

Investigation of Animal Health Problems on a Farm in Castlecomer,
Co. Kilkenny

Veterinary Laboratory Service
Department of Agriculture and Food

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Executive Summary

This is a Report on the investigations to date by the Veterinary Laboratory Service of the Department of Agriculture and Food (DAF) into animal health problems on a farm near Castlecomer, Co. Kilkenny. The farm in question is a mixed dairy/dry-stock enterprise with about 200 cattle on about 79 hectares. The farm is reported to have had a history of problems with high calf mortality rates, ill-thrift and growth retardation (stunting) in calves and growing animals, and poor milk production in cows. Calf mortality is reported to have been particularly high in the years 1998 to 2001 (approximately 70 calves reported to have died).

Kilkenny RVL first visited the farm in January 2003 following a request by the herdowner's veterinary practitioner. As the farm is located near an industrial source of atmospheric emissions – a brick factory – a Protocol (*Protocol for the Investigative Approach to Serious Animal and Human Health Problems*; EPA, 1997) was also activated in early 2004 to allow for a coordinated investigation involving DAF, the EPA, Teagasc and the South Eastern Health Board (Health Service Executive from January 2005). This Report, which is presented in two Parts, comprises the results of the DAF Veterinary Laboratory Service investigations to date. Part 1 of the Report covers the possible involvement of fluoride toxicity; Part 2 covers the general animal health investigations.

As fluorides are known components of brick-firing emissions, and are known to have caused environmental problems in other countries, the possibility that fluoride toxicity might be contributing to animal health problems on this farm has been the focus of the environmental component of the investigations to date. Characterisation of the emissions from the factory conducted by the EPA have not indicated any other pollutants of significance which might have contributed to animal health problems on the farm.

The Veterinary Laboratory Service investigations into fluoride toxicity comprised extensive clinical, pathological and analytical examinations on animals, carcasses and biological samples from animals on the affected farm. No evidence of fluorosis was found. Animals on the farm did not exhibit clinical signs of fluorosis – no lesions of fluorosis were found on pathological examinations of carcasses of animals from the farm. Fluoride concentrations in biological samples from animals on the farm were within acceptable limits. These findings are consistent with the environmental monitoring and analysis results to date which do not provide any evidence to suggest that animals on the farm have been exposed to concentrations of fluorides likely to lead to toxicity.

Investigations into wider animal health issues identified a number of significant disease conditions on the farm which are likely to have contributed to poor animal performance. The most important of these comprised infectious pneumonias, salmonellosis, mastitis, and bovine virus diarrhoea infection. Several cases of pneumonia identified in weanlings on post-mortem examination in 2005 were of a chronic nature and are likely to have originated in the calf-house. Mixing of age-groups in the calf-rearing house, as well as not isolating sick or ill-thrifty animals, were identified as factors likely to have contributed to the extent and persistence of the infectious conditions in young animals. Significant factors identified as contributing to the poor milk production were the high age-profile of the cows and mastitis.

Animal performance was generally poor in feed trials carried out on the farm in conjunction with Teagasc in the winters of 2003/2004 and 2004/2005. The latter trial included age-matched groups on a Control farm. According to the Teagasc report on the 2004/2005 trial, the poor performance of animals on the affected farm could not be explained by feed intake, management and housing.

Animal health and production on the farm have shown a definite improvement in the first five months of 2006. Calf health has been good – and this has been reflected in improved growth rates. Only two calf deaths have been recorded so far this year (mid-May) – one a stillbirth and the other a peri-natal mis-adventure. Milk production and quality are also reported to have improved. In this context, it is important that changes in disease management and bio-security procedures introduced on the farm continue to be fully implemented.

In conclusion, the examinations carried out in this investigation did not find any clinical, pathological, or analytical evidence of fluoride toxicity. These findings are also consistent with the results of environmental investigations to date which do not provide any evidence to suggest that animals on this farm have been exposed to concentrations of fluorides *via* air, water, or feed likely to lead to toxicity. On the other hand, the incidence and severity of a number of common disease conditions identified would undoubtedly have impacted negatively on animal performance.

Veterinary Laboratory Service investigations are continuing into the animal health problems on this farm. A multi-disciplinary group of scientists is also pursuing wider ecological and epidemiological investigations of the affected farm and surrounding area.

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Background and General Introduction

This is a report on the investigations carried out to date by the Veterinary Laboratory Services of the Department of Agriculture and Food into animal health and production problems on the farm of Mr Dan Brennan, Castlecomer, Co. Kilkenny. The Report is presented in two Parts: Part 1 covers the fluoride investigations; Part 2 covers the general animal health investigations.

The farm is reported to have experienced severe animal health problems since the early 1990s. Animal health and production is reported to have deteriorated further since 1997. The farm was first visited by staff of Kilkenny Regional Veterinary Laboratory (RVL) in January 2003. The main historical and on-going problems reported by the herdowner and his veterinary practitioner at the commencement of the Laboratory Service investigations in 2003 were:

Growth retardation characterised by animals that are small for their age – though normally proportioned. Calves are said to be of normal weight at birth but fail to thrive from about two months. Home-bred and bought-in calves are similarly affected. All of the calves (dairy and suckler) born in 2001 were reported to have been stunted.

General ill-thrift in growing animals. Although animals perform well in their first grazing season, once housed again, they become ill-thrifty and some lose weight. While performance improves when turned out the following spring, the net effect is that animals take a year longer to reach slaughter weight. Intermittent weight loss is also reported in cows.

Poor milk production. The herdowner reported low milk yields and poor butterfat and protein concentrations.

The Veterinary Laboratory Service (VLS) of the Department of Agriculture and Food (DAF) has been responsible for the investigations of animal health. Teagasc commenced investigations into animal nutrition, general management and husbandry on the farm in February 2003. It has also provided direct advice to the herdowner on farm management and production. It carried out an examination of farm housing, and assisted with the design and implementation of the two Feeding Trials (winters 2003/2004 and 2004/2005).

In line with the *Protocol For The Investigative Approach To Serious Animal/Human Health Problems*, and because the locality is subject to atmospheric emissions from a local industry (a brick factory), an inter-agency group was convened in early 2004 comprising representatives of DAF, the EPA, Teagasc, and the Regional Health Board.

The EPA has statutory responsibility for environmental monitoring associated with operation of the nearby brick factory. It also carried out environmental fluoride monitoring and deposition modelling in support of the overall investigation. As the brick factory is a known source of fluoride emissions, the inter-agency environmental investigations to date have concentrated on fluoride.

Besides fluoride analyses, the EPA also commissioned gaseous and particulate analyses for SO_x, NO_x, chlorides, heavy metals, particulate matter, VOCs, PCBs, PAHs and dioxins. These were found to be below the designated emission threshold - or below the limit of detection of the method of analysis - and thus further investigations with regard to animal health were not deemed necessary. Insignificant traces of aluminium, boron, copper, mercury, molybdenum, selenium, silicon, titanium and zinc were detected.

Staff from UCD Veterinary School (accompanied by personnel from the DAF-funded Centre for Veterinary Epidemiology and Risk Analysis based in UCD) first visited the farm in April 2004 at the request of Kilkenny RVL. A report on epidemiological aspects of the case was issued in March 2005.

The VLS investigations to date have concentrated on the Brennan farm. Two neighbouring farms were visited by Laboratory staff at Mr Brennan's suggestion in 2003 and 2004. Deaths due to pneumonias were reported to have occurred on one farm. *Salmonella dublin* was identified on laboratory post-mortem examination of a calf with lesions of pneumonia from the second. Recommendations regarding disease control measures and management were given to both herdowners. There have been few submissions to the laboratory since that time from either farm.

Description of the Farm

The Brennan farm is located on a north-west facing hillside about one kilometre from Castlecomer Co. Kilkenny. A brick factory adjoins one field at the north-western part of the farm (Figure 1).

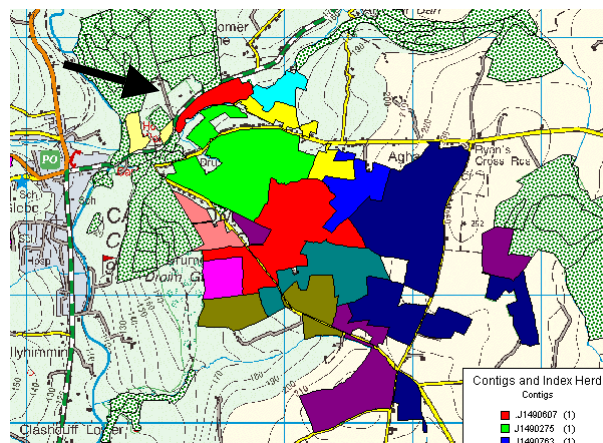


Figure 1: Farm map. The home farm is identified by the area marked in red. The factory is identified by the black arrow.

Farm size increased from about 32 hectares in the early 1990s to the current 79 hectares in the mid-1990s. Three parcels of land were farmed in 2003:

- The **Home farm** consists of 46.5 hectares. A lot of land reclamation had been done in the previous six or seven years and now there is very little waste ground. A number of fields were joined up by removal of hedges and were reseeded. The enlarged fields near the homestead are exposed and windy.
- The **'Brick Factory' farm** (rented) comprises 8 hectares adjacent to the brick factory. About five hectares have been farmed for 25 years and have been cleared of scrub. An additional adjoining three hectares has been farmed for the past three years.
- **Coolraheen:** 18.6 hectares had been rented for the past three years. This holding consists of small fields. Cattle normally thrive well there but did not perform as well as usual in 2002 due to the many wet patches in the fields in that wet year.

Part 1 - Report on Investigation into Suspect Fluorosis

Introduction

The possibility that fluoride toxicity might be contributing to the animal health problems on this farm has been considered from an early stage in the investigation. Parts of the farm adjoin a brick factory – a known source of fluoride emissions. In addition, the case history and early clinical examinations included features which might support a diagnosis of fluorosis – namely dental abnormalities and growth retardation.

Fluorine is widely distributed in nature, mostly in the form of fluorides, and originates naturally from rocks and soil, or as a pollutant from industrial sources (Cronin, 2000). The protective effects of fluoride on dental health in humans are well recognised. According to a European Food Safety Authority report (EFSA, 2005) fluoride is beneficial in the prevention of human dental caries when ingested in amounts of about 0.05 mg/kg body weight per day. Fluoride compounds were also once widely used in the prevention and treatment of human osteoporosis. While the benefits for animals have not been so clearly defined, safe limits for farm animal intake have been determined from controlled feeding trials (Shupe and Olsen, 1983).

Fluoride toxicity is also well described. It may be acute or chronic. Acute toxicity is due to excess intake of certain fluoride-containing substances over a short time-period – either by ingestion or inhalation - as industrial or household accidents in humans. Even in the presence of hospital facilities, the prognosis is poor and often results in death (Schulman, 1997; McIvor 1990). There is no evidence to suggest that acute fluoride toxicity has occurred on the Brennan farm - and it will not be considered further in this report.

Chronic toxicity is due to intermittent or continuous intake of fluorides in quantities above normal and is well described in both humans and farm animals. It may occur due to natural or industrial contamination of the environment. The former occurs in areas with rocks containing naturally high concentrations and has been reported from countries such as India (Choubisa, 1999; Dwivedi, 1997) and New Zealand (Cronin, 2000). Industrial contamination in an agricultural context is usually due to contamination of pastures in the vicinity of processes associated with high fluoride emissions. These include aluminium smelters and brickworks (Blakemore *et al*, 1948; Patra *et al*, 2000; Krook and Maylin, 1979).

The three main routes of exposure to fluoride are ingestion, inhalation, and percutaneous absorption. Only ingestion is likely to be of significance in a farm context - even in polluted environments (Weinstein and Davidson, 2004). While there are many reports of chronic toxicity due to inhalation in humans, these have been in enclosed environments subject to massive pollution, e.g. smelter plants.

For farm animals, the main source of fluoride in non-polluted environments is soil ingestion. In grazing cattle, for example, soil may account for up to 10 *per cent* of their total daily dry matter intake (Cronin 2000). Similar quantities of soil are taken in by cattle on silage. In an area subject to aerial pollution, fluoride deposited on herbage will also be an important source (Weinstein and Davidson, 2004; Krook and Maylin, 1979).

Fluoride in air exists in gaseous or particulate forms. In non-industrial areas it ranges between 0.05 and 1.9 µg/m³. Levels may be slightly higher in urban than in rural locations (EFSA, 2005). Surface water concentrations generally range from 0.01 to 0.3 mg/litre. In areas in which drinking-water for human use is fluoridated, the concentration generally ranges from 0.7 to 1.2 mg/litre. The upper safe limit for drinking water for cattle is 2.5 mg/litre. Normal

fluoride concentrations in herbages in this country range from 0.5 - 10 mg/kg dry matter; concentrations in soil range from 20 - 700 mg/kg dry matter (McGrath, 2001). These correspond with those reported internationally (Table 1).

Table 1: Internationally reported herbage fluoride concentrations (mg/kg DM¹).

Authors	Normal	High	Toxic
Parton <i>et al.</i> (2001)	10 – 20	50-100	100-300
Burns and Allcroft (1964)	2 - 25; most < 10		
Blakemore <i>et al.</i> (1948)	2 – 5		
Bunce H.W. (1985)	2 – 20		

¹ DM = dry matter.

International standards¹ permit fluoride concentrations in cattle-feed of up to 40 mg/kg dry matter² - which equates to an intake of approximately 1.0 mg/kg body weight/day for an adult cow. Dietary concentrations in excess of 100 mg/kg dry matter are toxic to cattle (Puls, 1994). This equates to a minimum toxic dose of approximately 2.5 mg/kg body weight/day for an adult cow – or a total daily intake of about 1.25 grams for a 500 kg cow. One group of workers report that permissible pollution standards may not protect cattle where these animals had previously suffered foetal exposure (Crissman *et al.*, 1980). However, clinical signs of severe classical fluorosis were evident in animals in the herd they investigated.

Air, soil and pasture fluoride concentrations in areas of known industrial contamination – and where clinical fluorosis has been confirmed - are well in excess of normal background concentrations. In China, for example, concentrations as high as 155 µg/m³ have been reported for samples of indoor air collected from homes where coal containing high amounts of fluoride was burned indoors (WHO Report, 2002).

EPA modelling of ambient air fluoride concentrations at the Brennan farm yard predicted annual mean ground level concentrations ranging from 0.031 µg/m³ to 0.051 µg/m³ for the years 1999 to 2004, inclusive. The model of ambient fluoride for the field adjacent to the factory predicted a maximum annual mean ground level concentration of 0.14 µg/m³. The model is based on continuous emission over a year-period at the maximum permitted by the licence (1 kg/hour). This model is in the process of being verified and will be reported on by the EPA.

The main clinical signs of fluorosis in cattle comprise symmetrical dental lesions consisting of mottling, pitting, staining and excessive wear of teeth, non-articular lameness³, exostoses and/or hyperostoses of long bones. Secondary signs reported as a result of inappetance and reduced feed intake include ill-thrift in all ages of cattle - and reduced milk production in cows (Suttie, 1957; Shupe and Olsen, 1987; Shupe *et al.*, 1992).

Absorbed fluoride is rapidly distributed in the circulation to the intracellular and extracellular fluids – regardless of route of intake. It is retained only in calcified tissues of bone and teeth. Ninety-nine *per cent* of the total fluoride content of the body is concentrated in calcified tissue – and the remainder is excreted in the urine (EFSA 2005; ATSDR, 2003).

The pathological basis of fluoride toxicity largely derives from interference by the fluoride ion with the normal processes of skeletal and dental mineralisation. This results in abnormal bone growth and remodelling – and defective tooth development.

¹ EC Directive 2002/32/EC. Nutrient Requirements of Dairy Cattle: Seventh Revised Edition, 2001.

² 1 mg/kg is equivalent to 1 ppm.

³ Lameness caused by damage to bone and not by damage to cartilage or joint.

Lesions in permanent teeth develop before tooth eruption - and are therefore confined to animals subject to toxic intake during the period of tooth growth, i.e. from about six months to three years of age for cattle. A congenital form of fluorosis has also been described where lesions developed before birth in deciduous teeth (Maylin, Eckerlin and Krook, 1987).

Bone lesions of fluorosis are most marked in growing animals.

The diagnosis of fluoride toxicity depends on the presence of clinical signs and pathological lesions – together with the demonstration of toxic concentrations of fluoride in animal fluids and tissues. Reported normal and toxic ranges in bovine blood, urine, and bone are given in Table 2⁴.

Table 2: Normal and Toxic Ranges for Fluorine (Puls, 1994)

	Feed (mg/kg)	Blood (ppb ¹)	Urine (mg/kg ²)	Bone (mg/kg ³ dry fat-free)
Normal	10-20	100-300	0.7 – 5.0	200-1,800
High	50-100	150-400	5 - 15	2,000-6,000
Toxic	100-300	>500	14 – 120	6,000-13,000

1 – parts per billion wet weight.

2 – equivalent to parts per million at specific gravity 1.04

3 - equivalent to parts per million dry weight fat-free.

High blood fluoride concentration is indicative of recent exposure (ATSDR 2003; EFSA 2005). Urine, on the other hand, can reflect both current and historic exposure – as it represents the quantities excreted without storage, as well as variable amounts released from storage during bone remodelling. The latter will depend both on the fluoride concentration of the bones in question, as well as the rate of remodelling underway. Urinary fluoride concentrations must, therefore, be interpreted with caution in the absence of bone analysis from the same animal (ATSDR 2003; EFSA 2005).

Bone fluoride reflects the historical body burden of fluoride of an animal - whether due to intermittent or continuous exposure, (Puls, 1994; ATSDR 2003; EFSA 2005). Fluoride retention in bone (and teeth) is proportional to the long-term exposure, i.e. it is cumulative - and is dependent on the turnover rate of bone (remodelling) - as well as animal age, sex and type of bone.

Normal fluoride concentrations in cattle bones range from about 200 to 1,800 mg/kg (fat-free; Puls, 1994). However, according to Jubb, Kennedy and Palmer (1993), no gross or microscopic lesions are seen in cattle at concentrations below about 2,500 mg/kg. Between about 2,500 and 6,000 mg/kg, microscopic changes may be seen without any gross changes. Above about 6,000 mg/kg, gross changes may be seen to a greater or lesser degree – depending on the duration of exposure and the ages of the affected animals.

A team of investigators at Cornell has also described a congenital condition of stunting in calves - which they say was due to transplacental fluoride toxicity in the offspring of fluoride-intoxicated cows. The identified source of fluoride in one case was environmental contamination from an aluminium smelter (Krook and Maylin, 1979). In a second, it was contaminated feed (Maylin *et al*, 1987). In both cases, cows on the affected farms also exhibited clinical signs of classical fluorosis, i.e. lameness and dental defects. While the congenitally-affected calves had tooth (deciduous) and bone lesions consistent with fluoride

⁴ Zipkin (1964). Note: Fluoride analyses in the present investigation were carried out on ashed bone. Ranges quoted by Puls (1994) are on fat-free bone samples and must be multiplied by a factor of about two to get an ash-equivalent value.

toxicity, bone fluoride concentrations were in the normal range when analysed post-natally. The authors speculate that this may indicate enhanced susceptibility of the foetus to fluoride toxicity due to rapid tissue production and turnover.

