particular antigen.

MATERIALS AND METHODS

ANIMALS

Cow study: Forty-three Friesian cows, ranging in age from 2-13 years were used. Cows of Askeaton origin were British Friesian and those of Abbotstown origin had a high Holstein Friesian component. The cows were assigned to four groups according to their origin and location (see table above).

Steer study: Twenty-four steers (mixed cross-breeds), matched according to size, were purchased outside the area of investigation. Twelve steers were placed on the VRL farm at Abbotstown and twelve placed on Index farm B in Askeaton.

Animals and their group assignments are given in Immunology Appendix B.

IMMUNISATION SCHEDULE

Keyhole limpet haemocyanin (KLH) (Calbiochem-Novabiochem, Nottingham, UK) was dissolved in phosphate buffered saline (PBS), sterilised by filtration through a 0.2 μm filter and precipitated on alum (Hudson and Hay, 1980). Each cow was immunised with 1 mg KLH administered subcutaneously, followed by 0.5 mg two weeks later. Steers were immunised with 1 mg KLH subcutaneously followed by 1 mg two weeks later. Control animals were given alum in PBS.

MEASUREMENT OF SERUM IGG AND IGM CONCENTRATIONS

Concentrations of total IgG were determined by radial immunodiffusion with a commercially available kit (The Binding Site Ltd., Birmingham, UK). Total concentration of IgM was measured by sandwich ELISA. ELISA plates (Greiner, Germany) were coated overnight with sheep anti-bovine IgM at 10 μg/ml in 0.05 M sodium

bicarbonate buffer pH 9.6. The plates were washed three times with PBS containing 0.1% Tween 20 (PBST). Dilutions of test sera and IgM standards were made up in 10% sheep serum PBST. After incubation for 1 hour plates were washed 6 times and a monoclonal anti-bovine IgM antibody (Lelystad, The Netherlands) in 2% sheep serum PBST was added at appropriate dilution. The plates were again washed after 1 hour and sheep anti-mouse IgG conjugated to horseradish peroxidase was added for 1 hour. Tetramethylbenzidine substrate (Sigma, Poole, UK) was added. The colour reaction was stopped after 30 minutes with 2 M sulphuric acid and absorbence at 450 nm determined using an ELISA reader (Spectra, Austria). Immunoglobulin concentration in test samples was determined by comparison of absorbence readings at 450 nm with standard curves produced using purified immunoglobulins of known concentration (Chemicon, CA, USA).

MEASUREMENT OF BOVINE SERUM COMPLEMENT ACTIVITY

Serum complement activity was determined by measuring the diameter of haemolysis induced by bovine sera in agar gel containing guinea pig erythrocytes sensitised with antibody. Guinea pig erythrocytes were collected in Alsever's anticoagulant at a 1:1 ratio. The blood was stored at 4°C for at least 4 days before being used. The blood was centrifuged and the anticoagulant and buffy coat removed. The erythrocytes were washed 3 times in PBS and made up to 1% in barbitone complement fixation test diluent (BCFT) (OXOID, Basingstoke, UK). The concentration was adjusted to 1×10^8 cells/ml. Rabbit anti-guinea pig erythrocyte antibody (Accurate Chemical and Scientific Corp., Westbury, USA) was added at appropriate dilution (1:8000). Erythrocytes were incubated for 45 minutes at room temperature with mixing every 10-15 minutes and washed twice, then resuspended in BCFT supplemented with 100-IU penicillin-streptomycin to a concentration of 1×10^8 cells/ml. A 2% solution of agarose (Sigma, Poole, UK) in BCFT plus 0.1% sodium azide was melted at 80°C and cooled to 45°C in a waterbath. Sensitised erythrocytes (EA) were warmed to 45°C and added to an equal volume of the agarose solution. Plates were poured immediately and allowed to solidify. Wells (3 mm in diameter) were cut in the agarose gel and 10 μl of undiluted serum was added to each well. The slides were returned to the refrigerator for 20 hours and then incubated at 37°C for 2 hours. A titration of a standard reference human complement serum (Sigma, Poole, UK) was included on each slide. The log of the concentration of reference serum was plotted against the diameter of lysis. From this titration curve the log of percentage lysis of test samples was measured.

IN VITRO LYMPHOCYTE PROLIFERATIVE RESPONSE TO MITOGENS AND ANTIGEN

Bovine peripheral blood mononuclear cells (PBMC) were prepared from heparinised venous blood by centrifugation over Ficoll-Paque (Pharmacia-Biotek, Sweden). All cells were washed twice in PBS and resuspended to a concentration of 1×10^6 in RPMI-supplemented with 5% fetal calf serum (Life Technologies, Paisley, UK) 2 mM L-glutamine, 0.5 μM 2-mercaptoethanol and 100 U penicillin-streptomycin. Lymphocytes were seeded at 10^5 cells/200 μl and set up in triplicate. Concanavalin A at 5 μg/ml, phytohaemaglutinin at 4 μg/ml, pokeweed mitogen at 1 μg/ml and KLH at 250 μg/ml were added. Negative controls consisted of cells grown under the same conditions without any mitogen/antigen. Plates were incubated for 48 hours at 37°C, 5% CO_2 prior to addition of 1 mCi/well of ^{3}H-thymidine (Amersham, Buckinghamshire, UK). The incorporation of ^{3}H-thymidine into cellular DNA was determined by use of liquid scintillation spectrometry. Results were expressed as net counts per minute (cpm) for mitogen response and as a stimulation index (SI) for antigen response. These were calculated as follows:

- Net CPM = cpm of mitogen stimulated cells minus cpm of unstimulated cells.

- SI = cpm of antigen stimulated cells / cpm of unstimulated cells.

γIFN PRODUCTION

Aliquots of 1 ml heparinised blood from each animal were added to individual wells of 24-well tissue culture plates (Costar, MA, USA) containing either KLH (0.1 mg/ml) or PBS (negative control). The cultures were incubated for 24 hours at 37°C, 5% CO_2 before harvesting the plasma supernatants. Samples were stored at -20°C prior to assay. γIFN production was measured in duplicate samples using a commercially available sandwich ELISA (CSL Limited, Parkville, Australia). Results are presented as the OD at 450nm.

ANTIBODY TITRES TO KLH

An indirect ELISA was used to measure anti-KLH IgG antibody responses in sera. The procedure used is a modification of that described by Pollock

et al., (1991). Briefly, ELISA plates (Nunc, Denmark) were coated with KLH at 10 µg/ml in PBS. Sera were diluted in 10% rabbit sera PBST and titrated on the plate in doubling dilutions. Monoclonal antibody specific for bovine IgG was added followed by a rabbit anti-mouse horseradish peroxidase conjugate. The plates were developed with tetramethylbenzidine-hydrogen peroxide. The reaction was stopped after 30 minutes by the addition of 2 M sulphuric acid. Absorbence readings were measured at 450 nm. For each serum sample titration curves were established and results expressed as the reciprocal of the dilution at which half-maximal OD was recorded.

PHENOTYPIC ANALYSIS OF PERIPHERAL BLOOD LYMPHOCYTES (PBL)

In the cow study, PBL were prepared by hypotonic lysis whereas for the steer study PBL were prepared by density gradient centrifugation. Isolated PBL were resuspended in PBS with 1% bovine serum albumin and 0.1% sodium azide (PBA) at approximately 2×10^7 cells/ml. 50 µl of the cell suspension were incubated for 30 minutes with 50µl of each of the following monoclonal antibodies which recognise cell membrane antigens; GC50A which recognises BoCD4 (Larsen *et al.*, 1990), CACT80C which recognises BoCD8 (Larsen *et al.*, 1990), CACT61A which recognises the TCR1 on γδ T cells (Davis *et al.*, 1990), VPM30 which recognises bovine B cells (Naessens and Howard, 1992) and MUC2A which recognises BoCD2 (Davis *et al.*, 1993). To determine background immunofluorescence attributable to non-specific binding, isolated PBL were incubated with normal mouse serum. After washing twice in PBA, cells were incubated in isotype-specific fluorescent conjugates. After 30 minutes cells were fixed in 1% formaldehyde-PBS and stored at 4 °C until analysed on a fluorescent activated cell sorter (FACS) (EPIX, Coulter, Fl, USA). Two-parameter analysis of forward scatter (FS) versus side scatter (SC) was used to gate the population of lymphocytes and data for 5,000 cells were collected.

POLYMORPHONUCLEAR FUNCTION - OXIDATIVE BURST ASSAY

This assay measures the oxidative burst activity of PMN. Dihydrorhodamine 123 (DHR123) is reduced to fluorescent rhodamine by the reactive oxidants produced by the oxidative burst. Aliquots of heparinised whole blood (50µl) were added to flow cytometer tubes and incubated on ice for 10 minutes. Cells were then incubated at 37°C with heat inactivated *E. coli* (1×10^7) for 10 minutes.

10µl of 100mM DHR123 were added and tubes incubated for a further 10 minutes. Red blood cells were lysed hypotonically and cells immediately fixed in 1% formaldehyde-PBS. Samples were analysed using a FACS to record the percentage fluorescent PMN and mean intensity of fluorescence. The relative intensity of oxidative burst (RIOB) was calculated by the formula:

(% fluorescent cells in sample minus % fluorescent cells in control) X (mean fluorescence intensity of cells in sample / mean fluorescence intensity of control cells).

Controls consisted of cells incubated with sterile PBS.

PHAGOCYTOSIS ASSAY

Using fluorescein labeled bacteria, the percentage of PMN engaged in phagocytosis was evaluated. Aliquots of whole blood from each animal were added to tubes and incubated for 10 minutes on ice. Fluorescein labeled *E. coli* (10^7) (Molecular Probes, The Netherlands) were added to each tube and incubated for either 0 or 10 minutes at 37°C. Trypan blue was added to quench external fluorescence (50µl at 3 mg/ml). Red blood cells were lysed hypotonically and cells immediately fixed in 1% formaldehyde-PBS.

Two-parameter analysis of FS versus SC was used to gate the population of PMN and data for 5000 cells were collected. The degree of phagocytosis was measured as fluorescent intensity. The percentage fluorescence and the mean channel number of fluorescence were used as a quantitative index of neutrophil response. The relative phagocytic index (RPI) was calculated as follows:

(% fluorescent cells in sample minus % fluorescent cells in control) X (mean fluorescence intensity of cells in sample / mean fluorescence intensity of control cells).

Daily standardisation was performed using FlowSet beads (Coulter, FL, USA).

SKIN TEST

Hair was clipped over a site in the mid-neck region. KLH (0.1ml at 1 mg/ml) was injected intradermally at this site with a McLintock syringe. A similar volume of PBS was injected at another similarly prepared site. Skin thickness was measured 72 hours after injection across two diagonals at right angles using constant tension calipers. Responses were recorded as mean post-injection thickness minus pre-injection thickness.

Control injection of PBS alone was used to ensure that the response was specific to KLH.

BLOOD SELENIUM LEVELS

These assays were conducted at the Abbotstown laboratories.

STATISTICAL ANALYSIS

Values are expressed as mean +/- standard error of the mean. Results were statistically analysed by UCD Statistics Department (Appendix 11).

RESULTS

The results for the cow and steer study are presented separately. When comparing variables for animals within herds, the normal ranges from large populations of cattle are useful indicators for assessing deviations. For some of the variables, normal ranges are available. However this is not the case for all the parameters measured as these assays are not routinely used in clinical assessment. Such data were statistically analysed. Results are presented with conclusions from the statistical tests. Statistical tests involved linear regression analysis of data available for animals at first and last blood samplings. General estimating equations were used to analyse the repeated measures of immune parameters. More detailed information on the statistical tests are included in an accompanying report (Appendix 11).

COW STUDY

Lymphocyte enumeration

The relative distributions of lymphocyte populations are presented in Table 7-1. The published values for the percentages of peripheral blood lymphocytes positive for CD2, CD4 and CD8 vary according to the source of reagents and design of the experiment. However, the values presented here for CD2, CD4 and CD8 correspond with those reported by Park *et al.*, (1992), Bensaid and Hadam (1991) and Wilson *et al.*, (1996). γδ T cells were identified using an antibody specific for TCR1 (Wyatt *et al.*, 1996). The values reported are in accordance with values published for cows aged 2-9 years (Wyatt *et al.*, 1996). There are no published values for B lymphocyte percentages with the antibody used in this study. B lymphocyte percentages were found to fluctuate widely.

Herd differences may account for some of the variations in percentages of B lymphocytes. When groups were compared according to their origin, the mean of combined observations for percentage B lymphocytes in peripheral blood was 23.8% for Abbotstown origin cows and 17.5% for Askeaton origin cows (Table 7-2). There was no difference in relative B lymphocyte percentages when compared on the basis of location. Thus differences in percentages of B lymphocytes appear to be related to origin rather than location. Animals 208 and 303 had consistently low numbers of B lymphocytes throughout the study (see Immunology Appendix B for the group assignment of animals). The mean number of B lymphocytes recorded for 208 was 4.9% and for 303 it was 6.4%. Both these animals are over 9 years old and had chronic mastitis.

The CD2, CD4, CD8 and B lymphocyte values for each group were statistically analysed. Differences were observed between the groups on both first bleed and last bleed analysis. First bleed analysis identified Group C animals as having higher B cell percentages than group A and D. Group B animals differed from group A animals with respect to CD8 levels and group D animals differed from group A animals on CD2 levels. On last bleed analysis only group C were found to differ from group D with respect to B cell and CD2 levels. Group C animals also differed from group A with respect to CD4 levels. Group D animals were not found to differ from group A animals on last bleed analysis. This would indicate that location is not responsible for the observed differences in proportions of lymphocyte phenotypes.

Serum immunoglobulin concentrations.

The normal range of IgG defined for adult Friesian serum is 17-23 mg/ml (Barta, 1993). The values recorded for cows in this study are higher than the normal range. Serum concentrations of IgG from May '97 to January '98 were lowest for group A, however, there appeared to be no difference between the other groups. The mean for the groups during the study period is given in Figure 7.1.

The normal values for IgM levels in cattle sera is given as 2.39-3.48 mg/ml (Barta, 1993) and 3.9 ± 0.3 mg/ml (literature from The Binding Site Ltd., 1995). These values are based on data collected from measurement of IgM levels by radial immunodiffusion. An ELISA was used to measure IgM levels in this study. ELISA is a more sensitive technique and this may account for the higher values recorded. The mean for the groups over time is given in Figure 7.1. From 20/1/97 to 11/5/97 higher values were recorded for animals in Group B. Increased levels are an indication of immunostimulation.

Lymphocyte responsiveness to mitogen stimulation

Blood samples from the Askeaton-based animals were subject to transport for 3 hours by train before assays were conducted. Samples from the VRL herd were not transported under the same conditions. An experiment was designed as part of the steer study (*see* below) to investigate whether agitation of blood samples while in transit had an effect on the variables measured. For convenience, the effect of train transport was simulated. However, the results of this experiment were inconclusive. This study found that simulated agitation had a significant effect on mitogen responses using paired samples in one experiment while no significant effect was noted in a repeat experiment. Because of these results, the possible effects of transport on the *in-vitro* mitogen response tests on samples from the Askeaton farms could not be determined.

Peripheral blood lymphocytes were tested for their proliferative response to various mitogens throughout the period of study. Results for each group following non-specific stimulation of lymphocytes with ConA, PHA and PWM are presented in Figure 7.2.

Group C and D animals showed a reduction in activity when tested with each mitogen on 5/12/96. This correlates with reduced CD4 counts measured (Table 7-1). This finding is consistent between the two groups for this date. This effect was noted on one occasion only. As these samples were taken when cows had been bled on three occasions over a short time period (2, 3 and 5/12/96) it is possible that the effect was due to stress. Corticosteroid hormones induced by stress, have been shown to alter the composition of blood mononuclear leukocyte populations and impair secretion of proteins critical to normal immune responses (Nonnecke *et al.*, 1997; Burton and Kehrli, 1996).

For statistical analysis, results for ConA and PHA were transformed and expressed as the ratio of the gross value to background on the natural log scale (values given in Figure 7.2 are expressed as the gross value minus the background). The PWM response was expressed in the same units given in Figure 7.2. Statistical analysis of the data revealed that only the PWM responses were consistently found to differ between group A and groups B, C and D. Even on first bleed analysis, significant differences were observed between the reference group A and each of the other groups. Thus, a baseline was not established for this variable. Because the PWM response varied considerably it was not a reliable indicator of immune function in these cattle.

For ConA and PHA stimulation, no differences were observed between the groups on first bleed analysis. On last bleed analysis group C had a significantly higher response compared to group A and D with respect to PHA and ConA. However, group D was not found to differ from group A. If the cause of these differences was due to location alone it would be expected that group D would also differ significantly from group A. Group A and D did not differ, thus the differences are not dependent on location. When the results were analysed over time, age was found to have a significant effect on the PHA and Con A response, while pregnancy had a significant effect on the ConA response.

Neutrophil assays

These assays were not conducted on the cow samples as pregnancy has an immunosuppresive effect on neutrophil function (Kehrli *et al.*, 1989; Crouch *et al.*, 1995). However, all cows were assessed for the expression of the CD18 molecule. In BLAD affected animals this molecule is absent. Expression of CD18 was observed on the neutrophils of animals studied.

Complement levels

Cow sera were screened on a few occasions for complement deficiencies using the haemolysis in gel method. This method identifies animals with gross deficiency in complement levels. It is not a sensitive technique for detecting absolute levels of complement activity. The mean percentage activity relative to reference sera for the groups is presented in Figure 7.3. All animals had detectable levels of complement activity in their sera.

Specific immune response to KLH

Proliferative lymphocyte response

All animals had a measurable lymphocyte proliferative response to the KLH antigen. Baseline levels of stimulation (i.e. Day 0) were less than a stimulation index of 2.5. Responses greater than 2.5 were measured in all animals immunised. However, there was wide variation in the magnitude of responses observed. Cows 92, 211, 213 and 313 had weak responses to KLH while cows 55, 63, 74, 83, 84, 85, 87, 91 and 103 had strong responses. The mean responses for the groups are presented in Figure 7.4. Statistical analysis of the data found that groups B and D had a similar mean response and that both differ significantly from group A. Group C cows did not differ in mean response from group A. Groups B and D animals originated in Askeaton but were located in Abbotstown and Askeaton respectively,

while group C represents VRL cows moved to Askeaton. Location did not have a significant effect on the response of group C cows. Thus, location cannot be established as a cause for the differences observed between group A and group D. When the data for all cows is analysed on the basis of location and origin, origin appears to contribute to the differences observed (Figure 7.5). Differences in origin may relate to breed differences between the cow groups.

Cytokine response to KLH stimulation

The cytokine γ–interferon, produced by lymphocytes in response to KLH stimulation was measured at three time points following immunisation. Maximal production of γIFN was measured 70 days after first immunisation. The results are presented as the mean response for the cow groups over background (Figure 7.6). Cows, 62, 63, 68, 89, 92, 103, 207, 208, 306 and 313 produced very low levels of γIFN. There were no significant differences between the groups in their ability to produce γIFN when compared at each time point.

Antibody response to KLH.

There was a detectable rise in serum anti-KLH antibody two weeks after the second immunisation. With the exception of animals 89, 92 and 99, sera from all animals showed increased antibody production to KLH immediately after second immunisation. As expected, the response wanes after the initial peak two weeks post second-immunisation. The mean measurements of anti-KLH IgG levels are presented for the groups in Figure 7.7. The statistical analysis of the results found that Askeaton origin animals located in Askeaton (group D) had a significantly weaker response compared to each of the other groups. However, location was not found to have a significant effect on the magnitude of the antibody response for cows moved from Abbotstown to Askeaton (group C). When cows were grouped according to origin and location, cows of Askeaton origin but not location had a weaker antibody response to KLH (Figure 7.8). This correlates with the earlier finding that origin of animals affected lymphocyte stimulation to KLH.

Skin test

Changes in skin thickness were recorded 72 hours after administration of the antigen. The mean for each group is presented in Figure 7.9. Negligible responses were measured in all animals. The kinetics of the skin response was not assessed and it may be that at 72 hours the reaction had declined

Lymphocyte enumeration

The mean percentage of peripheral blood lymphocyte subsets for the two steer groups sampled at various time points is presented in Table 7-3. All lymphocyte subsets were represented and deficits in animals were not identified. The lymphocyte subset profiles stained positive for CD2, CD8, and CD4 correlate with those reported for male cattle by Wilson *et al.*, (1996). The TCR1 subpopulation is known to be present in higher concentrations in peripheral blood of young ruminants compared to adults (Hein and Mackay, 1991). In comparison with the percentage TCR1 positive cells reported for the cow groups, the values for steers are significantly higher and this is a reflection of their age profile. The antibody used in this study has not been employed previously to enumerate B lymphocytes in peripheral blood of cattle. The percentages of B cells fluctuated widely (6 - 54%).

The three major subpopulations of lymphocytes are the T, B and TCR1 lymphocytes. The majority of T lymphocytes are CD2 positive and a subset of these is CD4 positive. As the values reported are expressed as percentages any increase or decrease in B lymphocyte numbers will alter the percentage values reported for CD2, CD4 and TCR1 lymphocytes. The wide fluctuations recorded for B lymphocytes may account for the fluctuations observed in CD2, CD4 and TCR1 lymphocyte percentages.

The values for B lymphocytes, CD2 and CD4 lymphocytes were analysed statistically. There were significant differences between the groups in their relative percentages on last bleed analysis and repeated measures analysis. Though significant differences between the groups were observed on repeated measures analysis no indication is given as to whether there was a progressive decline over time. From the data presented in Table 7-3, it is apparent that fluctuations in percentages of lymphocytes occurred within groups over time. There is no trend over time to suggest that location had a negative effect on proportions of lymphocytes.

Total immunoglobulin concentrations

The mean serum concentrations of immunoglobulins G and M (IgG and IgM respectively) are presented in Figure 7.10 for both groups. The reference range for serum IgG in adult cattle is 17.66 to 22.9 mg/ml (Barta, 1993). Concentrations for both groups were within this

normal range. Serum IgM levels were comparable in both groups.

Mitogen responses

The functional capacity of lymphocytes was measured using mitogens that induce lymphocyte blastogenesis. For each animal sample, Con A, PWM and PHA induced a significant degree of lymphocyte proliferation. This indicates the presence of functional lymphocytes in all the steers studied. Differences between the groups in the magnitude of proliferation varied with time (Figure 7.11).

For statistical analysis, results for ConA and PHA were transformed and expressed as the ratio of the gross value to background on the natural log scale (whereas the values given in Figure 7.11 are expressed as the gross value minus the background). The PWM response was expressed in the same units given in Figure 7.11. Statistical analysis of the data found that there were no significant differences between the two groups with respect to ConA and PHA responses. Statistical analysis of the PWM responses revealed that the two groups differed in magnitude of response on first bleed, last bleed and repeated measures analysis. Because differences were observed between the groups on first bleed analysis no baseline could be established. Thus, PWM was not a reliable variable for investigations into the effect of location on immune responses. A similar finding was reported for the cow groups.

Complement levels

Haemolytic complement levels in sera were measured by radial haemolysis in agar gel. No animal with a complement deficiency was identified. The mean values for the steer groups are presented in Figure 7.12. Levels were comparable between groups.

Phagocyte assays

The ability of neutrophils to ingest bacteria and initiate oxidative burst activity was assessed. The logistical problems of getting samples to the laboratory for analysis meant that samples were assayed up to 7 hours after sampling. As neutrophils are relatively short-lived cells, day to day differences in the time between blood collection and processing contributes to intra-assay variability. Thus it was not valid to make comparisons between the groups with respect to neutrophil function. No animal was identified with persistent neutrophil deficiency when inter-assay comparisons were made (results not shown).

Specific immune response to KLH

Proliferative lymphocyte response

Following immunisation the proliferative response of peripheral blood lymphocytes to KLH was measured. As expected, the animals given the adjuvant alone did not respond to KLH stimulation (SI less than 6). All the KLH immunised animals responded to KLH stimulation but there was wide variation in the magnitude of response. The mean responses for both groups are presented in Figure 7.13. There was a marked difference between the two groups in their ability to respond to KLH. Steers based on the Askeaton farm responded more slowly to antigen. The response between the two groups was significantly different.

Levels of blood selenium also differed between the groups and an additional study was conducted to investigate the possible effect blood selenium levels had on KLH responses. The results of this study are presented in the next section.

Antibody response to KLH

The mean anti-KLH IgG response for both groups is presented in Figure 7.14. Both groups of animals mounted antibody responses to KLH. Antibody responses reached maximum levels 2 weeks after the second injection of KLH. There was a significant difference between the two groups with respect to antibody response. Askeaton-based steers had a lower antibody response to KLH. These animals also had a lower lymphocyte proliferative response to KLH. Thus, it would appear that animals based on the Askeaton farm mounted a weaker cellular and humoral immune response to the KLH antigen.

THE EFFECT OF SELENIUM ON IMMUNE RESPONSE

In the steer study, animals located on the Askeaton farm had a lower response to KLH than those on the VRL farm. Blood selenium levels differed significantly between the two animal groups and this may have contributed to the observed differences in KLH response. Thus, a separate study was initiated to investigate the relationship between blood selenium levels and immune response following KLH immunisation.

Experimental design

Sixteen female animals were divided into two groups and one group received selenium supplementation. All animals were grazed on index farm B, Askeaton which is known to be marginal in selenium levels.

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Animals in the supplemented group were injected with barium selenate (1 mg/kg) two weeks prior to KLH inoculation. Animals were injected with 1 mg KLH in alum on two separate occasions two weeks apart. Two control animals receiving alum alone were included in both groups. Animal groupings are shown below.

Group	Se supplement	KLH Imm.
L (6 heifers)	+	+
LU (2 heifers)	+	-
M (6 heifers)	-	+
MU (2 heifers)	-	-

Blood was taken from each animal at monthly intervals. Blood selenium levels and the immune response to KLH were measured.

Results

KLH-specific immune response

The mean blood selenium concentrations are presented in Figure 7.15. Concentrations in the supplemented and unsupplemented groups (0.43 and 0.40 µmol/l respectively) were not significantly different on the day of selenium supplementation. By the day of first KLH immunisation (day 0), the mean selenium concentration of the supplemented group, at 1.23 µmol/l was significantly higher than that of the unsupplemented group (0.44 µmol/l). By the last day of sampling (day 111), the mean blood selenium concentration of the supplemented group had risen to 1.76 µmol/l while the unsupplemented group had fallen to 0.30 µmol/l.

The mean proliferative response to KLH for each group is given in Figure 7.16. No significant difference was observed between the two groups immunised with KLH. The mean anti-KLH antibody response for the groups is given in Figure 7.17. Again no significant difference was observed between the groups immunised with KLH. No correlation between specific immune response and blood selenium levels was observed. It was concluded that blood selenium within the range 0.2-1.8 µmol/l does not affect the specific immune response to KLH.

DISCUSSION

This study was designed to investigate the immune status of cattle on farms in the Askeaton area where it was alleged that environmental factors had an adverse effect on immune function. The immune status of cattle on farms in Askeaton was compared with cattle on the VRL farm. The investigation involved two studies, one with cows and a second with steers. It was not possible to match cows from Askeaton with those on the VRL farm; thus, cows were moved between locations and grouped according to their origin and location. The design of the cow study compared groups to determine whether immune differences observed were due to location alone and not a result of differences related to origin. With the second study, closely matched steers were purchased and twelve steers placed on both farms. In assessing the immune status of cattle, cells and humoral factors, which are an integral part of the immune system, were quantified and the functional capacity of various cells evaluated. The information gathered provides a comprehensive analysis of non-specific and specific immune parameters.

NON-SPECIFIC IMMUNE PARAMETERS

Percentages of lymphocytes

When investigating alterations in immune function, longitudinal measurements of the relative proportions of lymphocyte subsets are a useful indicator. A progressive decline in any particular lymphocyte population may indicate interference in immune function. This has been demonstrated in human immunodeficiency virus infection where a decline in CD4 positive T lymphocytes over time is observed and correlates with a state of immunodeficiency. No marked decline in percentages of lymphocyte populations was reported during the period of this study. While variability between the groups in the relative proportions of lymphocyte subsets occurred, no definite trend emerged which suggested an immunodeficiency.

The data suggest that lymphocyte subsets were fully represented in peripheral blood of all cattle studied. CD2 cells were the major subset. This subset can be further divided on the basis of CD4 and CD8 staining. The other populations represented were the TCR1 cells and B lymphocytes. Significant differences between the groups of cows in relative proportions of lymphocyte subsets were recorded. However, there was no evidence that these differences were due to location, as Askeaton-derived cows based in Askeaton (group D) did not have altered levels of the various lymphocyte subsets when compared to Abbotstown cows based in Abbotstown (group A). Statistical analysis of the data revealed that age had a significant effect on the distribution of lymphocyte subsets. With advancing age the percentage of CD2, CD8 and B cells increased. The statistical analysis found that pregnancy did not affect lymphocyte distribution. Breed and lactation may also account for differences observed

as these variables are known to influence the numbers and distribution of lymphocytes in blood. An additional problem in this study was the incidence of mastitis on the VRL and Askeaton (Index farm A) farms, which may have contributed to the variability in lymphocyte distribution.

Significant differences in relative proportions of lymphocyte subsets were recorded between the steer groups although these animals were closely matched. The Askeaton based steers had lower percentages of B cells and higher percentages of CD4 and CD2 cells compared to the Abbotstown steers. The statistical test of repeated measures also found significant differences between the groups in relative proportions of B cells, CD2 and CD4 cells. However, the statistical test of repeated measures does not indicate what differences occurred between the groups over time. There was considerable variation in the distribution of lymphocytes subsets recorded for the steer groups throughout the study period. This variability was recorded for both groups. No trend over time was apparent to indicate that location had specific effect on the percentages of lymphocyte subsets in peripheral blood.

Marked variability in proportions of bovine peripheral blood lymphocytes has been reported by other workers (Wilson et al., 1996). Cells may be sequestered extravascularily and blood levels may not reflect accurately relative proportions of the various lymphocyte subsets. Furthermore, Wilson et al., (1996) demonstrated a cyclical pattern in the concentration of bovine lymphocyte subsets during longitudinal studies. In this study, wide variability in relative proportions of lymphocytes was also reported and it was not possible to establish normal ranges for diagnostic purposes.

B Lymphocyte function

Assessment of B lymphocyte function generally relies on measurement of immunoglobulins. Levels of IgG recorded for the steer groups fell within the normal range reported for cattle. The range reported for the cows was higher than the normal range described. Cows used in the study varied with respect to age, breed, stage of pregnancy and lactation, which may account for the observed deviation from the normal range. Despite the higher than normal range recorded, there appeared to be no differences between the cow groups with respect to serum IgG concentrations. However, Abbotstown cows based in Abbotstown (group A) had reduced levels of serum IgG for the last three blood samples collected. Comparable serum IgG concentrations were recorded for the steers. No

effect due to location was observed for the cow or steer groups.

Serum levels of IgM were also measured. Measurement of IgM revealed that group B animals had increased serum levels of IgM from January to May 1997. This group represents Askeaton-derived cows located in Abbotstown. Raised IgM levels are indicative of immunostimulation. This observation which was of short duration (measured in cows located at Abbotstown) is unlikely to be related to environmental factors prevailing in Askeaton. Concentrations of serum IgM were comparable among the other cow and steer groups.

Neutrophil function

In assessing neutrophil function, conditions to allow inter-group comparisons were not established. Samples reached the laboratory at different times after collection due to distance from farms. As neutrophils are short-lived cells this variability had an influence on cell viability. This is likely to have adversely affected phagocytic and oxidative burst activity. By not controlling for cell viability, it was not possible to make inter-group comparisons. When intra-group comparisons of neutrophil function were made using samples collected on the same day, no steer was identified with consistently reduced phagocytic or oxidative burst activity.

Complement levels

Serum complement levels were measured by haemolysis in agar. This assay method is relatively insensitive (Nielsen et al., 1983) but it is a useful method of screening large numbers of samples for suspected complement deficiencies. No animal with complement deficiency was identified. Serum complement levels were comparable in all animals tested.

LYMPHOCYTE RESPONSE TO MITOGEN STIMULATION

The functional capacity of lymphocytes was assessed by measuring their proliferative response to mitogens. The mitogens ConA and PHA induce T lymphocyte proliferation. PWM induces B lymphocyte proliferation which is dependent on the presence of T lymphocytes (Stites, 1991). The PWM response differed between the cow and steer groups at most time points throughout the study. The broad range in relative proportions of B lymphocytes recorded may account for the variability in the observed PWM responses. There were observed differences in the magnitude of the PWM response for the cow and steer groups at first

bleed analysis. Thus, no baseline could be established in order to assess whether location influenced the response over time. For this reason, the PWM variable was not used as an indicator of immune competence. No significant difference was observed between the steer groups for ConA and PHA responses. Thus, location alone did not influence the responses to ConA or PHA. With respect to the ConA and PHA responses for the cow groups, Askeaton-derived cows in Askeaton did not differ significantly from Abbotstown cows based in Abbotstown. Again this would indicate that location did not have a negative effect on these responses. However, significant differences were observed between Abbotstown-derived cows based in Askeaton and Abbotstown. Abbotstown cows moved to Askeaton had a significantly greater response on last bleed analysis to ConA and PHA compared to Abbotstown cows in Abbotstown. It is unclear why only the Abbotstown animals based in Askeaton (groups C) differed significantly from those of the same origin based in Abbotstown.

From the studies investigating the non-specific elements of the immune system i.e. serum immunoglobulin levels, lymphocyte responses to mitogen stimulation, peripheral blood lymphocyte percentages and serum complement levels, location alone did not appear to have a significant influence on immune parameters. There were differences between the groups of steers and cows in the measurements of lymphocyte percentages and lymphocyte responses to mitogens. However, location could not be conclusively established as the cause of these differences. Pregnancy and age were factors included in the statistical tests for the cow study, which had a significant effect on some of the variables measured. Significant differences in blood selenium levels were recorded for both the cow and steer groups, which may also contribute to the differences observed in the non-specific immune parameters. Overall, with regard to measurements of the baseline indicators of immune status there was no apparent progressive decline. All animal groups had functional immune responses as determined by the tests of non-specific immunity.

SPECIFIC IMMUNE RESPONSE TO KLH

Analysis of the specific immune response was also undertaken. Animals were immunised with the antigen KLH, and humoral and cellular immune responses to KLH were assessed. Most animals had the ability to mount an immune response to KLH. A number of animals responded poorly but these were not associated with any particular group. The ability of the animals to mount both an antibody and cell-mediated immune response to the antigen

indicates that there was no defect in the recognition and presentation of KLH. The memory response to antigen was also effective, as demonstrated by *in vitro* assays. Despite the considerable variation in individual animal's ability to mount a KLH-specific response, statistical analysis of the data indicated that there was a significant difference between group means in the magnitude of this response. The mean antibody and cellular response to KLH for Askeaton-based steers were significantly lower than the mean for steers based in Abbotstown. Results from the cow study were not as clear-cut.

Cows of Askeaton origin, whether located in Abbotstown or Askeaton (groups B and D respectively) differed in the magnitude of their cellular response to KLH compared to cows of Abbotstown origin (group A). However, cows of Abbotstown origin moved to Askeaton (group C) were not found to have altered cellular immune response to KLH when compared with Abbotstown cows located in Abbotstown (group A). Furthermore, the cellular response to KLH for Askeaton-derived animals at both locations was similar. Thus, it appears that the differences observed in the magnitude of the cellular response to KLH are related to origin rather than location. In relation to the measured antibody response to KLH, only Askeaton cows located in Askeaton (group D) had a significantly lower response compared to cows of Abbotstown origin (group A). The anti-KLH antibody response of Askeaton-derived cows located in Askeaton was also significantly lower compared to cows of the same origin located in Abbotstown (group B). This suggests that location might have affected the ability of cows to mount an antibody response to KLH. However, location was not found to have an effect on the anti-KLH antibody response for cows moved from Abbotstown to Askeaton (group C). Thus, there is conflicting evidence with respect to the effect of location on the KLH-specific antibody response.

Possible explanations for observed differences in KLH-specific response

Apart from exposure to cytotoxic drugs or γ-irradiation few external influences cause permanent impairment of the immune system. It is possible that the differences between Askeaton- and Abbotstown-derived cows in measures of their immune response may have resulted from a permanent negative effect on the immune system that occurred prior to commencement of the study. However, if this were true then Askeaton-derived animals moved to Abbotstown (group B) would

also be expected to have altered immune function. Measurements of the non-specific immune response for group B animals were not found to differ significantly compared to cows based in Abbotstown (group A). In relation to the specific immune response to KLH, animals moved from Askeaton to Abbotstown (group B) were only found to differ from Abbotstown-derived animals in Abbotstown (group A) with respect to their cellular response to KLH. No differences were observed in relation to the KLH-antibody response. Whether the altered response to KLH is due to differences between the groups in breed or a consequence of some event that occurred prior to the commencement of the study is unclear. It was not possible to differentiate between differences due to breed or a permanent impairment to the immune system that occurred prior to commencement of the study, within the experimental design.

As blood selenium levels were found to differ significantly between the cow and steer groups, an investigation was conducted to establish whether selenium influenced the KLH-specific immune response. Blood selenium levels recorded for the steers based in Askeaton (0.44 – 0.84 µmol/l 119 days following immunisation) are considered to be marginal whereas levels recorded for Abbotstown steers (2.3 – 3.3 µmol/l 132 days following immunisation) are at the higher end of the normal range described (EPA, 1996). In assessing immune status of cattle on a marginally deficient selenium diet a measure of their immune response to KLH was compared with cattle on the same pastures given selenium supplementation. The results from this study indicate that blood selenium levels in the range, 0.25 – 0.57 µmol/l did not affect cell-mediated or humoral immunity to KLH when compared with animals whose blood selenium levels were in the range 1.11 – 2.12 µmol/l. Thus marginal blood selenium status does not appear to affect the immune response of cattle to the antigen KLH when compared to cattle with normal blood selenium levels. It was not established whether blood selenium levels in the range 2.3 - 3.4 µmol/l, reported for the VRL steers, had an effect on the specific immune response to KLH. It is possible that the higher levels of blood selenium reported for the VRL steers may have affected their response to KLH. However, it is worth noting that the cell-mediated KLH-specific response for Abbotstown cows in Abbotstown (reference group A) was of the same magnitude as the VRL steers and both of these groups had similar blood selenium levels (1.7 - 3.9 µmol/l and 2.9 - 4.6 µmol/l respectively). Furthermore, it has been reported that the increased blood selenium levels in cows receiving selenium supplementation over a 90 day period had

no effect on humoral immune responses (Ellis et al., 1997).

Another possible reason for the altered immune response to KLH recorded for the Askeaton-based steers may be due to an effect of transport. Blood samples taken from the Askeaton-based steers were subjected to transport for 3 hours by train before assays were conducted. Samples from the VRL herd were not subjected to the same conditions. A study was conducted whereby the effect of train transport was simulated using agitation. This study found that simulated agitation had a significant effect on KLH induced proliferative response using paired samples in one experiment while no significant effect was noted in a repeat experiment. Similar conflicting results were noted for the mitogen responses following agitation. However, the results of the cow study did not indicate that transport had a significant effect on the KLH response. Blood samples from Abbotstown-derived cows in Askeaton had similar responses in the KLH proliferation assays to Abbotstown-derived cows based in Abbotstown. Likewise, blood samples taken from Askeaton-derived cows at both locations had similar responses to KLH.

Conclusion

The immune system is required for both protection from infection and for clearance of infectious agents once infection has occurred. Any interference in immune function would be expected to increase the probability of infectious agents gaining a foothold in the host. Evidence that Askeaton-based steers had a weaker KLH-specific immune response is a significant finding. It is possible that these animals might be more susceptible to infections. However, the clinical data recorded for the steers (see Chapter Two) did not indicate that these animals suffered from chronic or recurring infections. In spite of the lower KLH-specific immune response, no direct correlation with increased prevalence of disease was reported. Indeed, all steers were considered to be in a healthy condition when measured in terms of production performance (see Chapter Two). This indicates that these animals did not suffer from immunosuppression.

In conclusion, there was no evidence of immunodeficiency from measurements taken to assess non-specific immunity. There was variability in the parameters measured but no consistent pattern emerged suggesting a progressive decline in immune function. Askeaton-based cows and steers did differ from control

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animals in their specific immune response to the KLH antigen. However, the animals showed no clinical evidence of altered immune competence. For the cow study these differences may have been influenced by differences in breed, age and pregnancy. The reasons for the differences in KLH responses in the steer study could not be determined. Although the results of a supplementary experiment showed no evidence of an effect of marginal selenium blood status on KLH-specific responses, the possibility that the high-normal selenium status of the Abbotstown steers had an enhancing effect on responses cannot be ruled out. The results of an experiment to investigate the effects of sample transport on the KLH tests were inconclusive.

Tables

Table 7-1. Mean percentage values of positively stained B lymphocytes and the T lymphocyte subsets, CD2, CD4, CD8 and TCR1 in peripheral blood[1].

Grp[2]	Date[3]	B lymphocytes	T lymphocyte subsets CD2	CD4	CD8	TCR1
A	7/10/96	11.28 ± 1.45 (11)	62.3 ± 4.23 (11)	36.05 ± 1.87 (11)	26.54 ± 1.47 (11)	14.68 ± 0.93 (11)
	9/12/96	16.88 ± 1.34 (11)	72.20 ± 1.99 (11)	35.73 ± 2.58 (11)	25.41 ± 1.78 (11)	nd[4]
	20/01/97	18.47 ± 1.21 (11)	63.71 ± 2.23 (11)	36.75 ± 1.62 (11)	24.31 ± 1.04 (11)	11.30 ± 1.21 (11)
	14/04/97	24.55 ± 1.97 (11)	64.14 ± 2.09 (11)	35.58 ± 1.89 (11)	33.43 ± 2.17 (11)	16.33 ± 1.49 (11)
	21/07/97	21.44 ± 2.13 (8)	69.74 ± 3.42 (11)	33.11 ± 2.04 (11)	33.43 ± 2.17 (11)	16.32 ± 2.58 (11)
	6/10/97	31.23 ± 2.74 (8)	58.69 ± 3.53 (8)	27.46 ± 0.73 (8)	29.46 ± 2.19 (8)	16.72 ± 1.25 (8)
	12/01/98	21.45 ± 2.84 (8)	58.53 ± 4.13 (8)	24.80 ± 1.37 (8)	27.64 ± 3.16 (8)	11.22 ± 1.55 (8)
B	7/10/96	11.39 ± 1.31 (11)	60.20 ± 3.00 (11)	37.29 ± 1.93 (11)	25.70 ± 2.40 (11)	13.04 ± 1.15 (11)
	9/12/96	16.53 ± 2.33 (10)	69.57 ± 2.67 (10)	33.43 ± 1.86 (10)	22.77 ± 2.25 (10)	nd
	20/01/97	17.00 ± 2.20 (11)	65.18 ± 1.99 (11)	35.03 ± 1.97 (11)	19.17 ± 1.10 (11)	13.89 ± 1.68 (11)
	14/04/97	22.77 ± 2.93 (10)	60.57 ± 3.02 (10)	31.78 ± 1.75 (10)	26.10 ± 3.09 (10)	15.83 ± 1.53 (10)
	21/07/97	25.32 ± 2.72 (10)	62.14 ± 3.80 (10)	34.24 ± 1.30 (10)	26.10 ± 3.09 (10)	17.43 ± 1.03 (10)
	6/10/97	22.36 ± 3.58 (10)	58.43 ± 3.23 (10)	27.59 ± 1.34 (10)	29.87 ± 2.73 (10)	16.20 ± 1.33 (10)
	12/01/98	17.15 ± 4.00 (10)	68.48 ± 5.90 (10)	28.2 ± 2.51 (10)	32.63 ± 4.17 (10)	17.03 ± 2.56 (10)
C	14/10/96	27.96 ± 1.50 (11)	55.14 ± 2.30 (11)	31.95 ± 2.55 (11)	22.68 ± 2.44 (11)	10.33 ± 1.42 (11)
	5/12/96	nd	60.83 ± 2.00 (11)	21.64 ± 2.98 (11)	25.96 ± 2.01 (11)	nd
	7/04/97	27.57 ± 1.42 (11)	61.03 ± 1.37 (11)	32.25 ± 2.07 (11)	25.65 ± 1.78 (11)	9.87 ± 1.70 (11)
	14/07/97	23.00 ± 1.91 (10)	66.66 ± 2.99 (10)	36.43 ± 1.70 (10)	36.23 ± 2.09 (10)	20.00 ± 1.78 (10)
	10/11/97	32.08 ± 2.27 (10)	53.80 ± 2.96 (10)	24.16 ± 1.49 (10)	31.20 ± 2.74 (10)	12.64 ± 1.27 (10)
	8/12/97	31.79 ± 4.02 (10)	54.15 ± 3.79 (10)	23.54 ± 1.53 (10)	29.17 ± 2.72 (10)	12.92 ± 1.92 (10)
D	14/10/96	19.93 ± 2.85 (10)	56.44 ± 1.90 (10)	36.94 ± 1.12 (10)	28.64 ± 1.47 (10)	14.14 ± 1.55 (10)
	5/12/96	nd	66.08 ± 2.37 (10)	27.21 ± 1.86 (10)	25.82 ± 1.52 (10)	nd
	7/04/97	11.95 ± 2.68 (10)	69.56 ± 2.85 (10)	33.67 ± 2.29 (10)	29.87 ± 3.66 (10)	14.04 ± 1.99 (10)
	14/07/97	9.46 ± 1.04 (10)	76.50 ± 1.99 (10)	36.42 ± 1.43 (10)	35.57 ± 2.58 (9)	20.02 ± 2.08 (10)
	10/11/97	18.13 ± 2.83 (9)	68.67 ± 4.27 (9)	29.80 ± 1.67 (9)	36.94 ± 2.35 (9)	16.72 ± 1.64 (9)
	8/12/97	nd	69.10 ± 4.00 (9)	33.35 ± 2.65 (9)	25.70 ± 3.60 (9)	11.25 ± 1.35 (9)

[1] Values represent the mean percentage positive ±standard error of mean. Number animals per group in brackets.
[2] A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton.
[3] Results are given for each group at the date of blood collection.
[4] Not done

Table 7-2. Mean ± SE of combined observations relating to percentages of lymphocyte populations for cows grouped according to their origin and location.

