****** Review December 2009

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Johne's Disease

A brief review of the pathophysiology

I know we reviewed this during the meeting but I thought it might be useful to briefly refresh ourselves here…

Johne's diseases is the result of chronic infection with the bacterium *Mycobacterium avium* subspp. *paratuberculosis* (Map). 80% of infections are contracted during the first month of life (Sweeney 1996) and particularly risk is the first 24hrs (Hayton 2007) as a result of one or a combination of the following:

1. Ingestion of infected faeces.
2. Ingestion of infected colostrum or milk.
3. Transplacental infection.

The classic clinical picture of Johne's is of calves suckling infected cows, faecal contamination of the dams' teats by other infected cows or of units practising pooled colostrum (usually as an attempt to control rotavirus scour through the use of Rotavec vaccine).

Once the calf has ingested the Map bacteria it colonises lymphoid tissue within the intestines and slowly reproduces. Over a period of several years the intestines become thickened and absorption/digestion impaired, at this point the immune system becomes aware of the infection and a response stimulated (at this point we can detect the rise in antibodies). The peak incidence of clinical disease is thought to be three to five years of age (Caldow, Low et al. 2003) the peak incidence of detection by laboratory tests is also three to five years (Jubb and Glavin 1999) (hence showing the delay in our ability to detect disease).

The Pourquier milk test used by NML has a sensitivity of ~20% in subclinically infected animals and a specificity of nearly 100% (Collins, Wells et al. 2005) - this means that we can confidently identify ~20% of infected animals, however those that we do identify we can be nearly 100% confident are truly infected. This leads us to the peculiar situation that if we re-test a positive statistically we are more likely to get a negative!

Recent results for ******

The 'highlights' of the recent Johne's NML individual cow screen are given below in Table 1.

Table 1 - Recent Johne's NML results

Line Number	Ear tag	DOB	Current Lactation Number	Current Status[A]	DIM at latest MR	Latest category (17/11/09)	Previous category (19/02/2009)
1024	0	26/03/2007	1	Preg (25/8)	151	High	--
1083	281491302375	05/02/2007	1	Served	82	High	--
883	W8262/05290	24/09/1997	9	Preg (23/9)	145	High	High
755	282354300314	16/07/2005	1	Served	739	High	Low
894	281491102044	05/09/2006	1	Preg (21/6)	286	High	Low
369	281491102051	04/06/2003	4	Preg (23/7)	218	Medium	High
887	323404600321	09/09/2006	1	Preg (26/6)	302	Low	High
964	2256191509	11/04/2006	1	Preg (24/7)	515	Low	High

All of the results are interesting, however of particular interest are:

1. High results, the majority of which are heifers.
2. The medium and two lows which were previously high. How do we interpret these? Most likely these are the result of the strange testing situation we find ourselves in (detailed

[A] Preg = pregnant (date of service); Served = served, not confirmed pregnant; NS = not-served

earlier); therefore the safest way forward (in terms of disease control) is to assume these individuals are truly positive.

Suggested Control Policies

Calves

We are currently recording the numbers of the cows contributing colostrum and accordingly the calves that receive it; therefore the first thing to do is determine whether we know of any calves who have received colostrum from the cows above (unlikely as most of the cows are >100days in milk). The freezer should also be checked to ensure that there is no colostrum already stored from these cows.
Currently both heifer and bull calves receive waste milk through the automatic feeders. As outlined above, 80% of infections are contracted within the first month of life therefore feeding waste milk during this period remains a risk (all be it a small one, 9-19% of subclinically infected animals will shed Map through the colostrum (Sweeney 1996)) – the question remains whether this is a risk we are willing to accept.

Cows

As there is a small risk of transplacental infection (20-40% chance of infection in clinically affected cows and 9% in subclinical (Sweeney 1996)) and the cows identified as infected pose a risk to other calves in the transition group there is an argument for the immediate cull of the identified animals. However as they are predominately in-calf heifers and potentially have several more profitable lactations ahead of them risk management seems a more appropriate strategy.
This might consist of:

1. Should any of the animals listed above become suspect clinical cases they should be culled immediately.
2. Serve them to beef only.
3. Minimise the risk at calving by isolating those animals detailed in Table 1.
4. Holstein-Friesian calves born to those dams should not be allowed to suckle and should be removed immediately at birth.