Suttie *et al* (1985), however, failed to demonstrate any post-natal tooth or bone lesions in calves of dams experimentally fed diets containing 40 to 50 mg of fluoride/kg dry matter of complete ration during pregnancy.

Investigation Methodology

Clinical Examinations

Animals of all ages were examined for signs of fluorosis on visits to the Brennan farm between 2003 and 2005. These comprised examinations for skeletal deformities, lameness and dental abnormalities.

Dental examinations were carried out on 29 cows in 2003, and 47 cows in 2005 (Appendix 2). In 2003, a comparative abattoir study was also carried out to determine the type and frequency of tooth abnormalities or staining in a normal population.

Deciduous teeth of all calves born in 2004 and 2005 were examined. The teeth and mandibles of all animals submitted for necropsy in 2005 were also examined.

Pathology

Post-mortem examinations were carried out at Kilkenny RVL on all farm deaths (bovine) since the start of the investigation in early 2003 (*see* Appendix 5 for details of submissions). Elective post-mortem examinations were also carried out on four animals identified by the herdowner in 2005 as representing typical examples of the growth retardation aspect of the problems. In addition to detailed general post-mortem examinations, the appendicular skeleton was closely examined for evidence of bone thickening and exostosis.

Sections of long bone were also collected for histopathology examinations from animals submitted for necropsy during 2005.

Radiology

The lower fore-limbs of two live animals were x-rayed in 2003 at UCD Veterinary Clinical Facility. Long bones from two elective post-mortem animals were examined radiographically in 2005. The mandible from one animal was examined radiographically.

Blood and urine samples were collected for fluoride analysis from live cows - as well as from a selection of animals submitted for post-mortem examination. For comparative purposes, samples were also collected for analysis from two cows and two weanlings on a neighbouring farm, and a farm about nine kilometres distant from the Brennan farm.

Fluoride Analysis

Samples of bone and teeth were collected for fluoride analysis from animals submitted from the farm for necropsy in 2003 and 2005 – and also from animals sent for abattoir slaughter from the Brennan farm in 2003. Two mandibles from elective post-mortem examinations, and a tooth from a cow with very worn incisors, were also analysed.

Urine samples were analysed for fluoride concentrations at the UK Veterinary Laboratories Agency Laboratory in Shrewsbury by selective-ion electrode analysis. Blood (serum) and bone samples (ashed) were analysed for fluoride by Direct Laboratories of Wolverhampton by the same methodology.

Environmental Investigations

Herbage and water samples from the Brennan farm were analysed for fluoride, by or on behalf of the EPA and Teagasc, at various times since early 2003. Detailed results will be reported by these agencies. Summary results for water, silage and grass samples for 2003 and 2004 are presented below.

Results

Clinical Examinations

The herdowner had reported that lameness was not a problem on the farm prior to 2003. This was confirmed by on-farm observations by laboratory staff in the period 2003 to 2005. No evidence of limb skeletal deformities, exostoses or hyperostosis was noted on live animals examined throughout the period.

Clinical radiography of the two live animals in 2003 did not demonstrate lesions suggestive of fluorosis.

Twenty nine cows of varying ages were subjected to dental examination in 2003. Eighteen had dental defects ranging from staining and pitting to severe wear. Four had teeth worn down to gum level – two of these were known to be over 10 years of age. Two other cows, aged 10 and 11, were missing teeth. Forty seven cows were examined in 2005. Eighteen of these, aged between eight and 12 years, had advanced wear of incisor teeth. Four of these had previously been recorded as having extremely worn teeth in the 2003 examinations.

No significant abnormalities were noted in the deciduous teeth of animals born in 2004 and 2005.

Results of the comparative abattoir survey of normal slaughter animals demonstrated that dental defects were similar in type and frequency to those observed on the Brennan farm. In addition, it was found that the tooth staining in the Brennan cattle could be removed by scraping with a blade. This indicates it was superficial – in contrast to the specific staining of fluorosis which is thought to be due to oxidation of the enamel matrix, and reportedly cannot be removed by scraping (Garlick, 1955; Jubb, Kennedy, and Palmer, 1993).

Pathology

General gross and microscopic pathology results are presented in Part 2 of this report.

With regard to the fluorosis investigations, no abnormalities suggestive of fluorosis were observed on gross examinations of bones and peri-articular surfaces of animals submitted for post-mortem examination throughout the period of the investigation. Periosteal surfaces of long bones were smooth and shiny and the shafts of the bones were not thickened. Neither exostoses nor hyperostosis were noted.

Erosion of joint cartilages in the large joints of the limbs (stifle, hock) were noted in one yearling which had been bought-in as a calf (Kilk/05/02909). The changes were not suggestive of fluorosis.

The results of the histopathological examinations of bone samples collected from post-mortem examinations in 2005 are presented in Appendix 3. While bones from several animals had changes consistent with their growth-retarded status, no lesions of fluorosis were seen.

Radiography

Radiographic examination of the bones from two animals submitted for elective necropsy showed normally proportioned bones - though small for their age. The physes were intact. There was no evidence of diffuse periosteal reaction on the surfaces of long bones, ribs, or mandible. No lines of interrupted growth were visible. No evidence of fluorosis was identified in either animal.

Fluoride Analysis

The results of serum fluoride analyses are summarised in Table 3. Serum concentrations were all within the normal range (Puls 1994).

Table 3: Serum Fluoride Analysis µg/L (ppb).

Number of animals	Range	Median	Mean	Std. Dev
26	29 - 144	42.5	56.5	32.19

The mean urinary fluoride concentration was within acceptable limits (Table 4). While three samples had values above the normal range (5.4, 5.6 and 8.5 mg/L), they were well below the minimum value that might suggest toxic intake (Table 2). However, it is recommended that bone samples be collected at culling for fluoride analysis from the animal with the value of 8.5 mg/L.

Table 4: Urine fluoride analysis results (mg/L).

Number of animals	Range	Median	Mean	Std. Dev
23	0.1 - 8.5	1.2	1.8	2.2

The results of bone fluoride analyses are given in Table 5. Concentrations were within the reported normal range.

Table 5: Bone (metacarpus) and teeth fluoride concentrations (mg/kg - ash)

	Number of animals	Range	Median	Mean	Std. Dev
Metacarpus	21	65 - 1380	264	534	506
mandible	7	72 - 1170	249	403	422
teeth	4	172 - 1540	267	561.5	656

Environmental Investigations

Herbage and water samples from the Brennan farm were analysed for fluoride, by or on behalf of the EPA and Teagasc, at various times since early 2003. Detailed results will be reported by these agencies. Summary results for water, silage and grass samples from the Brennan farm for 2003 and 2004 are presented in Table 6.

Table 6: Fluoride concentrations in water, silage and grass samples from Brennan farm 2003/2004.

Source	Year	Fluoride (mg/kg)
Deepwell	2003	0.17
Deepwell	2004	0.19
Quarry/stream	Various	<0.1
Silage	2003	3.8
Grass	2003	7.2

Summary results for herbage samples collected on behalf of the EPA from the Brennan farm, and two neighbouring farms, in 2005/2006 are presented in Table 7.

Table 7: Summary fluoride concentrations (mg/kg DM) in herbage samples collected by EPA¹.

Farm	Farm Mean (Range) June 05	Farm Mean (Range) Aug. 05	‘Factory’ Field² Mean (range)
Brennan farm	7.7 (1- 39) ³	6.7 (< 1 – 29) ³	25.5 (4 - 127)
neighbour 1	1.6 (< 1 – 3)	1.6 (< 1 – 3)	
neighbour 2	4.7 (1-16)	2.9 (< 1 – 9)	

1 - Results are based on samples collected from various sites on six dates between 13 and 20 June 05 and five dates between 2 and 12 Aug 05.

2 - ‘Brick Factory’ field - adjacent to factory. Sampled by EPA June, August, September, 2005 and February 2006.

3 – Includes figures for ‘Brick Factory’ field.

The ‘Brick Factory’ field showed consistently higher fluoride herbage concentrations than other locations. The mean of the samplings for June, August, September 2005 and February 2006 was 25.5 mg/kg dry matter (range 4 - 47). While a single sampling from this field by RVL staff in March 2005 gave a fluoride concentration of 127 mg/kg dry matter, this must be regarded as an outlier given the results of subsequent EPA samplings of the field. The area is subject to ongoing monitoring and results will be under continual review by the EPA.

Herbage was sampled on behalf of the brick factory at three sites adjacent to the factory (south of factory) monthly from April to August 2005. The mean fluoride concentration in the herbage was 7.2 mg/kg dry matter - with a maximum value of 48.3 mg/kg dry matter and a next-highest value of 8.1 mg/kg dry matter. Average monthly values ranged from 2.9 to 20.8 mg/kg dry matter.

Discussion

The case history and early clinical examinations on this farm included features which might support a diagnosis of fluorosis – namely dental abnormalities and growth retardation. However, in relation to the dental abnormalities – staining and pitting – these were observed to a similar degree and frequency in the abattoir study of normal animals. Garlick (1955), who studied teeth of thousands of normal animals at slaughter, also reported similar changes as being normal findings.

In relation to dental wear and missing incisors, this occurred predominantly in older cows and is consistent with normal age-related attrition. It should be noted that the age profile of this herd has been particularly high up to 2005.

Although growth retardation was undoubtedly a feature on this farm, it is debatable to what extent this might be considered indicative of fluorosis in cattle. Fluoride is known to stimulate rather than suppress bone growth – hence its widespread use until recently in the treatment of human osteoporosis (Balena *et al*, 1998). The classical lesions of fluorosis are, in fact, the result of excess – though abnormal - bone growth (Jubb *et al*, 1993).

Although fluoride toxicity directly affects bone growth and remodelling, this is usually expressed in bone deformities and exostoses rather than an uncomplicated suppression of bone growth in length. Choubisa (1999), for example, in describing widespread clinical fluorosis in cattle due to geochemical contamination of drinking water in India, specifically mentions that stunting was not observed.

Only one group of investigators has described stunting as a primary toxic effect of fluorosis in cattle – this is the case of congenital fluorosis described by the Cornell investigators

(Maylin *et al*, 1987). The authors suggest the stunting was - in part at least - due to *in-utero* effects of fluoride toxicity in calves of clinically fluorotic cows. The latter point is significant as these investigators also describe a clinical syndrome in the affected herds which is consistent with classical fluorosis, i.e. lameness, animals crawling on knees, and dental fluorosis in permanent and deciduous teeth (Maylin *et al*, 1987; Krook and Maylin, 1979). There is no evidence to suggest that this applies on the Brennan farm - where clinical, pathological and toxicological signs of fluorosis have not been observed.

While clinical fluoride toxicity can certainly have an impact on growth and production, the ill-thrift and poor milk production are generally considered to be an indirect effect due to inappetance and reduced feed intake secondary to dental defects and locomotor problems (Burns and Allcroft 1966; Patra *et al*, 2000). Although the Cornell investigators have ascribed milk production loss to a direct toxic effect of fluoride, it must be stressed that clinical fluorosis was also evident in the affected herds they report upon (Maylin and Krook, 1982; Eckerlin, 1986). Again, there is no evidence to suggest that this applies on the Brennan farm where clinical signs of fluorosis have not been identified.

The investigation for clinical and pathological evidence of fluorosis on the Brennan farm was extensive. Chemical analysis of blood, urine and bone from animals from the Brennan farm showed no evidence of fluorosis. Values were generally within normal ranges. Even the highest bone fluoride concentration of 1,620 mg/kg (ashed bone) – in an aged cow (*see* Appendix 1)– was well below the 2,500 mg/kg (fat-free) quoted in the literature as being the minimal value where microscopic, but not clinically significant, changes might be detected. Histopathological examination of bone sections did not show any changes suggestive of fluorosis.

It has been suggested locally that toxicity due to fluoride might occur following inhalation. However, while inhalation is an important route of toxicity for humans in enclosed industrial environments, it is not considered important for farm animals (Weinstein and Davidson, 2004). It is extremely unlikely that the levels of atmospheric fluoride to which the Brennan cattle might be exposed could lead to toxicity - either as a result of direct irritation of the respiratory passages, or of chronic systemic toxicity.

Experimental fluoride inhalation studies in rodents have shown that toxic gaseous concentrations lead to point-of-entry irritation lesions on the upper respiratory surfaces – i.e. the nasal and conchal mucosae and pharynx in nasal breathing rodents (ATSDR, 2003). Bovines are nasal breathers. No evidence of such lesions was identified on post-mortem examination of Brennan animals. In acute inhalation studies in rodents, generalised pulmonary oedema was also evident prior to death (ATSDR, 2003). Again, this was not a feature of the respiratory disease on the Brennan farm.

Although severe respiratory disease was identified in calves and weanlings on the Brennan farm in 2005, the cranio-ventral distribution of the lesions was consistent with the infectious agents identified - and such a distribution of lesions is not consistent with exposure to noxious gas or irritant particles (Jubb and Kennedy 1993).

It has also been suggested that fluoride acquired by inhalation might have a specific toxic inhibitory effect on the immune system – even in much lower doses than would affect the normal target organs of teeth and bone. It is suggested that such immune suppression might account for the high incidence of infectious respiratory conditions on the Brennan farm.

However, this is unlikely. In the first instance, there is no evidence in the literature to suggest that fluorides pass through an intermediate stage of metabolism whereby they might have enhanced toxicity for tissues other than teeth and bone (ATSDR 2003). So if

immunosuppression or other organ toxicity were to occur, then toxic effects in bone and teeth should also be evident (ATSDR, 2003). Secondly, a 1996 review of the literature on fluoride and white-cell function in humans (Challacombe, 1996) examined numerous studies and concluded that there is no evidence of any harmful effect on specific immunity following fluoridation of drinking water. Evidence from human *in-vitro* laboratory studies suggests that fluorides can have an enhancing, rather than inhibitory, effect on the immune system (Loftenius, 1999).

In conclusion, the examinations carried out in this investigation did not find any clinical, pathological or analytical evidence of fluoride toxicity. These findings are also consistent with the results of environmental investigations to date which do not provide any evidence to suggest that animals on the Brennan farm have been exposed to concentrations of fluorides *via* air, water or feed likely to lead to toxicity.

Part 2 - Report On Animal Disease

Introduction

This second part of the report deals with the wider investigations of animal health on the Brennan farm to date. Staff of Kilkenny Regional Veterinary Laboratory (RVL) first visited the farm in January 2003.

The farm is a mixed dairy/dry-stock enterprise. The farmer is the primary source of labour on the farm and carries out all milking, feeding, and calf rearing. The farmer also works part-time in a mart.

Animal Buildings consist of a new 'A-frame' building containing the milking parlour, cubicles, calf house and calving box, and an old lean-to off an old animal house. The lean-to has been expanded and added to with a slatted section. Another section, which has two large pens, was added recently to the A-frame building. Up to 2006, there had been no designated areas for isolation of sick and bought-in animals.

Stock numbers have increased from about 90 in the early 1990s to between about 150 and 190 in recent years. There were about 207 cattle on the farm during spring 2003 comprising:

- 38 dairy cows,
- 18 suckler cows,
- 14 cattle over 30 months
- 48 two year olds,
- 44 weanlings between 6 and 12 months
- 45 calves

The age profile for the dairy herd is high and culling has been low in recent years. In the two years 2003 and 2004, for example, where culling was under 10 *per cent*, between 50 and 60 *per cent* of the cows were over eight years old (Table 8).

Table 8: Age profile and culling¹.

Year	No. of cows ²	% over 8 yro ³	% over 10 yro ³	No. of culls ¹	Culling/year (%)
2000	48	-	-	15	31%
2001	57	-	-	2	4%
2002	55	-	-	6	11%
2003	51	57%	-	4	8%
2004	52	56%	-	5	10%
2005	57	40%	22%	12	21%

1 - Voluntary culls during each year based on the DAF CMMS database category 'FACTORY SLAUGHTER'.

2 - Number of cows at 31 Dec of each year from CMMS. As this is post-culls for each year, the culling rates are probably overestimated.

3 - Ages are calculated from Date of Birth recorded in CMMS. As these are not available for animals born prior to about April 1996 the 'over 8 yro' groups can only be calculated from 2003 onwards, and the 'over 10 yro' groups from 2005.

Replacement heifers are mainly reared on the farm - but because of difficulties in recent years in getting home-bred animals to target weight, replacements have also been bought-in. For the same reason, old cows have also been kept on in the milking herd.

A number of management changes have been made on the Brennan farm in the latter part of 2005 in response to ongoing findings of the DAF Laboratory Service investigations. These include:

- Milk recording which commenced in May 2005 to support a mastitis control program.
- Implementation of a comprehensive herd health program in conjunction with the herdowner's veterinary practitioners commenced in September 2005.
- Provision of calf-hutches for the 2006 calving season to allow calves be segregated and reared in age and health-based groups.
- General advice and literature has been provided with regard to herd health, biosecurity and disease management.

Background and History

Calves and growing stock

Newborn calves are generally left to suckle the cow for four to seven days (*See* Teagasc Nutrition Report, Appendix 8)⁵. Following this they are moved into the calf house. The calf house did not allow for separation of groups based on age or health status. Towards the end of the calf-rearing period, the house could contain a mix of age-groups from one week to four months. Later-born calves, which are not turned out to grass by August, may be housed for the entire period.

Calves are offered 5.5 litres of milk for approximately 90 days before weaning. A starter ration is offered from two weeks of age. Silage, hay and straw were used in various years as sources of roughage from about two weeks. Weanlings are fed silage and 1.8 kg/head/day of a home-mixed ration of equal parts of barley, distillers' grains and citrus pulp without added minerals. This is calculated to provide sufficient energy for 0.6 kg live weight gain per day.

Calf mortality was reported to have been high in the years 1998 to 2001. Approximately 70 calves were reported to have died in these four years – representing an overall mortality rate of about 40%. Losses were reported to have been associated mainly with diarrhoea. Five carcasses were submitted to Kilkenny Regional Veterinary Laboratory for post-mortem examination during this period – two in 1999 and three in 2000 (*see* Appendix 4). A *Pasteurella* species, a recognised respiratory pathogen, was isolated from a lung of a calf submitted in 1999. *Salmonella dublin* was identified as the cause of abortion in a foetus submitted in 2000. Rotavirus was also identified from a faecal sample and calf submitted to the laboratory in 2002.

Cows

The dairy herd comprises approximately 40 Friesian-type cows. Prior to 2003, a number of different proprietary commercial compound rations was used. From early 2003, a home-mixed ration – comprising citrus pulp, barley and distillers' grains - was fed to milking cows.

In March 2004, Teagasc observed that cow condition before calving was moderate (body condition score 2.70), and that the silage was deficient in energy for thin cows in late pregnancy. Milking cows were offered ad libitum silage and 5 kg proprietary concentrate.

Milk production is reported to have been poor since 2003. Few details are available on actual production prior to 2003. In 2003, production was reported to have peaked at 18.7 kg⁶, and

⁵ This has been reduced to 24 hours in 2006 following the introduction of calf hutches.

⁶ 1 gallon of milk = 4.68 kg.