	B lymphocytes	T lymphocyte subsets			
		CD2	CD4	CD8	TCR1
ORIGIN					
Abbotstown (A & C)	23.81 ± 0.84	62.26 ± 0.92	31.61 ± 0.72	27.64 ± 0.65	13.89 ± 0.56
Askeaton (B & D)	17.47 ± 0.93	65.47 ± 1.01	32.68 ± 0.58	28.04 ± 0.83	15.82 ± 0.54
LOCATION					
Abbotstown (A & B)	20.08 ± 0.78	63.97 ± 0.90	33.03 ± 0.58	26.02 ± 0.65	15.08 ± 0.46
Askeaton (C & D)	21.90 ± 1.17	63.72 ± 1.09	31.02 ± 0.73	29.99 ± 0.81	14.57 ± 0.67

Table 7-3. Mean percentage values of positively stained B lymphocytes and the T lymphocyte subsets, CD2, CD4, CD8 and $\gamma\delta$TCR1 – steer study.[1]

Date[2]	Group[3]	B Lymphocytes	CD2	T lymphocyte subsets CD4	CD8	TCR1	n
4/11/96	V	16.11 ± 1.54	56.63 ± 1.78	26.33 ± 1.22	16.02 ± 1.05	22.84 ± 2.85	12
11/11/96	R	13.32 ± 1.78	52.80 ± 0.03	23.72 ± 1.78	18.77 ± 1.18	23.52 ± 2.98	12
17/02/97	V	18.50 ± 2.77	57.17 ± 2.25	25.81 ± 1.23	17.10 ± 1.06	24.29 ± 2.24	12
24/02/97	R	18.20 ± 2.35	50.85 ± 3.14	17.64 ± 2.04	21.79 ± 2.51	25.55 ± 2.25	12
19/05/97	V	30.25 ± 2.76	48.13 ± 1.57	27.30 ± 0.89	20.44 ± 1.44	30.29 ± 2.47	12
26/05/97	R	31.44 ± 1.06	49.30 ± 2.72	23.06 ± 1.66	20.60 ± 1.84	24.58 ± 2.00	12
30/06/97	V	39.54 ± 2.42	41.45 ± 1.43	22.99 ± 0.80	20.37 ± 1.48	23.30 ± 2.51	12
7/07/97	R	22.03 ± 2.03	55.96 ± 1.70	24.86 ± 1.37	27.74 ± 1.87	25.52 ± 2.28	12
28/07/97	V	20.64 ± 2.39	42.56 ± 1.95	19.18 ± 1.06	21.23 ± 1.69	34.66 ± 3.31	12
18/08/97	R	13.36 ± 1.45	58.76 ± 3.43	26.47 ± 1.87	26.06 ± 1.61	28.66 ± 2.69	12
25/08/97	V	24.12 ± 1.93	44.57 ± 1.99	26.32 ± 0.79	22.94 ± 1.51	20.22 ± 1.82	12
22/09/97	R	23.30 ± 1.87	51.89 ± 3.17	24.42 ± 1.42	23.52 ± 2.27	23.89 ± 2.43	11
29/09/97	V	32.34 ± 1.96	52.10 ± 1.14	27.12 ± 1.18	21.57 ± 1.22	23.57 ± 1.38	12
28/10/97	R	35.97 ± 3.01	40.76 ± 3.61	22.99 ± 1.59	21.32 ± 1.97	nd	11
26/01/98	V	35.28 ± 2.10	42.06 ± 2.15	18.40 ± 0.98	21.25 ± 1.25	nd	12
2/02/98	R	21.76 ± 2.10	56.22 ± 2.28	26.63 ± 2.09	26.18 ± 2.73	21.41 ± 1.71	11

[1] Results are mean percentage positive ± standard error of the mean (n).
[2] Results are given for each group at the date of blood collection.
[3] V – steer group at Abbotstown, R steer group at Askeaton (Index Farm B).

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Total Serum IgG

Total Serum IgM
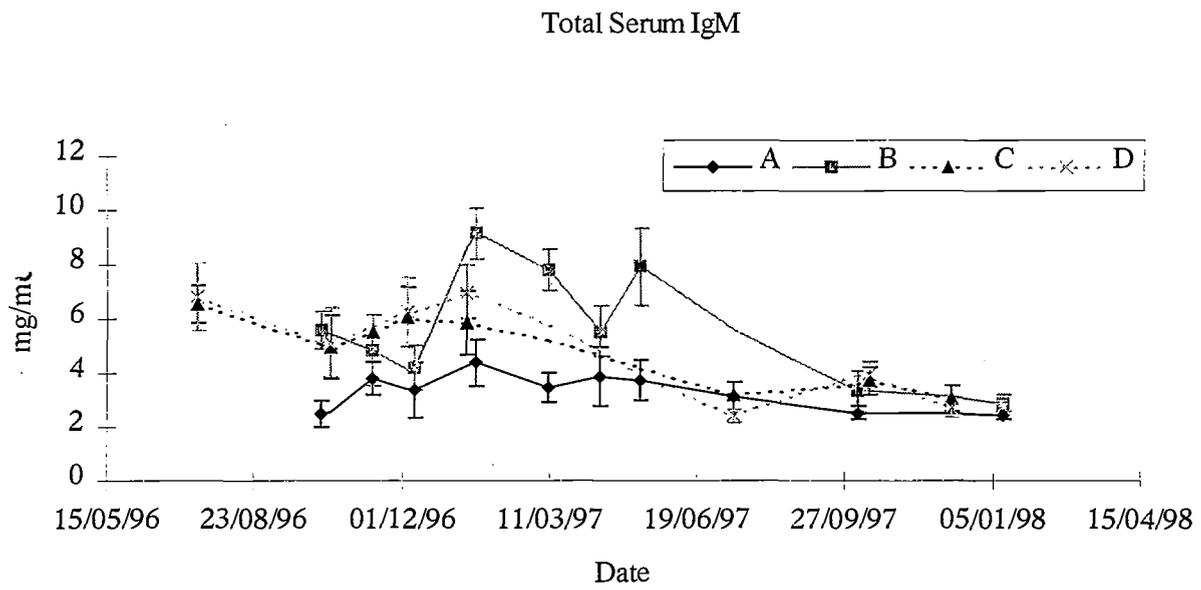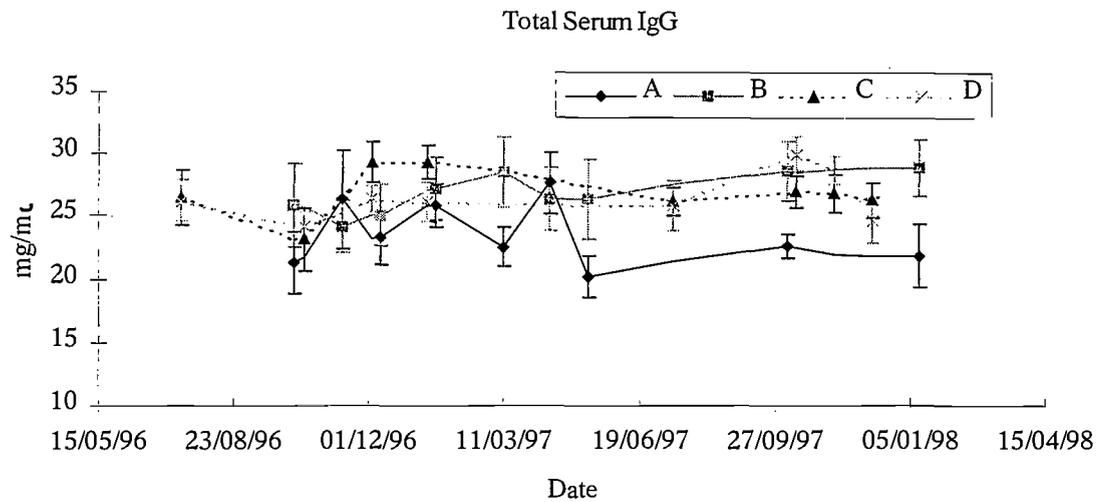
Figure 7.1 The mean of serum IgG and IgM for each of the groups of cows. The different groupings are: A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton
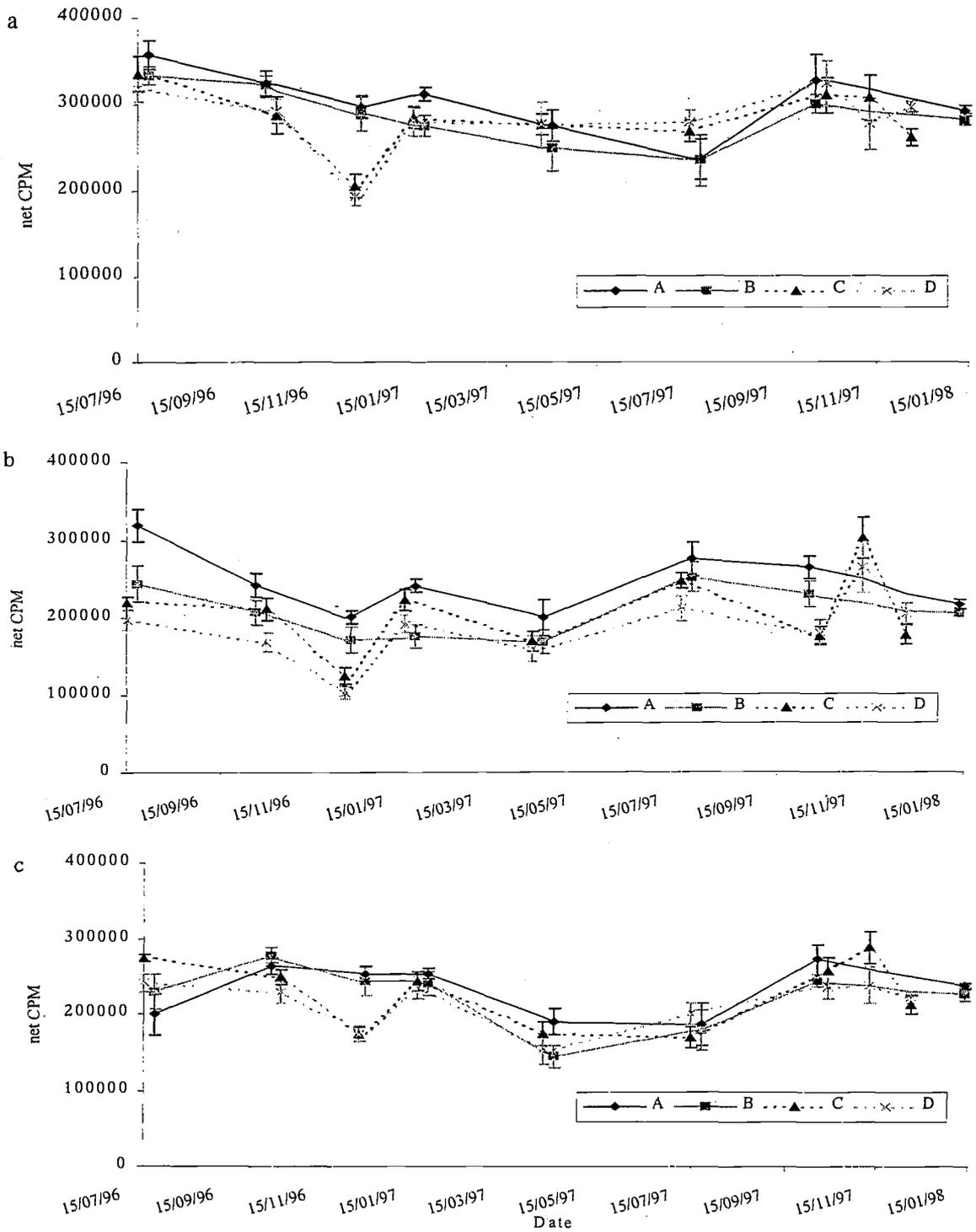
Figure 7.2. Mean proliferative response of peripheral blood lymphocytes to stimulation with the mitogens, Con A (a), PWM (b) and PHA (c) for the cow groups. The different groupings are: A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton. Results are presented as the group mean of ^{3}H-thymidine incorporation.

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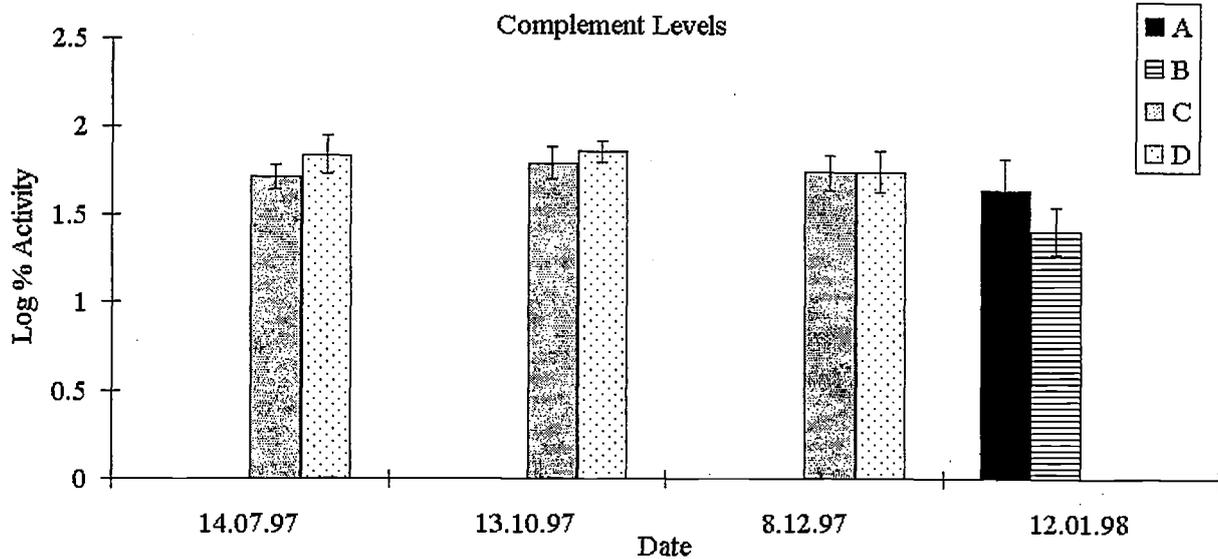
Figure 7.3. Mean serum complement activity for each of the four groups of cows, collected at various dates. The % activity was measured with reference to a human serum sample standard. The different groupings are: A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton.
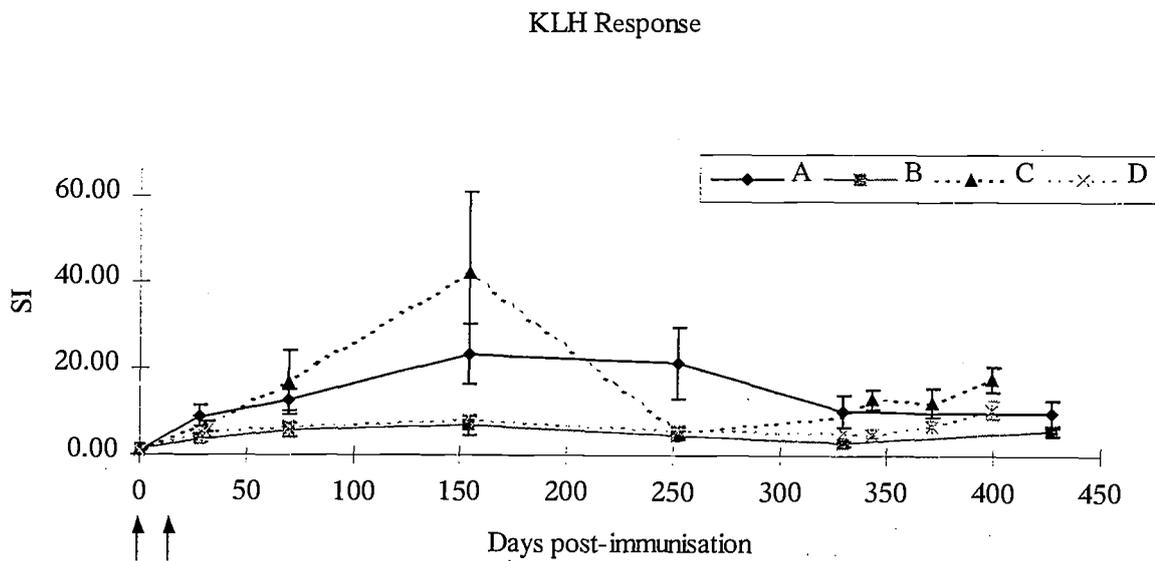
KLH Response

Figure 7.4. The in vitro lymphocyte proliferative response to KLH for cow groups. Assays were conducted on blood prior to immunisation and at intervals thereafter. The proliferative response is presented as the mean stimulation index (SI) for each group. Arrows indicate time of first and second immunisation. The different groupings are: A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton
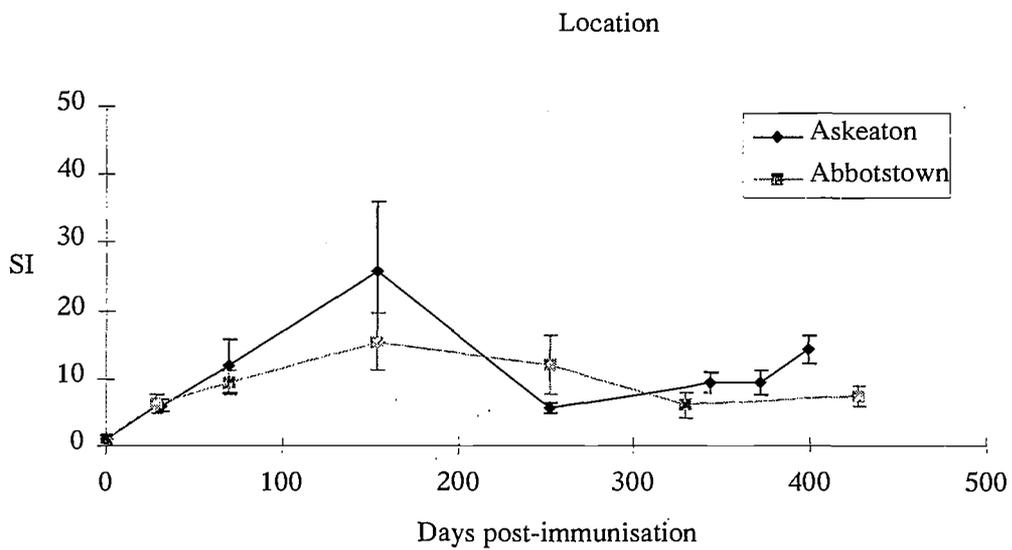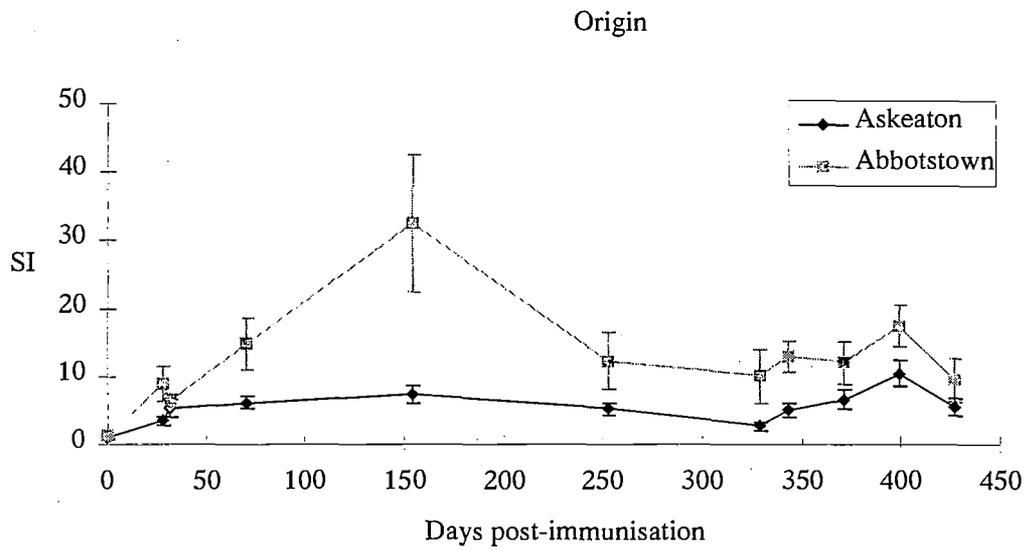
Figure 7.5. The mean proliferative response to KLH for animals grouped according to their origin and location. SI = stimulation index (*see* text).

Interferon Production
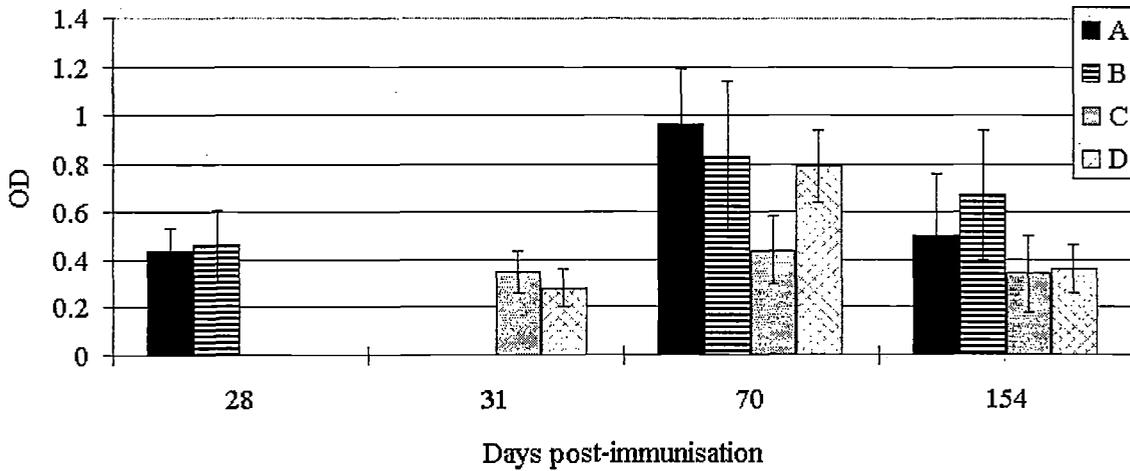
Figure 7.6. γIFN production following in vitro stimulation with KLH at days post-immunisation. Results are presented as the mean for each group. The different groupings are: A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton. OD = optical density.

Anti-KLH IgG
Response
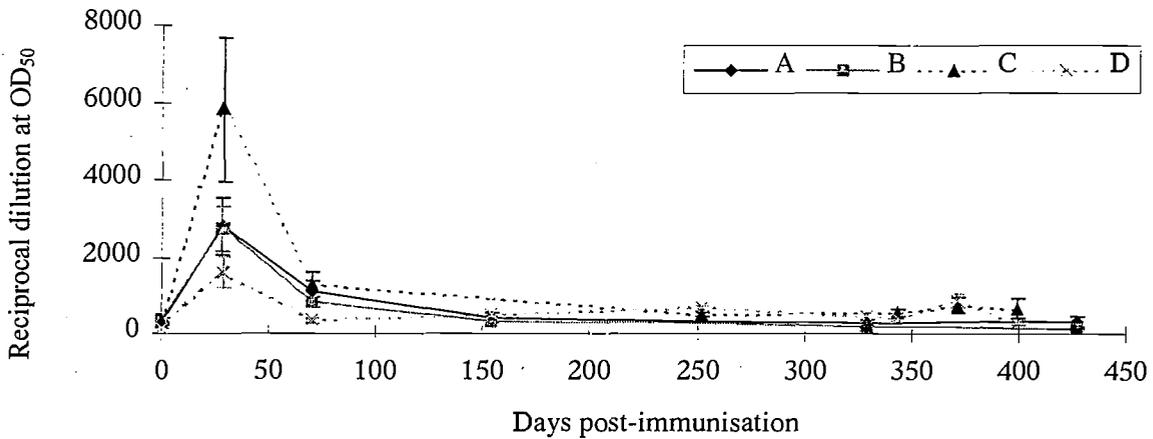
Figure 7.7. The mean anti-KLH IgG response for each group following immunisation. Values are presented as the reciprocal of the titration at half maximum OD. The different groupings are: A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton. OD = optical density.

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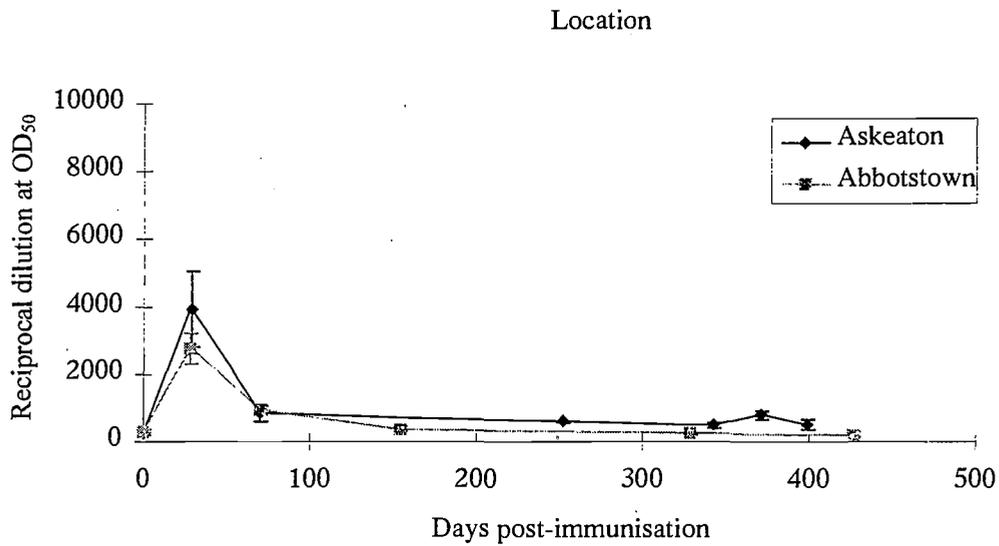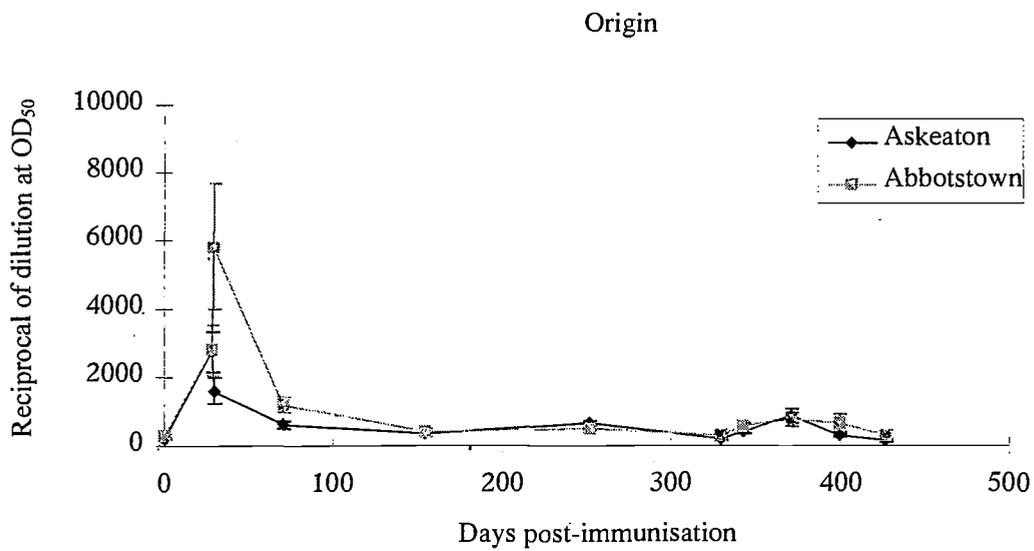
Origin

Location

Figure 7. 8. The mean antibody levels to KLH for groups of animals based on origin and location.

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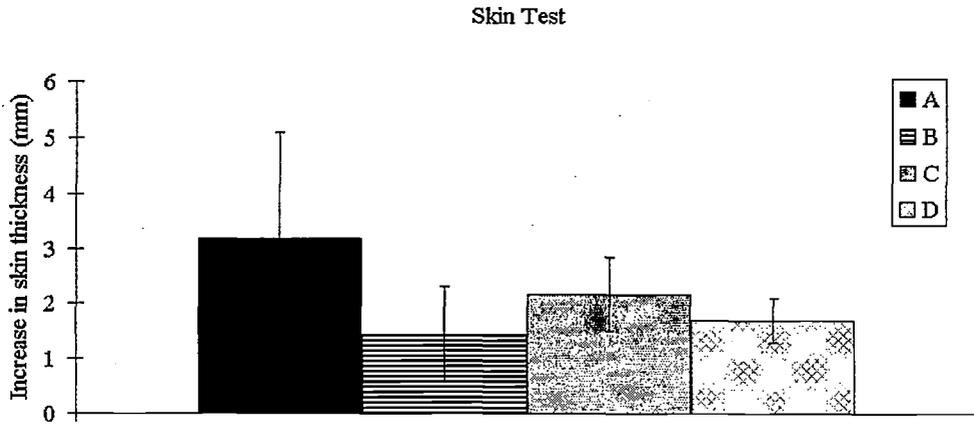
Figure 7.9. Mean increase in skin thickness for each group measured 72 hours after intradermal injections of KLH. The different groupings are: A - VRL cows located at Abbotstown, B - Askeaton cows located at Abbotstown, C - VRL cows located at Askeaton, D - Askeaton cows located at Askeaton
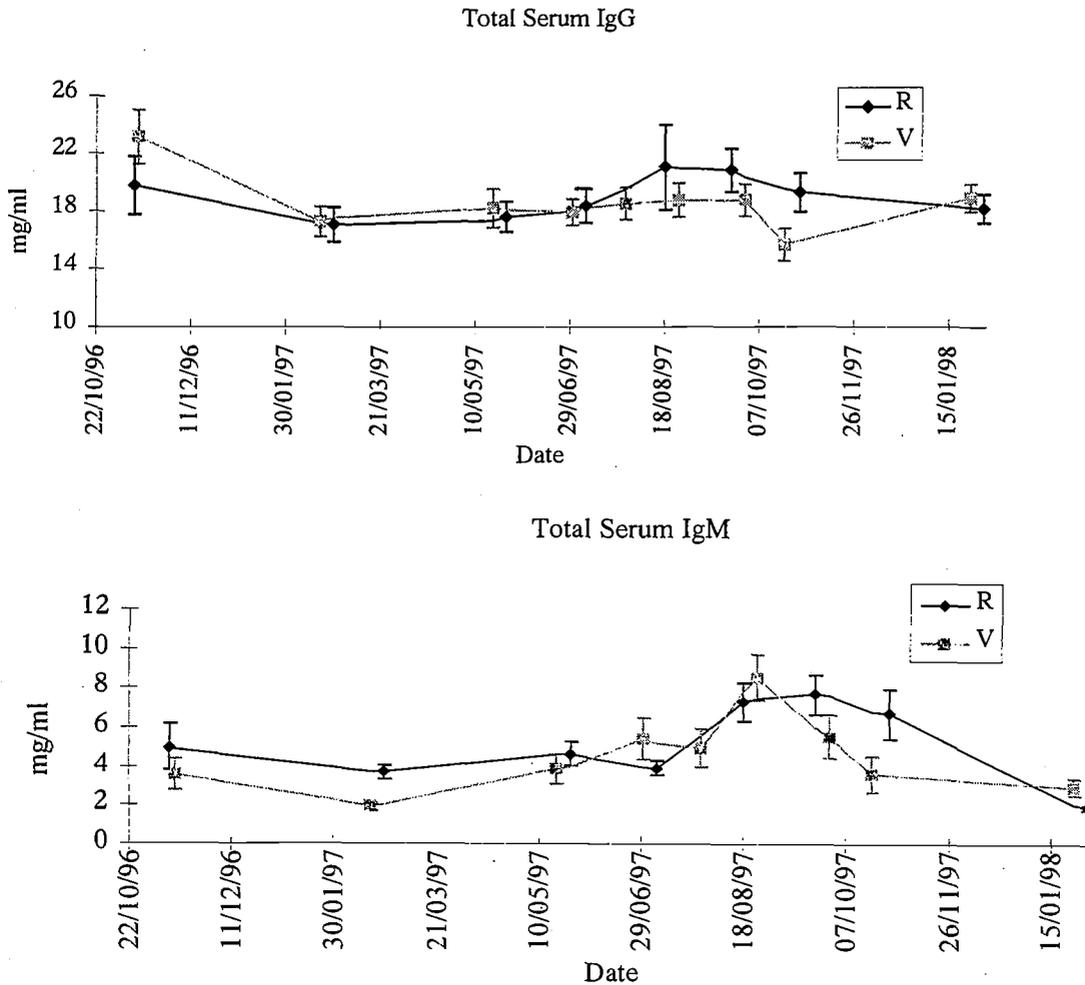
Figure 7.10. The mean of the total serum IgG and IgM concentration for the groups of steers on the VRL (V) and index farm B, Askeaton (R).
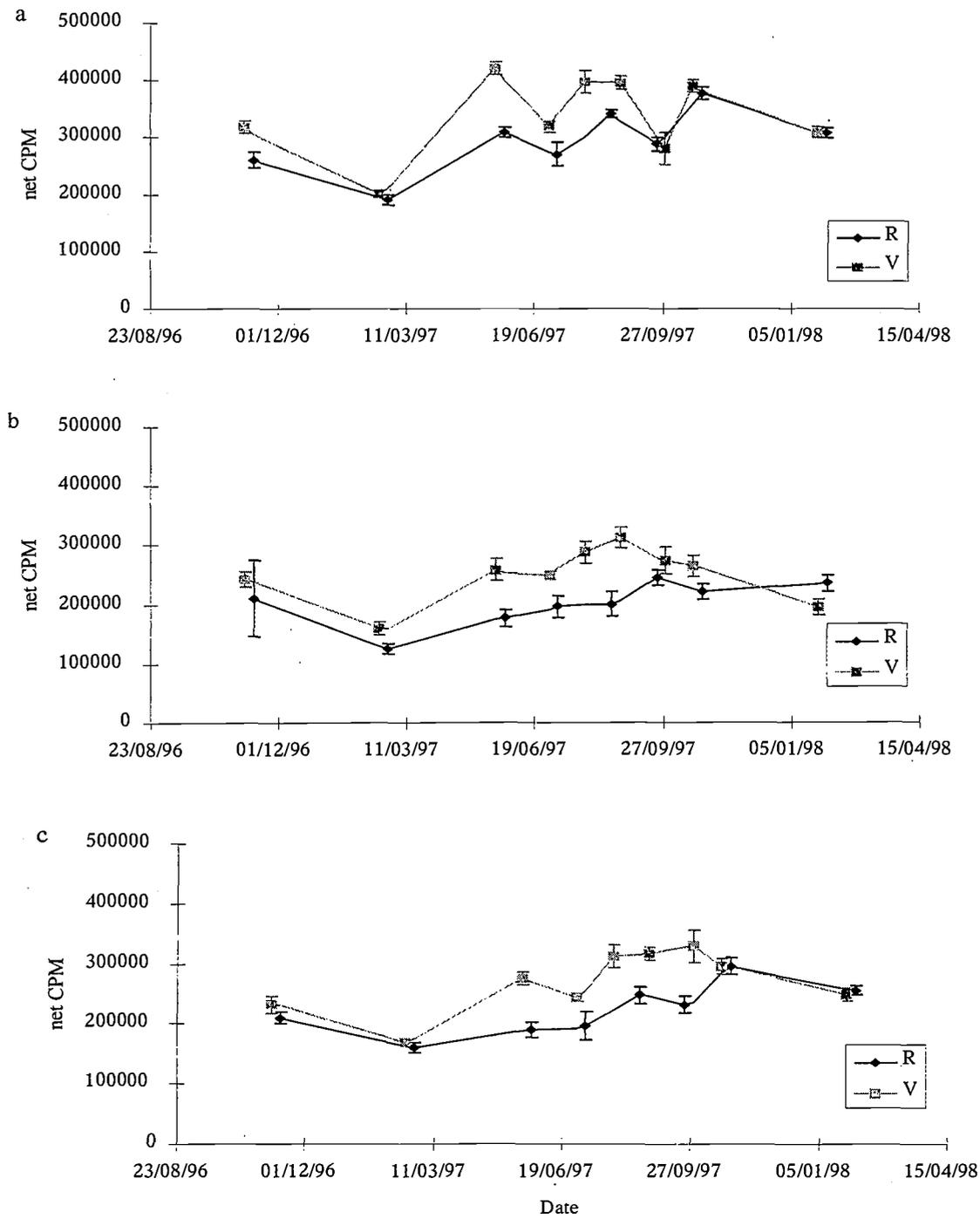
Figure 7.11. Mean proliferative response of peripheral blood lymphocytes to stimulation with the mitogens; Con A (a), PWM (b) and PHA (c) of steers in the study. The different groupings are: V - steers located on the VRL farm at Abbotstown, and R - steers located on index farm B, Askeaton. Results are expressed as the mean of the net counts per minute (CPM) of ^{3}H-thymidine incorporation

Complement Levels
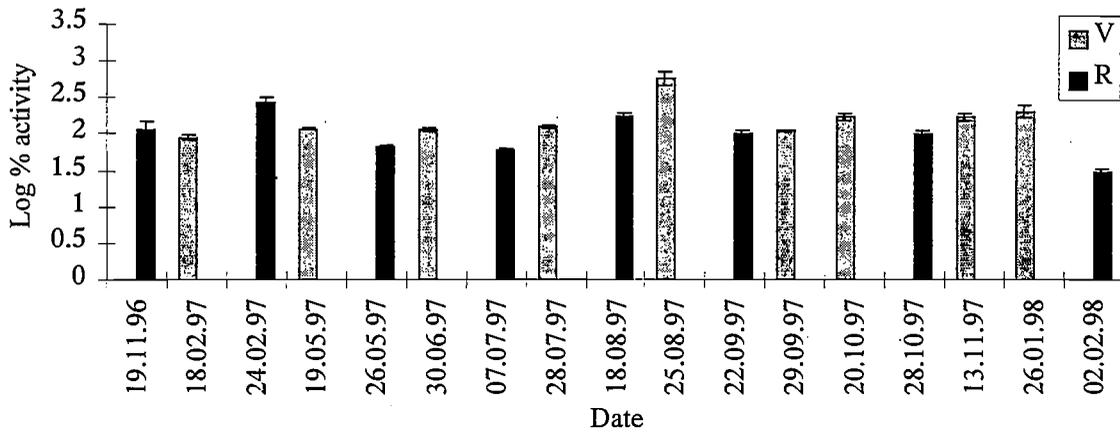
Figure12. Complement levels are presented as the log of the percentage activity of reference human serum for steers located on the VRL (V) and Askeaton (R) farms. Results are presented as the mean for the groups over time

KLH Response
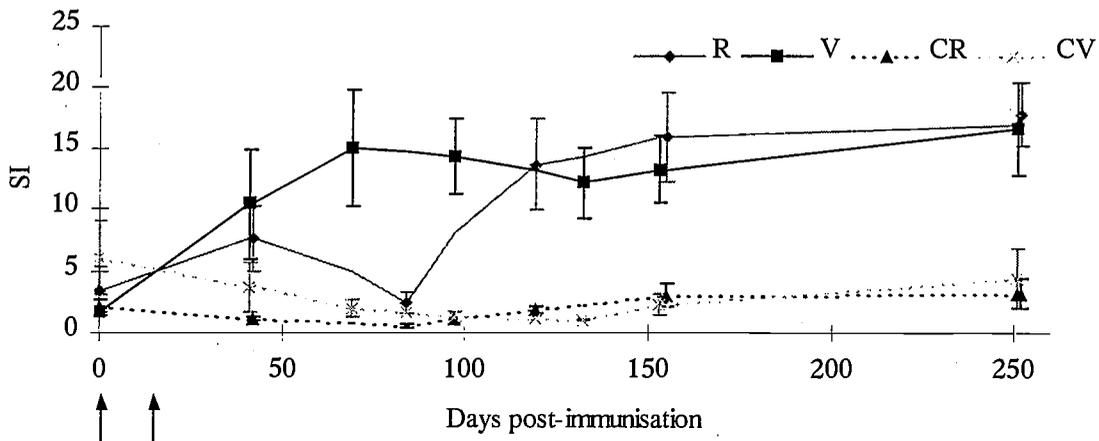
Figure 7.13. The mean in vitro lymphocyte proliferative response to KLH for steers on the VRL (V) and Askeaton (R) farms and the mean response for control animals on the VRL (CV) and Askeaton (CR) farm. Assays were conducted on blood prior to immunisation with KLH and at intervals thereafter. The proliferative response is presented as the mean stimulation index (SI) for each of the groups over time. Arrows indicate time of first and second immunisation.