Mastitis

Bacteriology

The bacteriology results for ****** are summarised in Figure 1 and for comparison the national clinical mastitis bacteriology results are shown in Figure 2.
The comparison between Figure 1 and Figure 2 is likely to be skewed by the over-representation of 'mixed growth' and 'no growth' results. 'Mixed growths' are suggestive of contamination during sampling, whereas 'no growths' can be the result of intermittent excretion of *S.aureus* or *E.coli* infections that have resolved prior to treatment of been denatured during transit.
To allow a more accurate comparison Figure 3 is a summary of the bacteriology results for ****** without the 'mixed or no growth' results. This demonstrates that the results for ****** are pretty much average for the UK. However as mentioned earlier 'no growth' results can be due to *E.coli* death during transit (or freezing), if we assume that the 19% 'no growths' are *E.coli* then *E.coli* is over-represented.

Given these results it seems likely (but in no way conclusive) that the pathogens on ****** are likely to be of an environmental origin. However also of concern is the number of mixed growths isolated suggesting that sampling technique could be improved (mixed growth results of <5% can be achieved (Bradley and Green 2001)).

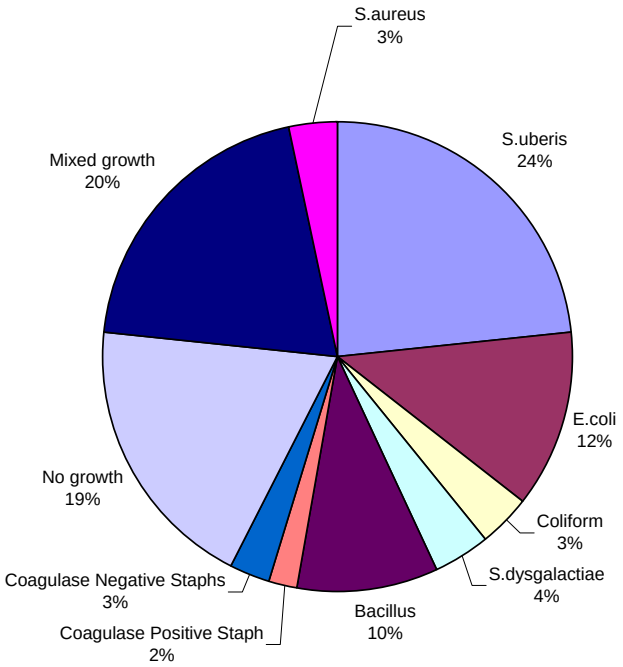


Figure 1 - Breakdown of bacteriology results for Oct 08-Nov 09

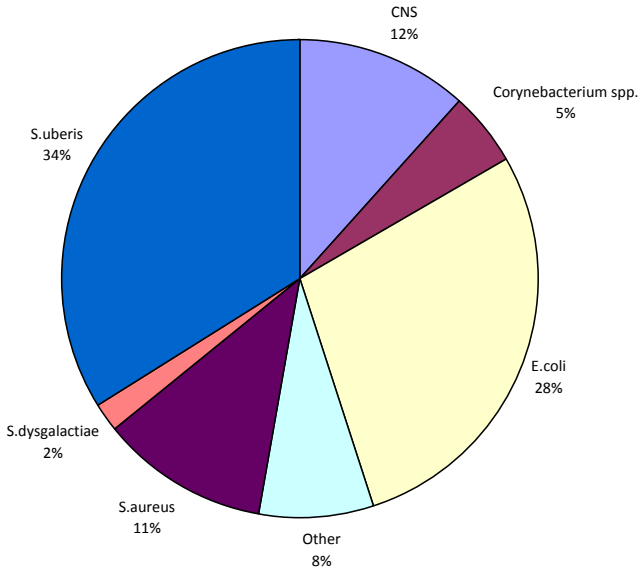


Figure 2 - National clinical mastitis results (Bradley, Leach et al. 2007)