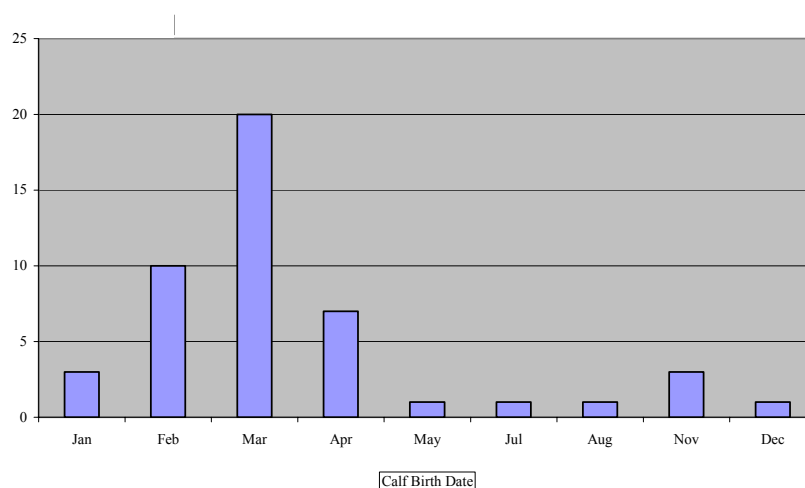
maintained 11.5 kg thereafter. It was reported to have peaked at 14 kg in 2004, falling thereafter to 9.4 kg per head per day. This was despite reportedly much better quality grass in 2004. Based on the feeding rate and genetic potential of the cows, Teagasc estimated that the cows should milk 16 - 18 kg/day.

Clinical mastitis was not reported to have been a significant problem prior to 2003. However, several well-recognised mastitis pathogens were identified from milk samples submitted to the Laboratory in 2002 – namely *Staphylococcus aureus*, *Streptococcus uberis*, and *Streptococcus dysgalactiae*.

Mastitis was also recognised as being a significant issue from an early stage of the DAF investigations. Recommendations for mastitis control were given in 2003. The bulk somatic cell count (a herd-level indicator of mastitis) continued to deteriorate until 2005 when a control program was implemented.

While fertility is not reported to have been a problem, few records are available. Bulls run freely with the cows. As a result, while the main calving season extended from January to April, births were recorded throughout the year in recent years (*see* Figure 2). Calving is likely to be more compact in 2006 due to removal of bulls for a period during 2005.

Figure 2: Births by month – 2004.



Scope and Methodology of VLS Investigation

Veterinary Laboratory Service involvement in this case commenced with a farm visit by Kilkenny RVL staff in January 2003 following referral by the herdowner's veterinary practitioner.

Investigations since then have included on-farm clinical examinations, investigations of animal health management and collection of clinical pathology samples for analysis. A comprehensive range of clinical pathology analyses was performed throughout the period.

Pathology

Post-mortem examinations were carried out at Kilkenny RVL on all farm deaths since the start of the investigation in early 2003 (*see* Appendix 5 for details). In addition, elective post-mortem examinations were carried out on four animals identified by the herdowner in 2005 as representing typical examples of the growth retardation aspect of the problems. Two animals from Feed Trial 2 (winter 2004/2005) were also necropsied.

Analysis of on-farm records

The farm drug treatment register and annual veterinary statements were examined in 2005 to assess antibiotic and other therapeutic drug usage. The purpose of this was to provide a proxy estimate of disease occurrence. While the value of this is entirely dependent on the comprehensiveness of the records, the results do allow some conclusions to be drawn regarding the scope and occurrence of certain diseases. Certain antibiotics, for example, are most likely to be used to treat clinical diseases such as pneumonias and septicaemias.

The farm animal movement register was also examined to provide annual data on on-farm deaths.

Milk recording

Milk recording commenced in May 2005. The records have been analysed to determine individual cow somatic cell count and new infection rates according to Bradley (2005).

Milk somatic cell counts (SCC) are a static measurement of udder health – the higher the count the greater the likelihood of an ongoing inflammatory process, i.e. mastitis. Counts below about 200,000 cells/ml are indicative of good udder health. Counts in excess of about 400,000 cells/ml are generally taken as indicative of mastitis.

However, SCCs do not provide an objective measurement of mastitis development and spread within the herd. To examine this, new infection rates were calculated as described by Bradley (2005) - which indicate mastitis spread and net mastitis prevalence in the dairy herd.

Toxicological Investigations

Because of its proximity to a brick factory – a known source of fluoride emissions – investigations focussed on the possibility of fluoride toxicity from an early stage. Details of the fluoride investigations have been presented in Part 1 of the present report.

Samples from animals were also analysed for potentially toxic concentrations of arsenic, lead, molybdenum and cadmium. Samples of feed were tested for mycotoxins by the Irish Equine Centre in 2003 and 2004.

Growth Measurements

Birth and Growth Weights

In order to ascertain calf birth weights, the herdowner was provided with weighing scales in 2005 to weigh newborn calves.

Calves were also weighed periodically to determine growth rates.

Feeding Trials

In order to investigate poor growth performance in yearlings, two feed trials were carried out in the winters of 2003/2004 and 2004/2005 in conjunction with Teagasc. The first of these (Feed Trial 1 – winter 2003/2004) was loosely structured and compared the growth of animals fed silage from the Brennan farm with that of animals fed silage from a neighbours farm. Groups were not age-matched.

The second trial (Feed Trial 2 - winter 2004/2005) was intended to investigate the effects of location (i.e. Brennan farm) and silage on growth rates. It comprised four groups of yearlings – two on the Brennan farm and two on a remote Control farm. An additional group on the Brennan farm comprised animals brought from the CVRL farm at Abbotstown to examine any effect on growth of brought-in animals (*see* Appendix 8, Teagasc Report on Feed Trial 2004/2005).

Results of Laboratory Investigations

Clinical Pathology

Biochemistry

Blood biochemistry results were generally within normal ranges in samples collected between January 2003 and the end of 2005. No clear mineral deficiencies were identified. Although some calves had marginal blood copper concentrations at the end of summer 2004, values were generally adequate and there was no evidence of significant copper deficiency.

Magnesium concentrations were also generally within normal ranges – low values at times being consistent with seasonal nutritional and production influences. Periodic low blood urea, and raised beta-hydroxybutyrate concentrations, were observed in old cows - two of which were heavily pregnant and recumbent at the time of sampling.

While analysis of samples collected on a number of occasions indicates the farm to be of moderate to marginal selenium status, it is unlikely that this is of particular significance in relation to the main animal health problems reported.

Raised activities of the serum enzyme γ GT in three heifers and two cows in summer 2004 may have been an indication of fluke damage to the liver. Raised activities of serum GLDH in three cows in spring 2005, and another three in spring 2006, may have had similar causation.

Mineral concentrations in tissue samples collected from animals at post-mortem examinations were also generally within normal ranges. Bone calcium, magnesium and phosphorus concentrations were normal. Except for one case of cobalt toxicity post-dosing with cobalt, liver cobalt and copper concentrations were in the normal range. Although five of nine tissue samples analysed for selenium were low, it is unlikely this was of clinical significance.

Blood immunoglobulin concentrations were below the recommended minimum in about a half of 32 calves sampled up to and including spring 2005. The low values indicate inadequate colostrum intake immediately post-calving, and, if representative of the entire annual calf crop, such a reduced immune status could have a significant negative impact on neonatal calf health.

A herd sampling for biochemistry analysis was carried out in March 2006. The findings indicate that while there were no specific deficiencies that would account for the clinical syndromes involved in this farm, values of certain biochemical parameters did indicate a requirement for supplementation in the diet.

Twenty-seven of the 186 animals sampled had low to marginal serum magnesium. Ten of these were cows, the rest were predominantly yearlings. Thirty five *per cent* of 182 cattle (cows and followers) analysed for serum copper had low concentrations. Fifteen animals were tested for whole blood selenium - these comprised cows, yearlings and calves. All but two animals had very low concentrations. Marginal to low thyroxine was identified in seven of 17 cows - all others were generally within normal ranges. Fifteen sera were low in urea. One was from a calf, two from cows and twelve were from yearlings.

Haematology

Samples from over 80 animals were subjected to haematology examinations between January 2003 and December 2005. While the majority of results are within normal ranges, there was a pattern of mild anaemia in some young animals and cows. In the young animals, this was generally consistent with the poor condition and ill thrift of the animals concerned. Samples from four weanlings in January 2003, for example, had mild anaemia consistent with poor

animal condition. There was also a mild to moderate neutrophilia in samples from three animals – suggestive of an inflammatory condition (possibly subclinical). Mild anaemia was seen in samples from some cows on grass in late lactation. This is described in the literature as a production-related finding. While it is probably nutritional in origin, the specific component(s) involved cannot be determined.

Mild anaemia in some heifers in the spring of 2005 - and cows in July 2005 - could also be due to fluke infestation. Fluke eggs were detected in faecal samples from two of three cows sampled in July 2005.

Raised white cell counts in some young animals at times were consistent with intercurrent infectious conditions.

Eosinophilia in samples from a group of cows in September 2005 is consistent with the diagnosis of re-infection hoose made at the time.

High white cell counts, with neutrophilia (i.e. suggestive of bacterial infection), were recorded in a group of two to four-week old calves sampled in April 2006. Some of these calves were in a multi-hutch to which an ill-thrifty calf had previously been added.

Salmonella dublin was isolated from the faeces of one of the calves. *Manheimia hemolytica* was also isolated from a nasal smear of this calf and another in-contact calf. *Pasteurella multocida* was isolated from a nasal smear of a third calf.

Microbiology

Faecal samples from calves were positive for cryptosporidia and rotavirus – known causative agents of calf diarrhoea – in the springs of 2004 and 2005. This is not an uncommon finding in calf-rearing establishments.

Of greater significance was the isolation of *Salmonella dublin* on a number of occasions from calves and young stock. It was isolated from faecal samples from a five day old calf in March 2004 and from a weanling in November 2004. Both had clinical signs of disease. It was isolated again in spring 2005 from a young calf with enteritis.

In a screen of eleven weanlings in November 2004, and 20 weanlings in Autumn 2005, *Salmonella dublin* was isolated from one animal on each occasion. Faecal cultures of nine cows sampled in January 2005 demonstrated *Salmonella dublin* in one cow. *Salmonella dublin* was also isolated from a faecal sample submitted in November 2004 from a recumbent cow.

Significant antibody titres to *Salmonella dublin* were also demonstrated in blood samples from two weanlings in January 2003 - and a variety of animals in 2004. All of these results indicate *Salmonella dublin* infection has been indigenous to the herd for some time.

Causative agents of mastitis – *Staphylococcus aureus*, *Streptococcus uberis* and *Streptococcus dysgalactiae* – were isolated from milk samples collected in the years 2003 to 2005.

Parasitology

Almost a hundred faecal samples from cattle on the farm were subjected to parasitological examination in the years 2003 to 2005. Occasional light coccidial infections were detected in calves. This is not a significant finding. Strongyle egg counts were generally low or negligible – however moderate to high counts were detected in three of a group of animals sampled pre-treatment in December 2004.

Evidence of fluke infection was detected in faecal samples from cows in March and July 2004 – and again in January 2005.

Faecal samples from calves in the Autumn of 2004 and 2005 were negative for lungworms (*Dictyocaulus viviparus*). However, investigation of an outbreak of coughing in cows in September 2005 showed some to be antibody-positive for *Dictyocaulus viviparus* (hoose).

Mange (*Chorioptes* and *Sarcoptes* species) was identified from two yearlings in 2005 – one of which was an Abbotstown-origin animal.

Virology

Antibody and virus tests were carried out on a range of blood and tissue samples in 2004 and 2005. While antibody results indicate herd exposure to the viruses of IBR, PI3 and RSV, only a small number of samples had results consistent with the presence of active viral infection at the time.

Because of the history of growth retardation in calves, tests for Bovine Virus Diarrhoea (BVD) virus were carried out on a number of occasions between 2003 and spring 2006. Samples from young stock collected in January 2003 were antibody and virus negative – indicating the animals sampled had not been infected prior to that time. However, some cows - and a single older stunted yearling - sampled at the same time, were antibody-positive. BVD virus was subsequently isolated from a two-week old calf with a congenital brain deformity submitted for post-mortem examination on 4 March 2004. Seventeen yearlings and eight cows sampled on 11 March 2004 were all antibody-positive.

BVD virus was also isolated from four of 58 yearlings sampled in the Autumn of 2004 in advance of the winter 2004/2005 Feed Trial (Nos. 308, 333, 339, 342). These animals were not used in the trial. Two of these, 308 and 333, were again virus-positive when tested in January 2005, i.e. they were persistently viraemic⁷.

Although all 10 of the Abbotstown animals on the Feed Trial were BVD-virus and antibody negative prior to departure from Abbotstown on 16 December 2004, and all were vaccinated against BVD on 8 December, one (703) was found to be virus-positive when sampled on 23 December 2005. The most likely source of infection was animals on the Brennan farm – as the Abbotstown farm was considered to be BVD-free on the basis of periodic testing.

A herd-test for the virus in March 2006 identified two new viraemic animals.

Pathology

A list of all carcass submissions for the years 2003 to 2005 is presented in Appendix 5. Eight bovine carcasses – ranging in age from aborted fetuses to cows - were submitted in 2003. The most significant findings from a herd health point of view were the isolation of *Salmonella dublin* from a yearling with haemorrhagic enteritis and the presence of hepatobiliary fibrosis in two cows. The latter is indicative of liver damage from fluke infestation. There were no specific findings on four adult heifer carcasses examined following routine abattoir slaughter.

Submissions in 2004 comprised three aborted fetuses and seven calves – the latter ranging in age from one week to three months. *Salmonella dublin* was isolated from two calves. This is a significant finding in the light of its isolation in 2003 and indicates it was probably endemic to the farm during this time. Four other calves also had lesions of systemic bacterial infections – in the two youngest these were associated with low colostral immunity. BVD

⁷ Herdowner was advised that persistently viraemic animals should not be sold on the open market for husbandry purposes. If retained on the farm that they should be isolated.

virus infection was identified as the cause of a congenital deformity in one calf (*see* above and Appendix 5).

Post-mortem submissions in 2005 were divided between non-elective and elective. The latter comprised four animals specifically selected for post-mortem examination on the grounds that they were identified by the herdowner as being representative of the ill-thrift and growth retardation problems on the farm. Two Abbotstown animals were also necropsied as they were in the feed trial and suffered weight loss (Appendix 5). A summary of histopathology findings for post-mortem examinations performed in 2005 is presented in Appendix 6.

Non-elective submissions for post-mortem examination in 2005 were substantially higher than in the two previous years. They comprised an aborted foetus, five calves up to a month old, four weanlings between three and nine months, and three cows. A further three aged cows were also examined at abattoir slaughter.

Lesions indicative of a protozoal cause of abortion – probably *Neospora* – were identified in the foetus. Three of the five calves had systemic bacterial infections associated with low colostral immunity. *Salmonella dublin* and *Mannheimia hemolytica* were isolated from a four-week old calf with ill-thrift; pneumonia was also identified on post-mortem. A two-month old calf had extensive bacterial pneumonia. One of these animals also had a fungal gastritis consistent with prolonged antibiotic usage (Kilk/05/01386). Five weanlings between four and six months old had varying degrees of bacterial-type pneumonias. The mixed bacterial isolates from lungs included *Mannheimia hemolytica*, *Arcanobacter pyogenes*, and *Haemophilus somnus*. *Salmonella dublin* was also isolated from one of these.

Two cows submitted for laboratory post-mortem examination had lesions indicative of fluke infestation and gastro-intestinal parasitism. One of these also had lesions of septic mastitis and a large inguinal abscess. Post-mortem changes were advanced in a cow suspected of having died of hypomagnesaemic tetany. Three cull cows examined at factory slaughter had liver lesions indicative of fluke infestation.

Four clinically normal - but ill-thrift or undersized - live cattle between four and 13 months of age were submitted for elective post-mortem examination. Three had lesions consistent with chronic gastro-enteritis (*Salmonella dublin* was identified in one of these animals). The two older animals had lesions of chronic bronchopneumonia. One of these also had erosions of the articular surface of the large limb joints. The youngest animal (four months) was chosen by the herdowner as being typical of the ill-thrift syndrome - and yet otherwise clinically normal. On post-mortem examination, this animal had a severe bronchopneumonia and an enteropathy. *Pasteurella* and *Salmonella dublin* were isolated.

Two of the Abbotstown yearlings from the 2004/2005 overwinter Feed Trial were also submitted for post-mortem examination. They were clinically normal - but in poor bodily condition - prior to euthanasia. The only findings of note on post-mortem examination were histopathological lesions of a chronic low-grade gastroenteritis.

Examination of on-farm records

Records of treatments in the drug treatment register from 2000 to 2005 were examined. According to the herdowner, entries prior to 2003 were under-recorded. Annual veterinary statements for 2003 and 2004 were also examined.

Using treatments as a proxy estimate of clinical disease occurrence, calf morbidity rates for January to May of 2002 to 2005 are given in Table 9. The records indicate a substantial number of antibiotic courses administered to calves from 2002 to 2005; occurring primarily in the early calf rearing period and spilling over into the grazing period. Calves treated ranged

from neonates to about five months of age. Depending on the year, between 30 and 64 *per cent* of calves underwent a course of antibiotic treatment each year since 2002. Repeat-treatment rates are also given in Table 9 as a proxy indicator of disease persistence and recurrence. Estimated repeat treatment rates for 2003 and 2004 were 36 and 23 *per cent*, respectively. This means that 50 *per cent* of animals that became ill in 2003, and 42 *per cent* in 2004, needed more than one course of antibacterial treatment.

Estimated mortality rates of calves for the rearing period January to August are also given in Table 9.

Table 9: Estimated morbidity, mortality and repeat treatment rates in calves and young weanlings from 1st January to 31st May.

Year	N	Estimated morbidity %	Estimated Mortality Rate % ⁵ (period January to August)	Repeat Treatment ¹ (% of population)	Repeat Treatment Rates ¹ (% Of sick animals)
2002 ²	45 ³	33	10	8	26
2003	39	72	4	36	50
2004	65	55	10	23	42
2005	70	64	12	17 ⁴	26 ⁴

1. Animals that underwent two or more treatment courses.
2. Under recorded according to the herdowner.
3. Estimated calf population.
4. Chronic pneumonia identified by necropsy in this group as weanlings, all animals were subsequently treated in September, figures not included in table.
5. Period January to August. 2002 figure from animal movement register; 2003 – 2005 estimated from laboratory submissions, not including elective post-mortems.

Calves bought-in and placed directly into the calfhouse - where the high level of antibiotic usage was occurring - also underwent multiple courses of treatment (Table 10). The first treatment courses commenced approximately nine days after purchase (*see* also Appendix 9).

Table 10: Treatment courses recorded from 31 March to 24 April 2005 for calves bought-in and placed directly into calf rearing house¹.

Bought-in calves	No treatment	One course	Two courses	Three courses	Four courses
N= 20	1	6	5	4	4

¹ Calves bought-in between 21 March and 6 April 2005.

There are limited entries in the records for housed yearlings. Records prior to 2002 are poor but an outbreak of pneumonia was recorded in yearlings in December 2000. Two of those animals were again treated for illness in February 2001, and one died in April 2001. In the spring housing period of 2002, two animals in the yearling age-group of animals each underwent two separate courses of antibiotic therapy consistent with treatment for pneumonia or septicaemia. In the housing period of winter 2003/2004, eight yearlings were treated with antibiotics normally used for the treatment of pneumonia and septicaemia. In 2005, three yearlings underwent a course of antibiotic treatment, these had previously been treated for pneumonia or septicaemia as calves.