Anti-KLH IgG Response
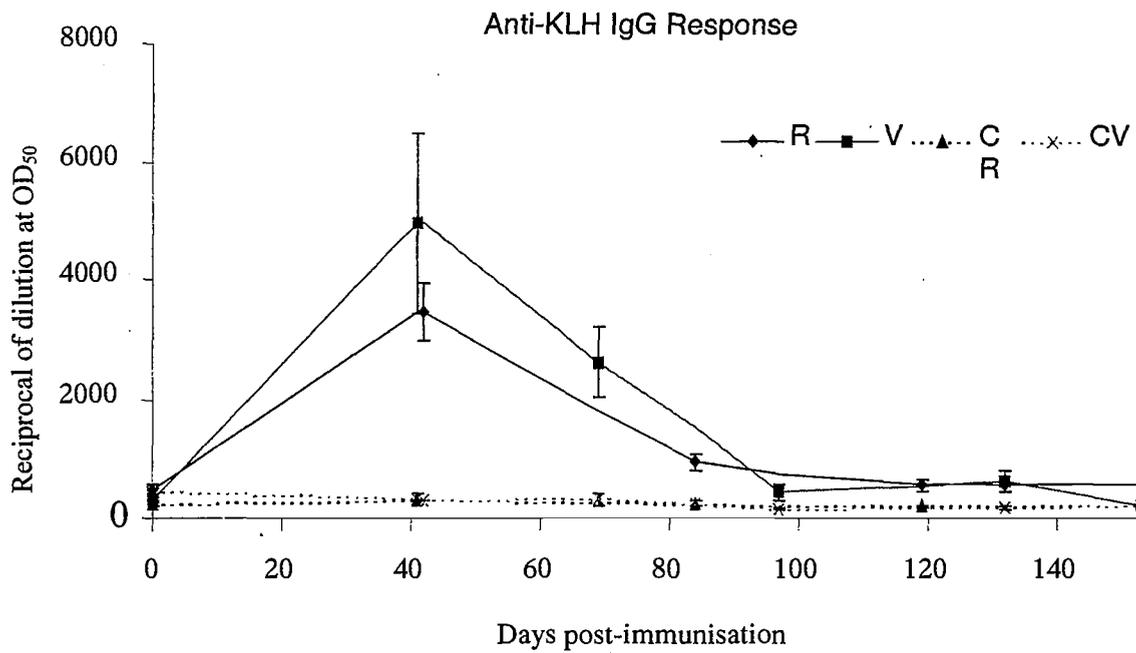
Figure 7.14. The anti-KLH IgG response for steers on the VRL (V) and Askeaton (R) farms. The mean response for control animals on the VRL (CV) and Askeaton farm (C) are also included. Assays were conducted on blood prior to immunisation with KLH and at intervals thereafter. The anti-KLH response is presented as the mean for the groups over time. Arrows indicate time of first and second immunisation.
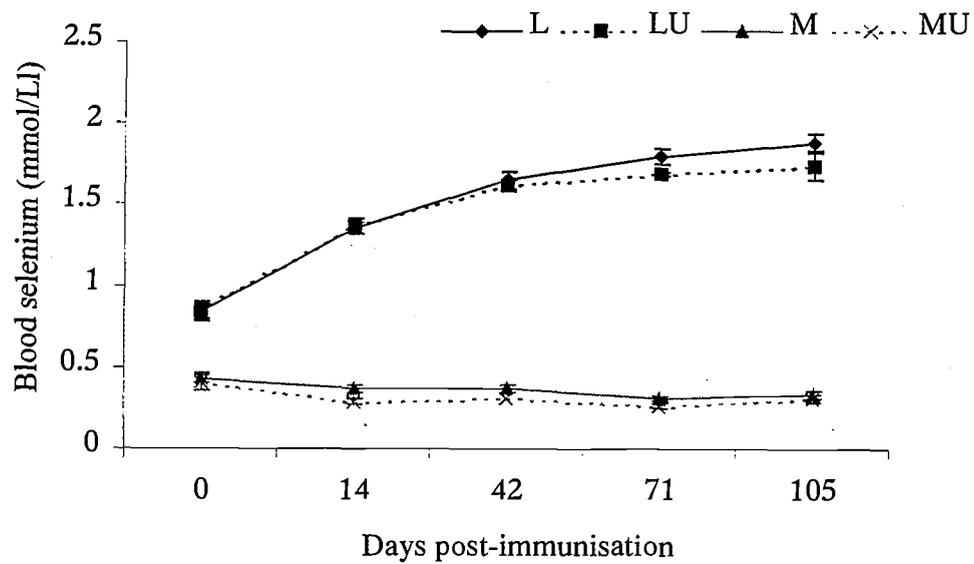
Figure 7.15. The mean blood selenium levels for each of the four groups prior to immunisation and at intervals thereafter. *See* table on page 184 for description of group codes (L, LU, M, MU).

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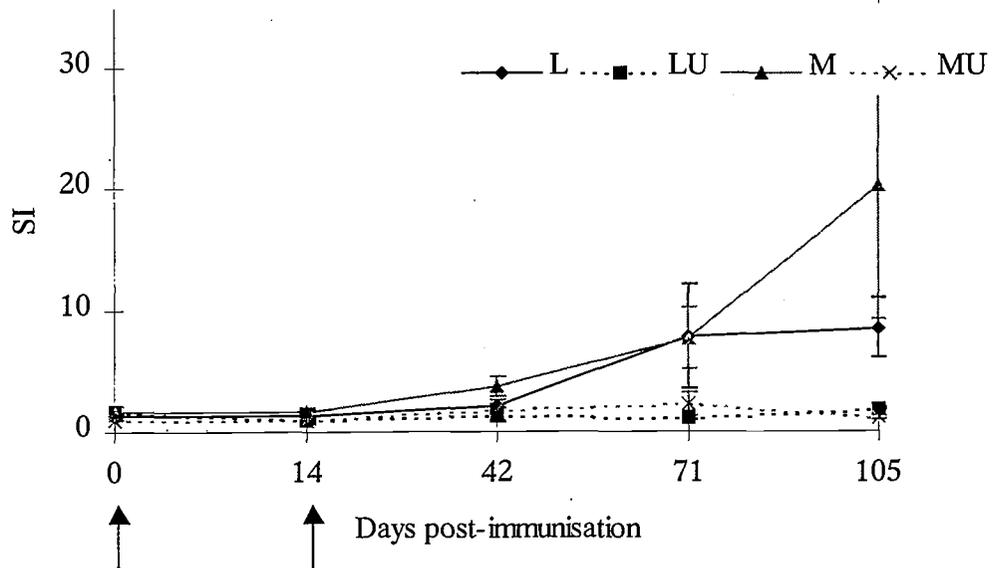
Figure 7.16 The mean lymphocyte proliferative response to KLH for each of the four groups. Arrows indicate time of first and second immunisation. *See* table on page 184 for description of group codes (L, LU, M, MU).
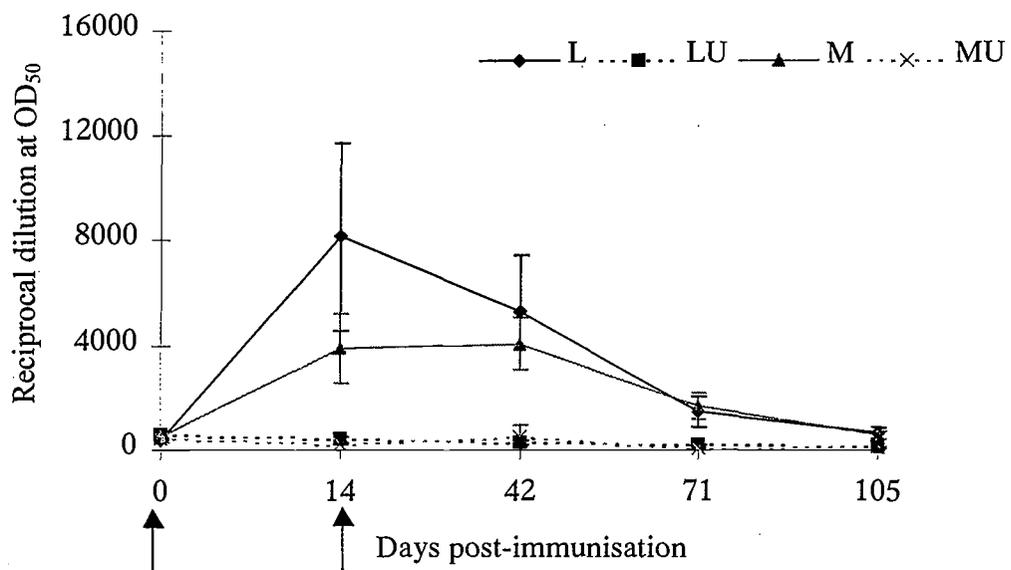
Fig 7.17. The mean antibody response to KLH for each of the four groups. Arrows indicate time of first and second immunisation. *See* table on page 184 for description of group codes (L, LU, M, MU).

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Immunology Appendix A

IMMUNE PARAMETERS EVALUATED

QUANTITATIVE STUDIES

Total white blood cell counts: Total white blood cell counts were carried out at the Veterinary Research Laboratories, Abbotstown and are the subject of a separate report.

Lymphocyte enumeration: The purpose of lymphocyte enumeration is to determine if animals have a full repertoire of defined lymphocytes. Absence of a particular cell population may provide a possible explanation for observed immunological defects. Lymphocyte populations are defined according to the expression of surface markers. Monoclonal antibodies have been developed that identify these markers on bovine lymphocytes. The more commonly studied bovine lymphocyte subsets identified by these monoclonal antibodies are B-lymphocytes and the T-lymphocyte subsets, designated CD2, CD4, CD8 and γδ TCR1. Using fluorescent labelled secondary reagents; lymphocyte profiles in blood were assessed by flow cytometry. Relative proportions of the lymphocyte subsets were monitored at intervals of three months.

FUNCTIONAL STUDIES

Complement: Complement is the name given to a group of proteins. Complement has an important non-specific role in defence through lysis of antibody-coated foreign cells, including bacteria. The ability of individual serum samples at lysing antibody-coated guinea pig red blood cells was used to measure bovine complement activity.

Neutrophil Assays: Tests for neutrophil function assess the ability of neutrophils to ingest bacteria. It is also possible to assay the oxidative burst activity following ingestion of bacteria. These assays are difficult to conduct, as neutrophils are relatively short-lived cells. The logistical problems of getting blood samples to Dublin for analysis meant that samples were up to 7 hours in transit before assays could be performed. Assays were conducted on steer samples only. Pregnancy is known to have an immunosuppresive effect on neutrophil function and it was decided not to conduct neutrophil function assays on the cow samples.

Serum Immunoglobulin measurements: Tests of B lymphocyte competence rely on detecting the products of B lymphocytes, the immunoglobulins. Two classes of immunoglobulins, IgG and IgM were measured. These represent the predominant immunoglobulins present in serum.

Mitogenic Response: The purpose of these assays was to determine the functional capacity of peripheral blood lymphocytes (T and B). The assays measure the ability of lymphocytes to proliferate *in vitro* in response to a stimulant. The stimulants used are referred to as mitogens and include concanavalin A (Con A), pokeweed mitogen (PWM) and phytohaemaglutinin (PHA). Each of these mitogens induces cell division. Depending on the type of mitogen used either T lymphocytes, B lymphocytes or both may be induced to proliferate. The assay measures the number of cells proliferating and surviving in culture following stimulation. Proliferation is measured indirectly by measuring radioactive nucleotide incorporation into cellular DNA.

SPECIFIC IMMUNE RESPONSE

An important aspect of the immune system is the ability of lymphocytes to specifically recognise foreign material. Animals were immunised with an antigen and the specific response to antigen measured. Haemocyanin derived from the keyhole limpet (KLH) was chosen as an innocuous antigen for immunisation. The following assays were conducted to measure the KLH-specific response.

Proliferation Assay: Once primed *in vivo* by immunisation, KLH-specific T lymphocytes are produced. In this assay, the ability of KLH-specific lymphocytes to proliferate on restimulation *in vitro* was measured.

Interferon-γ (γIFN) assay: Stimulation of lymphocytes with specific antigen results in increased production of certain cytokines. γIFN is one such cytokine. Levels of γIFN produced *in vitro* in the presence and absence of KLH was measured post-immunisation.

Antibody response: Immunisation also primes B lymphocytes to produce antibodies specific for antigen. Thus the antibody response to KLH was measured in animals.

Skin test: This is an *in vivo* method for assessing the animal's ability to mount a specific immune response to KLH. When KLH in saline is given intradermally to immunised animals a local reaction ensues leading to an

increase in skin thickness. The skin thickness at the site of reaction was measured with reference to a site where saline alone was injected.

Immunology Appendix B

ANIMALS AND THEIR GROUP ASSIGNMENTS

Cows

		Group	
A	B	C	D
55	201	62	301
57	204	63	302
68	205	81	303
71	207	84	304
74	208	87	306
83	211	89	307
85	212	91	308
92	213	96	310
95	214	97	312
98	313	101	314
99		103	

Steers

Group	
VRL (V)	Askeaton (R)
BMNL0003A	10THHRR
CZHY0005J	7GQHGY
WFCX0019U	15NTFOZ
HYHG0004N	17CSSVM
JMND0004W	17HHFWR
LGKY0001W	2LLVCH
MJXJ0003F	3SXBMS
PSA239492	5BMJXJ
PSA731055	731146PSA
TLA684732	18JGYKW
DJWB0003D	12PHYHG
YGPS0005M	612288PSA

CHAPTER EIGHT

Rat Feeding Trial

P. Nowlan - Bio-Resources Unit, Trinity College
Dublin

A laboratory-rat feeding trial was undertaken to
investigate the possibility that soil from the
Askeaton area might contain quantities of pollutant
which would have a significant negative impact on
rat health when compared to the effects of feeding
soil from a remote control site.

The trial, which involved supplementing the rats'
diet with Askeaton and non-Askeaton soils, was
intended to determine if there was any evidence
that soil from the Askeaton farm (Index Farm A)
had a negative effect on the growth or reproductive
performance of supplemented rats.

As a supplementary study to determine the effects
of the soil-feeding on the activities of the liver
enzymes phenylalanine hydroxylase and
glutathione peroxidase (see next section – vole
study), livers from some rat dams and new-born
male rats were analysed for these enzymes.

PURPOSE

The Bio Resources Unit in Trinity College Dublin
was asked by the Department of Agriculture to
undertake a trial in which feed mixed with soil
would be fed to rats. The soil was to come from a
farm in the Askeaton area of Co. Limerick where
there was a high incidence of cattle deaths and ill
health. Soil from another location was to be used as
a control. A third group of rats would be fed
standard laboratory diet without the addition of
soil.

HYPOTHESIS

The null hypothesis is that 'soil from the test farm
would be no more toxic then soil from the control
farm.' The null hypothesis would not be rejected
if health and performance in the rat group fed
Askeaton-origin soil was not significantly lower
than in the group fed soil from the control farm.

Two Trials would be performed:

- Trial I would examine the effects on the
  reproductive performance of rats.
- Trial II would examine the effects on the
  growth of rats born to mothers on the diet and
  maintained on the diet for 3 months.

PREPARATORY STUDIES

Thirty kg of soil were collected from the top 10
mm layers of soil from various sites on Index Farm
A at Askeaton. A similar quantity was collected
from a control site (Johnstown Castle farm). These
soils were added to a standard laboratory rat diet at
a rate of 15 per cent. Difficulties in milling soils
fine enough to incorporate evenly into a feed nut
delayed the start of the trials.

A pilot trial was performed to determine the effect
of feeding normal control soil to rats. This trial
which was controlled against normal rat diet
without soil showed no difference in weight gain
but showed that animals consumed more of the test
diet than the control. The trial duration was 24
days.

TRIAL I

Three groups of 16 female virgin wistar rats were
housed in a conventional animal unit. The unit
complied with all the legal requirements of
Statutory Instrument SI 17/94. The animals were
supplied from the stock (wistar) of the Bio
Resources Unit. Groups 1 and 2 were fed with a
diet containing 15% soil on a dry matter basis. The
third group was fed normal rat diet (Redmills). All
diets were made available ad lib. Water was also
available ad lib. The animals were bedded on peat
moss.

The females were fed the diets for 8 weeks. A male
was then introduced into their box for 10 days.
Food and water intake was measured daily and
animals weighed weekly. All animals were
observed daily for any abnormalities. The females
were removed to individual cages and again food
and water intake was measured daily and animals
weighed weekly.

All these figures were recorded on charts. When
litters were born, the total number in the litter was
recorded and the a daily check made of the litter till
day 21 on which day they were weaned. At
weaning the number of animals in the litter the
number and weights of females and males was
measured.

The young were euthanased with CO_2 and a post-
mortem examination performed on each pup. The

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livers were weighed and samples taken from 60 randomly chosen pups for analysis. The samples were stored on dry ice and held at -80⁰C until dispatch for enzyme analysis (phenylalanine hydroxylase and glutathione peroxidase – see Chapter Nine). The bodies of the pups have been stored in formal saline.

The females were allowed up to 3 weeks recovery in groups of 4 and were mated again. The second gestation proceeded as the first with the exception that the females were killed at the end of the trial and a post-mortem examination performed on them. The internal organs have been preserved in formal saline and samples of some livers sent for further analysis.

Two test diets were delivered to TCD Bioresources Unit and kept secure. The two test diets were colour coded Red and Blue. Forty-eight female wistar rats were allocated 4 per cage (NKP RB3) as per protocol. They were randomly allocated to 3 groups and fed the diets. The trial proceeded as per protocol. Diet was made up fresh as needed and delivered to the unit.

No rats died during the period of the trial.

DEVIATIONS FROM PROTOCOL

- One test diet had to be replaced due to mould contamination. As a result, the second part of the trial (growth measurement) had to be shortened to 85 days due to feed shortage. It is not considered that this had a significant effect on the interpretation of the results. Before the commencement of the trial it was agreed that the original intention to feed the rats in the two groups as matched pairs was impractical and little useful data would be obtained. The animals were therefore not paired for the feeding trial.

- In relation to weighing and sexing individual pups at birth it was decided that the possibility of increasing mortality and dam rejection was too high to justify this procedure. The total litter weight and number was taken instead.

- A lower than normal pregnancy rate in the first litter of some groups was attributed to the use of immature males. Because of this, the number of litters born on the first gestation was less then envisaged. In order to compensate for this, all females were mated a second time. Litters were chosen randomly from these latter matings to give 11 or 12 females in each group (Table 8-1).

RESULTS OF TRIAL 1

A full statistical analysis of the results of the trial was performed. Based on this, the following conclusions can be drawn:

- There was no evidence of a significant difference between first-litter groups in relation to food and water consumption.

- There was no evidence of a significant difference between first- and second-litter groups in relation to weight gain.

- A significant difference was noted between groups (blue and yellow) for the numbers of litters born at first mating. This difference was attributed to the use of an inexperienced male.

- There was no evidence of a significant difference between second-litter groups in terms of numbers of litters born.

- There was no evidence of a significant difference between first- and second-litter groups in relation to number of animals per litter.

- There was no evidence of a significant difference between first- and second-litter groups in relation to the ratio of male to female animals per litter.

- There was no evidence of a significant difference between first- and second-litter groups in relation to the number of animals weaned per litter.

- There was no evidence of a significant difference between first- and second-litter groups in relation to the weights of males and females per litter.

It can be concluded that the feeding of the diets led to no difference in reproductive capabilities. The lower number of litters in the blue group on the first breeding was attributed to the use of an inexperienced male. The problem did not recur on the second breeding.

TRIAL 2

The purpose of this trial was to determine if there were any differences in growth characteristics between diets when rats from the second litter from trial I were fed the test diets. Ninety-six animals were chosen at random from the animals of the second litter, evenly distributed by sex and group. The animals were weighed weekly and the food and water consumption was measured.

On completion of the study, post mortem examination was performed on the animals and tissues were preserved in 10% formal saline. Livers were collected frozen and sent for analysis to Dr John Donlon in University College Galway.

No significant differences in growth rates between the groups, as measured by weight gain, were noted (Table 8-2). Significant differences in liver weights of male rats were noted between those fed the soil diets and the controls (i.e. no soil in diet). However differences between the two soil-fed groups were not significant. The former finding, therefore, could not be attributed to differences between the Askeaton and non-Askeaton soils.

CONCLUSION

The results of this study showed no evidence that feeding of soil from Index Farm A in Askeaton had a significant negative effect on the reproductive or growth performance of rats when compared to rats fed soil from a control source. Based on the results of this study it was decided that there was no indication to carry out a soil-feeding trial on cattle.

Tables

Table 8-1: First and second breeding litter sizes

	Breeding #1	Breeding #2
Group Red	12	11
Group Blue	7	12
Group Yellow	8	12

Table 8-2: Mean litter weight gains (g)

Means	Mean Start Weight	Mean end weight	Mean weight gain	Sex: n=16
Blue	39.9	257	217	f
Red	44.9	263	218	f
Yellow	38.5	247	208	f
Blue	42.1	435	293	m
Red	45.5	427	382	m
Yellow	40.3	413	373	m

CHAPTER NINE

VOLE STUDIES

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Introduction

In a study of liver enzymes in feral rodents, Fallon *et al* (1997) reported that the activities of phenylalanine hydroxylase and glutathione peroxidase from the livers of voles collected in an area of supposed environmental pollution, i.e. Askeaton, West Limerick, were significantly lower than those from rodents collected at remote control sites (Coole Park, Co. Galway and Dromore Wood, Co. Clare). The authors postulated that the lower enzyme activities in the Askeaton voles were inter-related and were secondary to a suggested decline in herbage selenium in the area. Although no mechanism was proposed to support such an association, the authors also suggested that measurement of the activities of these enzymes in wild rodents could be used as biomarkers of pollution.

As herbage selenium concentrations are known to vary widely throughout the country (Fleming, 1962), and as these were not measured in the study by Fallon *et al.*, a further study was commissioned to determine if differences in liver glutathione peroxidase activity could be accounted for by differences in herbage selenium concentration. This study was a collaboration between the Biochemistry Departments of University College Galway and the Central Veterinary Laboratory Abbotstown, and the Johnstown Castle Analytical Services Laboratory.

The study involved the collection of voles, soil and herbage from the original Askeaton site and two remote control sites. Activities of the liver enzymes phenylalanine hydroxylase and glutathione peroxidase were measured in UCG as described by Fallon *et al.*, (1997). The activity of phenylalanine hydroxylase was measured in the presence of two of the cofactors for this enzyme, i.e. tetrahydrobiopterin (BH_4) and dimethyl-tetrahydropterin ($DMPH_4$). Liver selenium concentrations were determined by hydride vapour atomic absorption spectrometry at CVL Abbotstown. Selenium concentrations of soil and herbage samples were determined by fluorimetry at

Johnstown Castle. The report by Fallon *et al.,* on the results of the liver enzyme analyses is presented in Appendix 12. The results of the liver, soil and herbage selenium analyses are presented below.

Liver phenylalanine hydroxylase and glutathione peroxidase activities were also determined on rats used in the rat feeding trial (*see* Chapter Eight) to determine if there was any significant effect on the activity of these enzymes as a result of feeding soil from Index Farm A.

Results

Fifty voles were collected from the Askeaton and control sites (Coole Park, Co. Galway and Dundrum Wood, Co. Tipperary). Samples of soil and herbage were also collected from the Askeaton site and one of the control sites (Coole Park). Due to labeling errors, one sample each from the Askeaton and Coole Park site was omitted from the analysis of data. One herbage sample collected from outside a dwelling at the Askeaton site, and which had a selenium concentration of almost three times that of any other sample, was also omitted from the data analysis as it was considered that the high value probably reflected non-geochemical contamination.

As in the earlier study by Fallon *et al.,* (1997), liver glutathione peroxidase activity was significantly lower in voles from Askeaton than in voles from the control sites (Appendix 12, Table 1). However, in contrast to the earlier study, phenylalanine hydroxylase activity was higher in voles from the Askeaton site (Appendix 12, Table 1).

Liver (Table 9-1) and herbage (Table 9-2) selenium concentrations were also significantly lower at the Askeaton site. Although soil selenium concentrations were lower at Askeaton than the control site, the differences were not significant ($p > 0.05$).

No significant differences were noted in the activities of either enzyme in the livers of rats from the soil feeding trial.

Discussion

The results of the present study confirmed the finding of Fallon *et al* (1997) in relation to lower

hepatic glutathione peroxidase activities in voles collected in the Askeaton area of Co. Limerick than in voles collected at remote control sites. The results also demonstrated that selenium availability and intake (i.e. as indicated by herbage, soil and liver selenium measurements) were lower in Askeaton. This is consistent with the findings of Fleming (1962) who reported that soil and herbage concentrations vary widely throughout the country.

As selenium is an essential component of glutathione peroxidase (Rotruck et al., 1973), it would not be unexpected that variations in selenium availability would be reflected in variations in activity of the enzyme. This is in agreement with findings in other species (Beckett et al., 1990; Hafeman et al., 1974; Paynter, 1979; Whanger et al., 1977).

In direct contrast to the earlier study (Fallon et al., 1997), activities of the liver enzyme phenylalanine hydroxylase in the present study were higher in voles from the Askeaton area. This would not support their view that lower activities of this

enzyme in the earlier study were secondary to lower concentrations of glutathione peroxidase.

The suggestion by Fallon et al (1997) that variations in the activity of liver glutathione peroxidase in voles could be used as a marker of environmental pollution appears to derive from an assumption that a reported decline in soil selenium concentrations in the Askeaton area was due to environmental pollution. However, the authors of the present report are unaware of any published evidence to support either the suggestion of a decline in soil selenium in the Askeaton area or of an association between environmental selenium concentrations and pollution. In the circumstances, therefore, it is far more likely that variations in the activity of this enzyme in the livers of voles can be attributed to natural variations in the environmental availability of selenium than to environmental pollution.

Tables

Table 9-1: Selenium concentrations (μmol/kg) in livers of male and female bank voles from three sampling locations

Site	Males (n)	Females (n)
Askeaton	5.88 ± 1.04[1] (6)*	7.53 ± 1.16 (5)**
Coole Park	12.05 ± 1.13 (4)*	10.82 ± 0.90 (9)**
Dundrum	NA	8.02 ± 1.26 (8)

[1] standard error of mean.
[2] Not available.
* Differences significant at P < 0.01 (males)
** Differences significant at P < 0.01 (females)

Table 9-2. Selenium concentrations (mg/kg) in soil and herbage samples from Askeaton and Coole Park.

Site	Askeaton[1] (n)	Coole Park (n)	P[2]
Herbage	0.04 ± 0.003[3](8)	0.07 ± 0.009[3] (11)	< 0.01
Soil	0.40 (2)	0.45 (2)	NA[4]

[1] Roadside verge sites 0 - 400 metres from gate.
[2] Significance of differences between sites.
[3] Standard error of mean.
[4] Not applicable.

CHAPTER TEN

ANIMAL TISSUE ANALYSIS RESULTS

Introduction

Analyses for a range of potentially toxic metals were carried out on animal blood and tissue samples collected during the course of the animal health investigations. A list of all analyses, reference ranges, and the sample types on which the analyses were performed, is given in Table 10-1. Results on tissue analyses performed during the latter part of 1995 were included in the EPA Interim Report (EPA, 1995). The results of all animal tissue and blood metal analyses carried out throughout the course of the investigation are presented in this chapter. These include the results of analyses on tissues from nine animals from the two Index Farms in 1995 which were reported in the Interim Report.

Methodology

Tissues were collected from live animals by biopsy (liver), following slaughter at abattoirs or as part of post-mortem pathology examinations. Tissues collected comprised bone, liver, kidney and fat. Other samples were collected as required. Tissues were stored at –20ºC until required for analysis. Blood samples were collected from live animals.

Tissues and blood samples for analysis originated from animals on the two Index Farms in Askeaton, other farms in the Askeaton area with reported animal health problems, and animals on the Monitor Study control Farm at Abbotstown. From the time the DAF assumed responsibility for management of the two Index Farms, i.e. end of 1995, tissues were collected from as many animals as possible which died on the farm or were removed for slaughter for the duration of the Monitor Study. The same policy applied to indigenous and Askeaton-origin animals on the Control (DAF) farm at Abbotstown. Tissue collection from animals from Index Farm A ceased following cessation of the Monitor Study in March, 1998. Sample collection continued on Index Farm B and the Control Farm beyond the end of the Monitor Study – in the case of the former until its sale in 1999 and the latter until the removal of all study animals.

Tissue lead and copper analyses were performed in the Limerick RVL and Central Veterinary Laboratory (CVL) Abbotstown while other metal analyses were performed at CVL Abbotstown or ADAS laboratories in Wolverhampton, UK. Tissues for lead analysis were digested by nitric acid prior to quantification by a Varian SpectrAA 640 atomic absorption spectrophotometer (AAS). Tissues for copper, iron and zinc were digested with a mixture of three aggressive acids prior to estimation by AAS. Arsenic and selenium in tri-acid digests were assayed with the same instrument using a hydride generating accessory. Cobalt and molybdenum in tri-acid digests were analysed in a graphite furnace by electro-thermal atomic absorption spectrophotometry (EAAS). Aluminium in organs was estimated after digestion with Aqua regia by either EAAS or Inductively Coupled Plasma, depending on the concentration in the sample. Bone was ashed and then digested by perchloric acid for aluminium analysis by AAS. Bone fluorine was analysed in this digest by ion selective electrode. Ashed bone was digested with hydrochloric acid and its phosphorus content was estimated by colorimetry on a random access analyser.

The numbers and origins of animals sampled are included with the analytical results in Table 10-2 to Table 10-9.

Results

The results of all tissue and blood metal analyses performed on samples collected between November 1995 and October 1999 are given in Table 10-2 to Table 10-9. These include results already reported in the EPA Interim Report (EPA. 1995).

Aluminium
Normal reference ranges for tissue and blood concentrations of aluminium are given in Table 10-1. These are based on data from Puls (1994) and Allen *et al.,* (1991). The latter is included, as Puls' data for bone (tibia) does not include rib which, based on Allen's results, can be up to 50 *per cent* higher than tibia (reported mean values of 67 and 91 mg/kg for tibia and ribs, respectively). Rib and coccygeal bones were used for analysis in the present investigation. Although Allen's data were from sheep, the reported value for tibia aluminium (mean 67 mg/kg) is comparable to that of Puls (up

to 60 mg/kg) which, in any case, is stated as referring to both cattle and sheep.

The position in relation to tissue concentrations of aluminium which may be indicative of toxicity is less clear. 'Toxic' values reported by Puls (1994; cattle and sheep) overlap his 'normal' ranges and, although no sources are given, appear to indicate a range of tissue values from studies where animals were given above-normal amounts of aluminium compounds. However, reports in the literature of aluminium feeding trials for which tissue analysis results are available (Valdiva et al., 1978, 1982; Neathery et al., 1990a, 1990b), only refer to mild effects on feed conversion – which may have been a palatability effect rather than toxicity. They do not refer to the occurrence of clinical signs consistent with aluminium interference with phosphorus or magnesium metabolism. In the following analysis and discussion, therefore, aluminium analysis results can only be compared to reported 'normal' ranges.

Tissue aluminium results (kidney, liver and bone) for animals from the two Index Farms and the Control Farm are given in Table 10-2. All values were within reported normal reference ranges (Table 10-1). A rib from one of the eight animals sampled from Index Farm B in 1995 had a value of 76 mg/kg which is higher than the upper limit of 60 mg/kg reported by Puls (1994). However, as discussed above, Puls' value was for tibia which is not directly comparable. It was below the upper limit of 91 mg/kg reported for ribs by Allen (1991).

The sample in question was from a cow which was submitted for post-mortem examination in February 1995 (see Table 4-8 and Appendix A1 of EPA Interim Report 1995). As the animal had been outwintered on bare pastures (see Chapter Four), this value probably reflects increased aluminium intake associated with soil ingestion. Aluminium concentrations on herbage have been shown to be at their highest overwinter due to soil contamination (Cherney et al., 1983). This effect is particularly marked in wet weather and on pastures subject to poaching – both of which would have obtained on this farm over the winter of 1994/95 (see Chapter Four). Bone samples from seven other animals submitted for post mortem examination from this farm in March 1995 had aluminium concentrations ranging from 10 to 61.6 mg/kg with a mean of 33 mg/kg. Coccygeal bone samples collected from twenty further animals from the farm between 1996 and 1998 had aluminium concentrations ranging from 6 to 50 mg/kg.

Tissue samples collected from 14 animals on five other farms in the Askeaton area between 1996 and 1998 all had aluminium concentrations within the reference ranges (Table 10-1).

All of 85 blood samples collected from cows on the two index farms in Askeaton and the Control farm at Abbotstown in 1996 had aluminium concentrations within the reference range (Table 10-3).

Arsenic

Tissue arsenic concentrations were within reference ranges.

Cadmium (Table 10-6)

Liver cadmium concentrations were within reference ranges. Although elevated concentrations were detected in the kidneys of six cows from Index Farm B in 1995, they were well below toxic concentrations. They may have been associated with increased soil intake as a result of over-grazing during that year (EPA, 1995 - Teagasc Farm Report,). Most of the animals in question were aged and were moribund and in poor condition at the time of death.

Fluorine

The results of fluorine analysis carried out on tissue samples (bone) from nine animals from the two Index Farms in 1995 have already been reported in the EPA Interim Report (EPA, 1995). All values were within the reference range (Table 10-1). All of 113 blood samples collected from cows on the two Index Farms in Askeaton in 1995, as well as 85 samples collected in 1996 from cows in Askeaton and the Control farm at Abbotstown, had fluoride concentrations with the normal range (Table 10-4).

No other tissue fluoride analyses were undertaken as:

- The clinical signs of fluoride toxicity (fluorosis) are well recognised (Shupe, 1980). There was no historical or clinical evidence that this was a problem in the Askeaton area. No lesions suggestive of fluorosis were detected on post-mortem examination of any carcases from the area.

- There were no identified industrial sources of fluoride emissions in the area. Bauxite refining (Aughinish Alumina) is not known to be associated with significant fluoride emissions.

- EPA monitoring of rainfall in the area from 1995 to 1998 showed no evidence of fluoride pollution (Environmental Quality Volume).

- All tissue and blood analyses carried out in 1995 and 1996 (*see* above) showed values within reference ranges.

Copper and Cobalt

Tissue copper and cobalt concentrations were generally within reference ranges. No values indicative of toxicity were detected. Elevated but not significant cobalt concentrations were noted in four cattle from two farms in the Askeaton area (not Index Farms). They were probably the result of recent liquid drenchs.

Lead

An elevated lead concentration (38 μmol/kg) was detected in kidney tissue from a bull which originated from Index Farm A. The animal in question had been on the Abbotstown farm for nine months prior to slaughter and had shown no signs of lead toxicity. As the animal had been penned for some time prior to slaughter, the result reflected localised environmental concentrations.

Iron

Iron concentrations in liver and kidney samples vary widely with dietary content, amount of soil consumed and chronic infection. Reference ranges are given in Table 10-1. The results of tissue analysis for animals from the two Index Farms in Askeaton and the Control Farm are given in Table 10-9. Although some samples had concentrations above the reference ranges they probably reflected variations in soil intake and were not of toxicological significance.

Selenium

Elevated liver and selenium concentrations were detected in tissues from 31 of 51 animals sampled between 1995 and 1998. All except one, a cow on Index Farm A in 1995, were located on the Control Farm at Abbotstown. These reflect recognised elevated geochemical concentration on the Control Farm (Culleton *et al.*, 1997) and were not of toxicological significance.

Zinc

Elevated zinc concentrations were detected in kidney (35) and liver (6) samples from 35 of the 81 animals sampled. However, the values were well below toxic concentrations and are not of toxicological significance (Table 10-1). Some of the elevated concentrations may have been associated with systemic inflammatory responses as many of the animals in question had histories of inflammatory conditions prior to death e.g. chronic

purulent pneumonia or arthritis, cellulitis or muscular necrosis, salmonella septicaemia

Discussion

Owing to the proximity of an alumina production plant (aluminium oxide concentrated from bauxite), aluminium was one of the first potentially toxic substances to be considered in the Askeaton investigation. However, aluminium, which is the third most abundant element on the planet, is generally regarded to be of low toxicity and most of its compounds, including aluminium oxide, are relatively insoluble and therefore poorly bio-available. Owing to their relatively inert nature, aluminium compounds are widely used as in topical and oral medical preparations and as adjuvants in vaccines. Under normal conditions, grazing animals ingest significant quantities of aluminium in soil (Allen *et al.*, 1986). Concentrate feeds also contain considerable quantities of aluminium.

Were it to occur in farm animals, aluminium toxicity would be most likely to exhibit its effect *via* interference with the metabolism of essential metals such as phosphorus, magnesium and calcium (Allen *et al.*, 1986). However, while it is known that aluminium can form insoluble compounds with phosphorus - thereby reducing its absorption - the possible clinical significance of this is questionable. In experimental studies on cattle and sheep fed high levels of absorbable aluminium over periods of weeks or months, Crowe *et al.*, (1990) reported that while a reduction in blood phosphorus concentrations was noted, there was no evidence of clinical phosphorus deficiency. Valdivia *et al.*, (1978) reported no evidence of significant biochemical or clinical effects of feeding up to 1200 ppm of the soluble (and therefore highly available) aluminium chloride to steers over a three-month period.

The clinical significance of the potential effect of aluminium ingestion on calcium and magnesium is also open to question. Although both Kappel *et al.*, (1983) and Allen (1984) reported a reduction in blood magnesium in adult cattle following intra-ruminal inoculation of aluminium, the former failed to demonstrate any significant effect when aluminium was added to the feed. According to Cherney *et al* (1983), there is no evidence that environmental aluminium contamination has a significant effect on herbage magnesium availability or the incidence of hypomagnesaemic tetany in cows. There are also no reports in the literature of clinical hypomagnesaemia in cattle following aluminium administration.

Although Fogarty *et al.*, (1998), in a recent report on six cases of enteric conditions in horses from a farm in the Askeaton area, suggested a causative association between the lesions observed in the horses and uptake of environmental aluminium, the claim was purely speculative and no supporting evidence was presented. The issues raised in that paper have been dealt with in detail by Collery *et al.*, (1999).

The results of the present investigations in the Askeaton. area showed no evidence either of significant environmental contamination with aluminium from industrial sources (Environmental Quality Volume), or of toxic concentrations of aluminium in blood or tissue samples collected from animals within the area. Neither was there any evidence to suggest that either phosphorus or magnesium deficiencies (either direct or aluminium-induced) were a significant factor in relation to the reported animal health and production problems in the Askeaton area. Clinical phosphorus deficiency was not reported to have been a problem and the results of extensive blood analyses carried out on the two index farms, as well as over 20 other farms in the area, showed no evidence either of widespread hypophosphataemia or hypomagnesaemia (low blood phosphorus or magnesium; *see* Chapter Five). There was also no clinical evidence that the incidence of hypomagnesaemic tetany, a common problem in dairy cows, was unusually high in the area.

Analyses for fluoride were also undertaken on animal samples because of local concerns regarding associations between aluminium processing and fluorine. However, in the Askeaton context, these were probably largely unfounded as, while significant fluoride emissions have been associated with aluminium smelting (Krook and Maylin, 1978), they have not been reported as a by-product of the bauxite concentrating process (i.e. the process undertaken at the Askeaton plant). Interestingly, aluminium has been suggested for use in alleviating fluoride toxicosis in ruminants due to its ability to form insoluble complexes with fluorine (Allen, 1984).

Although elevated concentrations of naturally-occurring geochemical compounds of fluorine were identified in soil samples from the Askeaton area (EPA, 1995), these were largely present in a non-available (and therefore non-toxic) form.

The results of the present investigations provided no evidence to suggest that environmental fluorine toxicity was a problem in the Askeaton area. Environmental and animal blood and tissue concentrations of fluoride were well within acceptable limits. Neither were there any reports of disease outbreaks consistent with fluoride toxicity. Fluoride toxicity is a well recognised syndrome in animals with characteristic clinical signs, e.g. bone, tooth and hoof abnormalities (Krook and Maylin, 1978). There was no historical, clinical or pathological evidence that conditions of this type occurred on a significant scale in the Askeaton area.

The results of cadmium, iron, copper, cobalt, and selenium analyses on animal tissues showed no evidence of toxicity due to these metals. While occasional values were recorded above reference ranges, all were well below toxic concentrations.

Although over a half of the animals sampled had tissue zinc concentrations above the reference range, values were well below toxic concentrations and are not of clinical significance.

The only heavy metal analysis result on tissues in the entire investigation which specifically indicated toxic concentrations was a single case of lead in tissues from a bull. The animal in question originated from Index Farm A in Askeaton but had been located on the Control Farm at Abbotstown for seven months prior to slaughter. The animal had not shown any clinical signs of lead toxicity prior to slaughter.

In conclusion, the results of the blood and tissue analytical investigations reported herein show no evidence to suggest that the animals sampled had been exposed to toxic concentrations of the elements concerned. These findings are consistent with the absence of clinical reports of classical metal toxicity in animals in the area as well as of the environmental investigations which showed no evidence of significant heavy metal environmental contamination.

Table 10-1: Details of metal analyses performed on tissues and bloods.

Sample	Blood	Nrange	Bone	Nrange[1]	Trange[2]	Kidney	Nrange	Trange	Liver	Nrange	Trange
Aluminium	*	2-100 µg/l	*	6-60 mg/kg	NA[3]	*	<2.0-6.0 mg/kg		*	<1-7.5 mg/kg	
Arsenic									*	0-40.0 µmol/kg	>40 µmol/kg
Cadmium						*	0-2.0 µg/g	>100 µg/g	*	0-2.0 µg/g	>50 µg/g
Cobalt									*	0.7-5.0 µmol/kg	>50 µmol/kg
Copper	(*)	9.4 - 24.0 µmol/l				*	0-0.16 mmol/kg	>0.2 mmol/kg	*	0.06-2.5 mmol/kg	>4.0 mmol/kg
Fluorine	*	40 – 140 µg/l	*	400 – 1600 mg/kg	> 2,100 mg/kg						
Iron						*	0.54-2.69 mmol/kg	>5.4 mmol/kg	*	0.8-5.4 mmol/kg	>160 mmol/kg
Lead		0-2.5 µmol/l				*	0-2.5 mg/kg	>5.0 mg/kg	*	0-2.5 mg/kg	>5.0 mg/kg
Selenium	(*)	0.75-3.0 µmol/l				*	5.0-20.0 µmol/kg	>35.0 µmol/kg	*	0.5-3.0 µmol/kg	>30.0 µmol/kg
Zinc	(*)	5.0-25.0 µmol/l				*	0.27-0.38 mmol/kg	>1.99 mmol/kg	*	0.38-1.53 mmol/kg	>4.5 mmol/kg

1 Normal range.
2 Toxic range (estimated).
3 Not available (see text).
*Analysis performed.

Table 10-2: Tissue aluminium analysis results – Two Index Farms and Control Farm

Source			Index Farm A						Index Farm B					Control Farm		
Location			Askeaton				CVL		Askeaton				CVL	Askeaton	CVL	
Sample			1995	1996	1997	1998	1996	1998	1995	1996	1997	1998	1996	1996	1997	1998
Bone	Max		21.98	28.30	8.22	21.00	11.90	23.70	76.00	22.60	35.90	50.20	19.40	18.30		23.60
	Min		9.87	28.30	8.22	7.54	11.00	19.50	10.00	10.60	11.70	5.82	14.80	12.60		21.00
	Avg		15.49	28.30	8.22	14.00	11.45	21.97	29.12	18.53	23.80	13.53	17.10	15.45		22.30
	No.		4	1	1	7	2	3	18	3	2	15	2	2		2
Kidney	Max		1.40		1.52	3.52	0.97	2.91	2.80	1.69		3.23	0.95	5.28		1.11
	Min		0.73		0.75	0.23	0.80	0.86	0.57	0.92		0.73	0.85	0.81		0.83
	Avg		1.07		1.14	1.64	0.89	1.47	1.23	1.34		1.43	0.90	3.05		0.97
	No.		2		2	4	2	4	9	3		16	2	2		2
Liver	Max		6.00	1.13	1.03	5.02	1.05	3.84	2.62	2.31	1.82	5.11	1.62	3.75	1.31	1.07
	Min		1.34	1.13	1.03	0.25	1.02	0.84	0.85	1.12	0.64	0.59	1.10	3.75	0.75	0.99
	Avg		2.55	1.13	1.03	1.89	1.04	2.01	1.57	1.72	1.23	1.80	1.36	3.75	1.03	1.03
	No.		4	1	1	8	2	4	9	2	2	14	2	1	2	2

Table 10-3: Blood (plasma) aluminium analysis results – Two Index Farms and Control Farm

	Index Farm A	Index Farm B	CVL
Year	1996	1996	1996
Max	22.00	15.00	25.00
Min	2.00	2.00	2.00
Avg	12.53	6.22	11.94
No.	30	23	32

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Table 10-4: Blood (plasma) fluorine analysis results – Two Index Farms and Control Farm

	Index Farm A		Index Farm B		CVL	
Year	1995	1996	1995	1996	1995	1996
Max	68.00	83.00	74.00	117.00	130.00	121.00
Min	50.00	34.00	20.00	48.00	44.00	47.00
Avg	52.17	49.54	33.48	67.42	67.47	76.74
No.	37	24	46	24	30	38

Table 10-5: Tissue analysis results for samples from other farms in the Askeaton area.