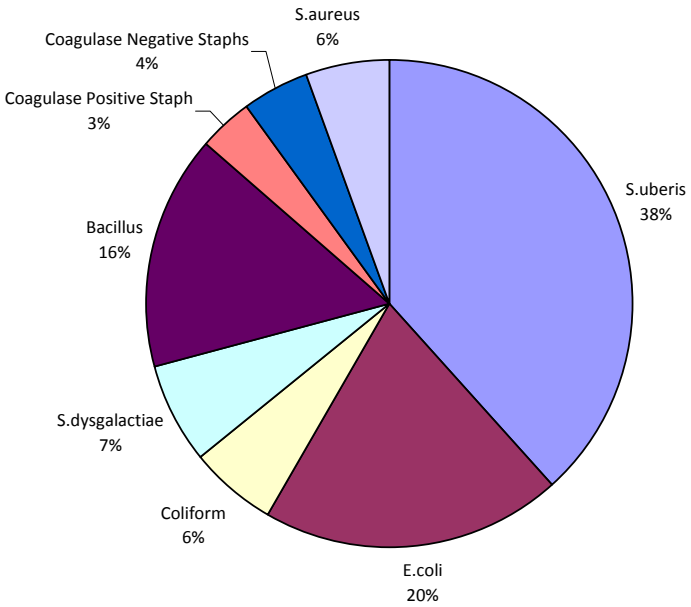


Figure 3 - Breakdown of bacteriology results for Oct 08-Nov 09 WITHOUT mixed and no growth results

Record Analysis

There are two common systems for reviewing cell count and mastitis data; Herd Companion/Interherd (NMR) and TotalVet (Sum-IT/QMMS). Herd Companion only reviews cell count data whereas TotaVet will examine both. The limitation of cell count data is that cows suffering mastitis will often be missing from a recording and accordingly not record a high cell count and be missing from the analysis whereas the limitation of clinical mastitis data is obviously whether identification and recording is accurate.

Dry Period Performance

Herd Companion's analysis of dry performance compares cell count at drying off with the first recording after calving, therefore as illustrated in Table 2 and Figure 4 cows can fall into one of four categories.

Based on the targets in Table 2 our high-to-high is within target whereas the low-to-high is above target – i.e. cows appear to be acquiring new infections during the dry period.
This is confirmed in TotalVet by Figure 5 where ~50% of mastitis cases originate from the dry period (mastitis within first 30d of lactation and recurrences).

Table 2 - Targets for dry period analysis

SCC	Interpretation	Targets
Dried off low, calved in low: *Low-to-low*	Dry period success, individual concerned dried off uninfected and calved in again uninfected.	As high as possible.
Dried off low, calved in high: *Low-to-high*	Dry period failure, individual concerned has dried off uninfected, but contracted a new infection during the dry period or around calving.	<5% (Bradley and Green 2005)
Dried off high, calved in low: *High-to-low*	Dry period success, individual has dried off infected, but during the dry period the infection has been eliminated.	As low as possible. Cows drying off with infections is undesirable.
Dried off high, calved in high: *High-to-high*	Dry period failure, the individual has failed to cure the infection present at drying off OR it has cured and a new infection has established.	<10% (Bradley and Green 2005)