Examination of the veterinary end-of-year statements from one veterinary practitioner indicates that between 100 and 130 individual treatment courses were administered to, or dispensed for, calves in each of the years 2003 and 2004 (based on quantity dispensed). The pattern of treatments and dispensing of antibiotics indicates that treatments generally involved single animals. The types of antibiotics used - MarbocylTM, NufloorTM, MicotilTM AlamycinTM and Tylosin 200TM - indicate specific treatments for pneumonias, septicaemias, or enteritis - rather than prophylactic blanket therapy with these drugs.

Reported usage of chlortetracycline oral antibiotic was also high. Chlortetracycline is generally used for the prophylactic therapy of pneumonia.

Milk Recording

Individual-cow monthly milk recording commenced in May 2005. Summary statistics for somatic cell counts (SCC) are given in Table 11. The most important feature of the data is the high proportion of cows in mid-2005 with counts in excess of 400,000 cells/ml (an accepted indicator of mastitis) - compared to zero animals in this category by the end of the year, and into 2006. This improvement was undoubtedly due to the mastitis control measures implemented in the second half of 2005, i.e. hygiene controls and culling high cell-count animals.

Table 11: Somatic cell counts (cells/ml) at milk recording.

Date of measurement	No. cows	% scc>1m	% scc >400,000	% scc>200,000	range
26/05/2005	37	5.4	12.2	29.7	8-1363
18/06/2005	31	22.5	32.3	32.3	25-4379
8/07/2005	39	12.8	28	33.3	25-6169
28/07/2005	38	18	28.9	42	49-3221
18/08/2005	37	8	32	51	30-2709
9/09/2005	29	10	24	44.8	19 - 3695
10/10/2005	32	12.5	28	43.8	40 - 2738
29/10/2005	25	16	44	60	74 - 3838
11/11/2005	23	4.3	8.6	30	39 - 1194
2/12/2005	28	0	14.2	29	14 - 638
28/12/2005	28	0	10.7	17.8	27 - 542
13/01/2006	12	0	0	33	31 - 222
4/02/2006	12	0	8.3	25	36 - 627
24/02/2006	17	0	0	11.8	27 - 375
28/03/2006	21	0	0	14.2	16 - 357

Table 12 provides information on the spread of mastitis in the herd during the period of monthly milk sampling. New infection rates are estimated according to the method of Bradley (2005) which is based on identifying animals whose cell counts have moved across the 200,000 threshold between consecutive recordings. Chronic infections are those with counts above the 200,000 threshold for two or more consecutive recordings. For comparative purposes rates are also given using 400,000 as the cut-off.

Table 12: Mastitis New and Chronic Infection Rates (*per cent*) in milking cows.

Date	New infection rate (200,000)	Chronic infection rate (200,000)	New infection rate (400,000)	Chronic infection rate (400,000)
18/06/05	9.3	12.5	19.4	12.9
8/07/05	17.9	17.9	10	15.3
28/07/05	10	31	5.3	23.6
18/08/05	16	35	10	24
9/09/05	10.3	34	7	17
10/10/05	6.2	34	18.8	9.4
29/10/05	32.0	32	32	12
11/11/05	0	26	0	8.6
2/12/05	7	25	7	7
28/12/05	0	14.2	7	3.5
13/01/06	33	0	0	0
4/02/06	8.3	16.6	8.3	0
24/02/06	5.8	5.8	0	0
28/03/06	9.5	4.7	0	0

While it is clear using both the 200,000 and 400,000 cell cut-offs, the reduction in new and chronic infection rates in the latter part of 2005 is emphasised in the 400,000 cell cut-off column (Table 12). This is consistent with the reduction in cell counts over the same period (Table 11).

Dental Examinations

An extensive examination of cattle teeth was carried out as part of the fluorosis investigation. Advanced wear of teeth was identified in 18 cows aged between 8 and at least 12 years old. These results are covered in more detail in Part 1 of this report.

Animal Toxicological Investigations

The results of blood and tissue fluoride analyses are presented in Part 1 of this report.

No tissue concentrations of arsenic, cadmium, lead, molybdenum, or zinc were of toxicological significance (Table 13).

Table 13: Tissue mineral analyses.

	Number	Mean	Median	Range	Std.Dev
Cd (mg/kg)	14	0.9	0.6	0.10 - 4.9	1.2
Pb (umol/kg)	12	4.1	2.1	1-9	2.7
Arsenic (umol/kg)	11	< 0.5	< 0.5		
Mo (umol/kg)	9	7.7	7.2	5.2 - 12	2.0
Zn (umol/kg)	9	0.8	0.63	0.3 - 2.5	0.7

Feed samples (silage and home-mix feed components) were submitted to the Irish Equine Centre Laboratory in 2003 and 2004 for mycotoxin analysis. While mycotoxins were

detected, concentrations were not at a level which would have had a clinically significant effect on animal performance.

Growth Rates

Only eight of about 45 calves born in 2005 were weighed at or near birth. Weights ranged from 36 to 46 kg. These are acceptable weights for the types of animals and are consistent with the herd history that low birth-weight has not been a feature of the problem.

There was a wide variation in average daily weight gains (ADGs) for calves in 2005. While it was not possible to age-match calves into discrete groups because of the wide spread in calving, a number of patterns were apparent. Early-born calves did better; calves housed for longer periods performed significantly less well. The latter observation is particularly important on a farm which tended to house calves for extended periods. For calves let out, while compensatory growth occurred, it tended to be restricted.

Feeding Trials

The Teagasc reports of the winter feeding trials of 2003/2004 and 2004/2005 are included in Appendices 6 and 7. In the unmatched (ages) trial of 2003/2004, weanling performance was poor at 0.2 – 0.3 kg ADG for the first 92 days of the trial. However, for the second period - days 92 to 142 - performance was acceptable at 0.5 – 0.6 kg ADG. Overall, however, performance was poor.

Performance of all three animal groups on the Brennan farm was poor in the age-matched Feeding Trial of winter 2004/2005. Animal weights increased for the first 70 days approximately - but levelled off thereafter. On the control farm, animals gained weight throughout. There was a variation in ADG between the groups on the Brennan farm. One group demonstrated acceptable gains until day 78 - but from 78 to 112 days declined dramatically. ADG was negative for this period. Overall, ADG for the whole trial for this group was 0.49 kg/day, whereas animals of the second trial group on the Brennan farm had poor performance from day one. Overall gains for that group were 0.27 kg/day for 112 days. This is in contrast to the groups on the control farm where performance showed acceptable average gains for the overall period at 0.65 and 0.67 kg/day.

The group of Abbotstown animals on the Brennan farm performed well for the first 49 days of the trial - but subsequently fell off and ended up with net weight losses for the period 78 to 112 days. Overall gains for these animals were poor.

Discussion

The Brennan farm has reported poor growth in cattle over many years resulting in extended rearing periods for animals. This has affected different aspects of the farm enterprise. Replacement heifers take longer to reach maturity – and some may never reach a suitable breeding size. This has contributed to the high age-profile of the herd as older cows are retained. Dry-stock also take longer to reach sale or slaughter weight. Some never reach commercially viable weights - even with rearing periods extended by up to a year.

Calves and growing stock

The main problem in calves and growing stock when this case was first referred to the Kilkenny RVL in January 2003 was growth retardation and poor performance – in particular of housed animals. Most of the calves born in 2001, for example, were reported to have become stunted. However, there was also clearly a historical problem of high mortality rates in calves - approximately 40 *per cent* over the period 1998 to 2001. The exact causes of most of these losses cannot be determined as there were relatively few submissions to the

Laboratory over this period (*see* Appendix 4). From 2003 to the present, mortalities have been relatively low in comparison. However, investigations since 2003 indicated that disease levels in calves were unacceptably high.

Some indication can be obtained as to the predominant causes of historical losses by examination of the farm therapeutic records. For example, the specific antibiotics used to treat calves in 2002 - the earliest date for which records are available for calves - indicate the presence of diseases such as septicaemias, pneumonias and enteritis. This was supported subsequently by the confirmation of these conditions on post-mortem examinations of calves and growing stock in 2005.

Passive transfer of immunity via colostrum is critical to calf health. While a relatively small number of tests was performed ($n = 32$), almost a half of calves tested had suboptimal to low passive immune status (zinc sulphate test < 20). If this were representative of the annual calf crop, then it indicates a relatively high proportion of calves at increased risk of infectious disease from birth. Where disease occurs, this would result in a build-up of infectious burdens and environmental contamination - especially where the isolation of sick animals is not practised. This increase in environmental contamination and infectious burden is commonly observed on farms in the intensive calf rearing period of spring when animals are housed.

The classical calfhood diseases of colibacillosis and colisepticaemia affect the susceptible calf and can cause death where passive immunity is low. The disease is contracted from the environment to which sick animals have shed the pathogen. Rearing of calves in single pens, on the other hand, helps to limit the spread of the organism. Rotavirus is another commonly encountered cause of enteritis in the young calf. The intervention to stem an outbreak is similar to that for colibacillosis and vaccination of cows in subsequent years should prevent further outbreaks because of localised immunity in the intestine provided by adequate colostrum.

Rotavirus, *Escherichia coli* and *Cryptosporidia* have been isolated from calves submitted to the laboratory from the Brennan herd. Enteritis (scour) is reported to have been the main cause of disease and death in calves during the period of highest mortality (1998 – 2001). In 2002 when a calf and faecal samples were submitted to Kilkenny RVL, rotavirus was identified. It is reported that further losses of calves were averted when milk from a rotavirus-vaccinated herd was provided to calves. In 2002 the mortality rate in calves was significantly lower than had been the case in previous years, but examination of the on-farm therapeutic records for 2002 indicates that there was still a high level of clinical disease present in calves at that time. The specific antibiotics used to treat these calves suggest the presence of diseases such as septicaemias, enteritis and pneumonia.

Although clinical respiratory disease was not reported to have been a major problem historically – other than in weanlings after housing - investigations in 2005 identified sub-clinical pneumonia as being a significant problem when elective post-mortems were performed. A severe infectious pneumonia, for example, was identified in an elective post-mortem examination of a weanling in August. This animal had been specifically identified by the owner as being typical of growth problems experienced. However, no other clinical signs had been reported. A severe pasteurella bronchopneumonia was also identified on post-mortem examination of another weanling calf which had a poor growth rate since birth (ADG 0.2 kg/day while housed – and which did not improve even after turnout).

Chronic broncho-pneumonia was identified in a housed six-week old calf in April 2005. Chronic pneumonia was also found on post-mortem examination of a four month old ill-thrifty weanling – which was still housed in August. These findings, together with the

information obtained from the drug treatment records, indicate that pneumonias were established in the calf-house.

Mixed infections were identified in some pneumonic animals at post-mortem; this is a common finding in enzootic pneumonias. The pathogens included *Mannheimia hemolytica*, *Haemophilus somnus*, and *Pasteurella multocida*. The lung lesions were generally quite severe. In these cases, large numbers of pathogenic bacteria may be recovered from the lung (Bryson, 1985). The animals are therefore highly infectious to their comrades - particularly while housed (Lundburg 2004). Chronic bronchopneumonia may develop if initial lung damage is severe and the lesions may remain for months with occasional flare-ups (Bryson 1985). Respiratory disease in young weanlings at grass on the Brennan farm was detected when elective necropsies were performed, and it is probable that this condition may have first arisen when they were housed.

In addition to the diseases described above, the isolation of *Salmonella dublin* is highly significant. *Salmonella dublin* was identified as the cause of abortion in 2000. An entry in the on-farm movement register at the time also indicates a calf with terminal gangrene of a leg. This is likely to have been the terminal dry gangrene form of septicaemic salmonellosis that occurs in calves. *Salmonella dublin* was also isolated in samples from calves with enteritis - and from a weanling with pneumonia - in 2005. These findings indicate that *Salmonella dublin* infection has been indigenous to the farm for some years.

Although Salmonellosis may present as differing syndromes in a herd, all have been described on this farm (i.e. abortions, acute diarrhoea, temporary acute milk loss, septicaemia which may be subclinical or manifest as acute pneumonias, and terminal dry gangrene). The majority of *Salmonella* infections in an affected herd are subclinical. In other words, the animals may not present with clinical signs of disease (such as diarrhoea) - but may still be detrimentally affected. Clinical infections are only the tip of the iceberg - even during outbreaks of clinical salmonellosis (Gay, 1999). Misunderstanding this 'iceberg effect' can lead to inappropriate management of individual animals in infected herds - meaning that attention is often only paid to the animals that are clinically ill - rather than instituting measures on a herd basis to limit the spread of disease (Gay, 1999).

The main complaint in dry stock (yearlings) in 2003 was ill thrift - which occurred predominantly when animals were housed. Although information is limited in this group of animals, pneumonias were also identified by elective necropsy in 2005. Likewise *Salmonella dublin* was isolated from a recumbent yearling with a haemorrhagic enteritis in 2004. The farm therapeutic records also indicate that over the years a number of yearlings underwent courses of therapy in the winter housing period with antibiotics similar to those used in calves and growing stock.

It is not possible to clearly define the role of BVD virus in the herd. It was certainly present since at least 2003 - and undoubtedly contributed to ill-health in younger animals. However, it is unlikely that it was a primary cause of the stunting problem. The limited weighings of newborn animals - and reports that newborn calves were of normal weights - tend to rule out the typical *in-utero* growth retardation of BVD virus. BVD can also cause immunosuppression - resulting in increased susceptibility to bacterial infections. While it is not possible to say to what extent this may have contributed to the disease problems on the Brennan farm, it cannot be ignored given evidence that the virus was actively circulating among susceptible animals over a number of years - and combined with the high incidence of infectious disease from calfhood.

Growth

The pattern of poor growth rates observed in some animals in 2005 - with variable but restricted recovery at turnout - is typical of what one would expect where a severe chronic unresolved infectious disease problem exists. The effects of disease on animal growth and future weight gain can be substantial and are dependent on the severity, type, and length of disease episodes (Virtala, 1996; Svensson, 2003; Donovan 1998). Respiratory disease has a greater effect on growth rates than enteritis (Virtala, 1996; Svensson, 2003; Donovan 1998). The difference in average daily gain (ADG) between healthy calves and calves that suffer from respiratory disease can be substantial. In a 90-day feeding trial, calves that experienced a single episode of respiratory disease had 0.18 kg lower ADG than healthy calves (Smith, 1998). Those sick two or more times had 0.33 kg lower ADG – which indicated absence of a compensatory gain. Virtala (1996) also observed absence of compensatory gain where prolonged or recurrent calthood disease episodes occurred. In other words, animals did not recover their growth rates. In general, animal growth rates recover once they have recovered from disease (Andrews, 1981). However, with extended or repeat disease episodes, growth rates may not recover (Virtala, 1996).

In the Brennan farm, it is likely that extended and multiple disease episodes, due to infectious pathogens, have been occurring with some regularity in calves and weanlings – possibly over several years. The frequency and severity of these episodes would have been contributed to by the failure to isolate sick animals – thus providing a continuing source of infection for other animals in the house. In the case of pneumonia, this would have been compounded by the mixing of different ages of animals in the same air space.

Feeding Trials

Animal performance in the informal winter-feeding trial of 2003/2004 is difficult to interpret due to the loose design and absence of age-matching in groups. Performance was initially poor – but improved in the latter part. Interpretation is made more difficult by the fact that some of the older animals were removed for sale in the latter part of the trial. The Teagasc Report is included in Appendix 8.

The results of the more formal structured trial of 2004/2005 undoubtedly demonstrated a negative effect of location on animal performance on the Brennan farm. Weight gains of the two age-matched groups - as well as the Abbotstown animals - ranged from poor to very poor. While it is not possible at this time to identify the nature of the location effects, the Teagasc Report (Appendix 8), concludes that feed intake, management and housing do not explain the poor performance of the three yearling groups on the Brennan farm over the winter of 2004/2005.

Milk Production

Milk yields were historically poor on this farm – based both on herdowner reports and records of milk sold. This continued to be the case during the VLS investigations since 2003 – and was confirmed when individual-cow recording was introduced in mid-2005. A number of factors have been identified by the present investigations which can go a long way to explaining such poor yields. Mastitis was recognised as being a significant issue from an early stage of the DAF investigations, and recommendations for mastitis control were given in 2003. While it is not known how long it had been a problem in the herd, a number of well-recognised mastitis pathogens were identified from milk samples submitted to the Laboratory in 2002 – namely *Staphylococcus aureus*, *Streptococcus uberis*, and *Streptococcus dysgalactiae*. It was only when individual-cow milk recording was introduced by DAF in mid-2005 that the full extent of the problem could be identified. About a third of the milking

herd had cell counts in excess of 400,000 – indicative of sub-clinical or clinical mastitis - in the months of June, July, and August 2005. There was also evidence of significant spread of mastitis in the herd in the second half of 2005 (Table 12).

Sub-clinical mastitis can lead to significant reductions in milk yields. Losses of around 1,200 kg in a lactation have been reported (Wilson 2004; Grohn, 2004). The negative impact of clinical and sub-clinical mastitis on yields can persist throughout a lactation - reducing yield by between 15 and 40 *per cent* (Beck *et al*, 1992). They may carry over to the next lactation to account for losses of 20 – 30 *per cent* (Wilson, 2004; Grohn, 2004). Milk quality is also adversely affected (Murphy, 1989; Saeman 1988; Urech, 1999).

This investigation has identified many high cell count cows in the Brennan herd - particularly old cows that had not been identified as clinically mastitic. This subclinical mastitis was predominantly due to *Staphylococcus aureus* within the milking herd. It is well-recognised that failure to identify and treat *Staphylococcus aureus* mastitis can result in substantial losses in milk yield (Grohn 2004). Infected animals also continue to provide a reservoir of infection for other cows.

Significant spread of mastitis was indeed identified in the herd. The new infection rate in June 2005, for example, was between seven and 10 *per cent*, and the chronic infection rate was between seven and 17 *per cent* - depending on the cut off used.

In contrast, cell counts - and the new infection rate - fell markedly in the latter part of 2005 and early 2006. This improvement was due to implementation of basic mastitis control measures, i.e. culling of old and high cell-count cows, and improved milking hygiene.

Nutrition critically influences the capacity of a dairy cow to attain peak milk yield and to maintain milk yield as lactation progresses. Peak milk is the point in the first hundred days of lactation where milk yield in a cow is at its maximum. After this point it declines normally by 8 – 10 *per cent* per month - and the term persistency refers to the rate of decline beyond peak (Leslie, 1994). For each kilogram of milk below that predicted for peak yield, there is a loss of 200 kg of milk in a 305-day lactation (Leslie, 1994) - and this cannot be recovered within that lactation. Therefore it is the objective of dairy enterprises to attain a cow's expected peak milk and to achieve persistent high yields after that point.

As peak milk is very sensitive to nutritional inputs in the transition stage from dry cow to lactation and in the early lactation period, any deficiencies in caloric density of feed in this period will affect peak yields. A Teagasc report on farm nutrition in March 2004 (Appendix 8) noted that “the silage was deficient in energy for thin cows in late pregnancy”. Pre-calving cows at the time were reported to have had an average body condition score (BCS) of about 2.7. Poor cow condition post-calving (BCS under about 3.0) can be expected to have a negative impact on milk production.

The high age-profile of cows in this herd will also have had an impact on milk yields. Over 50 *per cent* of cows in the herd were over eight years old in 2003 and 2004 (Table 8). Many, therefore, will have been past their productive peak.