Sample	Year	Al			As		Co					Cu	Pb	Se	Zn				
		1996	1997	1998	1997	1998	1995	1996	1997	1998	1999	1995	1997	1996	1995	1996	1997	1998	1999
Bone	Max	13.90	14.50	21.20	1.00	0.10													
	Min	11.20	14.50	6.83	0.00	0.00													
	Avg	12.55	14.50	13.17	0.18	0.05													
	No.	2	1	7	6	2													
Kidney	Max												3.35	19.10			1.36		
	Min												3.00	19.10			0.96		
	Avg												3.15	19.10			1.11		
	No.												3	1			4		
Liver	Max						1.08	3.58	11.74	8.90	1.07	2.07			2.45	3.48	2.43	2.59	0.38
	Min						0.60	1.18	0.25	0.08	0.75	2.06			0.72	0.60	0.51	0.28	0.22
	Avg						0.84	2.24	2.10	2.21	0.91	2.06			1.58	1.61	1.34	1.10	0.30
	No.						2	4	15	14	2	2			2	3	10	12	2

Table 10-6: Tissue cadmium analysis results – Two Index Farms and Control Farm

Source		Index Farm A			Index Farm B			Control Farm		
Location		Askeaton		CVL	Askeaton		CVL	Askeaton	CVL	
Sample	Data	1995	1996	1998	1995	1996	1996	1996	1997	1998
Kidney	Max	1.33	1.08	2.01	4.42	1.50	0.88	1.56		1.46
	Min	0.92	0.95	2.01	1.22	0.76	0.62	0.58		0.92
	Avg	1.13	1.01	2.01	2.81	1.13	0.75	1.07		1.19
	No.	2	2	1	9	2	2	2		2
Liver	Max	0.70	0.15	0.39	0.26		0.10	0.09	0.14	0.14
	Min	0.20	0.14	0.13	0.03		0.09	0.09	0.13	0.10
	Avg	0.35	0.14	0.23	0.14		0.09	0.09	0.14	0.12
	No.	4	2	3	9		2	1	2	2

Table 10-7: Tissue zinc analysis results – Two Index Farms and Control Farm

Source		Index Farm A					Index Farm B					Control Farm				
Location		Askeaton		CVL			Askeaton				CVL	Bckw[1]	Ask.[2]	CVL		
Sample.	Data	1995	1998	1995	1996	1998	1995	1996	1997	1998	1996	1996	1996	1997	1998	1999
Kidney	Max	0.71	0.55	0.55	0.85	1.00	0.91	0.64	0.17	0.16	0.45		0.37		0.33	1.05
	Min	0.65	0.34	0.55	0.45	0.30	0.34	0.54	0.17	0.12	0.38		0.36		0.29	0.63
	Avg	0.68	0.45	0.55	0.58	0.49	0.59	0.59	0.17	0.14	0.41		0.37		0.31	0.85
	No.	2	2	1	9	10	9	2	1	2	2		2		2	3
Liver	Max	2.60	0.55	0.55	1.12	3.25	2.77	1.05	0.71	0.54	0.24	0.34	0.43	1.99	0.33	0.63
	Min	0.99	0.34	0.55	0.18	0.20	0.80	1.05	0.56	0.36	0.22	0.34	0.32	0.36	0.26	0.56
	Avg	1.78	0.45	0.55	0.56	0.92	1.33	1.05	0.63	0.45	0.23	0.34	0.38	0.84	0.30	0.58
	No.	4	2	1	15	7	10	1	2	2	2	1	2	4	2	3

1 DAF farm at Backweston. [2]Askeaton.

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Table 10-8: Tissue selenium analysis results – Two Index Farms and Control Farm

Source		Index Farm A					Index Farm B				Control Farm			
Location		Ask	CVL				Askeaton		CVL	Bckw[1]	Ask.[2]	CVL		
Sample	Data	1995	1995	1996	1997	1998	1995	1996	1996	1996	1996	1997	1998	1999
Kidney	Max	22.87	11.31	37.77	27.09	29.73	18.70	16.48	24.07	36.02	23.20	15.67	23.98	18.44
	Min	17.53	11.31	13.95	27.09	18.16	9.85	8.83	23.74	36.02	17.61	7.93	20.61	4.97
	Avg	20.20	11.31	28.11	27.09	25.23	14.11	12.66	23.91	36.02	20.41	11.80	22.30	13.74
	No.	2	1	10	1	12	10	2	2	1	2	2	2	3
Liver	Max					4.86					27.06			
	Min					1.02					19.67			
	Avg					3.72					23.37			
	No.					5					2			

1 DAF farm at Backweston ²Askeaton.

Table 10-9: Tissue iron analysis results – Two Index Farms and Control Farm

Source		Index Farm A			Index Farm B				Control Farm			
Location		Ask.[1]	CVL		Askeaton			CVL	Ask.[1]	CVL		
Sample	Data	1995	1996	1998	1995	1996	1998	1996	1996	1997	1998	1999
Kidney	Max	2.55	3.29	3.72	4.71	1.58	1.92	1.48	2.12		1.21	1.73
	Min	1.92	1.30	1.08	0.82	1.43	1.24	1.48	1.94		1.02	1.38
	Avg	2.24	2.30	2.00	2.00	1.51	1.58	1.48	2.03		1.12	1.52
	No.	2	2	8	9	2	2	2	2		2	3
Liver	Max	7.12	2.97	2.71	3.93		2.04	1.62	2.91	2.19	1.35	2.94
	Min	1.66	1.20	0.92	1.25		1.54	1.52	2.91	1.52	1.14	1.29
	Avg	4.14	2.08	1.66	2.15		1.79	1.57	2.91	1.86	1.25	1.94
	No.	4	2	8	9		2	2	1	2	2	3

¹Askeaton.

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CHAPTER ELEVEN

CONCLUSIONS

The animal health investigations of the VLS in the Askeaton area were originally undertaken in response to local concerns regarding a possible link between industrial emissions and reportedly severe animal disease problems on two farms. The first of these (Index Farm A) was reported to have suffered animal disease problems of increasing severity since the late 1980s and reaching a maximum around 1993. A second farm (Index Farm B) reported severe animal losses in 1994 and 1995. Both of these farms were located close to the town of Askeaton and were approximately downwind (prevailing westerly wind) of a nearby bauxite refinery and two, more distant, oil-burning electricity generating stations.

Concerns regarding animal health problems in the wider Askeaton area were first raised at public meetings held in the area in the first half of 1995 to discuss the problems on the two Index Farms. Following this, the locally-formed Askeaton and Ballysteen Animal Health Committee provided the names of 25 herdowners who reported that they had experienced an excess of animal disease problems on their farms. These farms were bounded by an area of approximately 400 sq. kilometres extending eastwards along the Shannon from Foynes to Kildimo and inland and south towards Rathkeale.

The main questions to be answered by the animal health investigations were, therefore:

1. What were the causes of the animal health and production problems on the two index farms?

2. Was there any evidence that the incidence of animal health and production problems in the Askeaton area was significantly higher than comparable areas elsewhere?

3. If so, was there any evidence that this was due to common underlying factors. In particular, was there any evidence that environmental pollution had contributed to an excess of animal disease and production problems in the area.

The animal health studies which were subsequently undertaken in the Askeaton area comprised a retrospective survey of animal health and production on the 25 self-identified 'problem' farms as well as on the two Index Farms; a two-year longitudinal monitor study on the two Index Farms and a control farm; a longitudinal study on four of the self-identified 'problem' farms; a series of studies to investigate the immune responses of animals within and from the Askeaton area; a questionnaire survey of animal health; a feeding trial to determine the effects of soil from one of the Index Farms on laboratory rats; a comparative study on the concentrations of certain enzymes and selenium in the livers of voles from within and without the Askeaton area.

RETROSPECTIVE SURVEY

The purpose of the Retrospective Survey was to gather information on the type and incidence of disease on the 25 self-identified 'problem' farms, as well as the two index farms, and to determine a) if there was any evidence of an excess of disease in the Askeaton area as a whole and b) if there was any evidence that common underlying factors, such as environmental pollution, had contributed to an excess of disease in the area.

The findings of the survey in relation to disease type were that, for the most part reported diseases were similar to those reported on farms elsewhere, i.e. infertility, peri-parturient problems, infectious disease and animal deaths. There was no evidence of an unusually high incidence of uncommon or undiagnosed diseases.

With the exception of the two Index Farms, reported disease incidences on the Askeaton farms did not differ markedly from incidences reported in the international veterinary literature. While the conclusions of the report are at their most subjective in relation to disease incidence – owing both to the incomplete nature of the available data and to the lack of reference data from comparable populations elsewhere - there was insufficient evidence to suggest that the area had experienced an unusually high incidence of disease over the period of interest, i.e. from approximately 1988 to 1996. With one notable exception - which has been specifically dealt with in this report - re-visits to 17

of the 25 'problem' farms in 1997 did not provide any evidence to suggest that they were experiencing unusual or severe disease problems at that time.

In relation to disease causation, the wide range of reported problems, together with a lack of synchrony in relation to their onset or duration, provided little evidence to suggest that a single common underlying factor such as environmental pollution had contributed to disease incidence in the area.

Index Farms Monitor Study

The results of the Monitor Study provided no evidence to suggest that animal health or production on either of the two Askeaton Index Farms, or among the Askeaton-origin cows on the Control Farm at Abbotstown, were adversely affected by environmental pollution or other unidentified factors for the duration of the study, i.e. 1996 to 1998. Health and production performance of growing and adult cattle on the three Monitor Study farms were generally satisfactory throughout. Disease problems encountered comprised conditions which are common on farms elsewhere in Ireland and incidence rates were within acceptable limits. While differences in performance were recorded between the three farms - specifically in relation to body condition, fertility and milk production – these were consistent with the inevitable differences in weather, grass production and the day-to-day implementation of nutrition and fertility management.

Longitudinal Study of Four Farms

The Longitudinal Study comprised a one to two-year observational and investigative study of animal health and production, between 1996 and 1998, on four farms selected from among the 25 'problem' farms identified for the Retrospective Survey. The results of the study showed no evidence that animal health or production on any of the four farms were subject to unusual adverse influences during the periods of observation. While there was a wide range in production performance between farms, results were broadly compatible with expectations for comparable operations. The incidence of disease on each of the four farms was generally within acceptable limits and no serious outbreaks of disease were encountered. The conditions reported largely comprised those commonly-observed on commercial farms, i.e. stillbirths and neonatal diseases in calves, and mastitis, lameness and infertility in cows. While the

exact causes of a number of outbreaks of respiratory disease in cows and calves on one farm were not identified, the cases were generally mild in nature and clinical and laboratory examinations did not reveal any unusual features.

Immunology Studies

Reports of an increased incidence of animal disease in the Askeaton area inevitably raised questions regarding the immune competence of affected animals. Accordingly, a series of studies was undertaken on cows and growing stock - both within and from the area - to examine their immune systems and to measure their responses to test antigens. The results showed no evidence that animals in either the Askeaton or Control Farm locations were subject to immune suppression throughout the course of the studies. All measured cellular components of the immune system were fully represented in the animals under study and humoral responses were within acceptable ranges. Although blood samples from cows and steers on the Askeaton farms showed lower *in-vitro* lymphocyte responses than those from the Control Farm, there was no evidence that this was of any clinical significance and was not reflected in a higher incidence of disease in the animals concerned. The animals were generally healthy throughout and in relation to the steers, those on the Askeaton farm out-performed (weight gain) those on the Control farm.

Animal Health Questionnaire Survey

The purpose of the animal health questionnaire survey was to compare specific indices of animal health on farms in the Askeaton area and surrounds ('exposed' region) with farms elsewhere ('non-exposed' region). The most notable findings of the survey, however, were the clear differences between the exposed and non-exposed regions in terms of farm size and enterprise type. Farms in two of the three areas comprising the non-exposed region were significantly smaller, and a much higher proportion were described as suckler farms, than those in the exposed region. While these differences were a function of the survey design, it must be emphasised that the target population was originally selected as part of a larger human health survey.

The differences in population type between the exposed and non-exposed regions will undoubtedly have had a significant impact on the findings of the survey. While allowances were made in the comparative analyses for differences in farm size and type, the question must remain as to what management or other unidentified differences may have contributed to the differences in animal health performance.

In terms of animal health, the only areas where important differences were noted between the exposed and non-exposed regions were the rates of illthrift in cows and dry stock and mortality in suckler cows. While comparative figures are not available for incidences of ill-thrift, the reported suckler cow mortality in the exposed region in 1995 at 4.2 *per cent* was also high relative to rates reported in the international literature.

The reason for the higher rates of illthrift and suckler cow mortality in the exposed region cannot be determined from the results of the survey. The only related issue on which information was available was the significantly higher proportion of cattle outwintered in one of the two areas (Askeaton area) of the exposed region. Given the poor weather conditions of the winter of 1994-95, it is possible that this was a contributory factor.

RAT FEEDING TRIAL

Since cattle consume a significant quantity of soil in their diet, a laboratory rat study was undertaken to investigate the possibility that soil from one of the Index Farms in Askeaton contained material (e.g. pollutant) which was toxic to animals. The results of the study showed no evidence that the feeding of the Askeaton soil had a significant negative effect on the reproductive or growth performance of rats when compared to rats fed soil from a control source.

VOLE STUDY

The decision to fund a study on wild voles arose from a report in the literature which suggested an association between the lower concentrations of certain enzymes in the livers of voles from the Askeaton area and environmental pollution (Fallon *et al.*, 1997). The objective of the study was to determine if these differences could be accounted for by differences in the environmental availability of selenium which is a component of one of the enzymes - glutathione peroxidase. The results of the study confirmed the finding of Fallon *et al* in relation to the lower glutathione peroxidase activities in Askeaton voles. In addition, the study also demonstrated that environmental selenium

concentrations were lower at the Askeaton sites – which most likely accounts for the lower enzyme activities.

ANIMAL TISSUE ANALYSIS RESULTS

The results of the blood and tissue analytical investigations showed no evidence to suggest that the animals sampled had been exposed to toxic concentrations of the elements. Aluminium, fluorine, and arsenic concentrations were within reference ranges in all tissue and blood samples examined. Some tissue cadmium, cobalt, selenium, zinc and iron concentrations above published reference ranges were recorded. They were well below toxic concentrations and were not considered to be of toxicological significance. These findings are consistent with the absence of clinical reports of classical metal toxicity in animals in the area as well as of the environmental investigations which showed no evidence of significant heavy metal environmental contamination.

OVERALL CONCLUSION

In conclusion, the results of the retrospective studies of the Askeaton investigations confirm that certain farms in the area – in particular the two Index Farms - had an unusually high incidence of animal disease and production problems at times over a period of about eight years from 1988 to 1995. However, there is little evidence to suggest either that this was part of an area-wide problem or that unusual underlying factors such as environmental pollution were responsible. Although 25 other farms in the area were said also to have suffered an excess of animal disease problems, neither their number or distribution, nor the nature and severity of animal health problems reportedly experienced, would provide sufficient evidence to suggest that factors other than those normally considered, i.e. nutrition, management and infectious agents, need be cited to account for disease incidence in the Askeaton area.

While some differences were observed between the Index (Askeaton) and Control (Abbotstown) Farms in the prospective comparative studies, i.e. the Monitor Study and the Immunology Study, these were generally within the range of what could be accounted for by differences in location (i.e. weather, grass, housing) and the implementation of day-to-day management. Any suggestion that differences in health or production may have been due to environmental pollution must be balanced both by reference to the broad range of health and production parameters where performance was well within expectations, and to the failure of the

extensive environmental investigations of the other agencies involved in this investigation to show any evidence that the area was subject to industrial emissions injurious to animal health throughout the period of the studies.

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APPENDIX 1

Disease Incidence Tables

Abortion and Periparturient Conditions

Condition	Incidence (%)	Country/Region	Source
Abortion	2 - 2.5	International	Roy, 1990.
Abortion	1.6	Ireland	Crosse and Soede, 1988.
Abortion	2 – 4	Ireland	Mee, 1992.
Abortion	2.6	Ireland	Greene, 1978.
Abortion	2	UK	Murray, 1990.
Abortion	2.2	Switzerland	Frei et al., 1997.
Dystocia	10	US	Gearhart et al., 1990.
Dystocia	13.6	Switzerland	Busato et al., 1997.
Dystocia	9	Ireland	Crosse and Soede, 1988.
Dystocia	10	Canada	Erb et al., 1990. (lact incid.[1])
Dystocia	5.8 (median)	Canada	McDermott et al., 992
Dystocia (all ages)	13 (2 – 36)	UK	Peeler et al., 1994.
Dystocia (heifers)	18	UK	Peeler et al., 1994.
Dystocia (parity > 1)	9	UK	Peeler et al., 1994.
Dystocia	8.7 (1–27)	UK	Esslemont and Kossabiti, 1996.
Downer cow	2.1	USA	Cox et al., 1986.
Milk Fever	9	Canada	Erb et al., 1990. (lact incid)
Milk Fever	9.4 (6-18)	UK	Borsberry and Dobson, 1989.
Milk Fever	7.6	UK	Lucey et al., 1986.
Milk Fever	7.7	UK	Esslemont and Kossabiti, 1996.
Metritis	10.1	UK	Borsberry and Dobson, 1989.
Metritis	4.7	UK	Lucey et al., 1986.
Metritis	7.5	Canada	Erb et al., 1990. (lact incid)
Metritis	15	UK	Esslemont and Kossabiti, 1996.
RFM[2]	4.4 (1-25)	UK	Peeler et al., 1994.
RFM	4.7	US	Gardner et al., 1990
RFM	10	Canada	Erb et al., 1990. (lact incid)
RFM	1.3 (0-22)	UK	Borsberry and Dobson, 1989.
RFM	3.6 (0 – 19)	UK	Esslemont and Kossabiti, 1996.
Stillbirth	5	Ireland	Crosse & Soede, 1988.
Stillbirth	2.8 (median)	Canada	McDermott et al., 1992
Stillbirth	6.3	Switzerland	Busato et al., (1997)
Stillbirth	3.7	Switzerland	Frei et al., 1997.
Stillbirth	4.7	Ireland	Greene, H. 1978.
Stillbirth	6.3		Bruning-Fann and Kaneene, 1992.

[1]RFM = Retained foetal membrane. [2]lact. incid. = lactational incidence.

Twinning and Congenital Abnormalities

Condition	Incidence (%)	Country/Region	Source
Twinning	2.6	UK	Anon, 1988.
	3.3	UK	Peeler et al., 1994.
	4.1 (1– 10)	UK	Esslemont and Kossabiti, 1996.
	2.5	Ireland	Mee, 1991a.
	1 – 6	World-wide	Mee, 1991a.
Misc. defects	1.5	Ireland	Mee, 1991b.

Cow Morbidity and Mortality

Condition	Age/Type	Incidence (%)	Country	Source
Mortality	Dairy	2.4	Ireland	Menzies *et al.*, 1995.
Mortality	Dairy	2.0	US	Gardner *et al.*, 1990
Mortality	Suckler	1.6	Ireland	Menzies *et al.*, 1995.
Mortality	Calved	1.6	UK	Esslemont, 1992.
Non-detected oestrus	Dairy	37 (27-50)	UK	Peeler *et al.*, 1994
Non-detected oestrus	Dairy	46.4 (17 – 90)	UK	Esslemont and Kossabiti, 1996.
Ovarian cyst	Dairy	6.7	UK	Borsberry and Dobson, 1989.
Lameness	All	23	Ireland	Arkins, 1981
Lameness	All	17 (5– 48)	UK	Esslemont and Kossabiti, 1996.
Lameness	All	17 (8-28)	UK	Collick *et al.*, 1989
Lameness	All	25	UK	Whittaker *et al.*, 1983.
Mastitis	All	14.4	UK	Peeler *et al.*, 1994.
Mastitis	All	20.2	Switzerland	Frei *et al.*, 1997.
Mastitis	All	13	Canada	Erb *et al.*, 1990. (lact incid)
Mastitis	All	30	US	Gardner *et al.*, 1990
Mastitis	All	23	UK	Lucey *et al.*, 1986.
Mastitis	All	33	UK	Esslemont and Kossabiti, 1996.
Conjunctivitis	All	Present in 50% of herds	US	Gardner *et al.*, 1990
Culling rate	Dairy	30.5	IRL	DairyMIS 1996
Culling rate	Dairy	23	UK	Esslemont, 1992
Culling rate	Dairy	25	US	Gardner *et al.*, 1990

Mortality in Stores and Growing Animals

Condition	Age/Type	Incidence (%)	Country	Source
Mortality	1 – 5 months	1.02	Ireland	Menzies *et al.*, 1996
Mortality	6 – 24 months	0.82	Ireland	Menzies *et al.*, 1996
Mortality	6 – 24 months	1.2	US	Gardner *et al.*, 1990

Calf Morbidity and Mortality

Condition	Age/Type	Incidence (%)	Country	Source
Diarrhoea	Calf	15 - 20	International	Bruning-Fann and Kaneene, 1992.
Diarrhoea	birth to 8 wks	25	USA	Wells et al., 1997.
Diarrhoea	birth to weaning	20	Canada	Bruning-Fann and Kaneene, 1992.
Diarrhoea	0 – 90 days	15	US	Bruning-Fann and Kaneene, 1992.
Diarrhoea	0 – 14 days	10	US	Bruning-Fann and Kaneene, 1992.
Diarrhoea	15 - 90 days	5	US	Bruning-Fann and Kaneene, 1992.
Resp. dis.	Calf	7 – 15		Bruning-Fann and Kaneene, 1992.
Resp. dis.	0 – 8 wks	10	USA	Wells et al., 1997
Resp. dis.	0 – 90 days	7	US	Bruning-Fann and Kaneene, 1992.
Resp. dis.	0 – 7 days	1.6	US	Bruning-Fann and Kaneene, 1992.
Mortality	Calf	15.8 – 27.2	USA	Hartman et al., (1974)
Mortality	< 24 hrs	3.1	Switzerland	Busato et al., (1997)
Mortality	pre-wean calf	6.0	Switzerland	Busato et al., (1997)
Mortality	< 24 hrs	3.5 – 5		Roy, 1990.
Mortality	< 24 hrs	1.3 – 26		Drew, 1987.
Mortality	0 – 28 days	3		Roy, 1990.
Mortality	0 – 28 days	3	Ireland	Greene, 1978.
Mortality	0 – 6 months	21		Jenny et al., 1981
Mortality	pre-weaning (excl. stillbirth)	9.4	US (1,685 dairy herds)	Losinger and Heinrichs, (1997)
Mortality	pre-weaning	5	Switzerland	Busato et al., 1997.
Mortality	birth to 8 wks	7 – 8	USA	Wells et al., 1997
Mortality	0 – 24 hrs	6.1	UK	Peeler et al., 1994
Mortality	0 – 24 hrs	7.8 (1 – 20)	UK.	Esslemont and Kossabiti, 1996.

Target and intervention levels for fertility indices by herd size*.

Herd Size	Preg. Rate[1]	18-24 day return	S/C[2]	CSI[3]	CCI[4]	SR[5]	NDO[6]	IR[7]	HDR[8]	HDE[9]
Target:	60%	60%	1.65	65	85	80%	10%	10%	80%	50%
Intervention :										
10-20 cows	<30	<30	>3.13	>76	>94	<53	>24	>24	<53	<20
20-50 cows	<40	<40	>2.38	>75	>94	<62	>18	>18	<62	<30
50-75 cows	<48	<48	>2.08	>75	>93	<69	>16	>16	<69	<37
75-100 cows	<50	<50	>1.96	>75	>93	<71	>15	>15	<71	<39
> 100 cows	<52	<52	>1.92	>75	>93	<73	>15	>15	<73	<41

*Based on DairyMIS herds – (O'Farrell and Harrington, 1999).
[1]Conception to first service. [2]Services per conception. [3]Calving to first service interval. [4]Calving to conception interval. [5]Submission rate.
[6]Non-detected oestrus. [7]Infertile rate. [8]Heat detection rate. [9]Heat detection efficiency.

Average, target and intervention fertility performance for DAISY (UK) herds*

	Actual (average)	Target	Interference
Calve to first service	71.7	67	70
Submission rate	52	56	50
Overall conception rate	49	55	48
Conceived of served	90	93	90
Calving to conception	99	89	94
Calving interval.	380	370	377
Total culling	23	16	22
Culling for infertility	10	7	11

* Esslemont and Peeler (1993).

Haematology reference ranges for cattle

Test name	Abbrev.	Min.	Max.	Units
Packed cell volume	PCV	24	40	%
Red cell count	RBC	5	9	$10^{12}/l$
Haemoglobin	Hb	8	14	g/l
Mean corpuscular haemoglobin concentration		30	34	g%
Mean corpuscular volume	MCV	40	60	fl
White cell count	WBC	4	10.5	$10^9/l$

Blood biochemistry reference ranges for cattle

Test name	Abbrev.	Min.	Max.	Units
Calcium	Ca	2.1	3.1	mmol/L
Copper	Cu	9.4	24	µmol/L
Magnesium	Mg	0.65	1.2	mmol/L
Potassium	K	3.6	5.6	mmol/L
Selenium	Se	0.75	3	µmol/L
Sodium	Na	136	145	mmol/L
Zinc	Zn	5	25	µmol/L
Iodine	Iod	20	300	µg/L
Aluminium	Al	2	100	µg/L
Albumin	Alb	23	37	g/L
Total protein	TP	57	83	g/L
Urea	Urea	2.65	6.89	mmol/L
Phosphorous	P	1.4	2.5	mmol/L
Chloride	Cl	97	111	mmol/L
Glucose	Gluc	2.5	4.16	mmol/L
γGT	GGT	18	55	iu/L
Fluoride	F	40	140	µg/L
Aspartate aminotransferase	AST	38	120	iu/L
Thyroxine	Thy	45	120	nmol/L
Creatine phospho-kinase	CPK	50	130	iu/L
Glutamate dehydrogenase	GLDH	0	25	iu/L
β-hydroxyButyrate	βHB	0	0.9	mmol/L
Globulin	Glb	31	51	g/L
Albumin:globulin ratio	A/G	0.5	1.5	Ratio

APPENDIX 2

Monitor Farm Study Individual Cow Annual Milk Yields

Note: The lactation year includes animals which calved from after end breeding season of previous year to end breeding season of current year, i.e. approximately September to August.
All yields in the Appendices are given in gallons.

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Note : Milk recording began April, 1996. Estimated yields for cows calving over 75 days before milk recording commenced may not be reliable. These values are shown in brackets in tables below.

Indigenous cows 1996

ID	Calvdate	Yield[1]	Drydate	Lactation	Lactlen[2]	Comment
Short lactations:						
313	19/01/96	-673		8		moved to VRL July 96
First Calvers:						
308	10/05/96	866.7**	27/08/97	1	474	
Rest:						
312	10/05/96	848	29/12/96	3	233	
310	30/05/96	1066.4	07/03/97	3	281	
309	12/04/96	520.2	29/10/96	5	200	very excitable
304	02/03/96	1178.2	07/02/97	5	342	
314	18/02/96	1112.2	10/01/97	6	327	
306	22/03/96	1177.7	21/01/97	8	305	
301	23/03/96	923**	27/08/97	8	522	
311	15/05/96	693.4	23/12/96	9	222	3->2 quarters;mast,lame
303	15/01/96	758.5	13/10/96	9	272	mastitis x 2
302	10/03/96	1039**	27/08/97	9	535	
307	24/03/96	1216**	27/08/97	10	521	
Means		*958*		*6.8*	*342*	

[1]Gallons. [2]Lactation length in days. ** Projected 305-day yield.

VRL-origin cows 1996

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
96	25/03/96	1101.6	24/01/97	1	305	moved to Askeaton July 1996
97	02/04/96	1223.5	24/01/97	1	297	moved to Askeaton July 1996
63	31/03/96	748.1	13/10/96	6	196	mastitis winter 1995/96;lost qtr
89	01/02/96		16/10/96	2	258	to Ask July 1996
Calved > 75 days before 1st milk recording:						
65	04/11/95	(677.3)	08/07/96	6	247	lame, mastitis, ill-thrift winter '95/96
61	22/08/95	(662.6)	12/06/96	7	295	
52	08/08/95	(523.3)	06/06/96	8	303	pneumonia, mastitis, lame winter 1995/96
73	11/01/96	(530.8)	25/09/96	4	258	lame, mastitis, ill-thrift winter '95/96
81	14/01/96	(1153)	29/12/96	3	350	
62	03/09/95	(720.9)	08/07/96	6	309	teat-injury, mastitis, ill-thrift winter 1995/96
First Calvers:						
91	28/08/95	(919.7)	08/07/96	1	315	milked overwinter 95/96
101	12/05/96	977.6	24/01/97	1	257	moved to Askeaton July 1996
103	21/05/96	860.1	07/03/97	1	290	moved to Askeaton July 1996
104	21/05/96	816.1	07/03/97	1	290	moved to Askeaton July 1996
105	31/05/96	994.9	07/03/97	1	280	moved to Askeaton July 1996
Means		*912 (n=4)*		*1*	*279*	
Rest:						
87	20/01/96	1320.7	29/12/96	2	344	
84	28/01/96	1723.1	15/07/97	2	534	didn't go in calf in 1996

Indigenous cows 1997

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
303	11/02/97	738	27/08/97	10	197	?ketosis Apr/May
First Calvers:						
316	31/01/97	667.6	27/11/97	1	300	< 2 yro; small at calving
317	01/02/97	866.3	27/11/97	1	299	< 2 yro; small at calving
318	08/02/97	602.5	25/10/97	1	259	< 2 yro; small at calving
Means		*712.1*		*1*	*286*	
Rest:						
310	13/05/97	872.9	23/12/97	4	224	
304	26/04/97	899.5	23/12/97	6	241	
314	04/03/97	1182.8	23/12/97	7	294	
306	25/03/97	1163.2	23/12/97	9	273	
Means		*1030*		*6.5*	*258*	

VRL-origin cows 1997

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
62	15/09/96	663	01/09/97	7	165	severe masts; culled Feb. '97
First Calvers:						
107	25/11/96	968.3	26/09/97	1	305	moved to Ask July 1996
111	02/02/97	570.6	25/10/97	1	265	moved to Ask July 1996
115	11/02/97	805.1	25/10/97	1	256	moved to Ask July 1996
110	20/02/97	898.4	27/11/97	1	280	moved to Ask July 1996
108	24/02/97	780.9	27/11/97	1	276	moved to Ask July 1996
Means		*804.7*		*1*	*276.4*	
Rest (sorted by lactlen):						
105	08/05/97	766.4	27/11/97	2	203	
103	21/04/97	682.6	27/11/97	2	220	
63	04/05/97	886.9	23/12/97	7	233	3 quarters
81	03/04/97	936.4	27/11/97	4	238	
104	28/04/97	740.9	23/12/97	2	239	
97	31/03/97	1180.8	23/12/97	2	267	
96	28/03/97	972.3	23/12/97	2	270	
101	24/03/97	883.6	23/12/97	2	274	
87	17/02/97	1240.4	23/12/97	3	309	
89	18/12/96	1184.1	25/10/97	3	311	
91	21/09/96	1184.3	27/08/97	2	340	
Means		*970*		*3*	*264*	

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Indigenous cows 1996

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
417	20/02/96	390	04/05/96	7	74	
409	08/03/96	285.3	31/05/96	9	84	
404	04/03/96	151.6	31/05/96	11	88	
402	22/07/96	315.7	30/10/96	12	100	
422	07/06/96	190.3	16/09/96	6	101	
405	17/02/96	245.1	31/05/96	11	104	
413	26/02/96	387.4	27/06/96	7	122	
401	29/04/96	621.2	21/09/96	11	145	
414	30/03/96	546.9	16/09/96	7	170	
426	26/03/96	525.7	16/09/96	6	174	
418	18/07/96	469	31/12/96	6	166	
419	17/03/96	599.2	16/09/96	6	183	
Means		*387.1*		*8.5*	*122.3*	
First lactation:						
432	19/05/96	858.3**	08/11/97	1	538	
Rest:						
427	26/02/96	749.7	16/09/96	6	203	
423	11/02/96	846.9	16/09/96	6	218	
428	21/03/96	987.7	12/11/96	5	236	
433	05/08/96	969.6	29/04/97	3	267	
406	25/03/96	932	21/12/96	10	271	
429	05/06/96	949.4	07/03/97	5	275	
424	24/04/96	874.2	25/01/97	6	276	
472	10/03/96	1093	21/12/96	6	286	
420	26/03/96	989.2	17/01/97	6	297	
421	24/02/96	1095.9	21/12/96	6	301	
410	26/03/96	974.7	25/01/97	8	305	
415	26/04/96	1231	03/03/97	7	311	
430	25/06/96	1178**	15/11/97	2	508	
Means		*990*		*5.9*	*289*	

** projected 305-day yield

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Index Farm B (contd.)

Brought-in cows 1996

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
4235	16/02/96	646.3	29/07/96	3	164	
4023	06/05/96	671.1	19/11/96	4	197	
Rest:						
4255	22/06/96	745.1	25/01/97	3	217	
4227	15/01/96	912.7	21/09/96	2	250	
4251	13/02/96	814	30/10/96	2	260	
4123	10/02/96	987	30/10/96	2	263	
4165	26/04/96	1085.8	25/01/97	3	274	
4188	20/03/96	1003.3	21/12/96	2	276	
4132	28/12/95	1236.9	01/10/96	2	278	
4224	07/02/96	1180.6	16/11/96	4	283	
4198	03/02/96	1064.7	16/11/96	2	287	
4109	06/12/95	1274.7	21/09/96	3	290	
4218	29/01/96	1121.6	16/11/96	2	292	
4225	25/02/96	1153.8	21/12/96	3	300	
4347	01/05/96	1131.8	03/03/97	2	306	
4243	21/02/96	1211.4	26/12/96	2	309	
4256	27/04/96	1054.8	03/03/97	2	310	
4230	01/06/96	888.7	08/04/97	3	311	
4122	12/12/95	1366.3	30/10/96	2	323	
4172	29/01/96	1249.2	21/12/96	3	327	
4233	20/04/96	1216**	22/08/97	3	489	
Means		*1089*		*2.5*	*297*	

** projected 305-day yield

Indigenous cows 1997

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
420	15/04/97	642.5	13/10/97	7	181	
415	03/05/97	797.2	08/11/97	8	189	
414	05/04/97	546.9	13/10/97	8	191	
426	04/04/97	525.7	15/10/97	7	194	
433	03/06/97	737.4	19/12/97	4	199	
Means		*649.9*		*6.8*	*190.8*	
Rest:						
427	06/03/97	749.7	15/10/97	7	223	
419	21/02/97	599.2	13/10/97	7	234	
429	28/04/97	987.5	19/12/97	6	235	
410	27/04/97	990.8	19/12/97	9	236	
472	11/03/97	1250.3	19/12/97	7	283	
421	24/02/97	1164.3	19/12/97	7	298	
Means		*957*		*7.2*	*251.5*	

Brought-in cows 1997

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
4243	05/05/97	899	08/11/97	3	187	
4230	20/05/97	718.7	26/11/97	4	190	
4443	17/03/97	588.4		1	170	
Means						
First calvers:						
4447	10/04/97	805.5	19/11/97	1	223	
4444	28/03/97	743.7	08/11/97	1	225	
4446	27/03/97	724.4	22/11/97	1	240	
4439	24/03/97	872.2	26/11/97	1	247	
4436	06/02/97	1024	25/10/97	1	261	
4445	27/03/97	1072.4	19/12/97	1	267	
4437	23/02/97	1245.3	26/11/97	1	276	
4440	03/03/97	872.6	19/12/97	1	291	
4438	22/02/97	1157.7	19/12/97	1	300	
4435	19/02/97	1018.7	19/12/97	1	303	
4434	20/11/96	1633.7	19/12/97	1	394	
4441	22/03/97	727	19/12/97	1	272	
4442	20/03/97	862	26/11/97	1	251	
Means		*982*		*1*	*273*	
Rest:						
4067	01/02/97	1090.4	26/11/97	2	298	
4256	11/04/97	726.8	22/11/97	3	225	
4188	12/03/97	1013	25/10/97	3	227	
4347	06/05/97	1020	19/12/97	3	227	
4123	09/02/97	955.1	15/11/97	3	279	
4218	07/02/97	1222.6	01/12/97	3	297	
4198	20/02/97	1284.4	19/12/97	3	302	
4122	14/01/97	1267.3	15/11/97	3	305	
4227	30/01/97	1254.9	01/12/97	3	305	
4132	11/12/96	1206.3	15/11/97	3	339	
4233	01/04/96	1598.5	25/08/97	3		
4225	06/04/97	1174.4	19/12/97	4	257	
4172	10/02/97	1296.7	26/11/97	4	289	
4023	29/04/97	916.8	19/12/97	5	234	
4224	04/03/97	1252.1	19/12/97	5	290	
Means		*1152*		*3.3*	*277*	

VRL

Askeaton-origin (Index Farm A) cows 1996

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
213	29/04/96	397	30/09/96	4	154	
Long lactation:						
215*	17/03/96	(911)	07/08/97	1	508	
Rest:						
211*	08/06/96	(593)	06/02/97	4	243	
210	04/05/96	852	16/01/97	4	257	
206	14/04/96	937	10/01/97	4	271	
208	16/03/96	949	13/12/96	4	272	
202	04/05/96	1157	20/02/97	4	292	
204	28/03/96	1585	23/01/97	4	301	
205	17/03/96	1298	06/02/97	4	326	
212	07/02/96	1507**	26/03/97	4	413	
214	29/05/96	929**	07/08/97	7	435	
207	13/05/96	1091**	07/08/97	4	451	
Means		*1090*		*4*	*326*	

* first milk recording Aug. 1996

VRL-origin cows total milk yield 1996

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
68	01/09/95	1014	13/09/96	4	378	
71	05/09/95	929	30/09/96	5	391	
58	18/12/95	1103	30/09/96	6	287	
92	31/08/95	1432	04/10/96	1	400	
First Calvers:						
95	12/03/96	1438	15/01/97	1	309	
98	21/04/96	1178	14/01/97	1	268	
99	26/04/96	1049	06/02/97	1	286	
100	01/05/96	1364	23/01/97	1	267	
102	19/05/96	941	23/01/97	1	249	
Means		*1194*		*1*	*276*	
Rest:						
55	24/03/96	1250	16/12/96	7	267	
57	19/01/96	1281	28/11/96	6	314	
74	21/01/96	1315	16/12/96	5	330	
83	12/01/96	1504**	23/01/97	2	377	
85	15/01/96	1506**	20/02/97	2	402	
Means		*1371*		*4*	*338*	

** projected 305-day yield

VRL (contd.)

Askeaton-origin (Index Farm A) cows total milk yield 1997

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
First Calvers:						
217	30/01/97	977	10/11/97	1	284	
216	05/02/97	906	21/11/97	1	289	
218	11/02/97	1073	21/11/97	1	283	
Means		*985*	*35751*	*1*	*285*	
Rest:						
210	26/03/97	1106		5		
205	14/04/97	1412**		5		
211	17/04/97	868		5		
202	22/04/97	1173**		5		
212	22/05/97	1238**		5		
204	04/05/97	909	28/11/97	5	208	
201	09/03/97	1151	21/11/97	5	257	
206	12v/03/97	1068	13/12/97	5	276	
208	19/02/97	1103	28/11/97	5.	282	
313	08/02/97	1522	05/12/97	9	300	
213	01/04/97	1078**	06/07/98	5	461	
Means		*1148*		*5*	*297*	

** projected 305-day yields

VRL-origin cows total milk yield 1997

ID	Calvdate	Yield	Drydate	Lactation	Lactlen	Comment
Short lactations:						
68	31/10/96	573	06/05/97	5	187	sold 6/5/97
57	04/02/97	284	06/05/97	7	91	sold 6/5/97
First Calvers:						
106	23/09/96	1294	07/08/97	1	318	
109	01/02/97	1039	10/11/97	1	282	
114	01/02/97	1210	21/11/97	1	293	
113	15/02/97	1075	28/11/97	1	286	
Means		*1154*		*1*	*295*	
Rest:						
74	03/04/97	932	01/12/97	6	242	
83	02/04/97	1152	05/12/97	3	247	
58	21/03/97	1145	28/11/97	7	252	
95	22/03/97	1222	05/12/97	2	258	
99	08/04/97	1119	24/12/97	2	260	
100	07/04/97	1280	24/12/97	2	261	
71	09/03/97	1046	28/11/97	6	264	
102	01/04/97	1114	24/12/97	2	267	
98	15/03/97	1203	24/12/97	2	284	
85	22/04/97	1284	20/02/98	3	304	
92	04/12/96	1524	10/11/97	2	341	
Means		*1067*		*4*	*267*	

APPENDIX 3

Monitor Study Haematology – Cow Group Average Results by Month.

Note: IF_A = Index Farm A, IF_B = Index Farm B, C_F = Control Farm. Group indicates origin of cows, i.e. group IF_B on Farm IF_B = indigenous cows on Index Farm B; group B_I on Farm IF_B = bought-in cows on Index Farm B, etc.

(*See* Appendix 1 for test abbreviations, units of measurement, and reference ranges)..

PCV (Packed Cell Volume %)

Year	Farm	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	IF_B	IF_B					30.7	30.8	31.7	33.3	33.6	33.2	30.1	33.3
		B_I					32.4	30.3	30.8	33.4	29.8	31.5	28.0	34.2
	IF_A	IF_A						29.5	30.2	31.7	34.5	34.1	30.7	34.4
		C_F						32.2	32.0	34.2	36.0	34.1	30.9	35.4
	C_F	IF_A				35.9	35.5	35.8	36.4	37.1	37.5	37.1	37.6	36.3
		C_F				31.8	34.0	34.0	35.1	36.2	35.8	35.1	34.7	34.0
1997	IF_B	IF_B	31.0	31.6	33.5	32.5	32.9	32.4	33.0	33.3	30.4	31.0	30.9	30.1
		B_I	29.1	29.7	30.4	31.0	31.2	32.0	31.3	32.7	29.4	31.7	30.5	29.2
	IF_A	IF_A	30.1	27.6	30.2	31.6	31.8	31.2	31.0	30.7	32.5	32.4	33.3	32.3
		C_F	30.3	30.2	31.5	34.1	30.9	33.0	30.7	31.3	32.7	32.2	33.3	32.0
	C_F	IF_A	35.6	36.6	34.6	34.9	34.7	35.8	33.9	33.6	34.5	33.5	36.1	35.0
		C_F	33.3	35.0	31.9	30.6	31.3	33.8	32.4	32.5	33.0	32.7	33.9	32.9
1998	IF_B	IF_B	30.3	30.6		32.4						34.3		
		B_I	31.1	30.4		32.6						33.4		
	IF_A	IF_A	31.5	29.2	31.6									
		C_F	33.6	30.6	32.2									
	C_F	IF_A	34.6	33.1	34.1									
		C_F	35.1	35.7	30.4									

WBC (White Cell Count x 10⁹/l)

Year	Farm	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1996	IF_B	IF_B					8.0	8.4	7.8	7.3	7.0	8.4	7.4	7.3
		B_I					8.2	9.4	7.8	7.7	7.5	7.6	7.1	7.0
	IF_A	IF_A						7.5	7.0	7.7	7.2	8.6	7.9	7.6
		C_F						8.0	7.3	6.7	7.2	7.7	7.7	8.2
	C_F	IF_A				6.6	7.3	7.0	7.9	7.4	6.6	7.5	7.4	6.0
		C_F				7.7	8.8	8.6	9.6	9.1	7.7	9.2	8.5	8.1
1997	IF_B	IF_B	6.2	6.2	6.7	7.2	8.0	6.8	7.4	7.4	7.4	7.1	7.9	7.0
		B_I	7.4	6.7	6.6	7.3	8.1	7.8	7.9	7.9	8.5	7.9	7.6	6.7
	IF_A	IF_A	6.4	6.2	6.2	6.7	8.2	6.7	8.2	7.8	7.5	7.5	7.6	6.3
		C_F	6.7	6.9	6.1	6.4	7.9	7.4	7.3	7.6	7.4	7.3	7.4	6.3
	C_F	IF_A	6.5	6.1	6.3	6.8	7.4	7.2	7.2	7.6	6.6	7.8	7.7	6.4
		C_F	7.6	8.1	8.8	7.9	7.9	8.5	9.2	9.3	8.3	10.4	9.1	8.2
1998	IF_B	IF_B	6.5	7.7		7.9						8.6		
		B_I	6.9	6.9		7.5						8.2		
	IF_A	IF_A	6.2	6.1	6.5									
		C_F	7.0	5.9	6.2									
	C_F	IF_A	6.7	6.6	6.2									
		C_F	8.2	7.3	6.9									

APPENDIX 4

Monitor Study Blood Biochemistry – Cow Group Average Results by Month.