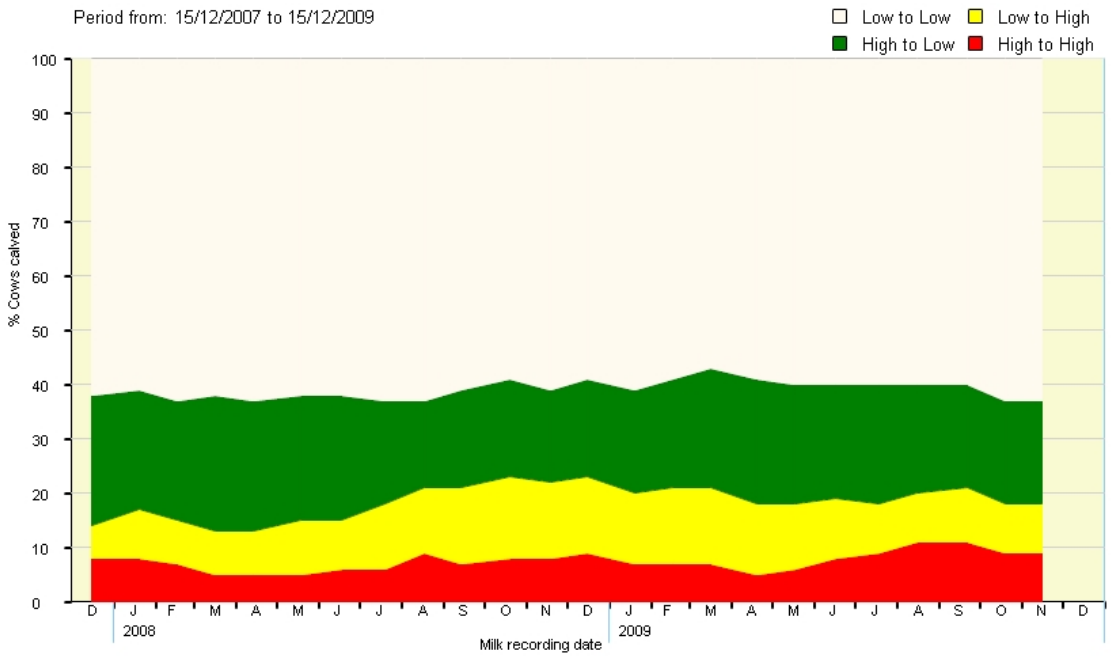


Figure 4 - Dry period performance from Herd Companion

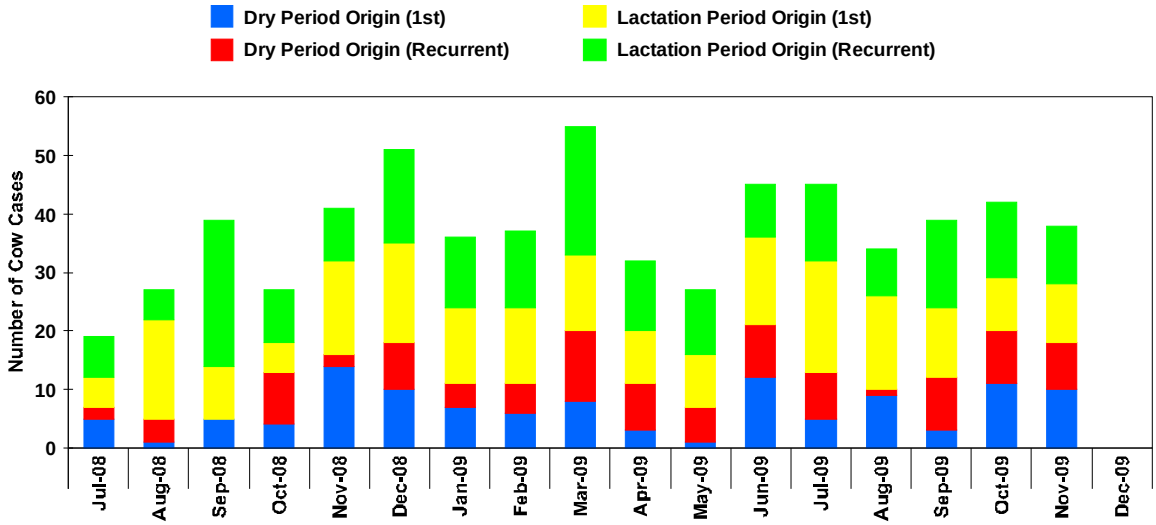


Figure 5 - Mastitis origin from TotalVet

General Mastitis Analysis

During our last meeting with Mike the question was posed – 'What is our mastitis incidence?' According to TotalVet the mastitis incidence for the last 12mths is ~100cases/100cows/year, whereas according to Interherd it is 86cases/100cows/year. The difference between the two values is likely to be due to 'rolling' and 'geometric' means which is essentially the difference in the ways TotalVet and Interherd calculate means.

The national mastitis incidence is thought to be around 47-65cases/100cows/year (Bradley, Leach et al. 2007) so regardless of which mean we follow the incidence is too high. Perhaps more concerning is the general rise in the rolling three month rate (Figure 6).