Liver fluke infestation was also identified in cows on post-mortem examination. Depending on the stage of lactation at which it occurs, even moderate infestation can have a significant negative impact on yields (Blowey, 2004).

Current Situation (up to mid-May 2006)

Animal health and production have shown a definite improvement in the first five months of 2006. Calf health has been good – and this has been reflected in improved growth rates (average around 0.6 kg/day). Of 39 calves born on the farm up to mid-May 2006, only two

have died – one a stillbirth and the other a peri-natal mis-adventure. While it is too early in the season to draw conclusions regarding milk yields, production is reported to have improved with individual cows milking up to 28 kg.

Conclusion

In terms of the initial case presentation in January 2003 – growth retardation, ill-thrift, and poor milk-production - the DAF Laboratory Service investigations outlined in this report have identified on-farm disease conditions which could account for much of their occurrence and severity. The farm has clearly had an on-going problem of infectious diseases acquired in the calf-house, persisting in individual animals and – besides losses due to deaths - restricting subsequent growth and performance in survivors. The most notable of these are the acute and chronic respiratory infections identified in calves and weanlings. *Salmonella dublin* infection has also been present in the farm for some time. Ill-thrift is a common feature of chronic salmonellosis in cattle. As these diseases are highly contagious, the practice of not isolating animals that are clinically ill is likely to have magnified these problems on the farm. While the impact of active BVD virus infection in the herd on the overall problems is not clear, this agent is associated with both ill-thrift and immunosuppression (Charleston *et al*, 2001). Mastitis, cow age, and liver fluke infestation were all identified as factors likely to have negatively impacted on milk production.

Some questions remain unanswered – most notably the outcome of the 2004/2005 winter Feed Trial. This and other issues are the subject of ongoing investigations. A group headed by staff from the DAF-funded Centre for Veterinary Epidemiology and Risk Analysis in UCD Veterinary School, and including scientists from the EPA and the University of Limerick, is currently considering wider ecological and epidemiological approaches to the investigation of the problems on this farm. DAF Veterinary Laboratory Services will continue to be available for diagnostic clinical pathology and post-mortem investigations as required.

Arising from a meeting in August 2005⁸, the Department of Agriculture and Food funded an animal health program for the farm - which included a wide-ranging vaccination program and provision of calf hutches for the 2006 calving season. The success of this programme depends entirely on the implementation of improved management practices. These include items such as provision of isolation areas, isolation of ill or poorly thriving animals, and generally improved biosecurity and disease management on the farm. It is hoped that continued adherence to such measures will help to maintain the improved animal health and production status recorded on the farm in 2006.

⁸ Interagency meeting (attended by EPA, Teagasc, DAF, and the herdowner's veterinary practitioners)

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Appendix 1 – Animal fluoride concentrations

Serum – blood samples collected from the Brennan and a neighbouring farm in 2005.

Animal	Kilk ref	F (µg/L)	Kilk ref	animal	F (µg/L)
SN	Kilk/05/02188-1	30	Kilk/05/02186-3	SLC	39
SN	Kilk/05/02188-2	44	Kilk/05/02186-2	SLC	47
SN	Kilk/05/02188-3	32	Kilk/05/03113-1	SLC	114
SN	Kilk/05/02188-4	29	Kilk/05/03113-2	SLC	108
SC	Kilk/05/02475-4	37	Kilk/05/03113-3	SLC	75
SC	Kilk/05/02475-3	43	Kilk/05/03113-4	SLC	111
SC	Kilk/05/02475-1	49	Kilk/05/03113-5	SLC	144
SC	Kilk/05/02475-2	56	Kilk/05/03113-7	SLC	73
SLY	Kilk/05/02186-5	36	Kilk/05/03113-6	SLC	96
SLY	Kilk/05/02186-6	33	Kilk/05/02534	SE	42
SLY	Kilk/05/02186-7	30	Kilk/05/03107	SE	33
SLC	Kilk/05/02186-4	40	Kilk/05/02909	SE	30
SLC	Kilk/05/02186-1	64	Kilk/05/02654	SE	35

SN; serum from neighbours animals, SC; serum from control animals, SE; serum from elective necropsy animals, SLC; serum from live cows on the Brennan farm; SLY; serum from live yearlings on the Brennan farm.

Urine samples collected from Brennan animals 2003 to 2005.

Animal	Kilk ref	F (mg/L)	Animal	Kilk ref	F (mg/L)
3 yrs	Kilk/03/02968-4	1.1	Cow, old	Kilk/05/03113-2	3.6
3 yrs	Kilk/03/02968-5	1.3	Cow, old	Kilk/05/03113-3	3.4
Yearling	Kilk/03/03392	0.6	Cow, old	Kilk/05/03113-4	5.4
Calf	Kilk/04/03603	<0.1	Cow, old	Kilk/05/03113-5	8.5
Cow, old	Kilk/05/02420	3.9	Elective	Kilk/05/03107	0.5
Calf	Kilk/05/02567	0.1	Elective	Kilk/05/03362	0.1
Yearling	Kilk/05/02654	1.6	Elective	Kilk/05/03425	0.1
Neonatal	Kilk/05/02860	<0.1	Old cow	Kilk/05/03671	1.2
Old Cow	Kilk/05/02420	1.3	Old cow	Kilk/05/03811	0.3
Yearling	Kilk/05/02909	1.1	Elective PM	Kilk/05/03962	0.4
Neonatal	Kilk/05/02936	0.2	Cull cow	Kilk/05/05613	0.8
3 yrs	Kilk/05/03113-1	5.6			

Appendix 1 contd.

Bone samples from Brennan cattle 2003 to 2005

Animal	Kilk ref	Metacarpus (F - mg/kg ash)	Rib (F - mg/kg ash)
3 yrs	Kilk/03/02968-2	1180	
3 yrs	Kilk/03/02968-3	758	
3 yrs	Kilk/03/02968-4	264	
3 yrs	Kilk/03/02968-5	1090	
Cow	Kilk/03/04287	1380	
Cow, old	Kilk/05/00173	1310	
Cow, old	Kilk/05/02420	800	1220
Yearling	Kilk/05/02909	243	309
Calf	Kilk/05/02567	147	247
Foetus	Kilk/05/02445	110	
Yearling	Kilk/05/02654	278	247
Neonate	Kilk/05/2936	68.2	69.3
Yearling	Kilk/05/03107	145	248
Yearling (Abbotstown)	Kilk/05/03362	111	215
Yearling	Kilk/03/03392	251	
Yearling (Abbotstown)	Kilk/05/03425	91.2	185
Cow	Kilk/05/03671	302	
Old cow	Kilk/05/03811	1200	1620
Weanling elective	Kilk/05/03962	73.6	152
Old cow	Kilk/05/05613	1350	
Calf	Kilk/05/02567	65.8	92.8

Fluoride concentrations in matched bone and urine samples from Brennan animals.

Animal	Kilk ref	Metacarpus (mg/kg ash)	Rib (mg/kg ash)	Urine (mg/L)
3 yrs	Kilk/03/02968-4	264		1.1
3 yrs	Kilk/03/02968-5	1090		1.3
Yearling	Kilk/03/03392	251	434	0.6
Cow, old	Kilk/05/02420	800	1220	3.9
Yearling elective	Kilk/05/02909	150	309	1.1
Yearling elective	Kilk/05/03107	145	248	0.5
Calf	Kilk/05/02567	65.8	92.8	0.1
Yearling	Kilk/05/02654	147	247	1.6
Neonate	Kilk/05/2936	68.2	69.3	0.2
Yearling (Abbotstown)	Kilk/05/03362	111	215	0.1
Yearling (Abbotstown)	Kilk/05/03425	91.2	185	0.1
cow	Kilk/05/03671	302		1.2
Old cow	Kilk/05/03811	1200	1620	0.3
Weanling elective	Kilk/05/03962	73.6	152	0.4
Abattoir cow	Kilk/05/05613	1350		0.8

Appendix 2 - Report on findings on permanent teeth in cattle of the Brennan farm and in control cattle.

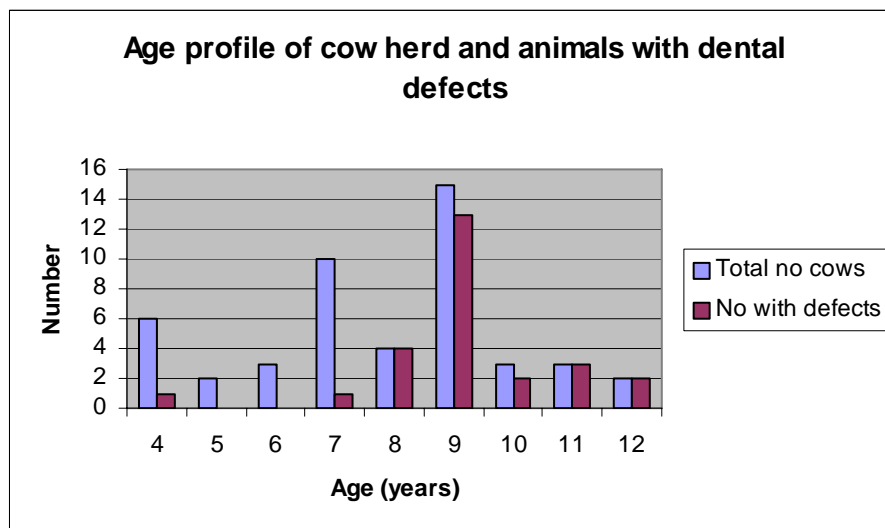
Materials and methods

Clinical examination of incisor teeth was performed in April or May 03 and in January 05. Mottling and staining of teeth, degree of wear, unusual wear or broken teeth, pitting of enamel and missing teeth were recorded. Some photographs were taken. Information on the ages of animals and whether they were home-bred or purchased was extracted from information in the Herd Register kept by the herdowner for animals born since 1997 or from estimations by the herd-owner for older animals. Teeth of animals originating from many counties and being slaughtered at a local abattoir were examined as controls in May 2003.

Results

Dental defects correlated with age of animals in 2005 are shown in Fig. 1.

Fig 1



Examination of teeth April / May 2003

The teeth of 29 dairy cows were examined in April / May 2003. Dental defects were found in 21 animals.

Fifteen had defects of the enamel. Seven had depressed brown defects (non-symmetrical in three and affecting corresponding teeth in four). Seven had non-depressed brown defects (non-symmetrical in three and affecting corresponding teeth in four). One had a white non-depressed spot that was non-symmetrical. Two had notches on the upper border of an incisor. Three had missing teeth (two were missing one tooth while one was missing two teeth). Seven had worn teeth.

Examination of teeth Jan 2005

Forty eight animals (47 cows and 1 bull) were examined in January 2005. Dairy and suckler cows were included. No significant abnormality was noted in 21 animals.

The incidence of dental defects was much higher in older animals and almost absent in younger animals.

Defects were found in 26 animals and included brown spots on teeth, loss of teeth and excessive wear.

One 12 year old animal had a brown spot with a depression in the enamel on teeth R2 and L2. Two years earlier, a brown spot without a perceptible depression in the enamel had been recorded in the same two teeth.

Four animals had dark spots on some teeth. Three of these animals had been examined two years earlier and defects had been found on the same teeth in most cases. Some of these earlier defects were more severe, being associated with pitting of the enamel.

Eight animals were missing one or more teeth. Six animals were missing one tooth while two were missing two. Four of these animals also had excessive wear of the remaining teeth while a fifth had chips broken off some of the remaining teeth. A sixth animal that was missing L2 and L4 had a crooked R1. Only two of the eight animals had no visible tooth defect apart from the missing tooth. Ages of affected animals ranged from 8 to 11 or 12 with five of the eight being either 8 or 9 years old.

Eighteen animals, between 8 and 12 years had advanced wear of incisor teeth. The teeth were worn to the gums in at least four animals. Ten of these 18 animals had been examined two years earlier, in spring 2003. Four had extremely worn teeth then while another four had a line or a spot on one or more teeth, consistent with damage to the enamel. No tooth defects were recorded in 2003 for the remaining two animals.

Purchased animals

The defect rates in homebred and purchased animals are compared in the table below:

	All ages	> 7 years	7 years or younger
Homebred	7/19=37%	5/6=83%	2/13=15%
Purchased	19/29=66%	19/21=90%	0/8=0%

Abattoir survey:

A survey of reactor animals slaughtered on 22 May 03 was conducted at a slaughter-house. Twenty-seven animals under 30 months and 40 animals over 30 months were examined. The younger animals came from 11 different counties while the older animals came from at least eight counties. Most herds were represented by only one animal. Brown staining was widespread in the over thirty month group. Findings are summarised below:

Tooth colour (Under 30 months):

Total	White teeth	Brown stain
27	17	10

* Incidence of brown staining was unrecorded once it was seen how widespread it was in the abattoir cattle.

Spots (brown or white) and depressions in enamel

	Under 30 months		Over 30 months		
Total	No defects	Defects	Total	No defects	Defects
27	20	7	40	19	21

The defects in the seven under-30 month animals consisted of :

- two animals with brown depressions in enamel of symmetrical teeth;

- two animals with brown non-depressed marks in enamel of symmetrical teeth;
- three animals with white non-depressed marks in enamel of symmetrical teeth.

No enamel stains or depressions were seen in the temporary teeth of the under-30 month animals.

The defects in the 21 over-30 month animals consisted of nine animals with white marks and 12 animals with brown marks on the enamel:

- four animals had white non-depressed non-symmetrical defects;
- five animals had white non-depressed symmetrical defects;
- two animals had brown depressed symmetrical defects;
- eight animals had brown depressed non-symmetrical defects;
- two animals had brown non-depressed symmetrical defects.

Damage to occlusal surfaces

Nicks in the outline of the top of the front surface of permanent incisors were seen in seven of the 27 under-30 month animals. Defects affected symmetrical teeth in two cases. Similar nicks were seen in six of the 40 over-30 month animals. Symmetrical teeth were not affected in any case.

Tooth loss and abnormal wear of teeth

No missing or abnormally worn permanent teeth were observed in the under-30 month animals.

Severe wear of permanent incisors was seen in 4 of 40 of the over-30 month animals and tooth loss was also seen in 4 of the 40 animals. Two animals had one incisor missing and two animals had three missing. The age of these animals is not yet known.

The defect rates in the two examinations of cows of Brennan farm are compared to the rates found in abattoir animals below:

	Brennan farm 2003	Brennan farm 2005	Abattoir <30 mth	Abattoir >30 mth
Total defects	21/29=72%	26/48=54%		
Enamel defects	15/29=52%	5/48=10%	7/27=26%	21/40=53%

Summary of findings in teeth of live yearlings and cows, examined April 2005

Yearlings born 2004 (Deciduous Teeth):

Eleven purchased animals were examined. Six had no or minimal tooth staining and no other defects. Animal 491 had moderate light brown staining of one central incisor and animal 334 had moderate light brown staining on right incisors 1 and 2. Animal 501 had a gap between its central incisors which had prominent necks and were mildly stained. Animal 612 had a very distinct black spot on left incisor 3. Both this animal and 629 had moderate staining of the incisors with a horizontal white band at gum level in all teeth. All of these animals were purchased at between 2.5 and 6 weeks, except the last three animals (tag numbers >600) which were eight months old.

Twenty four homebred calves were examined. Seventeen had little or no staining of the teeth. 3-0312 had a brown spot on each central incisor; 4-0338 had very obvious brown mottling on the centrals and less obvious brown staining on the other incisors; 5-0347 had symmetrical brown marks on both I-2 and a possible white mark on left I-3. There was a deep, dark depression in the enamel of left I-1 of 3-0329. The dam of this animal had spent only one

week on the farm before the birth of this calf. There was extensive mid- to dark brown staining of all incisors, with a white band at gum level, in the teeth of the latter animal (3-0329) and also 6-0348, 7-0349, 1-0351 and 9-0350. The latter four animals were all born over a short time period. (The date of birth of 9-0350 is uncertain: it was born sometime in May 04)

Conclusion: No lesions consistent with fluorosis were evident in deciduous teeth of animals born in 2004.

Cows

The teeth of 13 live cows were examined and photographed.

WGC 48 S had pinhead mottling of both central incisors; symmetrical brown staining on both I-3 and brown staining on left I-4. This animal was bred about three miles from the factory and came to Dan Brennan’s farm at 16 months old.

Seven animals (JRA 789949, 515516, 050043, 437825, 171062, 850827 and 367038) had a missing incisor. In all cases, the root as well as the crown was missing.

One animal (171062) had a crooked central incisor. This animal was purchased at about 4 yrs of age.

All of the incisors in two animals (JSA 691316 and 050043, aged 10 and 11 yrs) were worn to the gums.

Conclusion: Age related dental defects observed

Summary of findings in teeth of live weanlings, born 2005 and examined 16 Nov 2005:

The teeth of all the homebred weanlings born in 2005 were white or had minimal staining. No lesions suggestive of fluorosis were seen. Many of the animals purchased as calves had faint grey staining of the incisors.

Times of eruption of permanent teeth

The Cornell researchers report delay in eruption times occurring with fluorosis.

The permanent teeth of four heifers, slaughtered in April 03, aged 34 to 36 months, were examined.

Numbers of incisors erupted and ages for four of heifers from Brennan farm slaughtered April 05.

Age (months)	No. of incisors erupted
34	4
34.5	2
34.5	4
36	4

About 10 Brennan-farm heifers, aged about 27 months were examined in Spring 2005.

Conclusion: Eruption dates seemed normal.

Discussion:

The age profile is high in this herd. Almost half of the cows (23 of 48) are nine or more years old. Less than a quarter (11 of 48) are six years or younger. This is partly a result of ongoing problems with animal growth as in most years homebred replacements are not sufficiently

well grown to enter the herd. Similarly, the proportion of purchased animals is higher in recent as compared to earlier years.

No direct association between degree of tooth wear and body condition was noted. If grass length was short during the summer, such animals might not graze the required amount and might suffer in body condition as a result.

The defect rate in purchased as compared to homebred animals appears to be related to the age of the animals rather than to their farm of origin. Most of these animals were bought at between one and two years old so that some incisor development would occur after purchase leaving them as susceptible as homebred animals to any influence on Brennan farm affecting tooth development.

Most of the cows with enamel defects were born before the recording of birth dates became compulsory. Therefore it is not possible to use the expected date of eruption of individual teeth to calculate when the tooth damage occurred.

The abattoir survey showed an unexpectedly high incidence of dental defects, especially staining, brown or white spots on the enamel, often associated with pitting of the enamel. As these animals came from so many different herds in so many counties, it is unlikely that they are due to fluorosis.

Appendix 3 – Results of histopathological examinations of bone samples from Brennan cattle

Report on histopathological examination of bone sections from thirteen carcass submissions from premises of Mr Dan Brennan, Castlecomer, to Kilkenny RVL in 2005

(Note: gross findings on bone and teeth are described in Part 1 of the Main Report on the investigation.)

Microscopic examination of decalcified bone sections was undertaken as part of the investigations into the possibility that fluoride toxicity was contributing to the growth retardation observed in cattle on this farm – and also to characterise the histopathological changes in long bones associated with growth retardation irrespective of cause.