(*See* Appendix 1 for test abbreviations, units of measurement, and reference ranges; *See* notes Appendix 3 for group abbreviations.

Index Farm A

Test	Year	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Albumin	1996	IF_A						29.9	32.0	34.0	32.3	33.2	31.6	31.4
		C_F						32.9	32.2	34.2	32.8	33.1	32.0	30.8
	1997	IF_A	30.7	30.4	34.1	31.7	31.0	32.9	36.9	36.2	31.0	31.7	36.5	32.6
		C_F	30.1	29.6	33.1	32.6	32.1	34.3	37.4	37.3	31.5	31.9	36.1	30.8
	1998	IF_A	32.5	32.2	32.1									
		C_F	31.8	32.9	33.1									
Globulin	1996	IF_A						47.3	49.9	41.6	51.2	42.6	46.4	48.6
		C_F						45.9	49.2	40.8	51.0	41.7	43.7	48.6
	1997	IF_A	45.9	42.6	51.1	47.6	50.4	44.0	31.0	34.0	50.2	55.4	53.5	46.0
		C_F	48.1	47.6	52.9	44.8	45.7	39.6	27.5	31.5	48.3	48.8	50.1	45.8
	1998	IF_A	45.0	45.2	41.0									
		C_F	43.0	42.6	38.4									
AST	1996	IF_A						111.3	94.1	85.3	94.9	90.4	109.4	79.3
		C_F						84.6	83.2	71.1	78.3	82.6	92.6	77.6
	1997	IF_A	70.4	69.9	72.4	83.4	83.2	91.2	74.8	75.5	84.0	77.3	88.0	69.9
		C_F	72.5	71.8	72.9	81.8	82.7	91.4	76.5	70.8	87.4	75.2	84.7	68.3
	1998	IF_A	61.4	59.9	59.5									
		C_F	62.4	57.6	64.7									
βHB	1996	IF_A						0.5	0.9	0.7	0.5	0.5	0.5	0.4
		C_F						0.5	0.5	0.6	0.5	0.5	0.5	0.4
	1997	IF_A	0.7	0.6	0.5	0.9	0.7	0.7	0.5	0.5	0.5	0.3	0.4	0.3
		C_F	0.6	0.5	0.6	0.8	0.7	0.8	0.4	0.5	0.4	0.3	0.4	0.4
	1998	IF_A	0.7	0.5	0.5									
		C_F	0.4	0.5	0.5									
Ca	1996	IF_A						2.5			2.4			2.6
		C_F						2.6			2.4			2.7
	1997	IF_A			2.6			2.8			2.6			2.6
		C_F			2.6			2.7			2.6			2.5
	1998	IF_A			2.4									
		C_F			2.4									
CPK	1996	IF_A						157.3			192.8			218.7
		C_F						131.9			152.1			164.8
	1997	IF_A			181.2			206.7	117.5	245.4	163.5			102.4
		C_F			138.4			168.6		157.2	165.9			93.8
	1998	IF_A			118.4									
		C_F			114.3									
Copper	1996	IF_A						14.3	9.5	9.5	14.6	12.8	11.6	15.3
		C_F						12.6	9.1	9.3	13.5	12.5	10.6	13.8
	1997	IF_A	12.4	12.3	12.4	12.1	12.9	11.8	12.2	11.8	12.9	13.3	12.1	15.1
		C_F	12.3	11.6	11.8	10.8	10.8	9.9	10.0	10.8	12.1	12.0	11.5	14.6
	1998	IF_A	11.0	9.8	11.2									
		C_F	10.8	8.2	9.7									

Test	Year	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
GGT	1996	IF_A						21.8	21.4	16.0	16.2	19.1	14.6	16.0
		C_F						14.2	16.1	16.2	14.4	17.2	14.5	15.8
	1997	IF_A	19.6	20.3	19.1	19.3	17.5	19.1	16.1	15.5	22.9	21.0	20.1	25.6
		C_F	20.7	19.8	18.0	18.6	18.3	18.4	13.8	14.9	21.0	20.1	19.9	26.8
	1998	IF_A	21.1	21.1	19.2									
		C_F	20.1	18.6	19.0									
GLDH	1996	IF_A						27.0	15.8	7.3	18.1	17.0	31.5	11.1
		C_F						16.7	11.6	10.4	13.1	19.9	31.3	18.8
	1997	IF_A	8.5	9.7	10.4	11.7	14.8	14.6	12.8	19.3	13.9	12.5	13.8	8.4
		C_F	14.6	9.5	12.5	14.8	12.5	17.7	18.1	20.2	16.9	10.6	18.4	9.0
	1998	IF_A	5.5	8.8	7.6									
		C_F	6.3	6.3	7.9									
Glucose	1996	IF_A												3.5
		C_F												3.3
	1997	IF_A	3.3	3.4	3.8	3.3	3.7	3.5	3.2	3.5	3.4	3.5	3.3	3.3
		C_F	3.4	3.4	3.6	3.1	3.5	3.1	3.1	3.5	3.2	3.4	3.3	3.3
	1998	IF_A	3.2	3.3	3.4									
		C_F	3.4	3.4	3.4									
Mg	1996	IF_A						0.8	0.9	0.9	0.8	0.9	0.7	1.0
		C_F						1.0	1.0	1.0	0.9	1.0	0.8	1.0
	1997	IF_A	1.0	0.8	1.0	0.9	0.8	0.9	0.9	1.0	0.9	0.9	0.7	1.0
		C_F	1.1	0.9	1.1	1.0	0.9	1.0	0.9	1.0	1.0	1.0	0.8	1.0
	1998	IF_A	1.0	1.0	0.9									
		C_F	1.0	1.1	1.0									
P	1996	IF_A						1.8	1.7	2.0	1.6	1.8	2.0	1.8
		C_F						1.8	1.9	2.0	1.5	1.9	1.9	1.9
	1997	IF_A	1.7	1.9	1.8	2.0	1.6	1.7	1.3	1.6	2.3	2.0	1.8	1.9
		C_F	1.7	2.0	1.9	1.8	1.7	1.8	1.1	1.6 .	2.0	1.9	1.7	1.8
	1998	IF_A	1.7	2.0	1.8									
		C_F	1.6	1.9	1.9									
Se	1996	IF_A						2.5			3.0			3.0
		C_F						2.8			3.7			3.2
	1997	IF_A			2.4			2.7			2.3			2.0
		C_F			2.5			2.7			2.2			1.8
	1998	IF_A			1.2									
		C_F			1.2									
Urea	1996	IF_A						3.3	7.4	4.6	7.8	5.4	7.8	5.5
		C_F						3.0	6.0	4.2	6.9	4.8	6.9	5.2
	1997	IF_A	4.2	3.7	5.1	6.2	3.2	7.5	2.6	6.3	8.5	6.3	3.0	3.2
		C_F	3.7	3.3	4.9	5.9	3.4	7.1	1.7	5.8	8.5	5.7	2.8	3.1
	1998	IF_A	3.5	3.5	5.8									
		C_F	3.2	3.4	6.4									
Zn	1996	IF_A						10.9			14.4			14.4
		C_F						12.8			14.1			13.7
	1997	IF_A			14.5	16.3	13.2	14.0		25.1	15.9			18.7
		C_F			15.6	16.9	13.9	14.3		24.7	15.0			17.9
	1998	IF_A			15.2									
		C_F			15.5									

Index Farm B

Test	Year	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Albumin	1996	IF_B					31.6	32.4	32.9	35.6	32.9	32.6	31.7	31.0
		B_I					31.1	32.2	33.0	36.9	33.0	33.1	31.7	30.8
	1997	IF_B	31.2	30.4	31.1	30.1	32.6	32.7	38.3	36.1	28.8	31.7	31.3	30.3
		B_I	31.0	30.8	31.4	30.3	32.4	33.3	37.8	36.7	29.5	32.5	32.5	31.4
	1998	IF_B	30.8	33.4		31.9		32.2				33.2		
		B_I	31.3	34.7		32.0		32.0				33.8		
Globulin	1996	IF_B					48.3	50.5	46.0	51.8	46.8	40.5	45.7	42.7
		B_I					45.5	50.9	45.6	48.5	45.0	39.5	44.2	42.0
	1997	IF_B	48.0	47.9	46.8	45.9	45.6	47.9	34.0	32.2	50.7	47.8	47.1	48.9
		B_I	46.4	43.5	41.8	47.7	46.1	48.6	35.2	32.8	52.0	44.7	42.1	46.5
	1998	IF_B	41.8	40.1		42.6		42.6				48.4		
		B_I	41.1	41.3		40.9		44.2				48.4		
AST	1996	IF_B					78.7	85.8	79.3	88.6	92.1	107.6	130.7	75.7
		B_I					110.0	100.8	80.1	92.6	83.1	92.0	100.2	73.6
	1997	IF_B	63.2	58.3	63.1	69.7	84.1	90.4	87.6	84.5	83.5	68.7	90.6	68.0
		B_I	65.0	65.7	65.1	77.8	89.4	98.0	82.8	84.3	81.4	68.4	74.1	60.7
	1998	IF_B	64.7	58.8		71.6		87.1				89.0		
		B_I	60.0	59.6		74.5		78.1				81.5		
βHB	1996	IF_B					0.6	0.8	0.8	0.5	0.5	0.5	0.5	0.7
		B_I					0.5	0.6	0.6	0.5	0.5	0.4	0.5	0.7
	1997	IF_B	0.8	0.4	0.7	0.6	0.8	0.5	0.6	0.5	0.6	0.4	0.5	0.4
		B_I	0.9	0.6	1.1	0.6	0.8	0.5	0.6	0.5	0.8	0.5	0.6	0.5
	1998	IF_B	0.4	0.6		0.4		0.6				0.3		
		B_I	0.4	0.6		0.5		0.5				0.3		
Ca	1996	IF_B					2.6			2.4			2.6	
		B_I					2.5			2.5			2.6	
	1997	IF_B		2.5			2.3			2.3			2.4	
		B_I		2.5			2.3			2.3			2.4	
	1998	IF_B		2.2				2.6				2.5		
		B_I		2.3				2.5				2.6		
CPK	1996	IF_B								195.4			206.5	
		B_I								193.4			337.4	
	1997	IF_B		98.6			126.3		141.8	156.1			176.9	
		B_I		132.4			131.7		146.8	381.1			166.6	
	1998	IF_B		108.2				153.7				165.2		
		B_I		130.4				160.6				145.5		
Copper	1996	IF_B					10.8	10.9	10.0	14.4	12.4	12.5	12.5	12.5
		B_I					11.0	10.8	9.5	13.4	11.7	11.9	11.6	12.7
	1997	IF_B	11.3	11.6	13.2	11.2	11.7	11.5	13.0	12.8	11.2	13.6	11.5	12.6
		B_I	11.6	11.2	11.4	10.6	11.1	10.8	12.2	11.7	10.2	13.1	10.9	12.8
	1998	IF_B	10.0	10.1		11.4		12.3				12.2		
		B_I	10.1	10.0		11.5		10.9				10.1		
GGT	1996	IF_B					16.9	14.9	19.2	17.3	16.9	17.0	15.2	18.4
		B_I					24.6	21.7	19.5	16.1	17.1	18.1	16.4	17.4
	1997	IF_B	15.6	16.5	15.5	14.6	20.9	17.8	14.1	21.6	22.2	20.8	21.5	19.2
		B_I	19.6	17.2	14.9	17.4	25.0	20.6	33.7	18.0	23.6	22.1	22.6	20.1
	1998	IF_B	18.2	17.6		18.3		20.2				19.5		
		B_I	19.0	18.7		17.7		19.7				19.1		

Index Farm B contd.

Test	Year	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
GLDH	1996	IF_B					13.6	13.3	9.9	12.2	23.2	19.1	27.7	17.6
		B_I					29.5	23.1	11.0	14.3	14.9	19.9	22.2	17.2
	1997	IF_B	11.9	11.8	12.9	10.5	15.7	21.0	24.8	16.6	10.4	11.6	16.1	10.0
		B_I	15.7	15.5	19.7	14.3	24.0	25.3	23.2	15.4	14.4	12.3	12.2	8.0
	1998	IF_B	11.0	9.4		11.0		16.3				15.8		
		B_I	7.9	11.5		14.1		12.6				17.8		
Glucose	1996	IF_B												3.3
		B_I												3.3
	1997	IF_B	3.5	3.4	3.4	3.7	3.4	3.5	3.4	3.3	3.3	3.6	3.4	3.5
		B_I	3.2	3.2	3.0	3.7	3.4	3.5	3.5	3.3	3.4	3.5	3.4	3.5
	1998	IF_B	3.5	3.4		3.6		3.3				3.5		
		B_I	3.4	3.7		3.4		3.4				3.3		
Mg	1996	IF_B					1.0	0.9	0.9	0.9	0.8	0.9	0.9	0.9
		B_I					1.0	0.9	1.0	1.0	0.9	1.0	1.0	1.0
	1997	IF_B	0.8	0.9	0.9	0.9	0.9	0.9	1.0	0.9	1.0	0.9	0.8	0.9
		B_I	0.9	1.0	1.0	1.0	0.9	1.1	1.0	1.0	1.0	1.0	1.0	1.0
	1998	IF_B	0.9	1.0		0.9		1.1				0.8		
		B_I	1.0	1.0		1.1		1.1				0.8		
P	1996	IF_B					1.6	1.7	2.1	1.8	1.9	1.9	1.8	2.2
		B_I					1.4	1.7	2.2	2.1	1.9	2.1	2.0	2.2
	1997	IF_B	2.2	1.8	1.7	1.7	1.9	2.0	1.7	1.8	2.2	1.9	1.8	1.9
		B_I	2.1	1.8	1.8	1.8	2.0	2.1	1.8	1.9	2.3	2.0	1.9	2.1
	1998	IF_B	1.8	1.8		1.8		1.7				1.9		
		B_I	1.8	1.9		1.9		1.8				2.0		
Se	1996	IF_B					1.9			1.2			1.1	
		B_I					3.1			2.0			1.8	
	1997	IF_B		1.5			1.7			1.1			1.0	
		B_I		2.2			2.5			2.0			1.9	
	1998	IF_B		0.9				1.5				1.5		
		B_I		1.6				1.7				1.7		
Urea	1996	IF_B					2.2	2.7	7.0	6.7	6.3	7.6	8.1	5.1
		B_I					2.3	2.8	7.4	6.9	5.9	7.3	7.8	5.0
	1997	IF_B	5.5	4.2	4.1	5.2	5.4	6.5	12.4	7.0	6.8	6.1	5.6	5.0
		B_I	5.1	4.4	4.6	4.7	4.8	8.3	10.2	6.4	6.0	7.0	5.8	5.2
	1998	IF_B	3.7	5.2		6.8		5.2				8.7		
		B_I	3.5	4.9		6.6		5.1				7.9		
Zn	1996	IF_B					15.1	17.8	15.8	14.7		20.0		
		B_I					13.4	18.0	15.6	14.5		20.0		
	1997	IF_B	16.6	14.1	15.5	14.2	14.1	13.7	14.7	14.9			17.7	
		B_I	15.2	13.6	15.0	13.0	13.6	12.8	13.2	12.9			18.4	
	1998	IF_B						14.8				15.9		
		B_I						12.8				14.7		

Control Farm

Test	Year	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Albumin	1996	IF_A				31.1	33.5	32.4	33.9	33.1	34.4	33.8	33.8	33.4
		C_F				33.3	34.2	33.0	34.3	33.9	33.9	34.1	32.9	32.6
	1997	IF_A	33.1	32.5	30.6	32.3	32.3	32.8	39.2	38.8	32.3	32.9	34.3	33.3
		C_F	31.6	31.6	30.6	31.3	33.2	33.6	39.8	38.5	31.8	33.2	35.6	34.1
	1998	IF_A	34.1	34.0	33.0									
		C_F	35.2	35.7	34.2									
Globulin	1996	IF_A				50.7	33.2	48.9	53.2	50.0	45.0	42.8	44.8	45.4
		C_F				46.4	46.6	46.6	50.4	45.8	42.7	43.6	41.9	45.9
	1997	IF_A	41.9	44.8	47.8	45.4	42.6	43.4	32.6	33.1	51.6	48.8	42.8	47.0
		C_F	45.6	45.3	46.3	47.3	44.0	42.1	32.3	32.5	47.3	47.4	39.6	49.9
	1998	IF_A	39.1	44.0	42.2									
		C_F	37.8	41.2	38.1									
AST	1996	IF_A				83.0	88.5	106.2	103.4	94.8	119.3	125.9	142.8	94.6
		C_F				74.1	79.3	76.7	90.3	90.6	89.1	95.7	121.4	83.9
	1997	IF_A	59.6	63.2	66.5	67.9	88.5	92.2	107.8	90.7	116.4	103.3	93.6	128.7
		C_F	68.9	70.4	70.2	90.9	83.9	107.3	129.5	89.0	93.9	89.8	87.3	109.3
	1998	IF_A	86.2	81.7	100.2									
		C_F	67.5	74.9	91.2									
βHB	1996	IF_A				0.8	0.4	0.4	0.5	0.6	0.5	0.5	0.5	0.5
		C_F				0.4	0.4	0.4	0.5	0.6	0.5	0.6	0.5	0.5
	1997	IF_A	0.4	0.4	0.5	0.5	0.6	1.9	0.5	0.5	0.4	0.6	0.7	0.5
		C_F	0.3	0.4	0.5	0.6	0.6	0.8	0.5	0.5	0.3	0.4	0.4	0.4
	1998	IF_A	0.5	0.5	0.5									
		C_F	0.3	0.4	0.7									
Ca	1996	IF_A				2.7		2.6			2.6			2.4
		C_F				2.7		2.7			2.6			2.4
	1997	IF_A			2.5				2.5			2.7		2.6
		C_F			2.5				2.5			2.6		2.6
	1998	IF_A	2.4											
		C_F	2.6											
CPK	1996	IF_A				175.5		249.9			161.8			135.9
		C_F				131.0		130.1			131.6			108.4
	1997	IF_A	98.4		115.1				198.1	166.4		183.9		99.7
		C_F	102.7		105.3				198.7	155.0		129.2		116.9
	1998	IF_A	120.2											
		C_F	83.5											
Copper	1996	IF_A				15.2	11.1	12.9	12.3	14.0	16.3	12.0	14.5	14.3
		C_F				14.4	10.1	11.0	10.9	11.7	14.5	10.2	13.1	13.0
	1997	IF_A	14.2	16.3	15.2	14.4	14.3	14.6	14.8	14.4	12.2	12.7	13.2	15.7
		C_F	12.9	13.8	13.0	13.1	12.7	12.2	13.1	13.4	11.3	11.2	11.2	13.3
	1998	IF_A	12.6	12.4	13.0									
		C_F	10.7	9.7	10.9									
GGT	1996	IF_A				16.6	22.6	17.5	24.2	23.6	19.8	22.5	30.1	32.1
		C_F				14.9	18.7	13.3	21.7	20.0	18.1	16.1	22.9	30.8
	1997	IF_A	27.1	23.4	25.2	21.2	28.6	23.9	19.7	20.4	29.0	26.5	26.9	29.3
		C_F	23.0	18.2	18.7	25.7	18.9	22.5	20.3	19.4	23.7	22.6	26.5	23.8
	1998	IF_A	27.5	26.5	27.7									
		C_F	20.2	19.2	20.6									

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Test	Year	Group	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
GLDH	1996	IF_A				12.3	20.2	24.8	24.7	21.9	28.0	49.2	8.3	25.2
		C_F				11.4	17.6	16.7	20.7	19.4	17.6	35.5	4.8	40.3
	1997	IF_A	14.9	9.7	11.3	8.7	16.2	18.1	38.7	33.9	59.7	32.1	19.4	35.2
		C_F	25.8	15.0	14.9	28.4	20.1	30.6	68.9	46.8	56.4	29.6	15.8	22.5
	1998	IF_A	18.6	20.8	27.7									
		C_F	11.0	21.9	23.9									
Glucose	1996	IF_A									5.8	3.6	3.9	3.6
		C_F									3.5	3.4	3.7	3.4
	1997	IF_A	3.7	3.7	3.6	3.6	3.6	3.4	3.2	3.3	3.3	3.4	3.3	3.4
		C_F	3.6	3.5	3.4	3.4	3.6	3.3	3.3	3.2	3.2	3.4	3.4	3.5
	1998	IF_A	3.5	3.3	3.8									
		C_F	3.7	3.2	3.4									
Mg	1996	IF_A				1.0	0.9	0.9	1.0	1.0	1.0	1.0	1.2	1.1
		C_F				1.0	0.9	0.9	1.0	1.0	1.0	0.9	1.1	1.1
	1997	IF_A	1.0	1.0	1.0	1.1	1.1	1.1	1.1	1.0	1.0	1.0	1.2	1.0
		C_F	1.0	1.0	1.0	1.1	1.2	1.1	1.2	1.1	1.0	1.0	1.2	1.0
	1998	IF_A	1.0	1.0	1.1									
		C_F	1.0	1.0	1.0									
P	1996	IF_A				1.8	1.9	1.4	1.6	2.1	1.7	1.8	1.8	1.9
		C_F				1.8	1.7	1.6	1.4	2.0	1.7	1.8	1.8	1.8
	1997	IF_A	1.8	1.6	1.5	1.5	1.5	1.9	1.8	1.4	1.6	1.4	1.7	1.7
		C_F	1.8	1.7	1.6	1.5	1.4	1.9	1.8	1.4	1.6	1.3	1.5	1.9
	1998	IF_A	1.7	1.5	1.6									
		C_F	1.6	1.6	1.5									
Se	1996	IF_A				3.0		3.9			5.2			4.3
		C_F				2.7		3.9			4.8			3.5
	1997	IF_A			3.2				3.6			4.0		3.7
		C_F			2.4				3.6			4.2		3.3
Urea	1996	IF_A				7.3	6.7	6.3	6.0	8.2	7.5	8.9	7.8	5.3
		C_F				6.3	5.5	5.2	5.1	7.0	6.7	8.3	6.8	4.9
	1997	IF_A	3.8	3.7	3.1	5.6	5.7	5.6	4.5	6.0	6.4	8.4	8.1	5.5
		C_F	3.5	3.5	3.0	6.3	5.5	4.9	4.6	5.5	5.4	8.2	7.5	5.1
	1998	IF_A	4.4	4.2	6.8									
		C_F	3.5	4.1	6.1									
Zn	1996	IF_A				18.5	11.7	12.8			16.9	13.7	16.5	15.6
		C_F				15.2	11.9	13.4			14.6	13.7	14.9	15.9
	1997	IF_A	13.9	13.9	13.8	12.6	13.2	14.4	16.1	13.9	13.9	17.5	16.4	19.7
		C_F	13.2	13.4	13.5	14.4	13.0	15.3	15.3	14.4	14.4	16.8	16.8	19.6
	1998	IF_A	16.5	8.0										
		C_F	15.8											

APPENDIX 5

Longitudinal Study Haematology Results – Group Averages by Sampling.

(*See* Appendix 1 for test abbreviations, units of measurement, and reference ranges).

PCV (Packed Cell Volume %)

Year	Farm	Type	Jan	Feb	Mar	Apr	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1997	LS1	Cows			36.9		35.1	42.1					
		Growing[1]			36.0		34.8			30.6	32.0		33.7
	LS2	Cows				30.0		32.2	28.8	36.4			
		Growing				35.5		36.0	33.4	39.9			
	LS3	Cows		31.6			32.7			32.5			31.5
		Growing		33.9			31.9			37.0			34.0
	LS5	Cows	28.4			29.7		33.6				33.3	
		Growing	30.2			32.5		36.1				33.9	
1998	LS1	Cows							31.5				
		Growing					36.9	37.2					
	LS2	Cows		31.4	32.3				37.2				
		Growing		34.0	31.8				37.3				
	LS3	Cows				32.5			33.3		30.4		
		Growing				37.1			39.2				
	LS5	Cows	32.9			30.3			29.5				
		Growing	32.0			28.8			30.6				

[1]Growing stock (0 – 2 years old approx.)

WBC (White Cell Count x 10^9/l)

Year	Farm	Type	Jan	Feb	Mar	Apr	Jun	Jul	Aug	Sep	Oct	Nov	Dec
1997	LS1	Cows			7.5		8.2	8.8					
		Growing			8.0		9.2			9.9	10.1		9.5
	LS2	Cows				6.4		7.6	8.6	7.7			
		Growing				10.9		11.1	9.9	12.3			
	LS3	Cows		7.1			8.0			8.2			7.3
		Growing		9.8			5.9			8.8			8.1
	LS5	Cows	6.0			6.9		6.6				6.6	
		Growing	7.7			7.4		7.7				9.0	
1998	LS1	Cows							7.5				
		Growing					9.0	12.7					
	LS2	Cows		7.4	12.8				7.4				
		Growing		9.5	7.9				8.7				
	LS3	Cows				7.5			8.4		8.9		
		Growing				8.7			9.3				
	LS5	Cows	5.3			5.7			7.8				
		Growing	7.7			6.7			7.2				

APPENDIX 6

Longitudinal Study Biochemistry Results – Group Averages by Sampling.

(*See* Appendix 1 for test abbreviations, units of measurement, and reference ranges).

Test	Years	Farm	Type	Jan	Feb	Mar	Apr	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Albumin	1997	LS1	Cows			30.2		30.4						
			Growing		31.0			33.4			29.6	27.9		32.8
		LS2	Cows				29.7		37.6	26.2	30.9			
			Growing				28.6		35.8	30.8	30.5			
		LS3	Cows		31.1			32.5			31.4			34.9
			Growing		29.1			28.8			31.2			32.9
		LS5	Cows	31.1			31.9		41.2				34.6	
			Growing	28.7			32.0		40.3				33.1	
	1998	LS1	Cows							34.0				
			Growing					31.4						
		LS2	Cows		30.9	29.5	30.6			33.9				
			Growing		30.2	30.2	29.9			33.4				
		LS3	Cows				30.4			34.2		31.0		
			Growing				28.3			31.8				
		LS5	Cows	32.3			30.9			33.2				
			Growing	31.6			29.5			32.8				
AST	1997	LS1	Cows			97.0		85.5						
			Growing			75.0		74.0			80.0	78.9		91.2
		LS2	Cows				94.7		78.1	75.8	93.6			
			Growing				68.4		68.6	86.2	88.0			
		LS3	Cows		58.7			86.6			86.5			61.6
			Growing		62.4			78.4			72.2			57.8
		LS5	Cows	61.2			90.8		64.2				73.8	
			Growing	61.8			96.8		64.8				93.8	
	1998	LS1	Cows							101.0				
			Growing					75.2						
		LS2	Cows		81.0	77.0				97.3				
			Growing		97.6	97.8				101.4				
		LS3	Cows				70.7			80.4		76.0		
			Growing				69.6			86.0				
		LS5	Cows	53.4			64.0			79.3				
			Growing	58.5			79.3			76.7				
βHB	1997	LS1	Cows			0.3		0.7						
			Growing			0.5		0.5			0.4	1.1		0.4
		LS2	Cows				0.9		0.5	0.5	0.5			
			Growing				0.4		0.4	0.5	0.4			
		LS3	Cows		0.4			0.4			0.4			0.3
			Growing		0.2			0.3			0.2			0.3
		LS5	Cows	0.6			0.6		0.5				0.5	
			Growing	0.4			0.6		0.5				0.6	
	1998	LS1	Cows							0.4				
			Growing					0.4						
		LS2	Cows		0.4		0.5			0.4				
			Growing		0.3		0.3			0.4				
		LS3	Cows				0.3			0.5		0.4		
			Growing				0.3			0.4				
		LS5	Cows	0.5			0.5			0.5				
			Growing	0.5			0.4			0.5				

Longitudinal Study Farms Biochemistry Results contd.

Test	Years	Farm	Type	Jan	Feb	Mar	Apr	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Ca	1997	LS1	Cows		2.4			2.3						
			Growing		2.4			2.5			2.7	2.7		2.7
		LS2	Cows				2.7		2.3	2.3	2.4			
			Growing				2.8		2.6	2.4	2.5			
		LS3	Cows		2.6			2.8			2.7			2.3
			Growing		2.7			2.8			2.7			2.4
		LS5	Cows	2.5			2.6		2.5				2.5	
			Growing	2.7			2.9		2.6				2.6	
	1998	LS1	Cows							2.8				
			Growing					2.7						
		LS2	Cows		2.6					2.7				
			Growing		2.5					2.7				
		LS3	Cows				2.3			2.6		2.5		
			Growing				2.5			2.6				
		LS5	Cows	2.5			2.5			2.4				
			Growing	2.5			2.5			2.4				
CPK	1997	LS1	Cows			814.2		268.2						
			Growing			178.4		213.7			270.7	237.4		246.0
		LS2	Cows				483.7			117.4	162.7			
			Growing			213.8				260.3	200.8			
		LS3	Cows		109.6			175.3			162.0			108.8
			Growing		179.0			195.6			217.6			195.0
		LS5	Cows	90.5			154.7		202.2				169.5	
			Growing	185.8			326.0		208.0				229.8	
	1998	LS1	Cows							1586.0				
			Growing					240.4						
		LS2	Cows		87.1					199.0				
			Growing		109.8					150.8				
		LS3	Cows				208.3			185.1		131.7		
			Growing				160.0			221.2				
		LS5	Cows	89.4			131.0			117.0				
			Growing	101.0			249.8			144.3				
Copper	1997	LS1	Cows			10.0		11.2						
			Growing		12.6			7.8			8.7	10.5		10.3
		LS2	Cows				12.5		11.0	14.7	12.3			
			Growing				11.3		10.6	11.6	11.5			
		LS3	Cows		11.1			11.9			10.7			13.5
			Growing		11.7			11.1			9.4			12.7
		LS5	Cows	12.1			11.4		11.2				12.9	
			Growing	12.1			10.5		10.4				13.0	
	1998	LS1	Cows							10.6				
			Growing					9.9						
		LS2	Cows		12.9					13.0				
			Growing		13.1					11.6				
		LS3	Cows				13.1			11.0		10.3		
			Growing				12.2			10.0				
		LS5	Cows	13.2			12.3			11.5				
			Growing	14.0			15.8			12.5				

Longitudinal Study Farms Biochemistry Results contd.

Test	Years	Farm	Type	Jan	Feb	Mar	Apr	Jun	Jul	Aug	Sep	Oct	Nov	Dec
GGT	1997	LS1	Cows			25.5		27.0						
			Growing		21.3			16.0			17.7	20.0		17.3
		LS2	Cows				19.3		16.0	18.8	25.6			
			Growing				11.6		10.2	18.8	20.6			
		LS3	Cows		20.4			20.3			24.3			37.8
			Growing		12.8			11.8			21.8			34.8
		LS5	Cows	19.5			14.2		10.7				20.5	
			Growing	20.0			15.3		11.8				23.3	
	1998	LS1	Cows							23.0				
			Growing					17.2						
		LS2	Cows		62.3	27.0	40.7			17.1				
			Growing		94.5	91.5	85.9			15.8				
		LS3	Cows				26.3			20.4		20.0		
			Growing				29.6			21.8				
		LS5	Cows	16.6			16.2			17.8				
			Growing	29.8			20.3			19.3				
Globulin	1997	LS1	Cows			50.0		47.7						
			Growing		34.7			38.1			43.9	45.7		36.1
		LS2	Cows				35.3		33.6	49.4	49.5			
			Growing				26.3		30.6	36.3	48.5			
		LS3	Cows		37.3			47.7			46.8			45.6
			Growing		40.4			49.8			47.3			41.4
		LS5	Cows	37.2			42.2		21.7				44.1	
			Growing	36.8			39.4		20.7				44.3	
	1998	LS1	Cows							51.3				
			Growing					41.8						
		LS2	Cows		41.1	29.8	40.4			44.1				
			Growing		41.3	43.3	39.3			41.8				
		LS3	Cows				44.5			40.9		47.8		
			Growing				45.7			42.4				
		LS5	Cows	27.2			41.0			41.7				
			Growing	29.4			48.4			45.2				
GLDH	1997	LS1	Cows			19.0		25.0						
			Growing		39.7						31.5	19.0		19.7
		LS2	Cows				12.4		14.4	18.1				
			Growing				13.0		45.4	39.4				
		LS3	Cows		11.0			14.6			25.5			17.4
			Growing		10.4			10.2			19.8			13.6
		LS5	Cows	12.0			23.0		14.3				12.8	
			Growing	8.2			15.0		11.3				30.8	
	1998	LS1	Cows							17.0				
			Growing					13.8						
		LS2	Cows		35.8	26.0	17.3			10.6				
			Growing		86.3	108.8	108.5			29.0				
		LS3	Cows				10.0			13.0		12.8		
			Growing				25.6			28.0				
		LS5	Cows	6.4			10.4			16.3				
			Growing	15.5			22.3			14.7				

Longitudinal Study Farms Biochemistry Results contd.

Test	Years	Farm	Type	Jan	Feb	Mar	Apr	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Glucose	1997	LS1	Cows			3.4		3.9						
			Growing		4.3			4.0			3.9	4.8		3.9
		LS2	Cows				3.6.		3.5	2.9	3.4			
			Growing				3.8		3.8	3.4	3.3			
		LS3	Cows		3.4			3.3			3.4		•	3.4
			Growing		3.5			3.9			3.7			3.8
		LS5	Cows	3.4			3.7		3.2				3.2	
			Growing	4.2			4.4		3.6				3.3	
	1998	LS1	Cows							3.0				
			Growing					4.4						
		LS2	Cows							3.4				
			Growing							3.2				
		LS3	Cows				3.8			3.3		3.1		
			Growing				3.8			3.6				
		LS5	Cows	3.4			3.5			3.5				
			Growing	3.6			3.9			3.7				
Mg	1997	LS1	Cows			0.8		1.0						
			Growing			0.8		0.8			1.0	0.8		0.9
		LS2	Cows				1.1		1.2	0.9	1.0			
			Growing				0.9		1.0	0.9	1.0			
		LS3	Cows		0.9			0.8			0.7			0.9
			Growing		0.8			0.8			0.9			0.9
		LS5	Cows	0.9			1.1		1.0				1.0	
			Growing	0.9			1.1		1.1				1.0	
	1998	LS1	Cows							1.2				
			Growing					0.9						
		LS2	Cows		1.1					1.1				
			Growing		1.0					1.1				
		LS3	Cows				0.9			1.1		1.0		
			Growing				0.7			1.0				
		LS5	Cows	1.0			1.1			1.0				
			Growing	0.9			1.1			1.0				
P	1997	LS1	Cows			2.0		2.4						
			Growing			2.8		2.6			2.9	2.6		2.5
		LS2	Cows				1.9		1.2	2.0	1.9			
			Growing				2.7		2.1	2.7	2.3			
		LS3	Cows		1.8			1.9			2.0			1.9
			Growing		2.2			2.6			2.3			2.2
		LS5	Cows	2.2			1.5	1.9					1.9	
			Growing	2.5			1.5	2.6					2.4	
	1998	LS1	Cows							2.3				
			Growing					2.8						
		LS2	Cows		1.8					2.1				
			Growing		2.0					2.2				
		LS3	Cows				1.3			2.1		2.2		
			Growing				2.1			2.4				
		LS5	Cows	1.9			1.7			1.7				
			Growing	1.9			1.9			1.8				

Longitudinal Study Farms Biochemistry Results contd.

Test	Years	Farm	Type	Jan	Feb	Mar	Apr	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Se	1997	LS1	Cows			1.3		1.5						
			Growing		2.3			1.1			0.8	1.0		1.2
		LS2	Cows				2.8		2.3		2.5			
			Growing				2.6		2.0		2.8			
		LS3	Cows		2.2			1.7			1.3			1.3
			Growing		1.3			0.7			0.7			0.8
		LS5	Cows	2.4			2.1		2.4				1.9	
			Growing	1.6			1.4		1.6				1.5	
	1998	LS1	Cows							2.3				
			Growing					0.8						
		LS2	Cows		2.0					3.1				
			Growing		2.2					2.9				
		LS3	Cows				1.8			2.0		1.4		
			Growing				1.4			1.0				
		LS5	Cows	2.5			2.8			2.5				
			Growing	2.1			2.5			2.3				
Urea	1997	LS1	Cows			7.2		6.6						
			Growing		3.2			5.0			4.9	5.2		5.5
		LS2	Cows				6.7		7.0	8.5	8.5			
			Growing				5.4		6.1	6.2	3.4			
		LS3	Cows		4.5			5.0			7.9			4.5
			Growing		2.8			4.3			5.5			3.9
		LS5	Cows	4.8			4.3		4.9				7.4	
			Growing	3.5			3.8		5.9				7.3	
	1998	LS1	Cows							7.3				
			Growing					5.8						
		LS2	Cows		3.5	3.3	3.7			4.0				
			Growing		3.8	3.7	4.2			4.7				
		LS3	Cows				4.1			7.4		6.3		
			Growing				2.3			4.9				
		LS5	Cows	4.1			3.7			4.6				
			Growing	3.6			3.1			4.7				
Zn	1997	LS1	Cows			13.5		17.0						
			Growing		16.1			19.7			17.5	17.4		19.7
		LS2	Cows				14.7		15.6		13.6			
			Growing				15.6		15.0		14.2			
		LS3	Cows		13.7			13.6			12.6			16.7
			Growing		13.5			12.9			15.9			17.0
		LS5	Cows	12.5			15.0		17.4					
			Growing	14.3			16.5		17.8					
	1998	LS1	Cows							11.8				
			Growing					16.7						
		LS2	Cows							16.8				
			Growing							16.9				
		LS3	Cows				14.0			16.6		14.6		
			Growing				15.3			17.4				
		LS5	Cows	15.0			12.7			14.3				
			Growing	16.2			14.0			16.8				

No.	Service	Sire	Breed	Calving	Sex	Comment
26	19-6-84	CFO		4-4-85	M	Calf dead (288d)
52				18-3-86		Calf dead
55	26-7-85	BKY		4-5-86		Calf dead (282d)
	30-7-85		AA	18-3-86		Calf dead (231d)
36	17-8-85	BKY		10-6-86		Calf dead (297d)
51	31-3-86	TWB	Fr.	31-12-86	F	Calf dead, 2 weeks (274d)
55	14-7-86	BKY	Sim.	22-4-87	M	Calf dead, (250d); Cow went down, dead
5	5-8-86	BKY	Sim.	12-5-87	M	Calf dead (280d)
49(H)	28-5-86	IDG	Lim.	13-3-87	M	Calf dead (288d)
				30-5-88	F	Calf dead
31	23-4-87	BRD	Fr	3-2-88	F	Calf dead (285d)
59	6-5-87	PAL	Lim.	21-2-88	M	Calf dead (290d)
13(H)	6-5-87	KDS	AA	22-2-88	M	Calf dead (291d)
12(H)	1-6-87	KHS	AA	-3-88	M	Calf dead
22(H)	16-5-87	KHS	AA	28-2-88	M	Calf dead (287d)
17(H)	16-5-87	KHS	AA	-3-88	F	Calf dead
23	27-6-87	Fl10	Lim.	10-4-88	F	Calf dead (286d)
56	17-6-87	IOS	BB	2-4-88		Calf dead (288d)
39	16-7-87	GAL	Lim.	22-4-88	F	Calf dead (280d)
18(H)	26-5-88	KDS	AA	4-3-89	F	Calf dead (281d)
13	16-5-88	BRD	Fr.	1-3-89		Calf dead (288d)
19(H)	8-6-88	LSR	Fr.	18-3-89	M	Calf dead (282d)
15(H)	26-6-88	Hayes B	Fr.	–		Cow went over on back, dead
53	8-7-88			3-4-89		Calf dead, 'Leprosis'(268d)
17	3-7-88			30-4-89	M	Calf dead, 'Leprosis'(300d)
49	29-7-88			24-3-89		Calf dead, 'Leprosis' (238d) Cow dead
26	17-8-88		Lim.	31-5-89	F	Cow dead (287d)
29	10-5-89	WYT	Fr.	12-2-90	M	Calf dead (277d)
18	2-6-89		Lim.	12-3-90	F	Calf dead (282d)
25	18-7-89		Lim.	27-4-90	F	Calf dead (283d), Cow died in Oct.
5	21-8-89		Lim.	5-90		Cow went on back, dead
8	4-9-89		Lim.	27-6-90	M	Calf dead (296d)
42			Fr.	10-3-90	M	Calf dead
16				26-3-90		Cow sudden death, 15-4.
5	18-12-90	Cow down 9 days,				died 7-1-91
2	25-6-90		Fr.	1-4-91	F	Calf dead (279d)

381743	9-6-90	Lim.				Cow over on her back, died 20 Oct.
11(H)			12-4-91	F		Calf dead
51	5-7-90	Lim.	26-3-91	F		Calf dead (264d)
39	10-7-90	Lim.	8-3-91	F,M		Twins before time (240d) 2 calves dead

8   Cow died 20-12-91

30   Cow died 6-4-92

10	19-2-91	Lim.	1-2-91	M	Cow dead spring 1992, sore leg (285d)
20			24-1-92	F	Calf dead born
25	1-5-91	WYT Fr.	10-2-92	F	Calf dead (284d)
53	2-5-91	BUZ Fr.	16-2-92	F	Calf dead (289d)
850565		Fr.	29-2-92	M	Calf dead
50	30-6-91	Fr.	9-4-92	M	Calf dead (283d)
56	17-6-91	Fr.	1-4-92	M	Calf dead (287d)
4(H)	24-6-91	Lim.		M	Calf dead (26-5-92, 7 weeks old, A.)
17(H)	12-7-91	KDS AA	12-4-92		[a]Calf dead,1 month, (274d), Cow pined, died in spring
28(H)	22-7-91	KDS AA	14-4-92		Calf dead, 2 weeks (266d) Cow dead 15-12-92, pined away

Cow dead 20-1-93
Yearling dead 9-2-93
Heifer dead 10-3-93

56	8-6-92		4-2-93	Calf dead, 4 weeks before time (240d)
9	8-7-92		15-4-93	Calf dead (280d) Cow dead[b], down 2 weeks
092446	16-7-92	Lim.	29-4-93 M	Calf dead (287d)
Lim. Cow	12-8-92			Cow dead, Nov. 93
White Cow	10-8-92			Cow dead, Nov. 93, diarrhoea
59(H)	11-9-92		27-6-93 M	Calf dead (289d)
40			4-5-93	[c]Calf born dead, Cow died[d] 18-5-93, suddenly, tetany

[e]Calf dead 20-5-93,

2						Cow dead. Weanling died 4-11-93 [f]Weanling died 25-11-93
083916 MXA						Weanling (10 months old) died 2-10-94, rupture.
18	18-4-93	WYT	Fr.	2-2-94	F	[g]Calf died after 4 days (289d)
43	6-6-93	BOA	Char.		F	[j]Calf died 12 days old
7	11-7-93	FAO	Her.	24-4-94	M	[l]Cow dead 4-9-94, lame, lump (287d)
10(H)	25-7-93 1-9-93	LGE KDS	AA AA	14-6-94	M	[i]Calf dead at birth (286d)
26	26-7-93	LSR	Fr.	5-4-94		Calf dead, 5 hours (253d) Caesarean
29(H)	10-9-93	KDS	AA	26-6-94		[k]Calf born dead (289d)
45				18-5-94		[h]Calf died after a few minutes
19	18-5-94	BOA	Char.	6-3-95	F	Calf born dead (291d)
66(H)	20-8-94	LGE	AA	1-6-95	M	[m]Calf born dead (285d)

APPENDIX 8

Summary of Weather Patterns in the Mid-West 1990 – 1996

(Source Met. Eireann Monthly Weather Reports)

1990

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Sun	dull	dull	dull	normal	sunny	dull	sunny	dull	sunny	dull		dull
Temperature	mild	mild	warm	normal	warm	cool	warm	warm	cool	mild	cold (norm)	cold
Rain	wet	v. wet	dry	normal	dry	wet	dry	dry	dry	v. wet	dry	wet
Wind	windy	windy	moderate	blustery	light	moderate	light	moderate	mixed	windy	moderate	v. windy

1991

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Sun	sunny	normal	dull	dull		dull	dull	sunny	sunny	dull	dull	dull
Temperature	cold	cold	mild	normal	mild	cool	warm	warm	hot then cool	cool	normal	mild
Rain	normal (+snow)	wet (+snow)	wet	v. wet	dry	wet	rain down	dry	dry then wet	normal	wet	dry
Wind	stormy	moderate		windy	light	moderate			windy	windy		

1992

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Sun	dull	dull	dull	dull						bright		dull
Temperature	mild	v. mild	v. mild	colder	mild	warm	warm	cool	cool	cold	mild	normal
Rain	wet then dry	normal	v. wet	v. wet	below normal	dry	normal	wet	normal	dry	wet	dry
Wind	windy then calm		windy	moderate	windy	windy	windy	windy	windy	light then windy	windy	windy

1993

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Sun	dull	dull		dull	?dull	?dull	dull			sunny		
Temperature	mild	mild	mild	mild	mild	mild	mild	mild	cool	cold	cool	cool
Rain	wet	dry	below normal	wet	dry then wet	wet	normal	dry	normal	dry	dry	v. wet
Wind	windy	light	variable	light	strong east 5-d	light	light	light		light	variable – east	windy

1994

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Sun			dull		dull	dull	dull	dull	dull		dull	dull
Temperature	cool	cool	mild	cool	mild	mild	mild	mild		mild	v. mild	mild
Rain	wet	wet	v. wet	v. wet	normal	normal then wet	normal	normal	normal	dry	dry	v. wet
Wind	windy	windy	windy	windy	light							windy

1995

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Sun	dull	dull										
Temperature	mild	mild	cold	cold then mild	cool	warm	warm	v. warm	warm	mild	mild	mild then cold
Rain	v. wet	v. wet	wet - sleet/snow	sleet then dry	normal	dry	wet	v. dry	dry	wet	normal (wet)	dry
Wind	windy	windy	windy									

1996

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Sun		dull	dull	dull		sunny	sunny	dull	sunny	dull	dull	
Temperature	mild	cool	cool	mild	cold	mild	warm	warm	warm	mild	mild then cool	cold
Rain	dry	wet	wet	wet	dry then wet	dry	dry	normal	dry then wet	wet	wet	dry
Wind					mod. east			moderate	windy last week	windy	windy	

APPENDIX 9

Retrospective Survey Haematology and Biochemistry

(Average results from 1997 farm visits. *See* Appendix 1 for test abbreviations, units of measurement, and reference ranges).