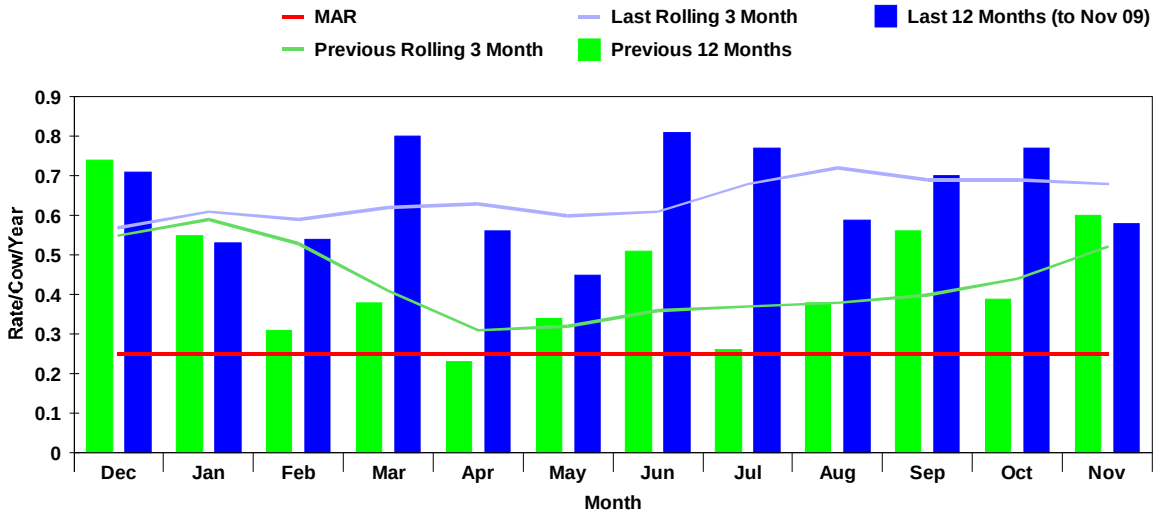


Figure 6 - Rolling 12mth incidence

Dry period infection vs lactation origin?

The next question is whether mastitis of lactational origin or from the dry period is more significant. Figure 7 and Figure 8 compare the numbers of cows at risk of infection plotted against the clinical mastitis rates attributable to that group, both the lactational origin and dry period origin are above the maximum acceptable rate (MAR). However when the proportion of at risk cows is compared to

the rate, the proportion of cows infected due to dry period infections is larger than that originating from lactation.
So it would seem that tackling the infections arising from the dry period is the place to start.

Environmental aetiology vs contagious

Figure 9 illustrates clinical mastitis events by their occurrence (i.e. how many times a cow suffers only a single case of mastitis), it clearly demonstrates a peak of cows suffering a single case of mastitis. This pattern is typical of environmental infections as they are simply 'random' events. Contagious problems are more likely to present widely distributed.
Figure 10 illustrates mastitis incidence by day in milk, Figure 10 is typical of an environmental pathogen problem (contagious pathogen problems tend to be skewed to the right towards higher days in milk).
Therefore given the Bacteriology results (p.3) together with the discussion above it appears that the mastitis occurring at ****** is primarily of an environmental origin.

Conclusions

******'s dry period performance is sub-optimal with a higher than acceptable number of new infections acquired. Overall the infections would appear to be of an environmental origin rather than a contagious.