Normal bone fluoride concentrations in cattle range from about 200 to 1,800 mg/kg⁹ (fat-free). Bone fluoride concentrations in animals from the Brennan farm have ranged from 65 mg/kg in the mandible of a calf to 1,620 mg/kg ash in a coccygeal vertebra of an old cow (Main Report – Appendix 3).

According to Jubb, Kennedy and Palmer (1993)¹⁰ no gross or microscopic lesions are seen in cattle at bone fluoride concentrations below about 2,500 mg/kg (fat-free).

Between about 2,500 and 6,000 mg/kg, the following microscopic changes may be seen without any gross changes:

- Brown mottling of some osteons (seen in un-demineralised sections only).
- Accumulations of poorly mineralized osteoid seams (seen in un-demineralised sections only).
- Enlarged and irregular osteocyte lacunae, canaliculi, and osteons due to incomplete ossification of the pericellular matrix.
- Increased numbers of irregularly distributed osteocytes.
- Complex mosaic pattern in cancellous trabeculae due to numerous cement lines from multiple surges of bone removal and replacement.
- Osteoporosis.

Above about 6,000 mg/kg, the gross changes described below may be seen to a greater or lesser degree – depending on the duration of exposure and the ages of the affected animals.

- Chalky-white, roughened bone surfaces due to formation of exostoses. Changes are usually bi-laterally symmetrical.
- Thickened spongy cortex with narrowing of the medullary cavity.
- Osteosclerosis (hardening) or osteomalacia.
- Joint surfaces are generally not directly affected.

None of the above gross lesions have been observed on animals from the Brennan farm.

The classical lesions of fluorosis in teeth are well described and can be recognised grossly. They include (in order of severity):

⁹ Puls R. (1994) Mineral levels in animal health, 2nd edition; pages 110-111.

¹⁰ Pathology of Domestic Animals, 4th edition, Jubb KVF, Kennedy PC, Palmer N, eds., Academic Press, Inc., Vol. 1, 1993.

- Initially chalky-white discolouration on the enamel surface.
- Later, yellow or brown discoloration of the enamel (cannot be removed by scraping).
- Mottling and pitting of enamel.
- Abnormal tooth wear.

Other than excessive tooth wear, which is not unusual in aged cows, the above lesions have not been observed in any animals from this farm. Although dark staining of teeth has been observed, similar staining has also been found in random animals from other farms examined at slaughter. The staining in question has also been shown to be superficial, i.e. can be removed by scraping. This is in contrast to the staining of fluoride toxicity which is embedded within the enamel.

The following is a report on the microscopic examination of demineralised bone sections from selected animals from this farm.

The standard procedure for the preparation of bone sections for microscopic examination involves demineralisation (decalcification) in acid solutions. This process softens the tissues so that thin sections can be cut, mounted and stained using standard histological apparatus and techniques. The stained sections can maintain a high degree of cellular and structural morphology – and this allows microscopic examination for a wide variety of pathological processes.

The main limitation of this procedure is that it does not allow visualisation of some pathological changes dependant on the presence of mineralised bone in sections. However, the preparation of un-demineralised bone sections is a non-standard technique which is not available to the DAF Veterinary Laboratory Service within its own resources. Because of this, the following histopathological assessment will only allow identification of microscopic changes of fluorosis in bone which can be visualised in demineralised sections (see bullet list above). However, a certain number of fixed bone and tooth sections have been stored in Kilkenny RVL should further processing be initiated.

Microscopic findings in bone sections

Depending on availability, bone sections examined were from the humerus, radius, femur, tibia, metacarpus and metatarsus.

Kilk/05/02445 – Foetus from cow reference Kilk/05/02420.

Findings

No specific lesions noted.

Conclusion

No specific changes.

Kilk/05/04807 – Weanling six months old.

Findings

Articular cartilage showed no specific change. Epiphyseal trabecular density low – i.e. osteoporosis. Trabeculae thin. Some trabeculae fractured and with associated microhaemorrhages. Physeal growth plate irregular with large cartilage down-growths into metaphysis. No primary trabeculae. Resting zone of cartilage thick in places – columns of

hypertrophied chondrocytes shorter than normal. Physis almost completely closed indicating little or no active growth in this bone at time of death.

Conclusion

Abnormal endochondral ossification with resultant osteoporosis and retarded bone growth.

Kilk/05/02420 – Cow. Dam of Kilk/05/02445.

Findings

Articular cartilage showed no specific change. Trabecular distributional density appeared normal¹¹. There was no evidence of abnormal remodelling of bone. Osteocyte numbers and distribution, and cement line patterns, did not appear unusual in bony tissue. Osteonal size and distribution in cortical bone was regular.

Conclusion

No specific changes.

Kilk/05/02860 – 10-day old calf.

Findings

Articular cartilage showed no specific change. Trabecular spaces contained largely adipose tissue – with widespread petechial haemorrhages and some fibrin formation. There was active haematopoiesis in the metaphysis. There was some evidence of interference with endochondral ossification characterised by presence of apparently unmineralised cartilage fragments (based on staining characteristics) in inter-trabecular spaces. Some primary trabeculae were thin or shortened.

Conclusion

Changes overall consistent with a terminal toxemia. Some evidence of defective endochondral ossification.

Kilk/05/02936 – 10-day old calf.

Findings

Articular cartilage showed no specific change. Trabecular distribution appeared normal. Trabecular spaces in the epiphysis and metaphysis contained haematopoietic and adipose tissue. Multiple fragments of apparently unmineralised cartilage were noted in the epiphysis between trabeculae. The physal growth plate was of variable thickness. The primary spongiosa appeared short and thick in places.

Conclusion

Some evidence of interference with endochondral ossification characterised by retention of growth plate cartilage.

Kilk/05/02909 – Yearling.

¹¹ Reference to ‘normal trabecular distributional density’ indicate relevant sections are not considered osteoporotic. However, as this is a relative measurement, and in the absence of suitable comparative material from similar-age animals, a degree of osteopenia could not be ruled out in some samples. Osteopenia of old age could be expected as a normal finding in a proportion of older cows.

Findings

Sections of articular cartilage examined showed no specific change. Epiphyseal and metaphyseal trabecular density low, and trabecular structure thin, indicating osteoporosis. Trabecular spaces contained largely adipose tissue. Physeal growth plate of irregular thickness with occasional cartilage downgrowths into metaphysis. Virtual absence of primary trabeculae indicating cessation of growth.

Conclusion

Abnormal endochondral ossification characterised by osteoporosis and premature closure of growth plate.

Kilk/05/03107 – Bullock 13 months.

Findings

Articular cartilage showed no specific change. Trabecular distribution was light indicating osteoporosis. The epiphyseal and metaphyseal cavities contained haematopoietic and adipose tissue. Fragments of apparently unmineralised cartilage were noted in the epiphysis between trabeculae. The physeal growth plate was of very irregular thickness. Cessation of growth was indicated by absence of primary trabeculae over most of section on slide – and presence of secondary trabeculae parallel to the growth plate. Fingers of growth plate cartilage extended into metaphysis from the physis in places. Some fragments of apparently unmineralised cartilage were noted between trabeculae in the metaphysis.

Conclusion

Abnormal endochondral ossification characterised by osteoporosis and premature closure of growth plate.

Kilk/05/03362 – Yearling. Abbotstown animal.

Findings

Articular cartilage showed no specific change. Trabeculae were much thicker than some of the other samples, e.g. Kilk/05/02909, and distribution appeared normal. The epiphyseal and metaphyseal cavities contained mainly adipose tissue. The physeal growth plate was of regular thickness appearance and had well-formed primary trabeculae.

Conclusion

No specific changes.

Kilk/05/03425 – Yearling. Abbotstown animal.

Findings

Articular cartilage of proximal metatarsus had uneven surface with breaks and an overlying adherent flap of cartilage. Remnants of inflammatory exudate adherent to part of articular surface. Trabeculae in epiphysis and metaphysis thick and quite densely packed in areas. Suggestion of compression of trabeculae in some metaphysis sections. The physeal growth plate (radius) was of irregular thickness with occasional fingers of apparently non-mineralised cartilage extending into the metaphysis. Metaphyseal trabeculae were lined by prominent osteoblasts in some areas.

Conclusion

Osteochondrosis. It is likely that this is an incidental finding in the context of the present investigations. Multiple factors are considered to be involved in osteochondrosis, including genetic predisposition, rapid growth, degree of physical activity, gender, and nutritional status.

Kilk/05/02654 – 1 Year old.

Findings

Articular cartilage showed no specific change. Epiphyseal and metaphyseal trabecular density low, and trabecular structure thin, indicating osteoporosis. Trabecular spaces contained largely adipose tissue. Only residual physeal growth plate present in sections examined.

Conclusion

Osteoporosis. Inadequate growth plate present in sections examined to comment on bone growth.

Kilk/05/03479 – Cow.

Findings

Trabecular density quite light indicating a degree of osteopenia. This would not be an unexpected finding in aged cows. Osteocyte numbers and distribution, and cement line patterns, in bony tissue appeared normal. Osteonal size and distribution in cortical bone was regular.

Conclusion

No specific findings.

Kilk/05/03811 – Cow.

Findings

Articular cartilage and bone structure appeared normal. Osteocyte numbers and distribution, and cement line patterns, were regular.

Conclusion

No specific findings.

Kilk/05/03962 – 4-month old calf.

Findings

Articular cartilage and bone structure appeared normal. Intertrabecular spaces in epiphysis and metaphysis contained mainly adipose tissue showing serous atrophy. Trabecular structure and distribution was thin indicating osteoporosis. There were multiple pieces of apparently unmineralised cartilage in the trabecular spaces. The physis showed almost complete cessation of growth along the metaphyseal border. There were areas of retention of apparently unmineralised cartilage well into the metaphysis.

Conclusion

Abnormal endochondral ossification with resultant retarded bone growth and osteoporosis.

Overall Conclusions

The most significant finding was of abnormal endochondral ossification characterised by premature growth plate closure and osteopenia or osteoporosis in five growing animals between about four and 18 months of age. On their own, these are not diagnostically specific findings. Premature growth plate closure is a microscopic finding consistent with a clinical history of growth retardation. Among the possible factors affecting bone growth in general are: nutritional imbalances (protein, energy, minerals, vitamins), enteric conditions affecting nutrient absorption, some acute or chronic bacterial infections, antibiotics of the tetracycline family, and virus infections such as BVD. Osteoporosis is a common lesion in farm animals – and the causes are generally nutritional in origin (Jubb, Kennedy and Palmer, 1993¹⁰).

Chronic enteric and respiratory infections are known to be common causes of growth retardation in humans and animals.¹² The effects are mediated by a negative impact on nutrient uptake and metabolism - as well as through direct influences of the inflammatory process on bone growth. Toxins produced by *Pasteurella* species for example - which are commonly involved in respiratory tract infections - are capable of inhibiting bone growth leading to stunting in farm animals¹³.

There is a well-defined history of quite widespread enteric and respiratory conditions in calves and growing cattle on this farm (Main Report – Part 2). Of the five cases above with signs of retarded bone growth, lesions of chronic bronchopneumonia were found in three animals, and four had lesions of enteropathy (Main Report – Appendix 5). These were chronic lesions and the associated conditions may have had a negative impact on bone growth. *Pasteurella* species were among the isolates from affected animals.

Tetracycline antibiotics are known to have a negative impact on bone growth in humans and animals¹⁴, and because of this their use is contraindicated in human newborns. However, it is not possible to determine to what extent, if any, they may have contributed to the growth retardation problems on this farm. Although there is evidence of their extensive usage in the perinatal period, we are not aware of any reports in the veterinary literature suggesting they could cause clinical growth retardation in cattle at normal dose rates.

Although congenital BVD virus infection is known to cause growth retardation – and BVD-infected animals have been identified on this farm – none of the five animals above were virus-positive. In addition, the characteristic growth arrest lines of BVD infection were not seen in any of the slides examined.

Bone sections from two 10-day old calves showed some signs of altered endochondral ossification. These may be secondary to intercurrent illness. One calf had cryptosporidiosis with associated toxæmia and dehydration. The second had a septicaemia.

There were no specific findings on examination of sections from the foetus, the three cows, or one of the two Abbotstown yearlings. The second Abbotstown yearling had lesions of osteochondrosis. As an isolated finding, this is unlikely to be of specific significance in the context of the present investigations of widespread growth retardation.

¹² Symposium: Causes and Etiology of Stunting. J Nutr. 1999 Feb;129(2S Suppl):529S-530S.

¹³ Ackerman *et al.*, Am. J. Vet. Res. **57**, 848-851; 1996.

¹⁴ Niebyl JR. Antibiotics and other anti-infective agents in pregnancy and lactation. Am J Perinatol. 2003 Nov;20(8):405-14.

In so far as can be determined from demineralised sections, none of the bone sections examined from these 13 animals showed microscopic lesions consistent with a diagnosis of fluoride toxicity. Osteocytes were regularly distributed and the appearance of cement lines did not suggest excessive remodelling. There was no evidence of hyperostosis, osteophytosis, or disruption of the normal cortical bone structure. Although osteoporosis was observed in some cases, this is not specific to fluorosis and is not an uncommon finding in farm animals. It can be caused by a variety of factors affecting nutrient intake and balance (Jubb, Kennedy and Palmer, 1993¹⁰). The possibility that other microscopic changes of fluorosis might be found in un-demineralised bone sections cannot be ruled out. However, given that fluoride concentrations in all bone samples analysed from this farm have been below the generally-accepted minimum concentration for the occurrence of microscopic lesions of osteofluorosis, the probability of this is considered to be very low.

CVRL Pathology Division

Appendix 4 – Submissions from the Brennan farm to Kilkenny RVL pre-2003.

Year	Blood samples	Clinpath (eg faeces, milk samples)	Carcass	Pertinent finding
1993	0	0	0	
1994	1	0	1	Lepto hardjo (foetus)
1995	0	0	0	
1996	0	0	0	
1997	8	0	0	Low iodine status
1998	4	0	0	Marginal GPX
1999	0	0	2	<i>Past. multocida</i> isolated from lung of calf with bloat.
2000	0	1	3	Salmonella Dublin (foetus); Crypto sporidiosis (calf). Enteritis (calf).
2001	0	0	0	
2002	0	15	1	Rota virus (calf & Faeces) Mastitis pathogens (esp. Staph aureus)

Appendix 5 – Post-mortem submissions from the Brennan farm to Kilkenny RVL
2003 to December 2005.

2003

Date	Lab Ref	Animal type	History	Main Findings
20 Jan 03	Kilk/03/0476	Foetus, 8 mth	Abortion	No diagnosis
10 Apr 03	Kilk/03/02968	4 heifers, 34 - 36 mths	Routine slaughter	No gross lesions
15/04/03	Kilk/03/03081	Calf, 2 weeks	Stomach upset, rapid death	Colibacillosis; inadequate colostrum.
17/04/03	Kilk/03/03136	Weanling	Sudden death	Intestinal torsion.
06/05/03	Kilk/03/03392	Yearling	Recumbent at turnout	Haemorrhagic enteritis. <i>S. dublin</i> , <i>Clost. sordelli</i> isolated.
13/05/03	Kilk/03/03546	Cow (10yr)	Recumbent due to accident.	Emaciation. Hepatobiliary fibrosis ¹
15/07/03	Kilk/03/04287	Cow (10yr)	Milk drop.	bacterial abomasitis. Hepatobiliary fibrosis.
01/09/03	Kilk/03/05307	foetus		No significant findings

1 – indicative of fluke infestation.

2004

Date	Lab Ref	Animal type	History	Findings
02/01/04	Kilk/04/0025	Foetus, 8 mths	Aborted	No significant findings
05/01/04	Kilk/04/0069	Foetus, 7 mths	Aborted	No significant findings
23/02/04	Kilk/04/01243	Calf, 10 days	Not drinking, emaciated.	Cobalt toxicity (drenched). Gram negative septicaemia.
29/03/04	Kilk/04/01926	Calf 1 wk.	Enteritis	<i>Salmonella dublin</i> isolated.
31/03/04	Kilk/04/01532	Calf, 2 wks	Recumbent.	Congenital BVD infection.
06/04/04	Kilk/04/02333	Foetus, 8m	Aborted.	No diagnosis.
13/04/04	Kilk/04/02567	Calf, 2 mth	Non-responsive pneumonia.	Peritonitis.
23/04/04	Kilk/04/02649	Calf, 10 days	Weak previous day.	Colisepticaemia. Low ZST. Cardiac defect.
19/05/04	Kilk/04/03064	Calf, 2 wks	Recumbent	Severe pneumonia. <i>H somnus</i> isolated.
9/07/04	Kilk/04/03603	Calf 3 months.		Encephalitis (brain abscess)

2005

Date	Lab. Ref.	Type	History	Main Findings
10/01/05	Kilk/05/00173	Aged cow.	Prolapsed vagina.	Chronic fibrosing hepatitis ¹
21/02/05	Kilk/05/01296	Foetus x 2	Abortion	No significant findings
24/02/05	Kilk/05/01386	Calf 10-day old	Recumbency	Meningitis, peritonitis, pneumonia. Hypogammaglobulinaemia.
8/04/05	Kilk/05/02420	Aged suckler cow. Pregnant	Recumbent, emaciated	Severe hepatobiliary fibrosis. Gastrointestinal parasitism.
15/04/05	Kilk/05/02567	Calf 6 weeks	Housed	Severe chronic bronchopneumonia (pasteurellosis).
20/04/05	Kilk/05/02654	Weanling 8-11m	Elective PM.	Enteropathy. Possible sequel to enteritis.
3/05/05	Kilk/05/02860	calf 2 wks.	Recumbent	Colibacillosis. ZST 19.
5/05/05	Kilk/05/02909	Yearling 14m. Housed.	Elective PM.	Chronic bronchopneumonia. Joint lesions. Enteropathy. Gastrointestinal parasitism.
6/05/05	Kilk/05/02936	Calf 10 days.		Meningitis. Hypogammaglobulinaemia.
18/05/05	Kilk/05/03107	Yearling 13m.	Elective PM. Home-bred. Undersize.	Focal bronchopneumonia and bronchitis. Enteropathy. Possible sequel to enteritis..
9/06/05	Kilk/05/03362	Yearling 16 months.	Elective PM. (Abbotstown)	Mild enteropathy. possible sequel to enteritis.
14/06/05	Kilk/05/03425	Yearling 16 months.	Elective PM. (Abbotstown)	Mild enteropathy. Possible sequel to enteritis.
20/06/05	Kilk/05/03479	Aged cow.	Suspect tetany.	Advanced pm changes.
12/07/05	Kilk/05/03671	Aged cow.	Abattoir slaughter.	Severe chronic fibrosing hepatitis. Chronic bronchitis.
2/08/05	Kilk/05/03811	Aged cow		Chronic fibrosing hepatitis. Suppurative mastitis. Gastrointestinal parasitism.
12/08/05	Kilk/05/03897	Calf 4 weeks.	Ill-thrift.	Systemic bacterial infection, bronchopneumonia. Isolates include <i>Mannheimia hemolytica</i> and <i>Salmonella dublin</i> .
19/08/05	Kilk/05/03962	Weanling 4 m/o	Elective PM.	Chronic bronchopneumonia. Enteropathy. <i>Pasteurella</i> and <i>Salmonella dublin</i> isolated. Fungal oesophagitis.
19/08/05	Kilk/05/03963	Weanling 4m	Ill-thrift.	Chronic interstitial pneumonia with bronchitis and abscessation. No isolates.
12/09/05	Kilk/05/04141	Weanling 6m	Ill-thrift.	Severe fibrinous bronchopneumonia. <i>Mannheimia hemolytica</i> isolated.
3/11/05	Kilk/05/04807	Weanling 6 months.	Ill thrift	Focal bronchopneumonia. Enteropathy. Arteriolar wall mineralisation.
18/11/05	Kilk/05/05051	Foetus	Abortion.	Protozoal-type encephalitis.
14/12/05	Kilk/05/05543	Weanling 9months	Ill thrift	Chronic bronchopneumonia consistent with pasteurellosis
16/12/05	Kilk/05/05613	Aged cow	Thin cow. abattoir slaughter.	Chronic fibrosing hepatitis.