Farm	PCV	WBC	Ca	Cu	Mg	Se	Zn	Alb	Urea	P	GGT	AST	CPK	GLDH	βHB	Glb
13	35.7	9.3	2.6	7.9	0.8	0.6	11.5	31.6	6.2	2.1	17.4	91.0	256.0	10.1	0.8	43.2
15	29.3	6.8	2.5	12.8	0.9	4.6	16.6	32.3	8.4	1.6	23.6	97.0	214.7	28.0	1.0	41.1
16	32.7	9.5	2.4	11.0	1.0	4.2	10.8	33.1	5.9	2.3	27.4	101.4	223.5	19.5	0.5	37.7
17	37.4	8.5	2.5	6.4	0.9	1.0	14.2	34.0	9.0	2.0	17.4	146.4	405.0	12.5	0.5	41.6
18	32.0	7.6	2.4	10.1	1.1	1.5	16.5	32.4	7.1	1.8	23.4	78.0	44.5		0.6	43.9
19	30.9	7.5	2.4	8.5	0.9	2.0	14.6	28.7	2.3	1.6	23.3	82.5	217.7	11.0	0.4	44.8
20	34.9	8.4	1.8	11.6	0.8	2.2	11.6	31.3	8.3	2.1	11.4	99.4	355.5	12.3	0.7	47.8
21	33.6	8.9	2.2	10.0	0.9	0.6	13.3	30.3	5.9	1.8	26.5	110.9	268.8	11.3	0.5	46.0
22	31.5	11.5	2.4	11.4	1.0	1.6	14.4	30.9	7.9	2.0	21.8	109.3	124.3	18.7	0.6	49.9
23	31.3	8.2	2.5	11.3	0.9	3.5	13.5	30.7	3.2	1.4	19.5	98.5	138.1	11.5	0.5	52.0
24	31.2	7.0	2.3	13.9	1.0	1.5	13.5	33.5	6.6	2.0	20.2	109.6	368.3	15.1	0.4	51.6
25	33.8	10.2	2.4	14.9	0.9	26.5	12.2	27.3	5.1	2.2	26.1	84.6	205.7	17.8	0.6	43.9
26	32.3	7.4	2.4	10.3	1.0	1.3	12.0	29.6	5.2	1.9	16.5	84.0	115.3	14.0	0.6	44.5
27	29.9	7.4	2.3	10.9	0.7	1.4	12.4	31.1	4.8	1.2	21.6	122.7	190.0	36.3	0.4	47.6
29	32.8	10.9	2.7	9.5	0.8	0.5	11.5	30.3	6.0	2.4	12.3	105.0	512.9	14.6	0.5	49.8
30	36.8	8.8	2.3	9.6	0.8	0.8	13.2	29.3	6.1	1.7	15.9	107.4	386.3	11.4	0.6	40.9
31	33.1	8.0	2.3	11.4	0.8	2.4	15.2	35.3	8.7	2.2	24.3	92.0	148.0		0.9	38.3

ANIMAL HEALTH - *to be completed by all adults and parents of children 1-14 years.*
Those 15 - 18 years parent / head of household to answer.

Thank you very much for your help in completing this much of the Questionnaire. We would be most grateful if you could answer some questions we have concerning animal health.

| 1 | 1 | 1-2

□□□□ 6

a. Do you live on a farm that contains cattle?

If no, skip rest of section

YES	NO
1	2

□ 7

b. If yes, are you the person responsible for managing the farm?

YES	NO
1	2

□ 8

If yes, please complete this section

If no, can you give details of the activities of the farm?

YES	NO
1	2

□ 9

If no, omit this section

If yes, please complete this section.

1. Farm animal deaths

Approximate number of farm animals that died in 1995 and in 1996 to the end of May

	Deaths in 1995	Average no. during 1995	Deaths from 1 Jan. to end May 1996	Approx. no. on farm at present
Dairy Cows				
Suckler Cows				
Heifers				
Bullocks				
Calves				
Sheep				
Horses				

□□□□□□□□□ 19
□□□□□□□□□ 29
□□□□□□□□□ 39
□□□□□□□□□ 49
□□□□□□□□□ 59
□□□□□□□□□ 69
□□□□□□□□□ 79

Definition
Dairy cow: an animal that has produced at least one calf and whose milk is supplied to the creamery.
Suckler cow: an animal that has produced at least one calf and which is suckled during its entire lactation.
Heifer: A female animal, aged more than six months, that has not calved.
Bullock: A castrated male animal, aged more than 6 months.
Calf: A male or female animal aged 6 months or less.

END OF
RECORD ELEVEN

HERD FERTILITY

What is the routine calving pattern in your herd? Is it

☐☐☐☐ 6

☐ 7

1 Spring Calving Only   2 Autumn Calving Only   3 Other   ____
(January – April)   (September – December)

If the answer to the previous question was 1 or 2, please complete the remainder of this section.
If the answer was 3, then please proceed to the next section (Question 3)

How many cows or heifers were inseminated by AI or a bull on your
farm in 1995?   ____   ☐☐☐ 10

How many of the above aborted their calves at least one month before the expected calving date? ____   ☐☐ 12

How many of the above were not-in-calf at the end of the 1995 breeding season?
(Exclude animals that aborted)____   ☐☐ 14

How many have calved late i.e. after January 1st 1996 for an autumn calving herd or after
May 1st 1996 for a spring calving herd   ____   ☐☐ 16

3. Calving Information For 1996 Calvings.

What is the total number of cows that have calved in
1996 to date?   ____   ☐☐☐ 19

Total number of cows that delivered twins.   ____   ☐☐ 21

Number of cows that needed assistance to calve.   ____   ☐☐ 23

Number of cows that went down within one month
of calving.   ____   ☐☐ 25

Number of cows that died within one month of calving.   ____   ☐☐ 27

Number of calves that were deformed at birth.   ____   ☐☐ 29

Number of calves that were born dead or died within
2 days of being born.   ____   ☐☐ 31

Number of calves that died between 3 days and 3 months of birth   ____   ☐☐ 33

Cattle diseases

We are interested in how many animals were treated by veterinary and non-veterinary personnel(yourself, relations, neighbours, farm relief personnel) between January 1st and May 31st, 1996 for the following conditions (exclude preventive treatments, e.g. routine dosing)?

Disease	Dairy Cows		Suckler Cows		Heifers		Bullocks		Calves	
	Vet	Non-Vet	Vet	Non-Vet	Vet	Non-Vet	Vet	Non-Vet	Vet	Non-Vet
Diarrhea										
Pneumonia										
Skin Condition										
Eye Condition										
Mastitis					N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Retained placenta/womb infections					N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Milk Fever					N.A.	N.A.	N.A.	N.A.	N.A.	N.A.
Lameness										
Fluke										
Mineral deficiency										

53

73

6

26

END OF RECORD TWELVE   1 3   1-2

46

54

62

6

END OF RECORD THIRTEEN   1 4   1-2

14

34

54

74

END OF RECORD FOURTEEN

269

Has pining (i.e. animals becoming thin) or ill-thrift (i.e. animal/s failing to thrive) been a problem in your herd in 1995 or 1996?

0 = no problem   1 = minor problem (< 5 cattle affected)

2 = major problem (5 or more cattle affected)

| 1 | 5 | 1-2

☐☐☐☐ 6

Dairy Cows   _____   ☐ 7

Suckler Cow   _____   ☐ 8

Heifers   _____   ☐ 9

Bullocks   _____   ☐ 10

Calve   _____   ☐ 11

5. General information

What is the total acreage of your farm? (Include outfarms and rented land)   _____   ☐☐☐ 14

If you keep a dairy herd, what was the average milk production per cow in 1995?

1 = less than 1,000 gals.   2 = 1,000 - 1,200 gals   3 => 1,200 gals   _____   ☐ 15

On average, how much concentrate did you feed to each of your cows in 1995?

1 = less than 500 kgs.   2 = 500 - 750 kgs.   3 = > 750 kgs.   _____   ☐ 16

What proportion of your cattle were outwintered during the 1995/96 winter period?

1 = none   2 = 1-50%   3 = >50%   _____   ☐ 17

On average, how much nitrogen did you apply to your land in 1995?

1 = less than 100 units/acre   2 = 100-200 units/acre   3 = > 200 units/acres   _____   ☐ 18

How many times have you had your silage analysed in the last three years?   _____   ☐ 19

END

THANK YOU FOR TAKING PART IN THIS SURVEY

I certify that I have interviewed the above named respondent in accordance with survey instructions.

SIGNED _____   DATED _____

Analysis of Immunological Data from Study of Askeaton and Abbotstown Cows

Summary

This report presents a statistical analysis of immunological data on 42 cows belonging to four groups (defined by their source and current location). The immune response measurements were recorded at approximately 10 separate "bleeds" from mid 1996 to early 1998. A preliminary analysis looks at the responses at first and last bleeds and compares the four groups of cows, making adjustments for differences in age and other factors. A "repeated measures" analysis then considers all observations, making appropriate adjustments for trends in time. In assessing the combined findings of these analyses, only PWM and Selenium were found to be consistently and significantly ($p < .01$) different than the reference group (A) in all of the comparison groups (B, C and D). Group B had lowered CD8, PWM, ConA, PHA and KLH compared to group A. Group D had lowered KLH and KLHIgG responses. Group C presented the most complex contrasts, with raised BCELL and PHA and lowered CD2, CD4 and PWM. This may be due in part to the lack of formal randomisation to select cows for moving, the variation in their exposure time to the new environment (see further discussion below), and the immune system's mechanism for coping with the change of environment.

We may conclude that the three comparison groups do differ from the reference group with respect to some immune responses, particularly PWM and Se, and groups B and D appear to have lower KLH. Comparisons of groups B and C to group D are also presented, indicating that cows moved to or from Askeaton differ in a number of responses to those Askeaton cows that were never moved.

Purpose of study

In recent years there has been a reported increase in morbidity in cows on at least two farms in County Limerick. A study was undertaken to determine whether there might be diminished immune function in cows on the affected farms possibly due to environmental factors.

It was decided to study the immune function of a number of cows from one of the affected farms, and compare these to a similar number of cows at the Veterinary Research Laboratory, Abbotstown. In order to help investigate the possible role and impact of environmental factors, approximately half of the cows in each of these groups were moved to the other location, thereby giving four groups of animals to be studied.

Group	Source	Location
A (11 cows)	Abbotstown	Abbotstown
B (11 cows)	Askeaton	Abbotstown
C (11 cows)	Abbotstown	Askeaton
D (10 cows)	Askeaton	Askeaton
Total 43 cows		

Objectives

a) To compare indicators of immune function in cows in the four groups.
b) To compare the four groups for their responses to a specific antigen.
c) From (a) and (b) to determine whether cows on or from the index farm have diminished immune function.

The animals were studied for a period of about 17 months, from July 1996 to January 1998. Animals were bled approximately every six weeks giving a total of 427 blood samples (see Table 1). In addition to the various laboratory measures of immune function (see next Section), age, pregnancy status, weight, length of time in current location, and the general condition of the cow were also recorded.

Table 1 Summary of available blood tests.

Group	Total Bleeds	cows X bleeds	Average bleeds per cow	Age in years at first bleed (min	mean	max)
A	112	8X11, 3X8	10	3.0	5.6	8.6
B	115	10X11, 1X5	10	2.6	6.4	10.6
C	103	6X10, 4X9, 1X7	9	2.7	4.8	8.5
D	97	9X10, 1X7	10	2.5	7.7	11.5
Total	427					

Materials and Methods

The analyses presented here were based on two data files: the first of these, IMAPR30A.XLS was received in early May '98, and the second, COWIMM1.XLS was received in early July. The purpose of the second data file was to correct a number of erroneous values which had been recorded for the dates on which cows had been moved to their new location. Hence any analysis involving this date (or time elapsed since this date) were re-done using the more recent data. However, it was also noted that two cows (#310, #57) had incorrect age information in the earlier file: this should be borne in mind when viewing results of analyses involving age-adjustments (but not time-since-moved), as these analyses were *not* redone using the updated data.

The immune response variables measured on the blood samples were divided into four categories: Lymphocyte subsets (**BCELL, CD2, CD4 and CD8**), Proliferation response (**CONA, PWM, PHA**), specific immune response (**KLH and KLHIGG**) and trace elements (**Copper, Selenium**). Not all of these responses were measured on every bleed date, with the result that there were varying numbers of observations available for analysis of each response (see Table 2).

For each of the response variables, Q-norm plots were inspected to establish whether these variables were approximately normally distributed, and to determine what

transformations of the data, if any, might be required. These plots indicated that **BCELL, CD2, CD4, CD8** were approximately normally distributed. The standardised PWM (obtained by subtracting background) was also approximately normally distributed. However, **CONA, PHA, KLH, KLHIGG, Cu** and **Se** required transformation and the transformed measures were used in subsequent analysis. For **CONA, PHA** and **KLH,** the ratio of the gross value to background, expressed on the natural log scale, was approximately normally distributed: for example, the measure of **CONA** used in analysis was **ln (gCONA/bckd)** and this variable was renamed **LNRTCON** to indicate that it is the natural log **(LN)** of the RaTio of **CONA** to background. For **Cu** and **Se,** a simple log transform sufficed. For KLHIGG, a log transform was sufficient to normalise the data for analysis of first and last bleed. For an analysis of the change in this variable over time (from immunisation), we expressed each value as a change from "baseline" as follows: all pre-immunisation values for a given animal were averaged on the log scale to give a baseline for that animal, and subsequent values were then expressed as differences from this baseline. This measure (labelled NEWLNIGG) had an approximate normal distribution.

Hence the response variables to be studied are:

- BCELL
- CD2
- CD4
- CD8
- PWM = gPWM-bkgd
- LNRTCON = ln(gConA/bkgd)
- LNRTPHA = ln(gPHA/bkgd)
- LNRTKLH = ln(gKLH/bkgd)
- LNKLHIGG = ln(KLHIGG)
- NEWLNIGG = LNKLHIGG - baseline (LNKLHIGG)
- LNCU = ln(Cu)
- LNSE = ln(Se)

and a summary of these is provided in Table 2 where the log-transformed variables are presented on the raw scale for ease of interpretation.

Table 2 Summary of response variables

Variable	number of observations	Minimum	Mean	Maximum
BCELL	196	2.7	20.8	48.5
CD2	214	37.9	63.9	88.2
CD4	216	14.4	32.1	52.3
CD8	218	9.9	27.8	47.5
PWM	324	52342	207743	415616
CONa/bkgd	323	8.2	221	2394
PHA/bkgd	319	2.2	163	1172
KLH/bkgd	289	.03	9.7	214
KLHIGG	266	78.8	931.1	21749
CU	399	7.1	12.8	21
SE	260	1.3	3.2	7.7

Analysis of the first bleed

As a preliminary analysis, we looked at the first available value for each of the response variables. Simple linear regression models were used to assess whether these responses depend on group, with adjustments made for age and pregnancy status. In all analyses, Groups B, C, and D were compared to group A (which was regarded as the reference group), and in addition, B and C were compared to D to assess whether animals moved to or from Askeaton differ from those that remained in Askeaton throughout.

The dates on which response data are available vary depending on the response in question and the completeness of data for each cow. Table 3 illustrates the variability in dates on which BCELL response data is available, indicating that there could be some confounding between Group and calendar time (or season). Similar variability in dates occurs with the other response variables. None of the eight response variables to be studied were available for one of the 43 cows (ID number B202), and so the analysis was carried out on 42 cows.

Table 3: Dates of first available BCELL and number of cows bled.

First available date	Group A	Group B	Group C	Group D	Total cows
07 Oct 1996	8	8	0	0	16
14 Oct 1996	0	0	5	6	11
09 Dec 1996	3	2	0	0	5
07 Apr 1997	0	0	6	4	10
Total	11	10	11	10	42

It should be noted that cows that were moved to a new location (groups B and C) might have been some time in that place before any measurements were available (see Table 4). All but one of the group B cows were 7 months in their new location before the first blood sample was taken. Group C had one cow with a bleed at the time of transfer, four cows with a bleed 2 months later, and the remaining six cows with no blood samples for approximately 7 months. The primary purpose of the analysis is to compare the groups for evidence of chronic impairment of immune function, and the purpose of the delay from relocation to first bleed was to standardise systems of management in the two sites. However, any immune response that has a time trend (e.g. if animals improve or disimprove steadily in their new location) may not be homogenous for all animals in group C due to the variation in their time in their new location. For this reason, we defined two subgroups of group C, depending on whether their "time-in-place" at first bleed was less than 3 months (group C0) or greater than 3 months (group C1). For each first bleed analysis (except KLH), in addition to comparing groups B, C and D to A, we also checked if there was evidence that the two subgroups of C were different. This was not possible for KLH, simply because there were *no* baseline values available for cows in subgroup C0.

Table 4 Summary of dates of transfer and dates of first blood sample.

Group	Number of cows	date transferred to new location	First available bleed date	Length of time in new location (nearest month)
B	9	20/12/95	22/7/96	7
	1	24/7/96	15/7/96	0
C	6	20/12/95	15/7/96, 22/7/96	7
	1	24/7/96	15/7/96	0
	4	24/7/96	14/10/96	2

The results of the preliminary analysis of first bleed data are presented in Table 5. Note that for KLH and KLHIGG we analysed the first available value *prior to inoculation*. For each response variable, we present two lines of results. The first line is simply the average response for each group, with the p-values for comparison with group A and group D given in parentheses: note that the p-value for comparison with group D is only reported when statistically significant. The second line (labelled ADJ) presents for each group the average response adjusted for differences in age and pregnancy status on the bleed date: the response given is the average response one would expect for a cow aged 6 years (the average age at first bleed) that is not pregnant. For KLH and KLHIGG, we also inspected the effect of adjusting for trimester of pregnancy instead of the yes/no pregnancy status, but the results were effectively unchanged.

For some responses there was a significant difference between the two subgroups of C, indicated by an asterix in the C column (note that the first available CD2, CD4, CD8 and Se were not re-analysed using the more recent data and so were not checked for such a difference).

The PWM variable showed significant differences between all three groups (B,C and D) and group A, with some of these differences being of relatively large magnitude and highly significant statistically. Groups C and D had lower selenium than group A. In addition, there was some evidence that group B had lower CD8 ($p=.03$) than the reference group (A), Group C had higher BCELL ($p<.001$), and Group D had lower CD2 ($p=.05$). None of the three groups B, C and D differed significantly from A with regard to CD4, PHA or KLH, although group C appeared to have higher KLH than group D.

Table 5 Analysis of first available blood test

	A	B	C	D
BCELL	12.8	13.4 *(.83)*	27.1 *(.000, .000)*	15.0 *(.43)*
ADJ	10.9	12.2 *(.62)*	25.2 *(.000 .006)*	16.0 *(.09)*
CD2	63.5	60.6 *(.43)*	57.9 *(.11)*	58.7 *(.18)*
ADJ	65.5	61.3 *(.24)*	59.5 *(.09)*	57.7 *(.05)*
CD4	33.8	35.3 *(.56)*	31.2 *(.28)*	35.9 *(.42)*
ADJ	34.0	34.8 *(.76)*	31.5 *(.32)*	35.0 *(.75)*
CD8	26.8	22.3 *(.07)*	23.6 *(.19)*	28.6 *(.45)*
ADJ	26.4	21.0 *(.03)*	23.7 *(.25)*	27.0 *(.82)*
PWM	289910	235328 *(.07)*	190630 *(.001)**	197192 *(.003)*
ADJ	306398	244072 *(.04)*	207285 *(.002)**	195708*(.002)*
RTCON	83.8	89.1 *(.89)*	107.8 *(.55)*	63.5 *(.52)*
ADJ	63.6	67.6 *(.89)*	94.6 *(.35)*	48.7 *(.58)*
RTPHA	51.5	61.2 *(.71)*	86.5 *(.25)*	48.5 *(.89)*
ADJ	39.8	47.1 *(.72)*	76.8 *(.15)*	37.5 *(.91)*
CU	11.3	12.2 *(.31, .005)*	10.6 *(.47)**	9.4 *(.03)*
ADJ	10.8	11.9 *(.24, .01)*	10.2 *(.48)**	9.4 *(.13)*
SE	3.94	3.73 *(.52)*	3.15 *(.01)*	2.4 *(.000)*
ADJ	4.05	3.82 *(.53)*	3.21 *(.01)*	2.43 *(.000)*
RTKLH	1.06	0.9 *(.45)*	1.28 *(.44, .05)*	0.77 *(.14)*
ADJ	0.99	0.83 *(.43)*	1.21 *(.41, .08)*	0.76 *(.28)*
KLHIGG	245.2	299.6 *(.44,.007)*	261.9 *(.80, .017)**	126.2 *(.02)*
ADJ	187.1	234.8 *(.38, .07)*	213.7 *(.61, .15)*	118.3 *(.13)*

Analysis of the last bleed

As with the first available measurements, a regression analysis was carried out to investigate responses measured on the last available bleed dates. The results are presented in Table 6 using the same format and notation as for first bleed. Note that the last available KLH values were measured a least 154 days after baseline (inoculation) while KLH response would normally peak at approximately 90 days post-inoculation. Except for a single value at 70 days, the last available KLHIGG values were also obtained at 154 days or later, while this measure would be expected to peak at approximately one month post-inoculation. For KLH, the response varied with trimester of pregnancy (p=.02 for trimester 1 or 2, p=.07 for trimester 3). For KLHIGG, trimester was also inspected but not found to be statistically significant. In contrast to the analysis of first bleed, only one response (CD2) showed differences in subgroups C0 and C1. This would indicate that by the time the last blood draw was done, other differences had 'evened out' over time.

No significant differences were found between the three comparison groups and group A with regard to BCELL, CD2, CD4, PWM and Cu, although the CD4 levels of group C tended to be low (p=.07). Groups C had higher ConA , PHA and KLH than group A, and all of these responses were lower in group B although only KLH achieved statistical significance. In addition group B also had lowered KLHIGG. The strongest result was observed for Selenium, with the differences between group A and each of groups B, C and D being highly significant statistically: the animals resident in Askeaton at the end of the study had lowered values while those transferred to Abbotstown had raised values.

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Table 6   Analysis of last available blood test.

|	A	B	C	D
BCELL	24.5	21.8 *(.55)*	31.5 *(.11, .003)*	17.5 *(.12)*
ADJ	19.4	19.9 *(.89)*	24.3 *(.17, .05)*	13.6 *(.14)*
CD2	59.2	61.9 *(.59)*	51.5 *(.13, .005)**	67.0 *(.13)*
ADJ	65.8	64.7 *(.8)*	60.7 *(.20, .02)*	72.9 *(.11)*
CD4	29.5	28.3 *(.64)*	24.6 *(.06)*	31.0 *(.59)*
ADJ	33.1	30.8 *(.33)*	28.8 *(.07)*	36.5 *(.20)*
CD8	27.1	29.4 *(.49)*	28.0 *(.79)*	33.9 *(.05)*
ADJ	27.9	29.0 *(.73)*	29.5 *(.60)*	32.9 *(.16)*
PWM	197326	204150 *(.72)*	178133 *(.30)*	210693 *(.48)*
ADJ	195154	205212 *(.62)*	175331 *(.30)*	212560 *(.40)*
RTCON	149.3	105.5 *(.35)*	324.2 *(.04, .006)*	125.1 *(.63)*
ADJ	229.0	121.8 *(.07)*	571.2 *(.008, .01)*	202.4 *(.73)*
RTPHA	116.6	84.0 *(.36)*	264.4 *(.02, .004)*	96.7 *(.60)*
ADJ	184.5	98.6 *(.06)*	483.4 *(.003, .009)*	164.3 *(.73)*
CU	13.1	13.3 *(.89)*	14.2 *(.38)*	13.6 *(.69)*
ADJ	12.9	13.2 *(.8)*	13.8 *(.44)*	13.4 *(.71)*
SE	2.9	4.0 *(.003)*	2.0 *(.000)*	2.0 *(.000)*
ADJ	2.8	3.8 *(.003)*	1.9 *(.000)*	1.9 *(.001)*
RTKLH	9.6	4.3 *(.05, .05)*	15.7 *(.22, .07)*	8.5 *(.76)*
ADJ	13.6	4.7 *(.008, .01)*	28.9 *(.05, .07)*	13.3 *(.96)*
KLHIGG	340.9	134.9 *(.02, .01)*	419.7 *(.58)*	338.6 *(.99)*
ADJ	408.0	151.2 *(.02, .004)*	490.6 *(.63)*	469.1 *(.74)*
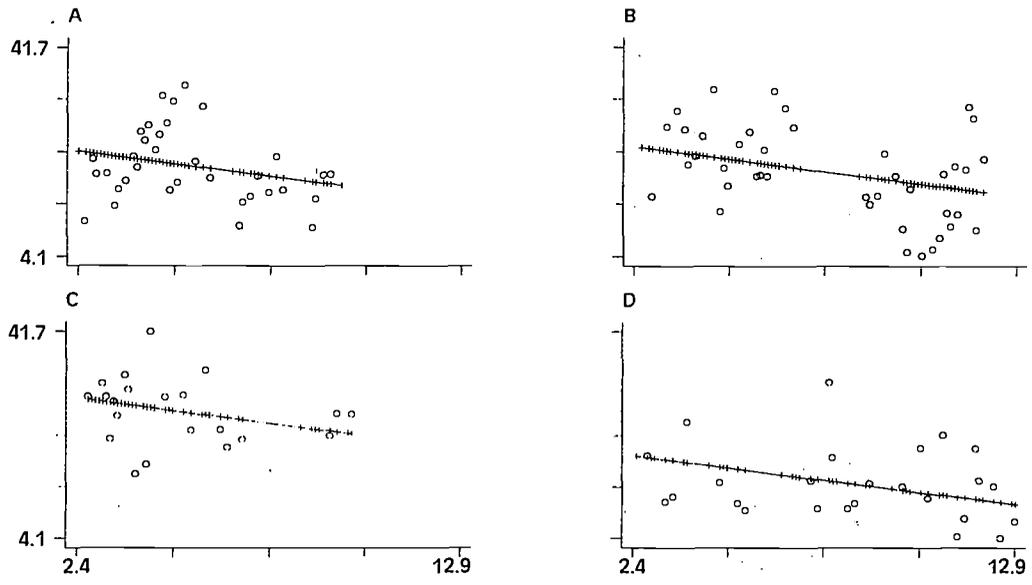
Fig 1. BCELL vs. AGE by group with regression lines

Longitudinal Data Analysis

The study of immune function in these four groups of cows is an example of a "longitudinal" study, where multiple measures are recorded for each animal at a number of time points during follow-up. Since many of the response variables appear to depend on age, we might expect to find a trend in the response over time for a given group of animals. For example, the plot in Fig. 1 shows the mean BCELL response versus AGE (in years) on the date of bleeding for the four groups of cows, with an age trend superimposed on the plot. We have already seen that for groups that were moved, the time in the current location might also be a factor. Furthermore, it is possible that the trend in time (or equivalently in age) may be different for the different groups, particularly those that were moved. To address all of these considerations requires an analysis of the full data: since there is an inherent correlation between repeated measures of the same response on the same animal, an analysis of all the observations must accommodate this correlation. One such method of analysis is GEE (generalised estimating equations), and we chose this as the method of analysis of all available observations. It must be stressed that GEE is a model-based method, and hence can only provide estimates of effect *under the assumption that the model is reasonable*. Since this assumption cannot be rigorously tested, any interpretations of the results produced should only be used as corroborative evidence for the more direct and simple results in the first- and last-bleed analyses, except for KLH and KLHIGG, as discussed briefly below.

For each of the responses BCELL, CD2, CD4, CD8, PWM, CONA, PHA, CU and SE (where ConA, Cu and Se are transformed to a log scale as explained previously) , we modelled the response as a function of Group, Age (a linear trend) and Pregnancy status. We also accommodated, where appropriate, a different trend in age in the different groups. For KLH, we fitted an additional quadratic term in DBASE (time since inoculation) to reflect the expected rise of the response to a maximum followed by a decay. Such a trend was apparent even in a crude examination of the mean KLH plotted versus DBASE (Fig. 2a). For KLHIGG, we used the NEWLNIGG variable which measures the change from baseline. The plot of this change does not exhibit the curvature seen for KLH but a decay from a maximum at the first post-immunisation bleed (Fig 2b). This is to be expected, as the first available measure was at 28 or 29 days post-immunisation, while the peak response would be expected at or before this time. The analysis of KLHIGG models this decay over time. For the KLH and KLHIGG responses, the GEE analysis is more appropriate than the first- or last-bleed analysis as the GEE approach models the profile over time of the specific response.

## Fig 2a. Mean of transformed KLH vs. time from inoculation
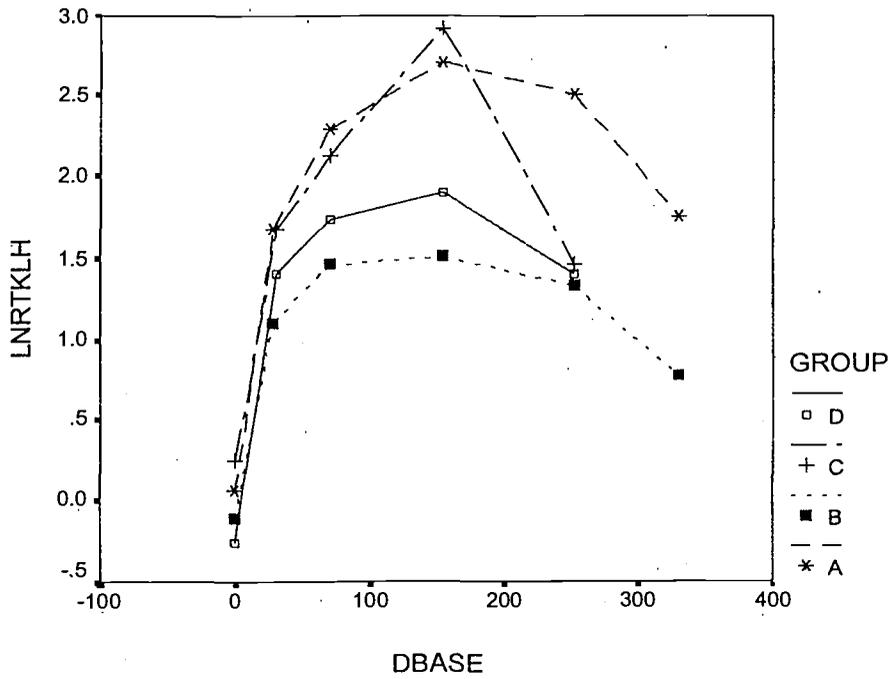
## Fig 2b. Mean NEWLNIGG (change of in KLHIGG from baseline on log scale) vs. time from inoculation
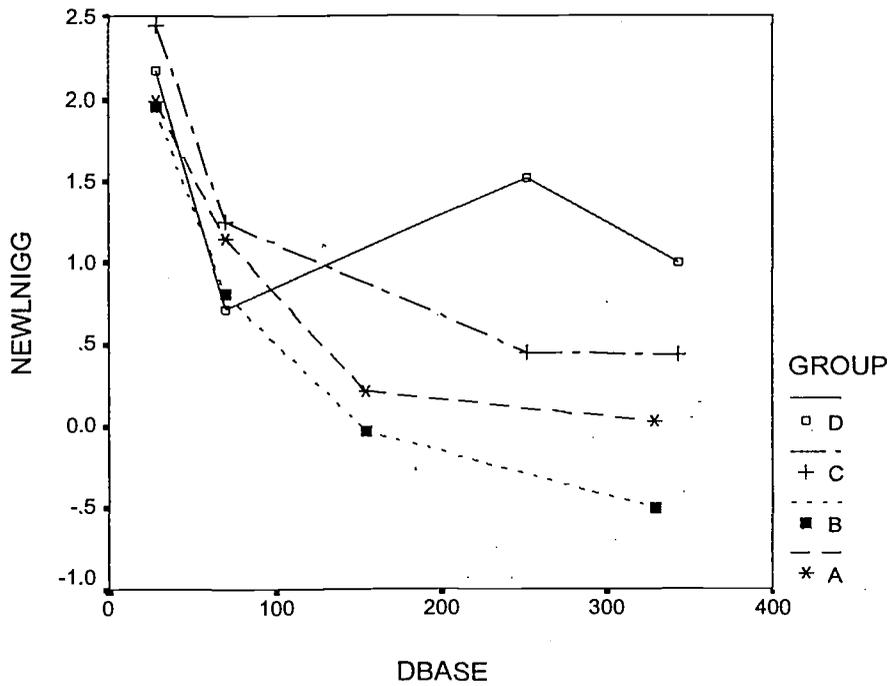
Table 7 presents a summary of the variables that were retained in each of the models. Note that each of the three group indicators (B, C, D) were retained in all models regardless of statistical significance. In addition, adjustment was made for pregnancy status where there was some indication of an effect that had not achieved statistical significance ($p < .2$). The adjustment term for Age is linear and for Dbase is quadratic as discussed above. For KLH, trimester of pregnancy was investigated but not found to be statistically significant, and for NEWLNIGG trimester 1 or 2 were significant (but not trimester 3) so we adjusted for the simpler pregnancy status variable in the final model. Where the KLH or KLHIGG responses are found to differ significantly between the groups, a further comparison of group D to groups B and C is performed.

Table 7 Variables included in models of response, with significance levels (*=05, **= .01) or p-values: the two entries for B and C represent comparison to group A and D respectively. Note: (a) AGE(B) is a term representing the fact that the time trend for group B differed from Group A, (b) The effect of Dbase is fitted as a quadratic curve.

	BCELL	CD2	CD4	CD8	PWM	CONA	Cu	Se	PHA	KLH	KLHIGG
B	.91	*	.45	**	**	**	.13	**	**	**, .52	.15, .**
C	**	**	**	.24	**	.09	.31	**	*	.32, .11	.18. .49
D	*	.98	.70	.28	**	**	.43	**	**	**	*
AGE	*	**	.69	*	.62	**	.49	.34	**		
AGE(B)				.06							
Pregnancy		.17			**	*			.14		**
Dbase (quadratic)										**	**

We will present the results of the analyses graphically as follows: for each response, the predicted value from the model will be plotted versus age for any of groups B, C and D which differed significantly from the reference group (A). These plots will be presented for a range of ages representing the ages of the cows during the time of the study, and in all cases the predicted value will be for a non-pregnant cow. Hence, if a group does not appear on the plot, it indicates that this group was not found to differ significantly from Group A, when adjustments were made for age and other factors. In the event that the nature of the trend over time differs for different groups this will be reflected by non-parallel curves. For KLHIGG, an additional plot will be presented to illustrate the significant difference between group B and D.

Figure 3 presents the predicted BCELL values for groups A, C and D, indicating that group C have higher values and group D lower values than group A. While age was found to significantly affect the BCELL value, the magnitude of this trend was negligible.

Fig 3. Predicted values of BCELL from the model (group B did not differ significantly from A).
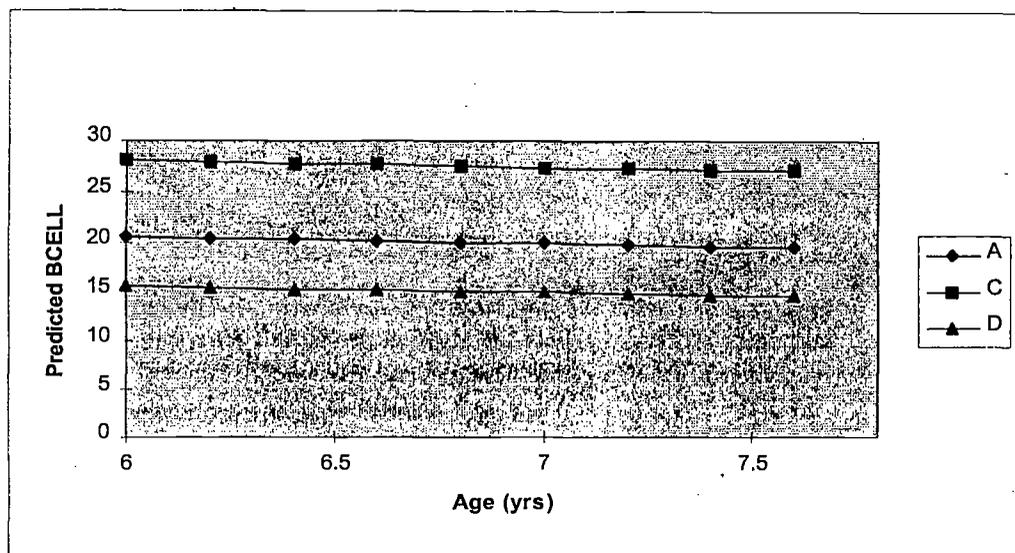

Figure 4 presents the predicted values of CD2, illustrating an age trend and indicating that groups B and C have significantly lower values than group A. Group D was not found to differ significantly from group A.

**Fig 4: Predicted values of CD2 from the model.**
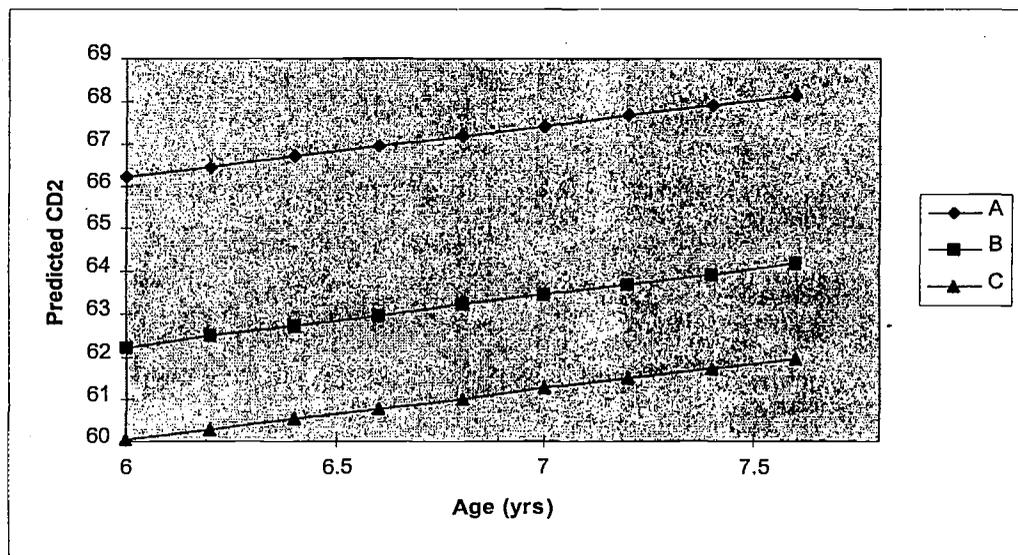
Figure 5 presents the predicted values of CD4 for groups A and C (the only group to differ significantly from group A). As can be seen from the plot, there was no significant trend with age for this response variable.

**Figure 5: Predicted values of CD4 from the model.**
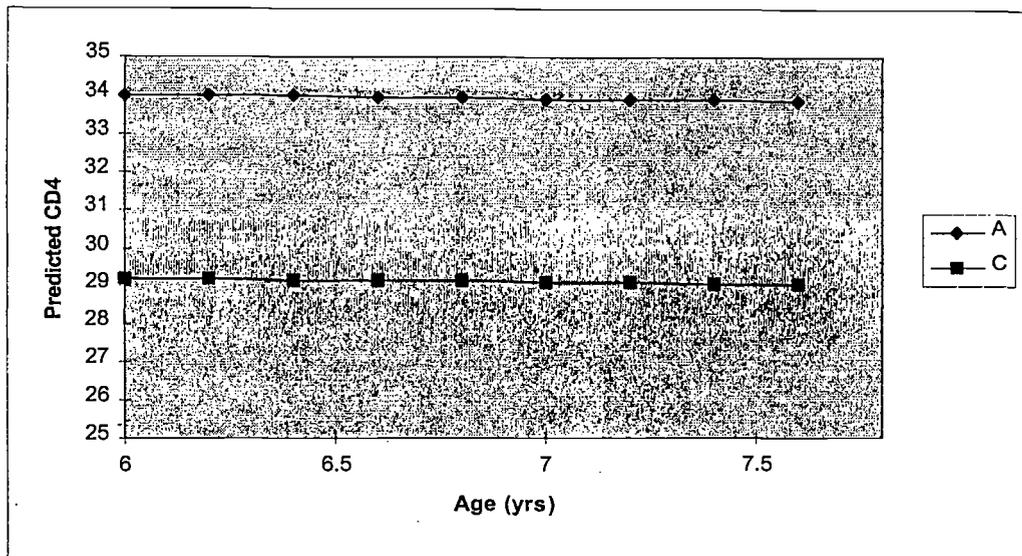
Figure 6 presents the predicted values of CD8, where only group B differed significantly from A. In this case, the time trend was found to differ in the two groups. Since the time trend in group A can be viewed as equivalent to an age trend, the increased slope for group B might be interpreted as due to the cumulative effect of time in the new location.

**Figure 6: Predicted values of CD8 from the model.**
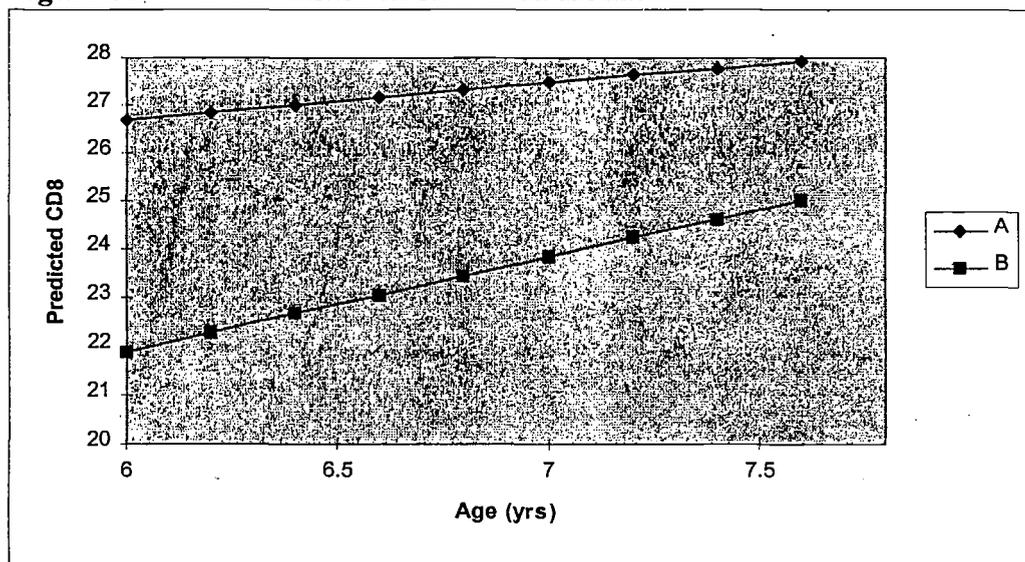
Figure 7 presents the predicted values of PWM, indicating no time trend, and all three groups (B, C and D) significantly different than group A. Group B and C (the groups that were moved) have almost identical predicted responses in this variable.

**Fig. 7 : Predicted values of PWM from the model.**
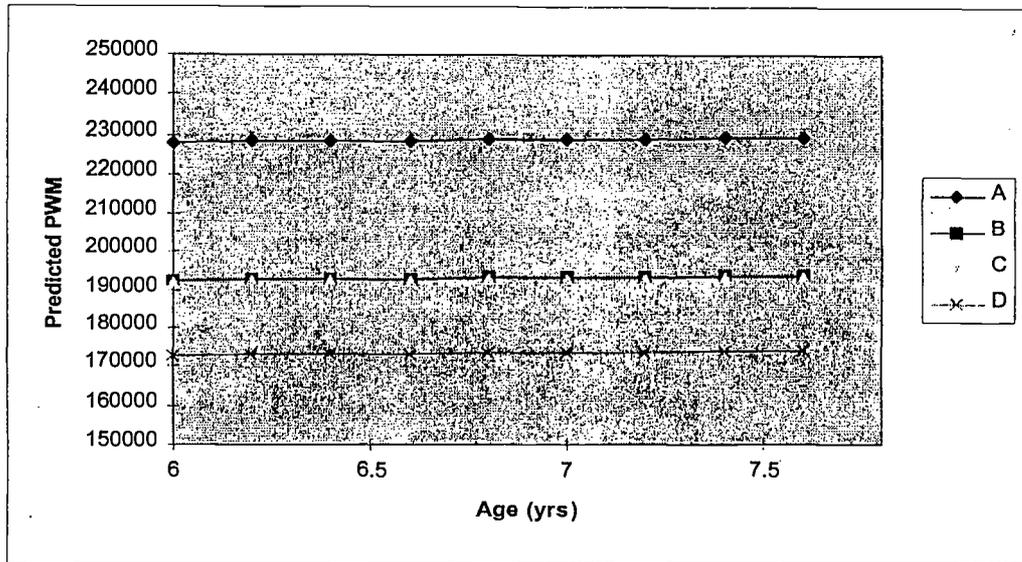
Figure 8 presents the predicted values for ConA, which is the ratio of gross ConA to background, on the log scale. There is some evidence of a time trend, and groups B and D, which are very similar to each other, differ significantly from group A.

Fig 8. Predicted values of ConA (ratio to background, on a log scale).

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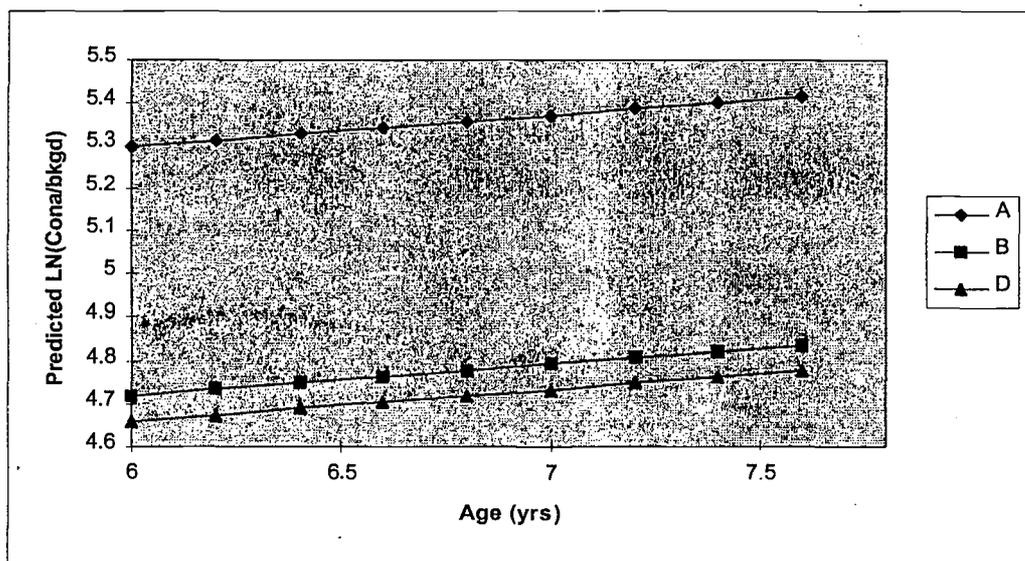
There were no significant differences between the groups with respect to Copper.
In contrast, as illustrated in Figure 9, the Selenium levels of all three groups (B, C and
D) differed significantly from group A, with group B having higher levels, and groups
C and D having similar levels that were significantly lower than A.

Fig 9. Predicted values of Selenium (measure on a log scale) from the model.
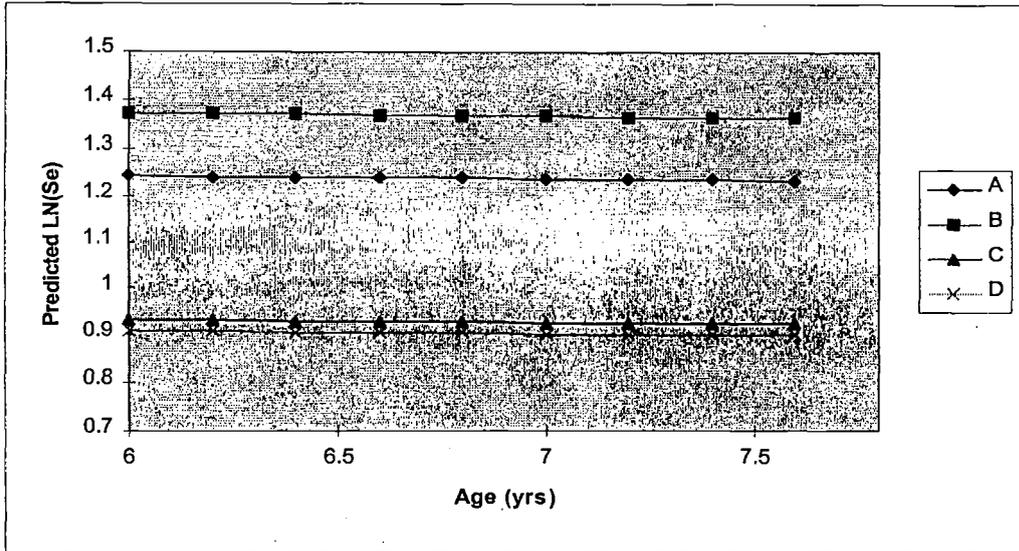
Figure 10 presents the predicted values of PHA, measured as the ratio of gross PHA to
background and expressed on a log scale. The time trend, while being statistically
significant, is of small magnitude. All three of groups B, C and D differ significantly
from group A, with group C having higher values and groups B and D having lower
(and very similar) values.

Fig 10: Predicted values of PHA (ratio of gross PHA to background on a log scale).
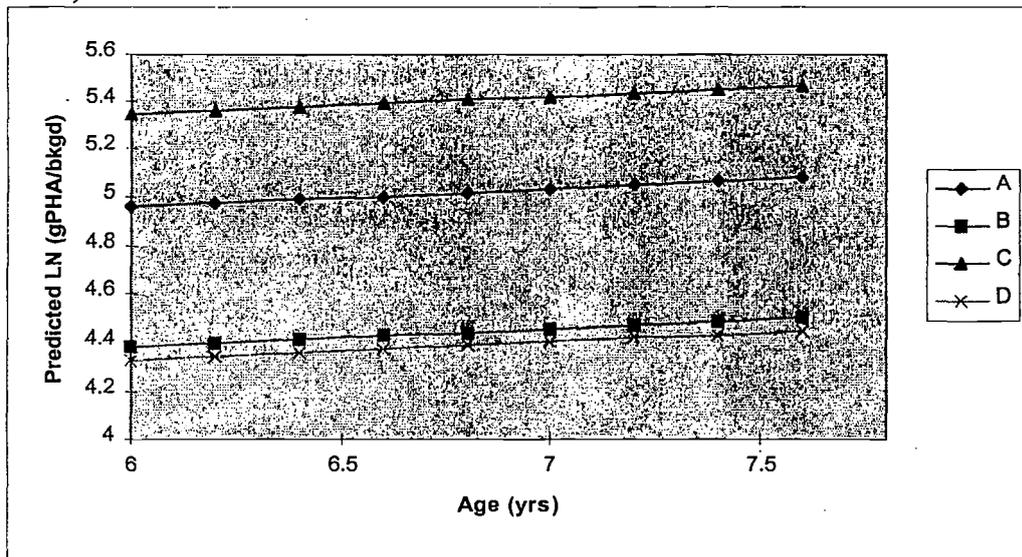
For the analysis of KLH, the time from inoculation (DBASE) was accommodated by using a quadratic curve, and the quadratic trend was found to be significant, which is not surprising in view of our simple graphical exploration of the data (Fig 2). Since there was no significant effect of age, we present the predicted values by time from inoculation (Fig 11). It can be seen that groups B and D are very similar and both differ significantly from group A.

Fig 11: Predicted values of KLH (ratio to background on log scale).
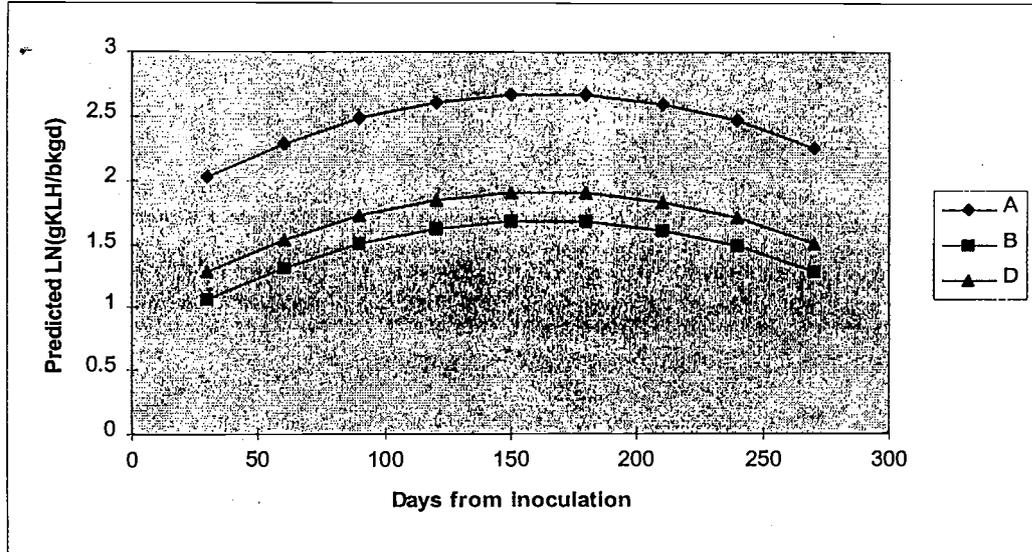
For KLHIGG, only group D was found to differ significantly from A, and the curve in Fig. 12 displays the decay in this response from the first available post-inoculation value at day 28 or 29. The comparison of group D vs. groups B and C indicated a significant difference between group D and group B, and the responses of these two groups is illustrated in Fig.13.

**Fig 12: Predicted values of NEWLNIGG (difference from baseline on log scale).**
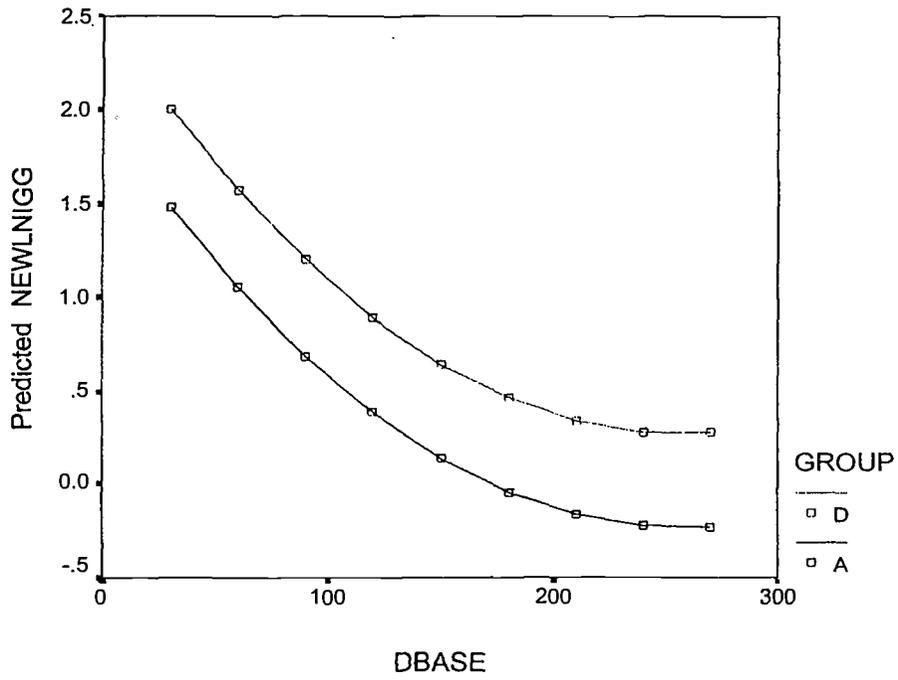
Fig 13. Predicted values of NEWLNIGG for groups B and D.
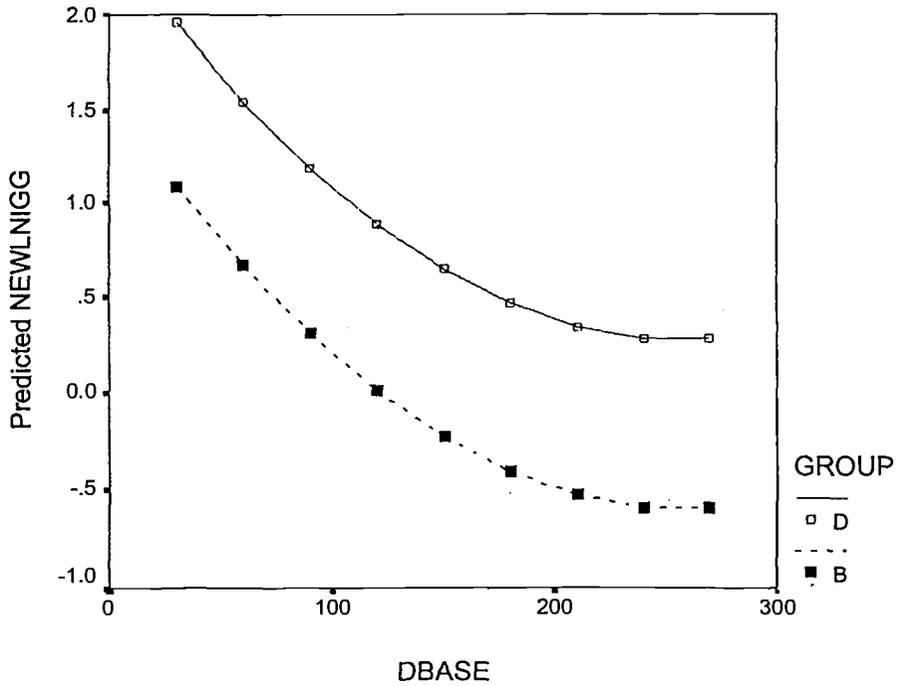
Analysis of Immunological Data from
Study of Askeaton and Abbotstown Steers

Summary

This report presents the statistical analysis of immunological data on 24 steers residing in two locations (VRL at Abbotstown and Ryan's farm in Askeaton). The steers were purchased in November 1996 and allocated to Abbotstown or Askeaton (12 to each location), in order to help address the question as to whether compromised immune function is associated with Askeaton. A total of 216 blood samples were collected from these steers between Nov. 1996 and Feb. 1998, and various indicators of immune function were measured.

An analysis of the first bleed from each animal indicated that the two groups were comparable for all but one of the immunological variables examined. The exception was PWM, which was significantly higher ($p < .001$) in the VRL group. Analysis of the last bleed, when the animals had been more than one year in their location, indicated that most of the immunological variables differed significantly between the two groups.

Introduction

In recent years there has been a reported increase of morbidity and mortality in cows on at least two farms in Askeaton, Co. Limerick. The question arose as to whether exposure to environmental factors in Askeaton may be associated with compromised immune function. A previous study begun in 1995 attempted to address this question by transferring some cows from Askeaton to VRL at Abbotstown and transferring a similar number of cows from VRL to Askeaton. The cows that were moved (together with a similar number that remained in their original location) were then bled periodically so that their immune function could be monitored. A number of difficulties with that study prompted the investigators to undertake an additional study, where steers would be purchased, allocated to Askeaton or Abbotstown and monitored over time for their immune function.

Twenty-four steers were purchased in November 1996 and allocated to VRL and Askeaton (twelve to each location). The animals were studied for a period of approximately 15 months (until February 1998), during which time a total of 216 blood samples were drawn for laboratory analysis of immune function (Table 1). The objective of the study was to determine whether there was evidence of a difference in immune function in the two groups.

Table 1

Group	Total Bleeds	Steers X Bleeds	Age at first bleed
R	96	8	0.8 yrs.
V	120	10	0.8 yrs.

Methods

The immune response variables measured on the blood samples were divided into four categories: Lymphocyte subsets (**BCELL, CD2, CD4 and CD8**), Proliferation response (**CONA, PWM, PHA**), specific immune response (**KLH and KLHIGG**) and trace elements (**Copper, Selenium**). Not all of these responses were measured on every bleed date, with the result that there were varying numbers of observations available for analysis of each response.

For each of the response variables, Q-Q plots were inspected to establish whether these variables were approximately normally distributed, and to determine what transformations of the data, if any, might be required. These plots indicated that **BCELL, CD2, CD4, CD8** and **PWM** were approximately normally distributed. However, **CONA, PHA, Cu** and **Se** required transformation and the transformed measures were used in subsequent analysis. For **CONA** and **PHA** the ratio of the gross value to background, expressed on the natural log scale, was approximately normally distributed. For **Cu** and **Se**, a simple log transform sufficed.

The **KLH** and **KLHIGG** variables refer respectively to the proliferation assay and the antibody response following **KLH** stimulation. These responses would be expected to rise to a peak (at approximately 3 months and 1 month respectively post-immunisation) and decrease over time thereafter to their pre-immunisation levels. We transformed **KLH** in the same way as **CONA** and **PHA** (i.e. the ratio of the gross value to background on the log scale) to create the variable **LNRTKLH**. We also computed the change from pre-immunisation "baseline" levels as follows: all pre-immunisation values for a given animal were averaged to give a baseline for that animal, and subsequent values were then expressed as differences from baseline. The standardised **KLH** variable thus created was called **NEWLNRTK**.

For the simple analyses of the first (or last) available **KLHIGG**, no transformation was required. For the analysis of change in KLHIGG over time we computed the change from pre-immunisation levels for the log-transformed KLHIGG, to create the variable **NEWLNIGG**.

It should be noted that 6 steers (3 in each group) were not immunised with **KLH**, so that these animals only contribute to the analysis of baseline **KLH** and **KLHIGG** and not to the changes from baseline over time.

The response variables used in the analysis are:

- BCELL
- CD2
- CD4
- CD8
- PWM = gPWM - bkgd
- LNRTCON = ln(gCONA / bkgd)
- LNRTPHA = ln(gPHA / bkgd)

- LNCU = ln(Cu)
- LNSE = ln(Se)
- LNRTKLH = ln(gKLH / bkgd)
- NEWLNRTK =LNRTKLH at time T - average LNRTKLH at baseline
- KLHIGG
- NEWLNIGG =ln(KLHIGG) at time T - average ln(KLHIGG) at baseline

where the prefix "g" indicates gross counts, "bkgd" indicates background.

A summary of the analysis variables is presented in Table 2, where log-transformed variables are presented on the raw scale for ease of interpretation.

Table 2 Summary of response variables

Variable	Number of Observations	Min	Mean	Max
BCELL	188	6	24.73	54.1
CD2	194	25.1	50.25	80.8
CD4	187	9	23.92	39.2
CD8	186	10.7	21.64	45
PWM	201	40545	226373	895692
gCONA/bkgd	201	0.65	176.22	1059.72
gPHA/bkgd	200	2.98	132.5	747.11
CU	95	8.10	13.04	26.10
SE	78	0.33	1.68	3.29
gKLH/bkgd	153	0.30	8.97	46.51
KLHIGG	189	96.39	904	15204

Analysis

The first data was available for the 24 steers from blood drawn in early November 1996 (Table 3). From Table 4, it can be seen that the first available laboratory tests showed that the two groups were similar for all variables except PWM. It is to be expected that the groups should be similar at this time, since they consist of animals of the same age that were purchased a short time before and so have had very little exposure to the two different test locations.

Table 3

First Available Date	V	R
04 Nov 96	12	0
11 Nov 96	0	12

On analysis of the last available blood test, however, it was found that for BCELL, CD2, CD4, CU, PWM, SE, and KLHIGG, the two groups differed significantly. For all variables except CU, these significant differences were also borne out by a repeated measures analysis of all bleeds from all animals.

Table 4   Comparison of average responses for two groups at first and last bleeds and p-values from repeated measures analysis.

	First Bleed			Last Bleed			Repeated Measures
Variable	R	V	p-value	R	V	p-value	p-value
BCELL	13.317	16.108	0.25	21.317	35.275	0.0001	**0.04**
CD2	52.8	56.625	0.29	57.1	42.058	0.0001	**<0.001**
CD4	23.717	26.333	0.24	27.067	18.4	0.0007	**<0.001**
PWM	147974*	243014	**<0.001**	233807	194688	0.04	**0.004**
LNRTCON	4.698	3.909	0.16	5.0282	4.7771	0.36	0.44
LNRTPHA	4.509	3.592	0.10	4.8892	4.5496	0.23	0.28
LNRTKLH	0.643	0.549	0.77	2.1886	2.0991	0.86	**.03** ⊗
KLHIGG	465.9	294.6	0.17	363.73**	210.95	0.005	**0.049** ⊗
CU	14.75	15.675	0.34	11.625	13.975	0.03	0.46
SE	1.691	1.533	0.52	0.5325	2.5548	<0.0001	**<0.001**

⊗ repeated measures analysis used the change from baseline of each of these variables, as discussed below.
*one extreme observation with PWM > 500000 removed
** 2 extreme observations with KLHIGG > 1000 removed

KLH and KLHIGG

The KLH values included in the first bleed analysis are baseline (i.e. pre-immunisation) values, while those in the last bleed analysis are at 251 and 282 days post-immunisation. The analysis of first and last bleeds indicated no significant difference between the two groups at these two times, and this was apparent from a plot of the average KLH response at each time point (see Fig. 1). However, this plot indicated that group R may have a more gradual KLH response, and this is even more apparent from a plot of the change in KLH from baseline (Fig. 2). A repeated measures analysis found significant evidence (p=.03) that the two groups do differ in their KLH response.

The analysis of KLHIGG at first (pre-immunisation) and last bleed indicated that the two groups are similar at first bleed but different (p=.005) at last bleed. These findings are apparent from a plot of the average KLHIGG versus time from inoculation (Fig 3), where it can also be seen that group R appears to have a lower maximum response . The KLHIGG response is expressed as the change from baseline on the log scale) in Figure 4, where the lower peak response for group R is also apparent. Since KLHIGG would be expected to peak at approximately 28 days post-inoculation, the first available blood after inoculation (at 41 or 42 days) should represent the maximum response measured for any animal. An analysis of the change from baseline, NEWLNIGG at day-41 and day-42 indicates a lower average response for group R, but this does not achieve statistical significance (p=.12), possibly due in part to the small sample sizes. A repeated measures analysis of NEWLNIGG however finds evidence (p=.05) of a difference in the KLHIGG response for the two groups.

Fig 1. Average LNRTKLH over time from inoculation for both groups.
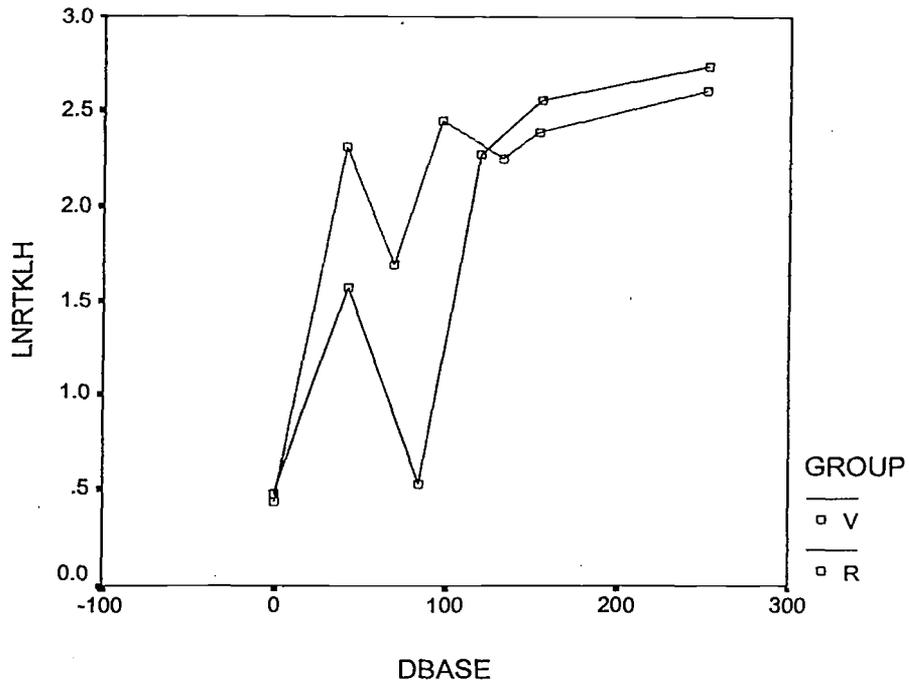

**Fig 2. Average change in LNRTKLH from baseline for both groups.**
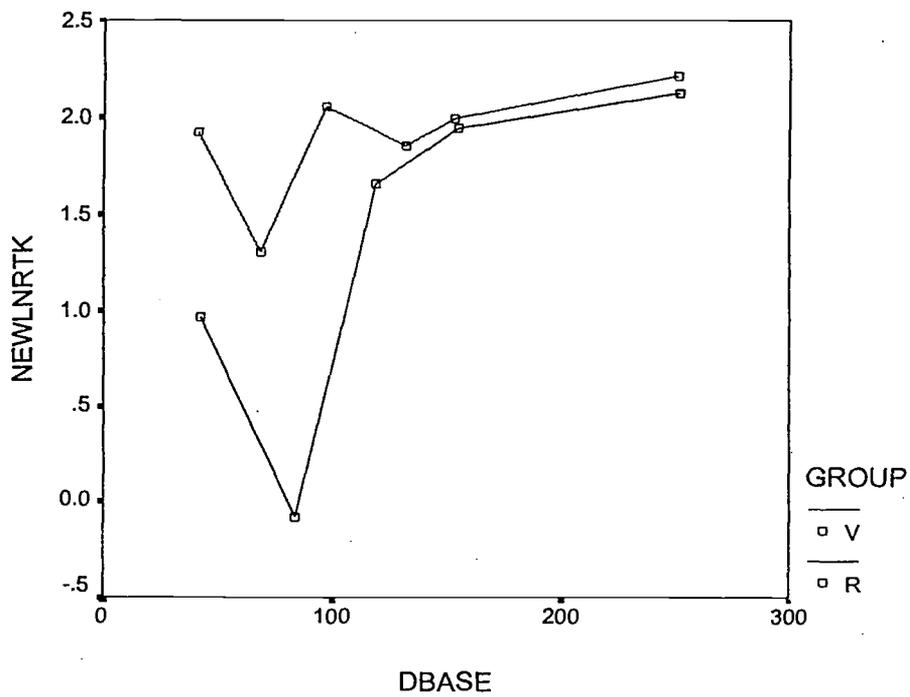
**Fig 3. KLHIGG versus time from inoculation for both groups**
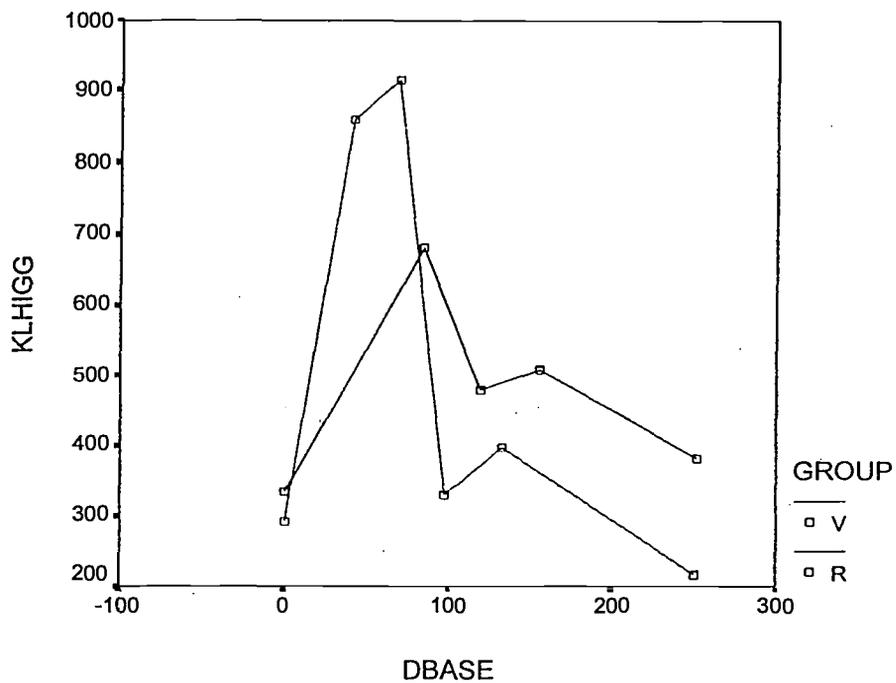
Fig 4. Change in KLHIGG (on log scale) from baseline for both groups.

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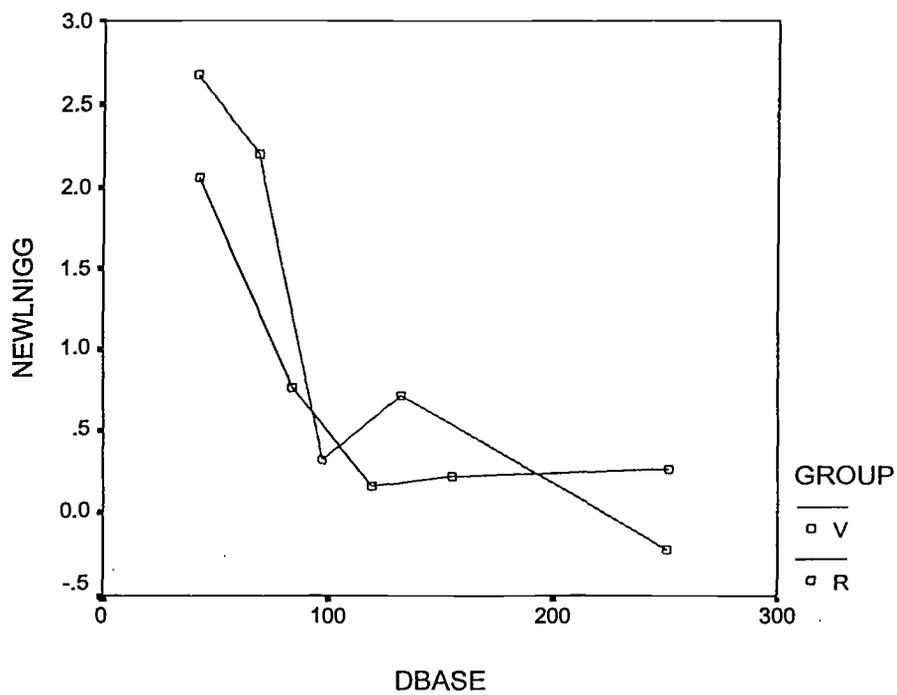
APPENDIX 12

Report on Vole Liver Enzyme Studies

Author: J. Donlon. Biochemistry Department, NUIG

We have previously reported [Fallon *et al.*, (1997) *Biology and Environment: Proc. Royal Irish Acad.* **97B**, 115-119] that in order to identify some novel parameters for monitoring environmental impact of industrial pollutants, we measured the hepatic phenylalanine hydroxylase and glutathione peroxidase activities of two species of feral rodents:- the bank vole *Clethrionomys glareolus* and field mouse *Apodemus sylvaticus* trapped at two isolated rural areas and at a study site adjacent to the Alcan bauxite refinery.

There was no difference in activities between mice from study site near Askeaton and reference sites (Coole Park, Co. Galway and Dromore Wood, near Ennis). However, enzyme activities of both male and female voles were lower at the Askeaton site, and males showed severe. diminution of hepatic tetrahydrobiopterin-dependent phenylalanine hydroxylase (27% of reference values). Western blotting experiments revealed normal abundance of phenylalanine hydroxylase protein, indicating loss of catalytic function. Loss of phenylalanine hydroxylase activity may have been due to oxidative stress resulting from an accompanying deficiency in glutathione peroxidase activity, which for voles of both sexes at the Askeaton site was half that of the references. We concluded that these two enzymes in bank voles may provide useful bioindicators of the impact of industrial pollutants.

For the above studies the animals had been trapped in the Autumn of 1995, Spring 1996 and Autumn 1996. For this more recent study, animals were trapped in the Autumn of 1997 and we set out to establish if the enzyme deficiencies were still extant at the same site near Askeaton and also included an additional reference site, Dundrum Forest, Co. Tipperary. Since glutathione peroxidase is a selenium-dependent enzyme, this current study also included determination of the selenium content of the livers of voles. An initial attempt was also made in order to establish the nature of the deficiency of glutathione peroxidase in the vole livers.

Liver phenylalanine hydroxylase activities and glutathione peroxidase activities were also determined in case of groups of laboratory rats that had been fed diets containing soils from three farms. These feeding experiments were conducted at another third level institution in Dublin.

RESULTS

Table 1 shows the specific activities of liver phenylalanine hydroxylase and of glutathione peroxidase from male bank voles trapped at Askeaton, Coole Park (one of the original reference sites) and Dundrum (additional reference site). The activities of phenylalanine hydroxylase were measured in the presence of two of the cofactors for this enzyme, i.e. tetrahydrobiopterin (BH_4) and dimethyltetrahydropterin ($DMPH_4$).
These results show that the deficiencies in phenylalanine hydroxylase activities originally found in voles near Askeaton no longer exist. In the case of the phenylalanine hydroxylase activities in the livers of male voles trapped in Dundrum Forest, the $DMPH_4$-dependent activities are significantly lower, but this is not considered to be of any significance *in vivo* since the BH_4-dependent activities do not differ from the other groups. In the case of glutathione peroxidase the activities are significantly higher for those animals trapped at Dundrum ($p < 0.005$) relative to the other groups of animals.

In an experiment designed to specifically determine the relative abundance of glutathione peroxidase present, we conducted western blotting experiments using three liver extracts, i.e. from (a) a male vole from Askeaton ('97); (b) a male vole from Coole Park ('97) and (c) a male vole from Askeaton ('96), which had displayed a very significant loss in glutathione peroxidase activity. The extracts were subjected to non-denaturing electrophoresis in order to resolve multiple forms of this enzyme. The affected sample did show diminution of both forms of glutathione peroxidase with a more marked loss of the slower migrating band. If other affected samples are found, this approach should be investigated further.

In relation to determining the reason for the loss of glutathione peroxidase activity, the selenium content of vole livers was also analysed using some of the samples collected at the same time as those used to generate the data of Table 1.

Tables 2 and 3 show the results obtained for the experiments where laboratory rats were fed diets containing soils. Two sets of three groups each were examined; Table 2 shows data from the first generation pups and Table 3 shows data for dams at completion of feeding experiments. The data are reported with groupings listed by their colour code. No differences were observed; the higher activities in new-born male rats are as expected.

TABLES

TABLE 1: Specific activities of phenylalanine hydroxylase and of glutathione peroxidase from livers of male bank voles.

Site	Phenylalanine (BH$_4$)	Hydroxlyase (DMPH$_4$)	Glutathione peroxidase
Askeaton	0.57 ± 0.10	5.13 ± 0.53	41.8 ± 3.5
Coole Park	0.40 ± 0.03	4.37 ± 0.43	51.2 ± 4.7
Dundrum	0.43 ± 0.03	2.27 ± 0.30	95.4 ± 15.3

The values are in nmol/min/mg protein, with 8 samples in each group.

TABLE 2: Specific activities of phenylalanine hydroxylase from male new-born rats whose mothers had been fed diets containing soils from three locations.

Site	Phenylalanine (BH$_4$)	hydroxlyase (DMPH$_4$)
Yellow	2.00 ± 0.17	8.93 ± 0.63
Blue	2.03 ± 0.13	11.20 ± 1.10
Red	1.87 ± 0.10	11.20 ± 1.10

The values are in nmol/min/mg protein, with 8 samples in each group.

TABLE 3: Specific activities of phenylalanine hydroxylase and of glutathione peroxidase from livers of female rats that had been fed diets containing soils from three locations.

Site (n)	Phenylalanine hydroxlyase (DMPH$_4$)	Glutathione peroxidase
Yellow (5)	5.07 ± 1.00	63.6 ± 5.5
Blue (5)	4.40 ± 0.67	60.2 ± 11.6
Red (7)	4.57 ± 0.67	63.0 ± 8.7

The values are in nmol/min/mg protein, with n indicating the number of samples in each group.

APPENDIX 13

Retrospective Survey - Reported duration and severity of disease incidents in individual Farm Reports

Note: Except for infertility, percentages indicate disease incidence. For infertility, percentages indicate percent cows infertile. NA = problem reported but insufficient information to estimate incidence or severity. Blank cell = no data or not reported to be a problem. In column 1, 'conditions' are generally listed as described in the individual Farm Reports. These may not always correspond with those used in Chapter Five where some attempt was made to apply a diagnosis.

Condition	Animal[1]	Farm	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
Abortion/Premature	Cow	2	~3%	~3%	~3%	~3%	~3%	~3%	~3%	~3%	~3%	
Abortion/Premature	Cow	5								12%	NA	~3%
Abortion/Premature	Cow	17								22%		
Abortion/Premature	Cow	25			(10%)*							
Abortion/Premature	Cow	27			~6%						~8%	
Abortion/Premature	Cow	29			12%			4%				
Abortion/Premature	Cow	31							3%	3%	5.30%	
Abortion/Premature	Mare	32							(30%)*		~10%	
Behavioural	Pup	32							100%			
Border disaese	Lamb	26								~2.5%		
BVD	Calf	26								~10%		
Congenital Deform.	Calf	6								1.30%	3.70%	
Congenital Deform.	Calf	7									2.40%	
Congenital Deform.	Calf	8		1.50%	1.50%	1.50%	1.50%	1.50%	1.50%			
Congenital Deform.	Calf	17				2 ~ 3 cases per year						
Congenital Deform.	Calf	20								~2%	~2%	
Congenital Deform.	Calf	21								~2%	~4%	
Conjunctivitis	Cow	15									11%	
Conjunctivitis	Cow	20									~2%	
Conjunctivitis	Cow	21					NA			NA	~90%	
Deaths	Calf	2				~10%	~10%	~10%	~10%	NA	NA	
Deaths	Calf	6								6%		
Deaths	Calf	17				~1%	~1%	~1%	~1%	~1%		
Deaths	Calf	29				(20%)*						
Deaths	Calf	30								5%	5%	

...\contd.

Condition	Animal	Farm	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
Deaths	Calf	32	NA	NA	NA	NA	NA	NA	NA	NA	-90%	
Deaths	Cow	2				-3%	-4%	-4%	-4%	-20%	-24%	
Deaths	Cow	1		-9%	-9%	-9%	-9%	-12%	-21%	-3%	-8%	
Deaths	Cow	14								14%	16.6%	
Deaths	Cow	16						7%	0%	2.70%	4.30%	
Deaths	Cow	24									12.5%	
Deaths	Cow	29				17%	-7%	-7%	-7%	-7%	17-25%	
Deaths	Growing	9						20%		2.10%		
Deaths	Growing	32	NA	NA	NA	NA	NA	NA	NA	NA	33-44%	
Deaths	Sheep	32								NA		
Deaths	Wildlife	2								NA		
Deaths	Wildlife	13	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Delayed calvings	Cow	16						NA	NA	NA	NA	
Diarrhoea	Calf	1				NA	NA	NA	NA	NA	NA	NA
Diarrhoea	Calf	9					NA	NA	NA	NA	NA	NA
Diarrhoea	Cow	20									-4%	
Diarrhoea	Growing	17	.				-6%	-6%	-6%	-6%	-6%	
Disease & deaths	Horse	32					-10%	-12%	-14%	-23%	-8%	
Downer	Cow	5	NA	NA	NA	NA	NA	NA	NA	NA	9%	
Downer	Cow	8			13-15%	13-15%	13-15%	13-15%	13-15%	13-15%	13-15%	
Downer	Cow	17								5 - 7%		
Downer	Cow	31									2.60%	
Dystocia	Cow	2									NA	
Dystocia	Cow	26									~100%	
Eye lesion	Cow	32		.							3.80%	
Eye lesion	dog/cat	6									NA	
Facial paralysis	Cow	20								-2%		-2%
Illness	Growing	13						100%	Depop.			
Illness	Sheep/goats	13						100%				
Illthrift	Adult	2				NA	NA	NA	NA	NA	NA	
Illthrift	Adult	21					> 50%	NA	NA			

...\contd.