1 – Chronic fibrosing hepatitis is consistent with damage due to liver fluke.

Appendix 6 – Histopathological Findings on Post-Mortem examinations in 2005.

Kilk/05/00173

Chronic fibrosing hepatitis, suggestive of fluke damage, was present in this 10 year old cow which also had a vaginal fibroma.

Sections of adrenal gland, lung, trachea, spleen, heart, uterus and kidney were also examined and did not contain significant histological changes.

Kilk/05/01386

This 10 day old calf had severe pyogranulomatous and necrotising meningitis, peritonitis and necrotising interstitial pneumonia. Cryptosporidiosis predominated in the small intestine and fungal gastritis in the rumen and abomasum. Neutrophilic enteritis was present in the large intestine and fat depletion in the serosal layer.

Sections of kidney, thyroid, lymphoid tissue, heart, liver, tongue and oesophagus were also examined and did not contain significant histological changes.

Kilk/05/02420

This cow had severe fibrosing hepatitis suggestive of liver damage by fluke. The animal also had evidence of gastrointestinal parasitism; nematodes were observed in the abomasum and small intestine. Occasional ciliate protozoa were seen in the rumen. Mineralisation of alveolar walls was present in the lung, the cause and significance of which are uncertain.

Tissue sections of the central nervous system (CNS), lymphoreticular system, endocrine system, urinary system, heart, pancreas and skin were also examined and did not contain significant histological changes.

Kilk/05/02445

There were no significant histopathological findings in the tissues of this foetus which comprised sections of liver, placenta, lymph node, lung, thyroid, heart, brain and kidney.

Kilk/05/02567

This calf had chronic bronchopneumonia and bronchitis. The foci of necrotic macrophages within the lung were reminiscent of those seen in *Pasteurella* pneumonia. The kidney contained changes suggestive of tubular nephrosis, the cause of which is unknown. The principal causes of acute tubular necrosis are ischaemia (for example, due to shock) and nephrotoxins (for example, nephrotoxic antibiotics such as tetracyclines).

Sections of liver, spleen, stomach and trachea were also examined and did not contain significant histological changes.

Kilk/05/02654

This one year old heifer had evidence to suggest the presence of an enteropathy (characterised by villous stunting), the cause of which was not apparent. Other minor histopathological findings included occasional clusters of coccidia in the large intestine, ciliated protozoa in forestomachs and occasional intraepithelial pustules in the rumen.

Tissue sections of the CNS, lymphoreticular system, endocrine system, urinary system, reproductive system, respiratory system, liver, muscle, heart, pancreas and skin were also examined and did not contain significant histological changes.

Kilk/05/02860

Sections of brain, liver, lung and kidney were submitted for histopathological examination and contained no significant histopathological findings.

Kilk/05/02909

This yearling bullock had chronic bronchopneumonia with bronchitis and lymphoid hyperplasia of bronchial associated lymphoid tissue (BALT). There was evidence of enteropathy (villous stunting and fusion) in the small intestine. A heavy focal aggregate of coccidia was seen in one section of the small intestine while some nematodes were seen in another. Occasional ciliate protozoa were observed in the GIT. This animal also had ringworm and mild interstitial myositis of unknown origin.

Tissue sections of the CNS, lymphoreticular system, endocrine system, urinary system, heart, testis, liver and pancreas were also examined and did not contain significant histological changes.

Kilk/05/03107

In the small intestine of this 13 month bullock with a history of illthrift, there was some suggestion of villous atrophy and fusion indicative of enteropathy, the cause of which was not evident. Ciliate protozoa were present throughout the GIT. This animal also had focal bronchopneumonia and bronchitis.

Tissue sections of the CNS, lymphoreticular system, endocrine system, urinary system, liver, heart, skin, genital system and muscle were also examined and did not contain significant histological changes.

Kilk/05/03362

There were no significant histopathological findings in the tissues submitted from this 15 month old animal. The sections examined comprised CNS, lymphoreticular system, endocrine system, urinary system, respiratory system, heart, skin, alimentary system (including pancreas and liver) and muscle. Scattered ciliate protozoa were seen in the rumen and abomasum.

Kilk/05/03425

The main histopathological findings were confined to the gastro-intestinal tract of this 15 month old animal with a history of illthrift. Despite autolysis of the mucosal layer of the small intestine, villi appeared blunted, suggestive of villous atrophy and enteropathy, the cause of which was not evident. Some ciliate protozoa were present in the lumen of the forestomachs.

Tissue sections of the CNS, lymphoreticular system, endocrine system, urinary system, respiratory system, genital system, heart, liver, pancreas, skin and muscle were also examined and did not contain significant histological changes.

Kilk/05/03479

The tissues submitted from this 7 year old cow, with a history of tetany, were very autolysed and comprised brain, mammary gland, liver, kidney, heart, muscle and lung. No significant histological changes were observed apart from some moderate portal tract fibrosis and bile duct hyperplasia in the liver.

Kilk/05/03671

This cow, with a history of having an inconclusive TB test result, had severe chronic fibrosing hepatitis, suggestive of damage to the liver by fluke. The animal also had chronic bronchitis/bronchiolitis with focal calcification of alveolar walls and mild rumenitis. Ciliate protozoa were present in sections of rumen and large intestine. The skin lesion seen on the dorsal fetlock at PM represented an ulcer containing foreign-body vegetable material.

Tissue sections of the CNS, lymphoreticular system, endocrine system, intestines, urinary system, heart and muscle were also examined and did not contain significant histological changes.

Kilk/05/03811

This cow had severe suppurative mastitis. Acute and chronic hepatitis were seen in the liver, most likely representing two separate conditions. The acute hepatitis may have been due to septicaemia (possibly originating from the mastitis). The severe fibrosing hepatitis is suggestive of previous liver damage by fluke. Gastrointestinal parasitism was also evident with occasional nematodes in the abomasum and coccidia in the large intestine. Changes suggestive of enteropathy were present in the small intestine (villous stunting and crypt abscesses), the cause of which was not evident. Multifocal intraepithelial pustules were seen in the oesophagus, the cause of which was not clear.

Tissue sections of kidney, pancreas, endocrine system, muscle, skin and respiratory system were also examined and did not contain significant histological changes. The sections of lymphoreticular tissue (lymph nodes) appeared normal apart from one lymph node in which the paracortex and medullary cords appeared depleted.

Kilk/05/03897

This 4 month old calf which died on-farm had necrotising bronchopneumonia associated with Gram negative bacteria. Interstitial changes were also seen in lung sections as well as calcification of artery walls, the cause of which is unknown. This calf also had pyogranulomatous lymphadenitis associated with Gram negative bacteria, affecting intestinal lymph nodes, and multifocal hepatitis, the appearance of which was consistent with that seen in Gram negative septicaemias. Although the sections of intestinal tract were autolysed, Gram negative bacterial colonies and crypt abscesses were present in the mucosal layer. Fat depletion was evident in this animal.

Tissue sections of the CNS, endocrine system, skin and muscle were also examined and did not contain significant histological changes.

Kilk/05/03962

The main histopathological findings were in the gastro-intestinal tract and lung of this 4 month old calf with a history of illthrift. Changes suggestive of enteropathy were present in the small intestine (villous stunting and crypt abscesses), the cause of which was not evident. Occasional coccidial structures were present in the ileum and rectum. Focal ulceration and intraepithelial pustules were seen in the omasum which also contained scattered ciliate protozoa. There was fungal invasion of the oesophageal mucosa (possibly due to antibiotic therapy). This calf also had evidence of chronic bronchopneumonia with bronchitis and lymphoid hyperplasia, the appearance of which was consistent with enzootic pneumonia of calves.

Tissue sections of the lymphoreticular system, CNS, endocrine system, skin, genital system, heart, kidney, liver, pancreas and muscle were also examined and did not contain significant histological changes.

Kilk/05/03963

The main histopathological findings were in the lung and liver of this 4 month old calf with a history of bloat. This calf had evidence of chronic interstitial pneumonia with bronchitis, secondary bacterial infection and abscessation. While the cause of the interstitial changes in lung was not evident, previous viral damage cannot be ruled out. The liver contained bridging portal tract fibrosis and periacinar congestion (possible consequences of the bloat).

Tissue sections of the spleen, kidney and CNS were also examined and did not contain significant histological changes.

Kilk/05/04807

This 6 month calf had focal bronchopneumonia, hepatopathy (characterised by hepatic atrophy, portal tract fibrosis and marked arteriolar proliferation) and enteropathy (characterised by villous atrophy). According to the gross description of the liver, the hepatopathy observed was localised and was “3cm in width extending the length of the margin of the left lobe of the liver”. The cause of this localised hepatopathy is unknown. Occasional coccidia were present in the small intestine. Mineralisation was also seen in this calf, in arteries/arterioles of the liver, lung and bladder and focal alveolar wall deposition. This mineralisation is most likely metastatic in origin.

Tissue sections of the lymphoreticular system, CNS, endocrine system, skin, heart and pancreas were also examined and did not contain significant histological changes.

Kilk/05/04141

This 7 month old calf had fibrinous pleurisy and severe pneumonia characterised by the infiltrates of necrotic macrophages associated with bacteria. The appearance of this pneumonia was consistent with that caused by *Manheimia* species. There was also evidence of rumenitis, the cause of which was not evident. Other observations included apparent tonsillar depletion with foci of mineralisation within the tonsillar epithelial lining (the cause and significance of which are unknown) and muscle fibre atrophy.

Tissue sections of the CNS, endocrine system, kidney and heart were also examined and did not contain significant histological changes. The sections of pancreas and intestine examined were autolysed.

Kilk/05/05051

This 7 month old foetus had evidence of non-suppurative encephalitis. Although protozoal cysts were not observed in the brain sections examined, the changes in the brain are consistent with those seen in protozoal encephalitis. Myocarditis and hepatitis were also observed in this foetus, the causes of which were not evident.

Tissue sections of kidney, thymus and lung were also examined and did not contain significant histological changes.

Kilk/05/05613

This old thin cow had renal amyloidosis and chronic fibrosing hepatitis. Changes seen in the lymph node were consistent with fatty infiltration of the node, the significance of which is unknown. Histopathological changes were not observed in the other tissues submitted i.e. lung and adrenal gland.

CVRL Pathology Division

Appendix 7 – Teagasc Nutrition Report March 2004

Dan Brennan, The Spa, Drumgoole, Castlecomer, Co. Kilkenny Nutritional Status of the Farm, March 2004

Prepared by Dr. Siobhán Kavanagh, Nutrition Specialist, Teagasc Kildalton
& Mr. Michael Cody, Dairy Adviser, Teagasc Kilkenny

1. Silage quality

A single core from any pit or baled silage is not adequate to critically analyse a silage but in general silage quality on the farm is as to be expected given silage making conditions in 2003. In general silage quality on the farm is representative of the quality of silage on many farms this year. Dry matter, preservation and crude protein being good and digestibility moderate. Visual assessment of the silage would agree with laboratory results. The baled silage analysis was based on bulked cores from eight bales and the pit silage analysis was also based on multiple cores from the part of the pit expected to be used during the feeding trial.

2. Winter feeding programme 2003 – 2004

An assessment was made of the winter feeding programme on the farm in the winter of 2003-04. One of the objectives of the winter feeding programme in 2003-04 was to explore the possibility of a nutritional problem specific to home-grown forages.

a. Calves

Calves are reared as follows:

1. Calf is left with dam for about 4 – 7 days. If cow does not seem to have been drunk out, colostrum is offered by bottle and teat or calf is tubed if it will not drink.
2. Calves are offered 1.44 gals of milk for approximately 90 days before weaning.
3. Calves are offered a starter ration from 1-2 weeks old. Calves need to be offered *ad libitum* meals from 10 days old. They need to be eating at least 1.0 kg meals / hd / day at weaning. This should be gradually increased to 2.0 kg / head / day plus good hay or silage when calves are indoors.
4. It is important that when animals are turned out to grass that they are offered a good supply of fresh grass. It should be possible to phase out meals one month after turnout. This is reliant on good grassland management.

b. Weanlings

To compare home-grown and bought-in forages, one pen of animals was placed on each of two silages: home-grown pit silage or bought-in baled silage (See Table 1 below for analysis). Animals were weighed individually both at the beginning and end of the winter period. Animals were offered silage *ad libitum* + concentrates at 2.0 kg and 2.5 kg for the home-grown and bought-in silages, respectively. The concentrate consisted of a home mixed blend of barley (33.3%), citrus pulp (33.3%) and distiller grains (33.3%). Animals were also offered 50g / hd / day of Superchoice General Purpose Minerals (Glanbia). Overall crude protein content of the diets was 15.1% and 14.7% for home-grown and bought-in silages respectively (Table 1).

Table 1. Feeding Weanling 2003 / 2004

	Home-grown pit silage (SE18302)	Bought-in baled silage (SE16311)
Silage quality		
DM%	24.8	37.4
pH	3.7	4.3
Ammonia N	6.2	6.2
Crude protein %	14.2	13.1
DMD %	68.4	67.5
Concentrates fed kg*	2.0	2.5
Expected live weight gain kg / day	0.60	0.65
Actual performance		
Weight kg		
Initial	222	216
Day 92	239	242
Day 142	253	262
Average daily gain kg		
Day 0-92	0.20	0.30
Day 92-142	0.54	0.65
Day 0-142	0.27	0.34

*Initially it had been expected that the quality of the bought-in silage (intake factor and digestibility) would be significantly poorer than the home-grown silage and for this reason these animals were offered 0.50 kg additional concentrates.

The performance of these weanlings was unacceptably low at 0.2 – 0.3 kg average daily gain (ADG) for the first 92 days. This is significantly poorer than the target of 0.6 kg ADG. However, for the second period Day 92 – 142, performance was reasonably good and as expected for weanlings on silage and meals. There is no clear explanation for this improvement in performance over the second period. Overall performance was poor. In general there was considerable variation in the weight gains of animals within pens.

Younger weanlings (c. 148 kg LW) were offered 3 kg meals and hay. Average daily gain of these animals was -0.02 kg over a 3 month period. The expected gain for these animals is 0.80 kg ADG. Again these animals performed substantially better for the next 50 days (ADG = 0.56 kg), even though concentrate feeding levels were reduced to 1 kg / head / day. Other weanlings on 3 kg meals and 0.5 gallons milk + hay had a daily gain of 0.09 kg (Day 0 – 92) and 0.53 for Day 92 – 142 when meal levels were reduced to 1kg. In both cases over the whole period (142 days) gain was unacceptably low.

c. Finishing cattle

Seven friesian steers were purchased in December 2003 for winter finishing. These animals were offered 4.5 kg of concentrates and 12 kg fodder beet and bought-in baled silage *ad libitum*. Animals were offered 100 g / hd / day of a fodder beet mineral. Expected dry matter intake of these animals is 10.8 kg DM (1.95% of BW). Based on this intake, a daily gain of 1.0 kg / hd / day would be expected. These animals performed very poorly and achieved a daily gain of 0.43 kg / hd / day. Daily gain for the second period (Day 92 – 142) was moderate (ADG = 0.79 kg).

Similarly a group of home reared animals were offered the same diet but with home-grown silage. Again expected gain was 1.0 kg / hd / day but recorded gain over a 92 day period was 0.27 kg per day.

The improved live weight gain in all groups of animals in the second measurement period (Day 92 – 142) is difficult to explain, if we are to assume that management and feeding regimes were not changed during this period. But 50 days is a relatively short period on which to draw conclusions and the total period of 142 days is more representative. It would be recommended however that live weights be continued to be monitored to establish the true extent of the problem.

Observations from both the weanlings and finishing cattle in the winter of 2003-04 would suggest that the problem is not specific to the home-grown forages or home-reared animals. Does this rule out a genetic problem in the herd?

d. Dry cows

Dry dairy cows were offered *ad libitum* access to silage (68 DMD) and were offered a pre-calver mineral (Glanbia) for 4 – 6 weeks before calving. Cow condition before calving was moderate (Body condition score (BCS) = 2.70). This silage was deficient in energy for thin cows in late pregnancy. Approx. 2 kg concentrates / day would be required for an ADG of 0.90 kg.

Sucklers were offered 74 DMD silage during the winter period and likewise were in poor condition (BCS = 2.30).

e. Milking cows

Milking cows were milking very poorly at 11.5 kg per cow per day. Low milk protein is also a problem on this farm and this is generally an energy deficit problem i.e. underfeeding or incorrectly balanced diet. It may be expected that peak yield in these animals will be low. These animals were being offered *ad libitum* silage and 5 kg of a concentrate (Ultra Dairy 18%, Glanbia). Based on this feeding rate and the genetic potential of the animals, cows should milk c. 16 - 18 kg / day.

3. Mineral nutrition

Castlecomer soil type is not ideal in terms of mineral balance. It is a known fact that there are numerous mineral interactions in soils of this type and a number of trace elements are likely to be tied up and not available to the animal. Silage and soil analysis showed low levels of copper, selenium, zinc, phosphorous and high levels of molybdenum, aluminium, iron and sulphur. Blood levels of copper, T4, glutathione peroxidase, zinc, calcium, phosphorous and magnesium were normal. Please find attached blood, forage and soil mineral analysis for the farm.

Phosphorus is lacking in the soil (5.75 mg/l) and forage (2.8 g/kg) samples taken. However blood levels are normal and in general supplementation rates are adequate for milking cows and finishing cattle but somewhat low for weanlings and dry cows (Table 2). It is unlikely that this deficit is sufficient to explain the severe stunting.

Table 2. Calcium and Phosphorous Balance for Winter Feeding for Livestock on the Farm¹

	Ca g/kg DM	Phos g/kg DM
Weanlings	8.3	3.7
Dry cows	6.9	3.2
Milking cows	8.0	4.5
Finishing cattle	7.3	4.6

¹Based on forage mineral analysis and mineral supplementation

Deficiency of phosphorous is rare on most well-run farms. The main signs of phosphorous deficiency are infertility, agalactia, anorexia, dryness of skin, lameness, stiffness, inability to stand, lack of growth or weight gain, pica, reluctance to move, underweight, poor condition, ill-thrift and weight loss. While animals on this farm exhibit some of the symptoms mentioned above including ill-thrift, weight loss and poor condition, there are many that they do not exhibit.

Mineral supplementation is essential to overcome deficiencies in the soil and forage. It is important that all animals receive a mineral supplement during the indoors period and in some cases at grass (e.g. young animals). Routinely pre-calver minerals should be offered to all cows. Pre-calver minerals have been offered on an ad hoc basis in the past. However, during the winter of 2003 / 2004 minerals were offered to cows pre-calving and post-calving, as per Teagasc recommendations. Likewise, drystock have received mineral supplementation according to the Teagasc recommendations in 2003-04.

Minerals need to be eliminated from the equation totally by adopting a mineral feeding programme as per the Teagasc recommendations.

4. Other comments

a. Intake

Inappetance does not appear to be a problem on the farm. Occasional intake measurements over the past 2 years would suggest that intake is not an issue on the farm.

b. Mycotoxins

The risk of mycotoxins in the feed was assessed in the Autumn 2003. Samples of feed analysed included pit grass silage, baled grass silage, rolled barley, citrus pulp, distillers grains. Please find attached these results. Expert opinion would suggest that while mycotoxins are present these are not at levels that would cause any negative effect on animal performance.

c. General Management

The ill-thrift that is observed on this farm could be related to a number of issues including nutrition, housing, management and environment. Forage quality is moderate, housing is reasonable and management on the farm is adequate. These three factors alone are not sufficient to explain the serious nature of the stunting and ill-thrift present on this farm.

d. Feeding Management

Weanlings and finishing cattle are offered silage in an easy feed system on a daily basis. Stale feed is not removed from in front of animals on a routine basis. It is recommended that animals are not expected to clean up stale feed regularly as this can have a negative effect on intake and live weight gain. Animals are offered meals twice a day and minerals are sprinkled on top of silage. Both meals and minerals are measured out.

e. Feed storage

Coarse ration for the weanlings and finishing cattle is stored alongside a pelleted ration for the milking cows. This store is not bird or vermin proofed but it is reasonably enclosed. While ingredients for the coarse ration are purchased individually, these are dumped into a trailer for transport and are "semi" mixed. Further mixing is done on farm with a shovel. There are no signs of chemicals occupying the same space as the meals.

f. Water supply

Examination of the water troughs in the wintering shed would suggest that there is a clean supply of water available to animals. The quality of the drinking water is being investigated.

Report on Feeding Trial 2004 / 2005

Dan Brennan, The Spa, Drumgoole, Castlecomer, Co. Kilkenny

Personnel

Michael Cody (Teagasc), Donal Toolan (DAF), Jimmy McLaughlin (DAF), Siobhán Kavanagh (Teagasc)

Introduction

Problems of ill-thrift and stunting of cattle on the Index farm, at Drumgoole, have occurred since the 1990s. Similar problems have been reported from two adjoining farms but have not been investigated in detail. Intensive investigations by Teagasc and the Regional Veterinary Laboratory, Kilkenny have been continuing since January 2003.

A preliminary study in 2003/2004 showed weanling performance was unacceptably low at 0.2 – 0.3 kg average daily gain (ADG) for the first 92 days of the winter. However, for the second period Day 92 – 142, performance was reasonably good (0.55 – 0.65 kg ADG). Overall performance was poor.

As a result of the preliminary findings of that study, a number of objectives were set for the current study:

1. To quantify dry matter intake of the weanlings on the Index farm
2. To investigate the effect of source of silage on animal performance
3. To investigate the effect of location of housed animals on animal performance

Materials and Methods

Treatments

Four pens, each containing nine weanlings from the Index farm, matched as far as possible for age, weight, breed and sex were assigned at random to each of the four treatments. Two pens were on the Index farm (Drumgoole) and the other two on a farm at Cruttencrough, three miles away. There was no reported problem of ill-thrift on the Cruttencrough farm. All animals on the trial were reared on the Index Farm.

Treatment 1. Animals housed on the Index farm and offered the Index silage (II)

Treatment 2. Animals housed on the Index farm and offered Cruttencrough silage (IC)

Treatment 3. Animals housed on the Cruttencrough farm and offered the Index silage (CI)

Treatment 4. Animals housed on the Cruttencrough farm and offered Cruttencrough silage (CC)

Ten weanlings from Abbotstown farm were housed on the Index farm and offered the Index silage (ABB/I). These were not part of the randomised trial but were present for observation purposes to see how bought-in animals would perform, relative to home-reared stock.

Forages & Feeds

Silage from both sources (from the clamp on the Index farm and from baled bags from the Cruttenclough farm) were analysed for nutritive value - DMD, crude protein, pH & dry matter- prior to commencing the trial. The results of the initial analyses were used to calculate the concentrate supplementation required to achieve a live weight gain greater than 0.5 kg per day. The concentrate composition was 0.28 barley, 0.21 maize gluten feed, 0.186 maize distillers grains, 0.074 molasses, 0.073 palm kernel meal, 0.067 soya hulls, 0.05 beet pulp, 0.025 citrus pulp and 0.037 minerals + vitamins + fat.

Measurements

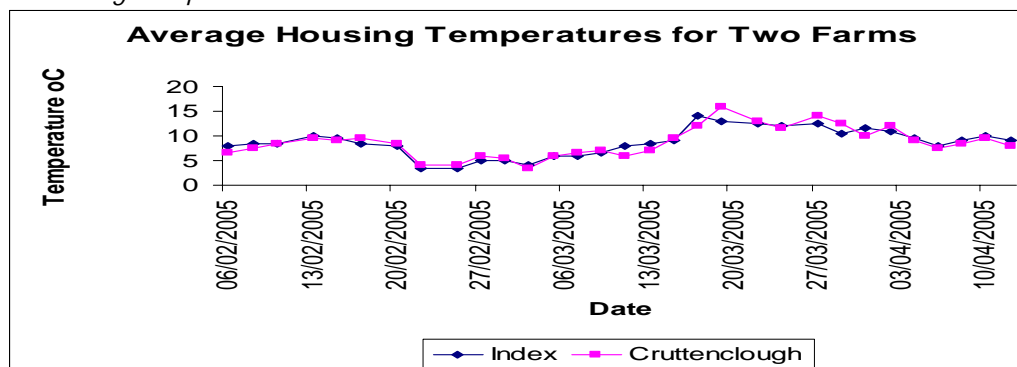
1. Animals were weighed at housing to allow randomisation of the animals based on weight and breed. The trial began on the 21st December. Animals were weighed on day 0 (21st December) on day 49, day 78 and day 112. Animals were weighed at similar times each day, after feeding.
2. Weanlings were fed on four days in the week (Monday, Wednesday, Friday and Saturday) using a mixer wagon. From mid January onwards animals were fed every second day.
3. Initially, forage and concentrates were weighed and dispensed from a mixer wagon but due to issues of calibration with such small quantities of concentrates, concentrates were weighed separately using a spring balance but added into the wagon and fed as a mixed feed. The weight of forage and concentrates offered to the animals and volume of water dispensed in each pen was recorded on each feeding day.
4. Unconsumed feed was gathered and weighed as required. This was recorded as 'refused'
5. The identity of any ill animals was recorded by the stockman.
6. On the day of feeding, maximum and minimum temperatures were recorded at both sites.
7. The weanlings were sampled prior to the commencement of the trial for serological evidence of BVD. No BVD virus positive animal was included in the pool of 36 weanlings to be allocated to the trial pens. Cattle reared on the Index Farm were vaccinated for RSV, Salmonella and Blackleg. Abbotstown cattle were vaccinated for Salmonella, IBR, PI3, RSV and Pasteurella, BVD and Blackleg.
8. An operative was contracted in from the Farm Relief Service. He carried out the feeding and management of the stock. He recorded the weights of silage and meals offered, weigh back of unconsumed feed, temperature and flowmeter output.

Results

Animal Health

One animal on IC was lame but was not treated. This animal was removed from the pen for a number of weeks. Three animals on II were treated for 3 days but were not removed from the pen, apart from one animal for 2 days. Ambient temperature is shown in Figure 1. The two sites matched one another over the period of the trial.

Figure 1. Housing Temperature



Forage Analysis

Table 1. Forage Analysis for the Winter 2004 / 2005

	Index Farm	Cruttencloagh farm
DM %	30.9	26.6
Crude protein %	12.9	15.6
DMD %	70	74
UFL	0.78	0.82
pH	4.7	4.1

Forage analysis is presented in Table 1. The original assumption was that silage was 66 DMD and 76 DMD, on the Index Farm and Cruttencloagh farm, respectively. Concentrate requirements were then calculated to be 2.5 kg and 1.0 kg as fed for the Index Farm and Cruttencloagh farm, respectively. This was based on the assumption that the energy requirement of 200 kg weanlings is approximately 3.7 UFL to achieve 0.6 kg ADG.

Measured average concentrate consumption was 2.4 kg as fed on the Index Silage and 1.0 kg as fed on the Cruttencloagh silage. Actual energy intake and predicted gain is presented in the table below (Table 2)

Table 2. An Estimation of Predicted Gain, based on forage analysis and expected energy intake

Treatment	Energy Intake UFL / day	Av. Live weight kg	Predicted Gain kg / day
II	5.1	229	0.80+
IC	4.1	203	0.6 – 0.7
CI	5.1	223	0.80+
CC	4.3	212	0.6 – 0.7
Abbotstown Cattle	7.0	370	0.80+

Intake

Dry matter intake

Dry matter intake was normal or above normal on all treatment pens and the Abbotstown cattle, ranging from 4.5 kg DM to 6.7 kg DM (Table 3). As a percentage of live weight it ranged from 2.24% to 2.71% of live weight. Generally, based on research work from Teagasc Grange, it is assumed that weanlings on grass silage + concentrates will consume 2.0 – 2.2% of body weight. Intake peaked in the period Day 49 - 78. This would not be unusual as there is a period of adjustment to a conserved forage diet.

Water intake is presented in Table 4. NRC (2001) guidelines on the water requirements of beef cattle over the winter period range from 16 – 22 litres / day. Water intake was similar for II, IC and CI. Intake on CC was exceptionally low at 1 litre / day. Was there a problem with the water meter on the CC treatment? As a general observation, water intake was lowest on the Cruttenclough silage on both sites.

Table 3. Dry Matter Intake (kg DM / day), as measured on a per pen basis

Treatment	Period			Overall % of LW
	Day0_49	Day49_78	Day78_112	
II	5.2	6.7	5.9	2.68
IC	4.5	5.2	4.8	2.40
CI	5.4	6.3	5.9	2.71
CC	4.8	5.5	5.0	2.37
Abbotstown Cattle	8.4	7.3	9.0	2.24

Water Intake

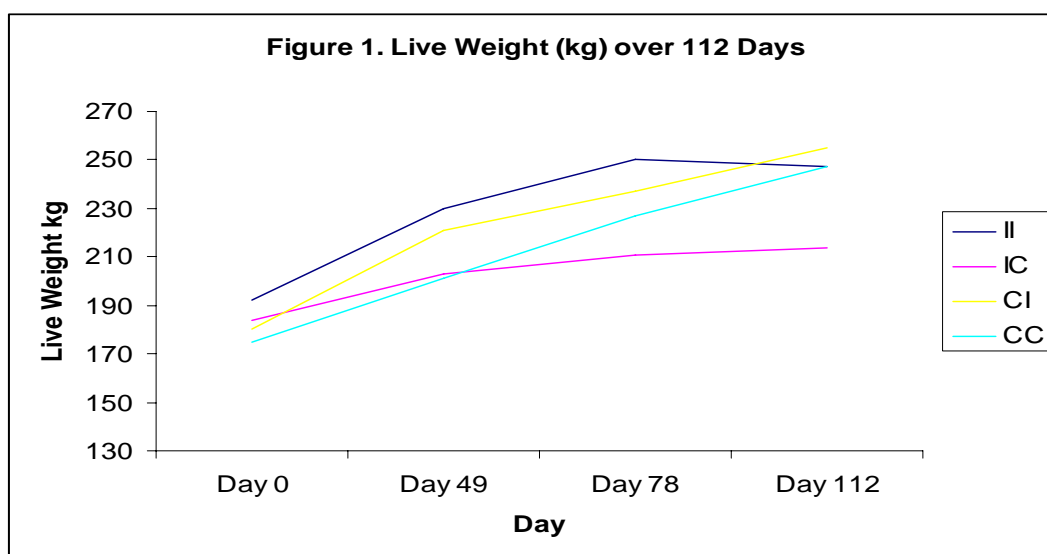
Table 4. Water Disappearance, as measured using a flow meter on a per pen basis

Treatment	Average Water Disappearance litres / animal / day	
	Day18_70	Day71_112
II	15	15
IC	9	9
CI	13	13
CC	1	1
Abbotstown Cattle	19	19

Performance

Figure 2 shows the live weight of the four groups over the 112 day period. It is evident from the diagram that on the Index farm treatment (II and IC), live weight increases for the first 70 days approximately but plateaus off thereafter, whereas on the Cruttenclough farm treatments (CI and CC),

liveweights continue to increase. Intake decreases slightly on all treatments during the period day 78 to 112 but this is insufficient to explain the observations on the Index Farm.



Live Weight Gain (kg/day)

Table 5. Live Weight Gain (kg/day) for the 4 Treatments and the Abbotstown Animals

Treatment	Period				
	0_49	49_78	78_112	Day 49_112	Day 0_112
II	0.78	0.71	-0.08	0.28	0.49
IC	0.40	0.36	0.10	0.22	0.27
CI	0.84	0.5	0.53	0.54	0.67
CC	0.54	0.88	0.61	0.73	0.65
Abbotstown Cattle	0.66	0.30	-0.48	-0.13	0.21

The II treatment showed good gains until day 78 but from 78 to 112 declined dramatically and was negative for this period. Overall daily gain for the whole trial on II was 0.49 kg / day. Animals on the IC treatments had poor performance from day one. Overall gains were 0.27 kg / day for 112 days. Animals on CI and CC showed good average gains for the overall period at 0.67 and 0.65 kg / day. Performance on the CC treatment dropped in the period 78_112 slightly but this of little consequence. Abbotstown animals performed well for the first 49 days but subsequently fell off and ended up with live weight loss for the period 78_112. Overall gain of these animals was poor, considering the quality of the Abbotstown animals presented on the farm on Day 1.

Least square means and statistical analysis is presented in Table 6. Only the main effects of location and silage type are presented. There was no interaction between location and silage type, apart from the gain in period 49_78. Silage type had no significant effect on weight gain apart from the period 0_49 when animals on the Index silage performed significantly better than the Cruttenclough silage. Location had a significant effect on weight gain in every period, apart from the period 0_49, with animals on the Cruttenclough farm performing significantly better than on the Index farm.

Table 6. Least Square Means and Statistical Analysis of the Location and Silage Effects on Live Weight Gain (kg / day)

	<u>Location Effect</u>		<u>Silage Effect</u>		<u>P-value</u>	
	Index	Crutten clough	Index	Crutten clough	Location	Silage Type
Weight Gain						
0_49	0.59	0.69	0.80	0.47	0.27	0.001***
49_78	0.49	0.72	0.63	0.58	0.03*	0.56
78_112	0.001	0.57	0.22	0.35	0.001***	0.09
0_112	0.39	0.66	0.58	0.47	0.001***	0.07

Feed Conversion Efficiency (FCE)

Table 7. Feed Conversion Efficiency for the 4 Treatments and the Abbotstown Animals, (kg DM feed / kg live weight gain)

Treatment	Feed Conversion Efficiency (kg DM / kg LW gain)		
	0_49	49_78	78_112
II	7.3	7.3	-83.75
IC	11.25	12.5	52
CI	6.67	9.82	11.89
CC	8.33	5.45	9.02
Abbotstown Cattle	12.7	24.3	-18.75

For periods 0_49 and 49_78, there were relatively small differences in feed conversion efficiency between the treatments (Table 7). However, for the period 78_112, FCE deteriorated dramatically on the II and IC treatments. The poor feed efficiency observed with the Abbotstown animals is consistent with that noticed with the treatment pens on the index farm (II and IC).

Summary of Results

- Intake was normal or above normal on both forages on both farms
- Water intake was variable across silages and sites. Intake was lowest on the CC treatment at 1 litre / animal / day and highest with the Abbotstown animals
- Live weight reached a plateau on the Index Farm after Day 78 approximately, regardless of forage used. Live weight continued to increase on the Cruttencrough Farm, regardless of forage quality
- Live weight gain was normal for periods 0_49 and 49_78 on the II, CI and CC treatments. Performance was poor on the IC treatment for all periods. Live weight gain declined to below normal levels on the Index Farm in the period 78_112, regardless of forage used. Live weight gain remained good on the CI and CC treatments for the period 78_112 and overall (0_112)
- Feed conversion efficiency was poorest on the Index Farm, regardless of forage used. The major deterioration occurred during the period 78_112.
- Abbotstown animals were 345 kg when moved onto the Index farm. These animals were only 370 kg after 112 days. This poor performance cannot be explained by feed intake (2.24% of body weight) or water intake.

Conclusion

Feed and water intake was normal on the Index Farm. Management on both farms was similar with an external operative doing the feeding and management. General observations on the housing at the Index farm by a Teagasc expert showed the housing facilities to be adequate for the weanlings. Feed intake, management and housing do not explain the ill-thrift observed on the Index farm over the winter of 2004 / 2005.

Appendix 9 – Antibiotic treatment courses for bought-in calves in 2005.

Details of purchased calves and their antibiotic treatments shortly after purchase.

Tag A	Tag B	Date of purchase	No. of Treatment courses excluding CTC and 4September	Treatment Course Commenced	Treatment Course Commenced	Treatment Course Commenced	Treatment Course Commenced	Treatment Course Commenced
2210732	403	21/03/2005	1	31/03/2005			CTC	
2210732	405	21/03/2005	1	31/03/2005			CTC	
1820262	369	23/03/2005	2	31/03/2005	08/04/2005		CTC	
1820262	370	23/03/2005	2	31/03/2005	08/04/2005		CTC	
1820262	372	23/03/2005	2	31/03/2005	08/04/2005		CTC	
1820262	374	23/03/2005	3	31/03/2005 ¹	08/04/2005	12/04/2005 ¹	CTC	
1820262	375	23/03/2005	2	31/03/2005	08/04/2005		CTC	
1820249	264	23/03/2005	1		03/04/2005		CTC	
1820249	265	23/03/2005	1		03/04/2005		CTC	
1820262	269	23/03/2005	0				CTC	
1820837	563	31/03/2005	4		08/04/2005	15/04/2005	CTC	24/04/2005
1820837	565	31/03/2005	3		08/04/2005	15/04/2005	CTC	24/04/2005
1820837	566	31/03/2005	3		08/04/2005	15/04/2005	CTC	24/04/2005
1820837	567	31/03/2005	4		08/04/2005	15/04/2005	CTC	24/04/2005
1820837	568	31/03/2005	3		08/04/2005		16/04/2005	24/04/2005
1820837	569	31/03/2005	4		08/04/2005		16/04/2005	24/04/2005
1820837	570	31/03/2005	4		08/04/2005		16/04/2005	24/04/2005
1919350	276	06/04/2005	1			15/04/2005	CTC	
1919350	289	06/04/2005	2			15/04/2005	CTC	24/04/2005
1919298	165	06/04/2005	1			15/04/2005	CTC	

1. Treatment dates for animals with a duplicated tag number whose identity has not been clarified. Entry is applied for each animal bearing that tag

Errata:

Page 7 Line 23, delete 'have been cleared of scrub'

Page 24, para 3 end of second line should read
"one aborted foetus, *seven* calves up to 6 months"

Page 53, Table.

Row 1, Column 4 add "abattoir examination".

Row 2, Column 3 (type) should read calf x2 (3-5d)

Row 2, Column 4 (history) add 'were classified as foetus/ stillbirth'

Row 6, Column 3 (type) replace with "11 mths old"