Condition	Animal	Farm	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
Illthrift	Cow	2				NA	NA	NA	NA	NA	NA	
Illthrift	Cow	1						~27%				
Illthrift	Cow	6								NA	~18%	
Illthrift	Cow	7						NA	5.50%			
Illthrift	Cow	24									~40%	
Illthrift	Growing	5			NA	NA	NA	NA	NA	NA	80-100%	
Illthrift	Growing	27									80-100%	
Illthrift	Lamb	21		NA	NA	NA	NA	NA	NA	NA	NA	
Illthrift	Lamb	32									NA	
Illthrift/deaths	Growing	9						1.70%	1.70%	NA		
Infertility*	Cow	1			~9%	12%		27%	5.9%	12%	18%	12%
Infertility	Cow	5					~19%	~35%	~10%	~10%	~13%	
Infertility	Cow	6								~8%	~16%	
Infertility	Cow	7							10%	10%	10%	
Infertility	Cow	8			(20)*	(20)*	(20)*	(20)*	(20)*	(20)*	(20)*	(20)*
Infertility	Cow	9	NA	NA	NA	NA	NA	NA	NA	CR[2]	CR	CR
Infertility	Cow	12			NA	NA	NA	NA	NA	24%	9%	NA
Infertility	Cow	15							NA	NA	NA	NA
Infertility	Cow	16							NA	NA	7.3%	NA
Infertility	Cow	18			NDO[3]	NDO	NDO	NDO	NDO	NDO	NDO	NDO
Infertility	Cow	19				NDO	NDO	NDO	NDO	NDO		
Infertility	Cow	21								~25%	NA	
Infertility	Cow	22					~5%	~5%	~5%	~5%	~5%	~5%
Infertility	Cow	25							NA	NA	NA	NA
Infertility	Cow	27								27%		
Infertility	Cow	30									10%	
Infertility	Cow	31								10%		
Infertility	Sheep	32							~11%	NA	~29%	
Irritability at milking	Cow	1						(30%)*	(30%)*	(30%)*	(30%)*	
Irritability at milking	Cow	5										NA
Irritability at milking	Cow	6										(100%)*

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Condition	Animal	Farm	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
Lameness	Cow	2	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Lameness	Cow	6								11%	NA	
Lameness	Cow	8			NA				NA	NA	25-30%	NA
Lameness	Cow	25								~15%	~8%	
Locomotor	Calf	6									40%	
Locomotor	Calf	23							~60%			
Low milk yield	Cow	17					NA	NA	NA			
Mag. tetany	Cow	21			~12%	~3%	~3%	~3%	~12%			
Mag. tetany	Cow	24								12.5%		
Mastitis	Cow	2	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Mastitis	Cow	6								4.30%	15%	
Mastitis	Cow	9			34%	29%	30%	23%				
Mastitis	Cow	25			~20%		~40%	~45%	~20%	~5%		
Milk drop	Cow	22	0%		0%	0%	0%	0%	0%	0%	100%	
Neoplasia	Cow	12										5%
Neoplasia	Cow	25						~2%				
Pneumonia	Bull	9								1 case		
Pneumonia	Calf	9									50%*	
Pneumonia	foal	32	NA					NA	80-100%			
PNM[4]	Calf	1	NA	NA	NA	NA	NA	NA	NA	NA	NA	
PNM	Calf	6								7%	13%	10%
PNM	Calf	7						~11%	~25%	~25%	~11%	
PNM	Calf	9	8%					NA	NA	35%	17%	~3.3%
PNM	Calf	16						13%	5.60%	8%	15%	
PNM	Calf	17				16 - 20%	16 - 20%	16 - 20%	16 - 20%	16 - 20%	Depop	
PNM	foal	5									1 of 2	
PNM	Neonate	25			4%*	~22%						
Poor milk yield (gals)	Cow	2	NA	NA	NA	717	720	709	690	645	NA	
Poor milk yield (gals)	Cow	6					1185	1277	869	908	806	
Poor milk yield (gals)	Cow	7				474	486	405	270	574	556	
Redwater	Adult	31									18%	

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Condition	Animal	Farm	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996
Resp-Ent	Adult	24	NA	NA	NA	NA	NA	NA	NA	100%	100%	
Resp-Ent	Calf	2	NA	NA	NA	NA	NA	NA	NA	NA	NA.	
Resp-Ent	Calf	8			80%*	80%*	80%*	80%*	80%*	80%*	80%*	NA
Resp-Ent	Calf	21									NA	NA
Resp-Ent	Calf	25							high	High		
Resp-Ent	Calf	32	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Respiratory	Adult	31								58%		
Respiratory	Calf	17					~100%	~100%	~100%			
Respiratory	Calf	20									-26%	
Respiratory	Growing	14							7%	9%	20%	
Respiratory	Growing	23					High	High	High	High	High	
Respiratory	Growing	31								100%		
Respiratory	Horse	20									NA	
Retained placenta	Cow	21									-30%	
S/C abscess	Cow	21								-30%		
Salmonellosis	Calf	29	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Salmonellosis	Cow	29			12%			4%				
Skin lesions	Cow	2								>50%**	>50%*	
Skin lesions	Cow	1				NA	NA	(30%)*	(30%)*	(30%)*		
Skin lesions	Cow	6									13%	
Skin lesions	Cow	32							NA	NA	NA	
Skin lesions	Horse	32					(-8%)*	(-20%)*	(-6%)*	NA	NA	
Skin lesions	Sheep	32							-5%	NA	NA	
Trauma	Adult	31	NA	NA	NA	NA	NA	NA	NA	NA	2.00%	
Twinning	Cow	2	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Twinning	Cow	6							-2.5%	-2.5%	-2.5%	

[1] Animal type: bovine unless otherwise stated. [2] Reduced Conception Rate [3] Increased rate of Non-Detected Oestrus. [4] Perinatal calf mortality.

* Data unreliable.

APPENDIX 14

Pathology and Clinical Pathology Submissions to Limerick RVL from Askeaton area.

September 1995 to end 1996

Tissues and carcasses *(amended version of* Table 3.8-3 *EPA 2nd Interim Report, 1998)*

Ref No.	Submission	Herd ID	Result
1495	Foetus	1	Pulmonary atelectasis, anoxia
1808	Foetus	19	No specific findings
328	Weanling	12	No specific findings
2514	Stillbirth	2	Asphyxia assoc. with dystocia
2534	Cow	2	Obturator paralysis
3636, 3637	Foetus & bloods	2	No specific findings
3284, 3285	Placenta & bloods	19	No specific findings
2277	Calf	9	Virus pneumonia (RSV)
604, 1131,	Foetuses	12	*Campylobacter foetus,*
1810, 3823			anoxia/atelectasis
2495	Calf	12	Lead poisoning
103	Cow	28	Downer cow
485	Foetuses x 2	28	No specific findings
839	Stillbirth	28	Atelectasis, thyroid hyperplasia
914	Weanling	28	BVD virus positive
1157	Cow	28	Periparturient death
3922	Foetus	28	No specific findings
3687	Ram	18	Parasitic gastro-enteritis
3990	Ewe	3	Parasitic gastro-enteritis
1178	Cow	21	Mastitis
2682	Calf	21	BVD virus positive
2595	Calf	21	*Salmonella dublin* & IBR
3312, 3313	Calf	28	BVD virus positive
3261	Calf	11	Pneumonia

Clinical Pathology (bloods, faeces, milks, swabs). *(*Table 3.8-4 *EPA 2nd Interim Report, 1998 - amended).*

Ref No.	Date	Submission	Herd ID	Result
2121		Blood	1	Liver dysfunction (photosensitization)
Various	Various	Milks	1	Staphs, Streps, Coliforms, etc.
Various	Various	Milks	2	Streps & Staphs.
Various	Various	Calf faeces	2	Cryptosporidia (1 submission) & no specific findings
Various	Various	Cow faeces	2	No specific findings
2838		Bloods	2	Babesiosis
Various	Various	Bloods	2	No specific findings
2306		Milk	1	Streps & Staphs.
3025		Bloods	3	BVD virus positive
2565		Skin scraping	28	No specific findings
3261		Swabs	9	Virus negative
2596		Calf faeces	21	No specific findings
2413		Bloods	28	Herd BVD test
557 *et al*		Bloods etc.	28	No specific findings
2909		Bloods	12	No specific findings
378, 3298, 3299		Bloods & placenta	11	No specific findings
Various		Milk, faeces & sheath washings	12	No specific findings

All Submissions from Askeaton area January to June 1997 (inclusive).

RefNo.	Submission	Herd	Result
57	Milks x 76	1	*S.aureus* x 1, *S.dysgalactiae* x 2, *A.pyogenes* x 1
65	Stillbirth	9	Foetal atelectasis/hypoxia, 6 fractured ribs.
91	Stillbirth	9	Foetal atelectasis/hypoxia
92	Faeces x 4	9	Neg. Fluke, worms, coccidia
150	Foetus	31	No specific findings.
160	Calf 1 day	31	Atresia of anus and posterior rectum, no tail.
176	Milks x 64	2	*S.aureus* x 8, *S.dysgalactiae* x 2, *S.uberis* x 4
208	Faeces x 2	9	Positive Cryptosporidia (x 2)
325	Blood x 1	6	Serology: BVD virus - Antibody Pos. Virus Neg.
423	Blood	6	BVD bleed 35. Rebleed
459	Blood	6	No specific findings
463	Blood x 4	9	ZST = 13; 10; 23; 22 – calves
470	Blood x 6	22	Thyroxine = 101; 61; 78; 60; 79; 45
497	Stillbirth	1	Histopathology: Lung – Amniotic debris present in bronchi and bronchioles.
498	Stillbirth	9	Histopathology: Lung Diffuse exudative pneumonia sequel to inhalation of amniotic debris
545	Stillbirth	6	Amniotic fluid in bronchioles and alveoli with atelectasis of lungs i.e. evidence of intrauterine anoxia/asphyxia.
546	Blood	6	Ca, I, P, Cu slightly low; Albumin subnormal (Cow post calving – stillbirth.
547	2x Uterine discharge	6	No. 2731 – *Actinomyces pyogenes*. No. 43 NSF
548	Bloods x 3	7	ZST = 28; 10; 23 Calves biochemistry.
593	Foetus	5	Brachygnathia inferior. Bilaterial renal agenesis.
595	Blood	5	Post-abortion biochemistry, serology. Low Ca, Mg, Cu, Thyroxine.
608	Cow head	6	No specific findings.
637	Blood x 5	1	Rebleed. Previous Low PCV in 3; 2 hair loss.
645	Milk	6	No specific findings.
663	Milk x 2	7	*S. dysgalactiae* & *E. coli* x 1
686	Faeces	1	Neg. Fluke, Worms, Coccidia
687	Calf faeces	1	*E. coli* only
688	Skin Scraping	1	Neg. Ectoparasites, fungal spores & hyphae
689	Faeces	2	Neg. Fluke, Worms, Coccidia
702	Blood	9	γGT 238.8 Raised. BVD virus negative Cows stillbirth (See 729)
723	Bloods x 3	14	Serology BVD virus PLA-2/3 positive
724	Bloods x 1	14	Heifer illthrift. Biochemistry and Haematology - normal values.
729	Stillbirth x 2	9	Amniotic debris in lungs. Evidence of anoxia/asphyxia - Delayed calving
748	Milk	9	*E. coli.*
813	Skin	1	Biopsies Cow No. 87 – Non-specific dermatitis
863	Stillbirth	2	Decomposition advanced suggesting prepartum intrauterine death.
905	Stillbirth	7	foetal atelectasis – hypoxia
934	3 Calf Faeces	8	*E. coli.*
957	Milk	1	NSF
963	Milk	2	*S.dysgalactiae.*
964	Calf faeces	2	*E. coli.*
1097	Calf faeces	7	*E. coli.*
1135	Calf faeces	7	Positive rotavirus.

1298	Blood x 1	2	Cow Pneumonia. Raised White Cell Count (see 421).
1415	Milk x 12	2	*S.aureus* x 2; *S.dysgalactiae* x 1
1465	Milk x 1	8	*A.pyogenes*
1485	Blood x 1	7	Cow recumbent post calving. Ca, Mg, T_4, Albumin – Low. CPK, GOT Raised. Anemia, Low WCC.
1506	Cow	7	Muscle damage right thigh - trauma from nerve damage and milk fever. Also Ostertagiasis (*see* 1485).
1513	Blood x 1	6	Heifer not thriving. Haematology - check infection.
1541	Blood x 1 ex bull	5	Biochemistry, haematology, serology-routine check
1542	Blood	5	Biochemistry (Blood copper) – normal
1543	Blood x 6	7	Follow up on comrades - see 1485 & 1506
1618	Faeces x 6	7	Negative Fluke, Worms, Coccidia
1644	Blood x 1	6	Cow Chronic, pneumonia and lameness Haematology and biochemistry
1678	Blood x 5	1	Biochemistry Copper - 1/5 slightly subnormal
1685	Pus sample	2	*A. pyogenes*
1717	Milks x 16	6	*S. aureus* x 1
1718	Bloods x 4	6	Cows illthrift. Biochemistry, Haematology - routine check
1816	Stillbirth	6	Foetal atelectasis. Lung histopathology - mild diffuse exudative pneumonia. Enlarged thyroid wt 35.3 gm.
1817	Blood x 1	6	Mg subnormal 0.60 mmol/l - Cow stillborn calf
1818	Blood x 1	6	Glucose 3.6 mmol/l (Rebleed) had high glucose previously
1822	Bloods	26	Retrospective Survey ReVisit
1853	Blood	6	Anemic, Positive Babesia
1876	Blood	2	Follow up haematology. Cow haemorrhage had low PCV Now normal
1894	Viscera	7	Vitreous humor Mg 0.45mmol/l – Low
1906	Calf	24	Widespread petechiation and cerebral congestion suggested anoxia/asphyxia delayed calving?
1921	Blood	5	Serology Leptospira hardjo 1/400
1939	Milk x 1	2	No specific findings
1975	Milk x 1	1	*S. dysgalactiae*
1978	Bloods	24	Investigation Perinatal mortality Haematology, Biochemistry various abnormal parameters
1980	Bloods	23	Retrospective Survey ReVisit
2038	Milk x 1	9	*S. aureus*
2039	Calf faeces	1	Positive Rotavirus
2116	Stillbirth	6	Dead in utero for some time
2117	Blood	6	stillborn calf (+ dam)
2122	Bloods	27	Study – Self Reporting Farms
2150	Milk x 24	1	*Corynebacterium bovis* x 4. *S. aureus* x 1
2151	Faeces x 3	1	Negative fluke, strongyle & lungworm
2174	Bloods	20	Retrospective Survey ReVisit
2213	Bloods	13	Retrospective Survey ReVisit
2243	Bloods	5	Cows coughing. Haematology, Biochemistry, virology - results pending.
2280	Bloods	19	Retrospective Survey ReVisit
2285	Bloods	27	Haematology, Serology, Biochemistry - Hypomagnesemia
2326	Milk x 34	7	*S. aureus* x 31

Industrial Pollution in the Shannon Estuary: the Possible Harmful Effects of Sulphur Dioxide Emissions on Livestock in the Askeaton Area of Co. Limerick

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Industrial Pollution and Animal Health in Co. Limerick

Considerable industrial development along the Shannon estuary has raised fears of the harmful effects of pollution of atmosphere, soil, pasture and crops on the health of both livestock and human populations. Planning considerations led the Limerick County Council (LCC) to commission baseline surveys of soils and crops (Fleming & Parle, 1982) and animal production (Rogers & Poole, 1984) before several new enterprises began operation, including Auginish Alumina on the island of that name. The Environmental Protection Agency (EPA) was asked to coordinate the assessment of subsequent changes in environmental quality and in both animal and human health, taking direct responsibility for monitoring the environment and guidance on health matters from toxicology experts from the US and the UK. Two interim EPA reports published in 1995 and 1997 provided the information for the present external assessment of industrial pollution which was asked to provide particular guidance on the environmental and nutritional significance of SO_2 emissions from Auginish Alumina and two large power stations at Moneypoint and Tarbert, using the considerable data base which had been accumulated in 1994-6.

1. Significance of SO_2 emissions in Co. Limerick

The EPA reports (1995, 1997) provide a comprehensive description of SO_2 in and deposited from the atmosphere at a number of sites, including two farms (Ryans and Somers, Askeaton) where major health problems had been allegedly caused by industrial pollution beginning in the mid 80's.

1.1 Direct atmospheric effects at Askeaton. The general levels of SO_2 in the atmosphere have declined slightly since reaching a peak in 1990 (Figure 5.1; EPA, 1997) due largely to reduced emissions from the major source, ESB, Moneypoint. The 24-hourly atmospheric SO_2 levels at the monitoring site closest to Ryans farm were never more than $1/_6$th of the national standard of acceptability (350 µg/cubic metre) between 1987-1994 (Figure 6.7; EPA, 1995). To cover the possibility that episodic peaks on the Ryan and Somers farms were not detected by 24-hourly monitoring elsewhere, hourly monitoring facilities were set up on both farms. The WHO limit of 325 µg/cu. m. was exceeded on only one occasion (on Ryan's farm) between October 1995 and December 1996 and values are generally low (<40) and stable (Appendix C; EPA, 1997). Episodic peaks were rare, small and little different from those recorded on a healthy control farm (Abbotstown). These levels of SO_2 emission are unlikely to have caused direct harm to local livestock.

1.2 Direct effects of SO_2 on plants. There is little to add to the interpretation of the data given in the EPA (1995, 1997) reports in which the levels of SO_2 detected near Askeaton are thought to be too low to harm plant life. The only aspect which has been overlooked is the exacerbative effect of high wind speed on SO_2 phytotoxicity. Mansfield and his associates have shown that the phytotoxic concentration of atmospheric SO_2 to S23 perennial ryegrass declines as wind speed rises (Ashenden & Mansfield, 1977). At a level below the WHO limit (110 v. 123 ppb), SO_2 was phytotoxic at a wind speed of 25 mmin⁻¹ but not at 10 mmin⁻¹. Such information has yet to influence legislative limits for SO_2 but could be relevant to the Co. Limerick situation. For

example, the "scorched" conifers which have caused concern at the problem farm(s) (EPA, 1995) may have been damaged by relatively low SO_2 concentrations carried at high speed onto the leaf surface. However, extrapolation from Mansfield's glasshouse wind tunnel experiments also raises problems. There is probably a "double dilution" effect of high wind speed at sites such as Askeaton. It would be surprising if there was not an <u>inverse relationship</u> between windspeed and <u>both</u> the <u>non-marine sulphur concentration downwind</u> from the point sources and the <u>proportion of non-marine sulphur present in the atmosphere</u>. Such relationships could be deduced by obtaining (if EPA does not already have it) wind speed data from nearby Shannon airport. If the relationships are confirmed, they would "defuse" any future argument that industrial activity has led to SO_2-rich wind damage to plant life since the damage could just as easily and more legitimately be attributed to wind-borne marine sulphur or marine sodium.

1.3 Indirect effects via soil and plant at Askeaton. Indirect harm may theoretically be caused by sulphur deposition which raises soil and pasture sulphur concentrations and induces deficiencies of selenium and copper in grazing animals. These two possible antagonisms were given prominence by the report of Rogers & Poole (1984) which highlighted risks of both selenium and copper deficiencies occurring in Co. Limerick due to local geochemical features BEFORE industrial expansions occurred. However, the levels of non-marine sulphur deposition recorded or predicted on the two farms are insufficient for such antagonisms to be potentiated. The additional inputs of 4 kg S/hectare for dry deposition predicted in the Askeaton area (p. 39; EPA, 1995) and the 5 kg non-marine S/ha deposited in rainfall at Somers farm (p. 41; EPA, 1995) <u>are of micro-nutrient proportions</u> and orders of magnitude less than those employed when sulphur - a macro mineral - is used as a fertilizer to raise forage sulphur concentrations (Table 1) or lower plant selenium in seleniferous areas (200 kg S/ha; Dhillon & Dhillon, 1992). The total atmospheric inputs of sulphur are barely sufficient to replace annual exports of sulphur in farm products and excreta with all-year-round grazing (Goh & Nguyen, 1997) and would not be expected to change background herbage sulphur or selenium levels, particularly with large sulphur outputs in silage which are incompletely returned via excreta. McClaren et al (1975) calculated sulphur balances for a site in S.E. Scotland where grass was cropped and inputs of sulphur from rain and dry deposition were similar to those found in Askeaton (5.3 and 6.3 kg S/ha, respectively): an additional 30 kg fertilizer sulphur/ha was required to achieve a sulphur balance. The most recent measurements of herbage sulphur and selenium concentrations at Ryan's and Somers' farms (p. 18-19; EPA, 1997) provide no convincing evidence for major changes for either element. Sulphur concentrations are not exceptional for Ireland (Table 4.1; EPA, 1997) though higher than most of the values recorded for Area C (which included Askeaton) in the baseline survey (2.4-4.2 g S/kg DM; Fleming & Parle, 1982) and for the Somers farm in the 25-farm study of Rogers & Poole (1984; 2.8 g S/kg DM - Farm 14, Table 3). However, the latter value was for summer only and the recent results show that sulphur values can vary widely from site-to-site and with season (Figure 4.1; EPA, 1997). Similar difficulties arise when trying to assess "before and after" changes in herbage selenium. Values for the two problem farms in 1995/6 fall within the wide range recorded in the baseline survey. If such selenium levels are associated with ill-health and poor milk yield, then similar problems should be commonplace in Co. Limerick.

A further assessment of indirect effects on the basis of indices of copper and selenium status in the livestock has been made (Table 2) but interpretation again raises difficulties. Levels of both copper and selenium in the blood have increased at both sites since the baseline survey was conducted, particularly on Ryan's farm where the initial values were subnormal and indicative of risks of copper and selenium deficiencies. The mean liver copper concentration in recently post-

mortemed stock (Appendix Table A1-5; EPA, 1995 - all but one value from Ryans stock) confirmed an adequate copper status even in casualties (mean value 1299 μmol/kg: minimum 691: adequate >105), assuming values are on a fresh weight basis. However, the improvement in copper status on Ryan's farm is probably due largely to improved, direct copper supplementation of the herd. There is no indication of whether similar changes were made in selenium supplementation. The observed changes on both farms are the net result of beneficial management or dietary changes and any harmful environmental effects: if the latter occurred, their influence was exceeded by positive effects.

1.4 Direct and indirect effects elsewhere in Co. Limerick. It has already been pointed out (EPA, 1995) that if SO$_2$ or other emissions from either power stations or Auginish Alumina are responsible for past ill health and poor production on the Ryan and Somers farms in Co. Limerick, sites other than Askeaton should be more affected. The modeling studies confirm that other sites, notably Kildysert on the opposite side of the Shannon estuary, should receive two to three times more sulphur from the combined emissions from the three point sources. Agreement of model predictions and field observations for SO$_2$ in the air at Askeaton suggests that model predictions for other sites can be trusted. The question which then arises is whether these higher inputs could be sufficient to harm livestock. It might be argued that any addition of sulphur to the already high background levels (mean 3.0 g S/kg DM: range 1.8-8.9; Fleming & Parle, 1982: Appendix 16) is a matter for concern and intervention. However, the effects of herbage sulphur concentration on copper availability (the cornerstone of both Cu x S and Cu x Mo x S antagonisms whereby copper deficiency is induced in livestock; Suttle, 1986) fall off exponentially in conserved forage as sulphur levels rise (see Figures 1a and 1b). In grazed pasture, the effect of sulphur declines as herbage molybdenum concentrations rise (Figure 1c). Thus, even the maximal additions of sulphur to the environment north of Co. Limerick are unlikely to cause any significant, detectable lowering of copper status in cattle (or sheep). The same can probably be said of selenium status although there is little or no published information on the quantitative relationship between soil sulphate status and selenium uptake.

2. Causes of ill-health and poor performance on the Ryan and Somers farms at Askeaton

The establishment of causal relationships in alleged incidents of non-specific disease outbreaks in farm livestock is notoriously difficult and requires a careful analysis of ALL relevant variables.

2.1 Management. It is noteworthy that *on each farm, a major management change had preceded complaints about animal health problems.* On Somers farm, there was an application of lime in 1986 which is known to increase risks of molybdenum-induced copper deficiency and manifested as increases in mortality, at least in sheep (Suttle & Jones, 1986). On the Ryan farm there was a switch from hay to big bale silage in 1986: analyses of subsequent silages indicated that it was generally of low quality. The feeding of poor quality silage during late pregnancy to the spring calving herd may have caused many of the problems attributed to pollution on the Ryan farm. The natural and wholly understandable reaction of the farmers, forewarned by the conduct of a baseline survey, is to blame external changes for any new problems, particularly when they are detectable by the visual and olfactory senses. Everyday occurrences such as white encrustation on dandelions and the purple tinge of N$_2$ deficiency become linked in the farmers mind to the same cause - atmospheric pollution by new industry. While a final conclusion should await the completion of animal trials and monitoring studies at both farms, the recent abrupt improvement in many aspects of health and the restoration of normal milk yields in indigenous stock with no

contemporary change in environmental quality (EPA, 1997) suggests that crucial management deficiencies had either spontaneously improved or been corrected.

2.2 Nutrition. Both management changes (liming and the switch to silage) would exert their influence by impairing the quality of nutrition (lowering either copper or energy status) but it is impossible to gauge the degree of management improvement from the interim reports (EPA, 1995; 1997). The reports give no indication of how the new, well recorded copper treatments and levels of dietary supplementation compare with what was done earlier. Furthermore, the all-important details of silage and pasture quality in 1994-6 are not presented in the interim reports. In the final report, it will be necessary to sharpen the focus on these and other contrasts. In view of the weight given to the farmers earlier opinions, it would be worthwhile recording their latest impressions of how much the quality of management, pasture and winter diet have changed and contributed to the upturn in animal performance. Regardless of any changes after the baseline survey, there is a strong possibility that the health of some stock on Ryan's farm would have been adversely affected by combined copper and selenium deficiencies. However, there was no indication of such deficiencies on Somers' farm. If the ill-health syndromes on the two farms from the mid 80's were clinically similar and causally related, copper and selenium deficiencies are neither the common cause nor contributory factor.

2.3 Culling. The second interim report (Appendix A, p. 24; EPA, 1997) rightly draws attention to the need to cull aged and sick animals. Both indigenous and imported stock were culled in a wholly sensible and normal way between 1994-6. Again it will be necessary to make comparisons with the previous culling policy. There is often a reluctance to cull sick animals when owners suspect that herd illnesses have been caused by external factors because culling removes "evidence" for their claims. From the data for Somers farm given in Table 1.1 (EPA, 1995) it would appear that no adult cows left the herd unless they died and animal wastage was low (8.3% for 1986-94) by normal culling standards for the area (16.4%; p.22, Rogers & Poole, 1984). A more rigorous culling policy with early replacement by good-yielding cows would probably have contributed to yield and condition score improvements, irrespective of any adverse effect of pollution on past performance. However, if chronic pollution has occurred, increased culling would tend to remove the oldest and most affected stock.

2.4 Local geochemistry. The US toxicologists pointed to the possibility of interactions of pollutants or other factors with anomalies in local geochemistry. The one factor which does manifest itself in abnormal blood or tissue composition is fluoride. The values for bone fluorine given on page A1-5 of EPA (1995) were dismissed as being well below the level used to diagnose fluorosis. Furthermore, absence of characteristic dental lesions appeared to confirm that fluorosis was not present. However, dental lesions are only seen if cattle are exposed to fluoride prior to tooth eruption (see Table 3); only replacements reared by Ryan or Somers would have been affected and dental lesions would not be a feature of fluorosis in bought-in adult stock. There is a highly significant correlation of bone fluorine with age (Figure 2) indicating chronic fluorine exposure. Furthermore, the levels recorded in the herbages, hays and silages on both the Somers and Ryan farms in 1995 are often into double figures (Table 5.1 B2-53; Table 5.3 B2-54) and consistently higher than values recorded in Area C in the baseline survey (maximum 2.4 mg/kg; Appendix 9, p. 84; Fleming & Parle, 1982). The increases in forage fluorine to mild fluorosis levels (Table 3) may reflect soil contamination rather than environmental pollution effects. The bone fluorine levels regarded as diagnostic are derived from experimental studies with continuously exposed but otherwise well-nourished dairy cows. It is known that intermittent

exposure is more harmful than continuous exposure and that undernutrition increases the danger (Shupe, 1980). Comparisons with the literature are complicated by the sampling of tail vertebrae and expression of results on an ash basis. Assuming that metabolically active bones (e.g. mandible and rib) contain twice as much fluorine as tail vertebrae and ash constitutes 50% of bone dry weight, the results are equivalent to rib fluorine/kg DM and as such indicative of mild exposure in the oldest stock (Table 3: Shupe, 1980). Seasonal (winter) peaks in exposure via soil-contaminated silage in undernourished cows may be worth monitoring. It is recommended that seasonal fluctuations in plasma fluorine be measured and the importance of minimising soil contamination during silage harvesting be stressed amongst local farmers. Milk fluoride concentrations, the method chosen for monitoring fluorine exposure in the live animal, are less sensitive to fluctuations in fluorine intake than plasma (Table 3: Shupe, 1980).

3. Quality of early advice

The EPA have rightly emphasised the need to introduce expert surveillance at the earliest possible stage of such sensitive issues as the impact of industrial pollution on animal and human health. From the earliest stage it is important that the facts are precisely recorded. Simple mistakes in describing prevailing wind directions (SW not W) and locations (Auginish Alumina is NW not W of Ryans farm) can unnecessarily feed suspicions. With so many possible influential factors, it is important to get advice from specialists as well as generalists and to assess by international, not local standards. Two examples are given below

3.1 False warnings of low selenium status. The baseline survey of Rogers & Poole (1984) highlighted a high incidence of herbage and blood selenium (or GSH Px activity) levels which were deemed to be "low". Since sulphur was a known potential selenium antagonist, fears of SO_2 emissions inducing selenium deficiencies were aroused. The standards used by Rogers & Poole (1984) are uniquely high and based on national relativities rather than associations with clinical disease or production responses. Recent studies in Australia and New Zealand (e.g. Langlands et al, 1989; Wichtel et al, 1997) confirm that grazing animals of adequate vitamin E status tolerate blood and herbage selenium values of 30 µg/l and 0.05 mg/kg DM, respectively, whereas Rogers & Poole (1984) considered corresponding values of <70 µg/l and <0.2 mg/kg DM to be low. Ireland contains many seleniferous regions and the chosen limits were based on national experience which is atypical.

3.2 Imprecise advice on risks of copper deficiency. By contrast with selenium, risks of sulphur and molybdenum-induced copper deficiency in Co. Limerick are very real due to outcrops of molybdeniferous shale and above average herbage sulphur concentrations. Both sulphur and molybdenum have continuously variable influences on copper availability which were known before the baseline survey was conducted (Figure 1: Suttle, 1978). To suggest that molybdenum was unlikely to cause problems below an arbitrary level of 4 mg Mo/kg DM (Fleming & Poole, 1982; p. 42) was to underestimate the likelihood of pre-existing copper problems. Fortunately, Rogers & Poole (1984) used a better index of risk (Cu:Mo ratio in herbage) and concluded that 80% of farms in the area were vulnerable to Mo-induced copper deficiency. Had copper deficiency surfaced as a biochemical feature of livestock in the 1994-6 survey, unnecessary confusion could have been caused.

Conclusions

1. The levels of SO_2 in the atmosphere in Co. Limerick in general and around Askeaton in particular show minimal evidence of pollution and are unlikely to harm man or his livestock.

2. The amounts of sulphur (S) deposited from the atmosphere from combined industrial and marine sources are unlikely to raise herbage S or lower herbage Se to extents which have either physiologically or clinically detectable effects in livestock.

3. There is no evidence that industrial emissions in Co. Limerick have adversely affected the health of livestock of the chief complainants, Ryans and Somers.

4. Managerial factors probably contributed to the poor performance and health on Ryan's and Somers' farms: foremost and common to both was a reluctance to cull cows which under-performed for commonly encountered reasons (mastitis; lameness). Introduction of new management strategies to both farms, including more rigorous culling, was associated with an abrupt improvement in performance.

5. Anomalies in the natural geochemical environment may have contributed to past poor performance on Ryan's farm where there is long-standing proneness to combined copper and selenium deficiencies

6. The possibility that seasonal, subclinical fluorosis, due to local geochemical or environmental features, adversely affects stock in Co. Limerick should be investigated.

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Table 1. Responses in sulphur (S) concentrations in forages to applications of S in fertilizers.

S Application (kg/ha)	Source	Forage type	Forage S (g/kg DM) Untreated	Treated		Reference
100	$CaSO_4$	Pasture	1.5	4.2		Murphy, 1975
138	$(NH_4)_2SO_4$	Sorghum silage	1.0	1.3		Ahmad et al, 1995
67	$(NH_4)_2SO_4$	Maize silage	2.9	4.3	Dressing Single	Buttrey et al, 1986
				4.3	Split	
132	$CaSO_4$	Tall fescue	3.3	4.0		Spears et al, 1985
		Orchard grass (both hay)	2.9	3.7		

Table 2. Mean values for the copper and selenium status of "indigenous" cattle on two "problem farms" in the baseline survey (1979-81) and during recent (1994-6) surveillance.

Farmer		1979-81*	1994-6[#]
		(μmol/l)	
Ryan	Cu	5.6	12.0
	Se	0.25	1.29
Somers	Cu	8.9	12.1
	Se	2.03	2.88

* Data from Rogers & Poole (1984) (Table 8, farms 14 (Somers) and 15 (Ryan); values converted to "new units").

Data from Tables 2.5-2 and 2.5-4, EPA (1997).

Table 3. A guide to the diagnosis of fluorosis in dairy cattle based on chronic experimental exposure studies and field cases (after Shupe, 1980).

Fluorosis State	Age	Normal	Marginal	Mild	Moderate	Severe
F in diet (mg/kg DM)		>15	15-30	30-40	40-60	60-109
Incisor Score		0-1	0-2	2-3	3-4	4-5
F in milk (mg/l)		<0.12	<0.12	0.12-0.15	0.15-0.25	>0.25
F in blood (mg/l)		<0.30	<0.30	0.3-0.4	0.4-0.5	0.5-0.6
F in bone*	2	400-700	710-1600	1600-2100	2100-3000	3000-4200
(mg/kg fat free	4	700-1100	1140-2400	2400-3100	3100-4500	4500-6600
DM)	6	650-1220	1220-2800	2800-3800	3800-5600	5600-8700
F in urine	2	2.3-3.8	3.8-8.0	8.0-10.5	10.5-14.7	14.7-19.9
(mg/l)	4	3.5-5.3	5.3-10.3	10.3-13.3	13.3-18.5	18.5-25.6
	6	3.5-6.0	6.0-11.3	11.3-14.8	14.8-21.0	21.0-30.1
	2	0-1	0-1	0-1	0-1	0-3
Molar Score	4	0-1	0-1	0-1	1-2	1-4
	6	0-1	0-1	0-1	1-3	1-5
Periosteal	2	0	0-1	0-1	0-2	0-3
Hyperostosis	4	0	0-1	0-1	0-3	0-4
Score	6	0	0-1	0-2	0-4	0-5

* rib: for tail veterbrae or metacarpal and metstarsal, divide by 2.0; to obtain values/g bone ash, multiply by 2.0.

FIGURE 1a Effects of sulphur concentration g/kg DM) in silage on the availability of copper (A_{Cu}·%) from that source to sheep, predicted from the equation $A = 10.6 - 6.65 \log_e S$ (r = 0.81: 7 d.f.)
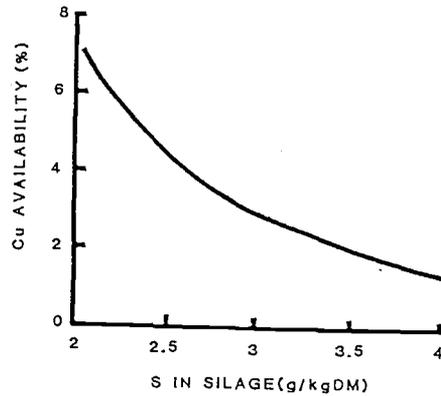

FIGURE 1b Effects of molybdenum (mg/kg DM) and sulphur (g/kg DM) concentration in hay on the availability of copper (A_{Cu}·%) from that source to sheep predicted from the equation $A = 8.9 - 0.70 \log_e Mo - 2.61 \log_e S$ (r = 0.88: 6 d.f.)
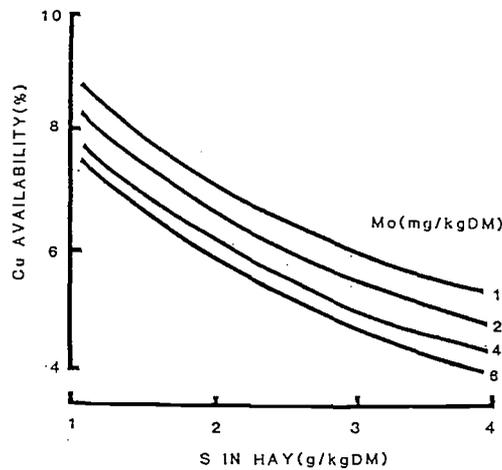

FIGURE 1c Effects of molybdenum and sulphur concentrations on the absorption of copper from summer herbage by sheep as predicted by the equation

$$A_{Cu} (\%) = 5.72 - 1.297S - 2.785 \log_e Mo + 0.227 Mo \times S$$

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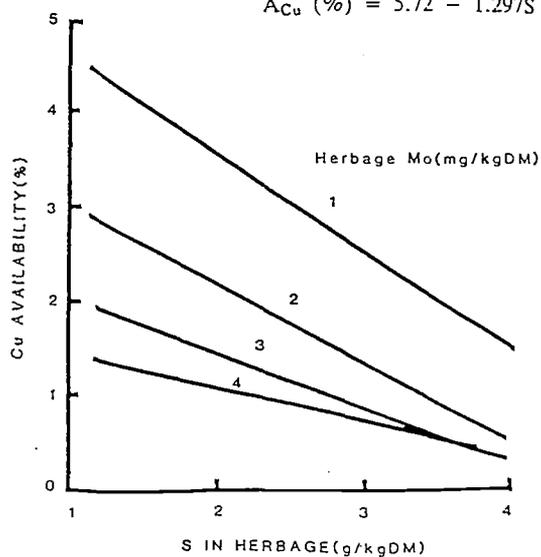
Figure 2.Bone fluoride increases with age at post-mortem
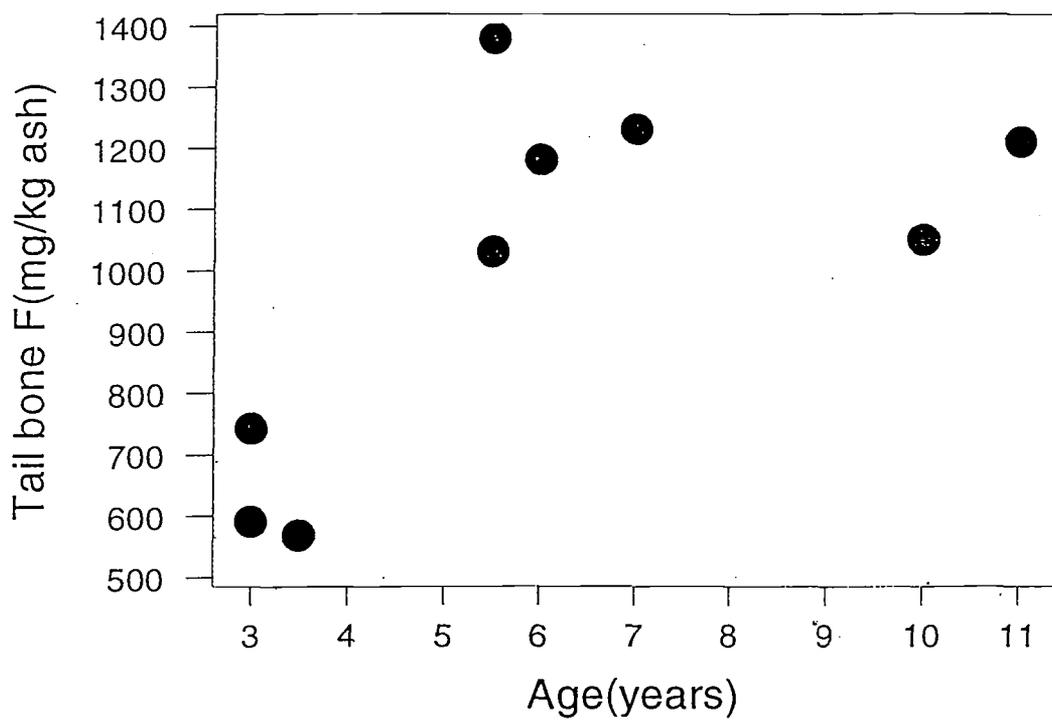
NOTES

An Ghníomhaireacht um Chaomhnú Comhshaoil

Bunú

Achtaíodh an tAcht fán nGníomhaireacht um Chaomhnú Comhshaoil ar an 23ú lá d'Aibreán, 1992 agus faoin reachtaíocht seo bunaíodh an Ghníomhaireacht go hoifigiúil ar an 26ú lá d'Iúil, 1993.

Cúraimí

Tá réimse leathan de dhualgais reachtúla ar an nGníomhaireacht agus de chumhachtaí reachtúla aici faoin Acht. Tá na nithe seo a leanas san áireamh i bpríomhfhreagrachtaí na Gníomhaireachta:

- ceadúnú agus rialáil próiseas mór/ilchasta tionsclaíoch agus próiseas eile a d'fhéadfadh a bheith an-truaillitheach, ar bhonn rialú comhtháite ar thruailliú (Integrated Pollution Control-IPC) agus cur chun feidhme na dteicneolaíochtaí is fearr atá ar fáil chun na críche sin;
- faireachán a dhéanamh ar cháiliocht comhshaoil, lena n-áirítear bunachair sonraí a chur ar bun a mbeidh rochtain ag an bpobal orthu, agus foilsiú tuarascálacha treimhsiúla ar staid an chomhshaoil;
- comhairle a chur ar údaráis phoiblí maidir le feidhmeanna comhshaoil agus cuidiú le húdaráis áitiúla a bhfeidhmeannas caomhnaithe a chomhlíonadh;
- cleachtais atá fónta ó thaobh an chomhshaoil de a chur chun cinn, mar shampla, trí úsáid iniúchtaí comhshaoil a spreagadh, cuspóirí cáilíochta comhshaoil a leagan síos agus cóid chleachtais a eisiúint maidir le nithe a théann i bhfeidhm ar an gcomhshaol;
- taighde comhshaoil a chur chun cinn agus a chomhordú;
- gach gníomhaíocht thábhachtach diúscartha agus aisghabhála dramhaíola, lena n-áirítear líontaí talún, a cheadúnú agus a rialáil agus plean náisiúnta bainistíochta um dhramháil ghuaiseach, a bheidh le cur i ngníomh ag comhlachtaí eile, a ullmhú agus a thabhairt cothrom le dáta go tréimhsiúil;
- córas a fheidhmiú a chuirfidh ar ár gcumas astúcháin COS (Comhdhúiligh Orgánacha Sho-ghalaithe) a rialú de bharr cáinníochtaí suntasacha peitril a bheith á stóráil i dteirminéil;
- na rialúcháin OMG (Orgánaigh a Mionathraíodh go Géiniteach) a fheidhmiú agus a ghníomhú maidir le húseaid shrianta a leithéad seo d'orgánaigh agus iad a scaoileadh d'aon turas isteach sa timpeallacht;

- clár hidriméadach náisiúnta a ullmhú agus a chur i ngníomh chun faisnéis maidir le leibhéil, toirteanna agus sruthanna uisce in aibhneacha, i lochanna agus i screamhuiscí a bhailiú, a anailisiú agus a fhoilsiú; agus
- maoirseacht i gcoitinne a dhéanamh ar chomhlíonadh a bhfeidhmeanna reachtúla caomhnaithe comhshaoil ag údarás áitiúla.

Stádas

Is eagras poiblí neamhspleách í an Ghníomhaireacht. Is í an Roinn Comhshaoil agus Rialtais Áitiúil an coimirceoir rialtais atá aici. Cinntítear a neamhspleáchas trí na modhanna a úsaidtear chun an tArd-Stiúrthóir agus na Stiúrthóirí a roghnú, agus tríd an tsaoirse a dhearbhaíonn an reachtaíocht di gníomhú ar a conlán féin. Tá freagracht dhíreach faoin reachtaíocht aici as réimse leathan feidhmeannas agus cuireann sé seo taca breise lena neamhspleáchas. Faoin reachtaíocht, is coir é iarracht a dhéanamh dul i gcion go míchuí ar an nGníomhaireacht nó ar aon duine atá ag gníomhú thar a ceann.

Eagrú

Tá ceanncheathrú na Gníomhaireachta lonnaithe i Loch Garman agus tá cúig fhoireann chigireachta aici, atá lonnaithe i mBaile Átha Cliath, Corcaigh, Cill Chainnigh, Caisleán an Bharraigh agus Muineachán.

Bainistíocht

Riarann Bord Feidhmiúcháin lánaimseartha an Ghníomhaireacht. Tá Ard-Stiúrthóir agus ceathrar Stiúrthóirí ar an mBord. Ceapann an Rialtas an Bord Feidhmi úcháin de réir mionrialacha atá leagtha síos san Acht.

Coiste Comhairleach

Tugann Coiste Comhairleach ar a bhfuil dáréag ball cunamh don Ghníomhaireacht. Ceapann an tAire Comhshaoil agus Rialtais Áitiúil na baill agus roghnaítear iad, den chuid is mó, ó dhaoine a ainmníonn eagraíochtaí a bhfuil suim acu i gcúrsaí comhshaoil nó forbartha. Tá réimse fairsing feidhmeannas comhairleach ag an gCoiste faoin Acht, i leith na Gníomhaireachta agus i leith an Aire araon.