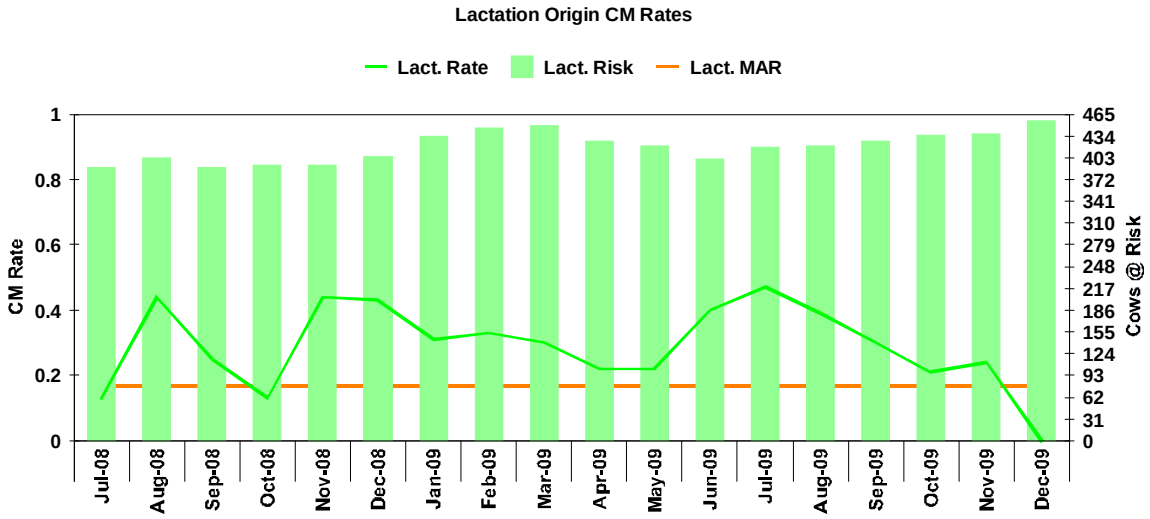


Figure 7 - Clinical mastitis of lactational origin

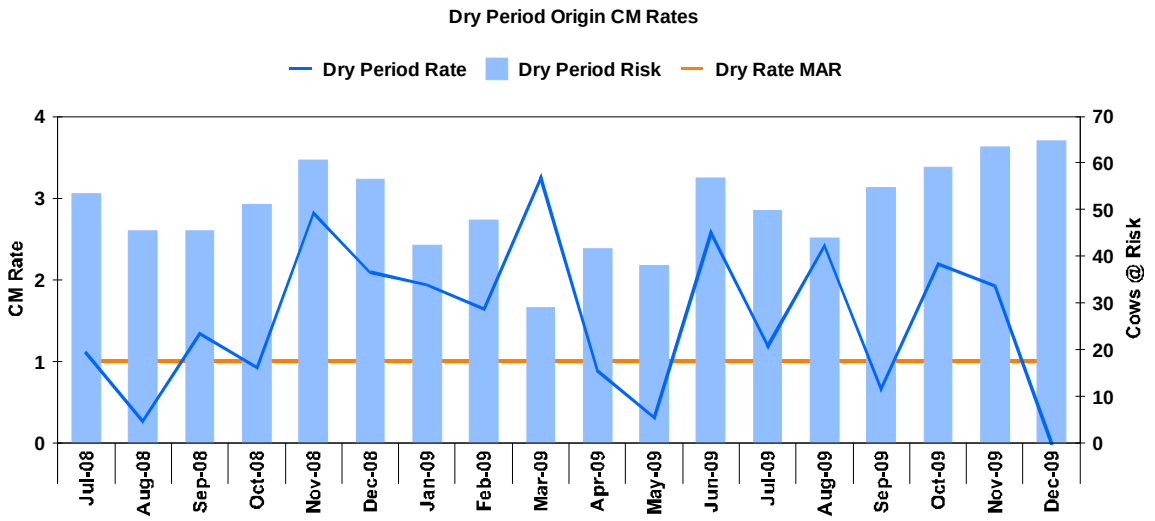


Figure 8 - Clinical mastitis of dry period origin

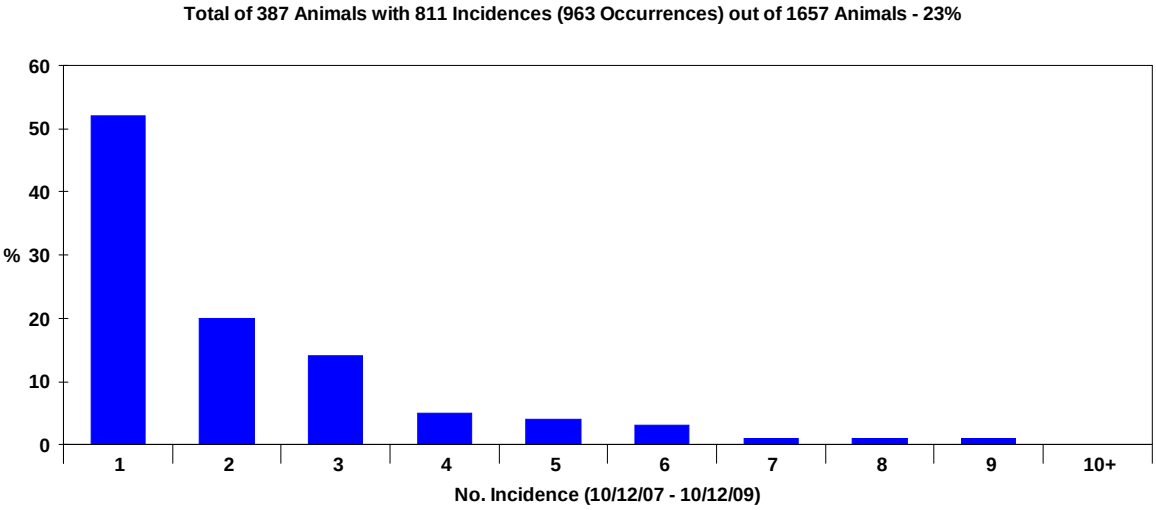


Figure 9 - Mastitis incidence by occurence

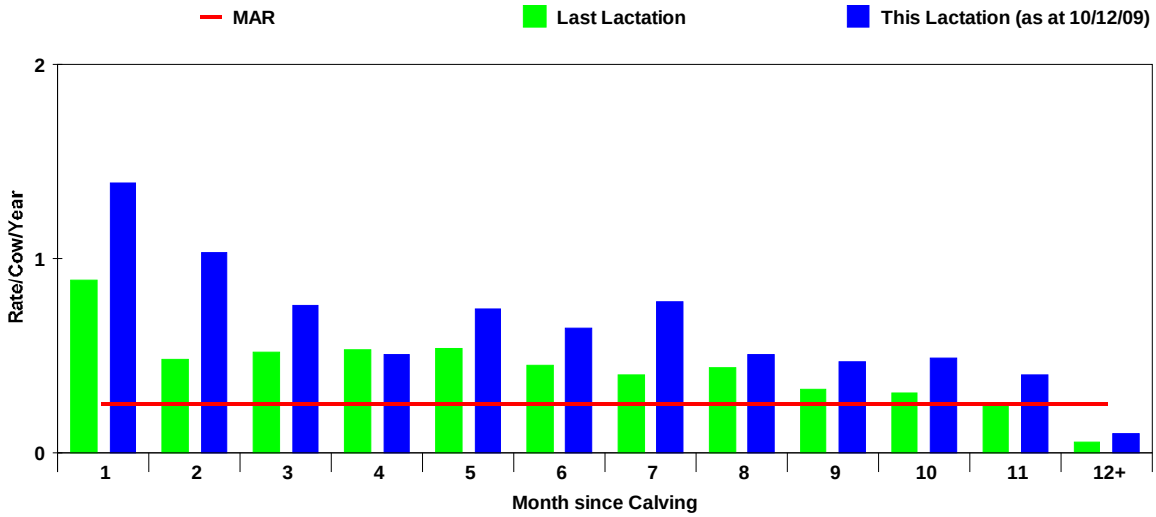


Figure 10 - Incidence by DIM

Hypocalcaemia

We have recently experienced a number of clinical milk fevers together with a rise in associated diseases (uterine prolapses, retained foetal membranes, toxic *E.coli* mastitis), a series of urine pHs were taken (urine pH is used as a measure of the degree of metabolic acidosis the transition cow ration is inducing, the induction of a metabolic acidosis ensures better control of clinical and subclinical milk fever) and were higher than the target (6.2-6.8 (Goff 2008)). Urine pHs were taken again on 17/12/09 and the results are shown in Table 3.

Table 3 - Urine pHs taken on the 17/12/09

7.95
3.01
7.87
7.97
7.31

If we ignore the spurious result these remain above the target range of 6.2-6.8 and as a consequence remains unlikely that we are obtaining effective control of milk fever.

During the treatment of down cow **** and I were discussing the 'ideal' treatment of milk fevers. A clinical case of milk fever requires 12g of i/v calcium over a period of 12mins (Goff 2008), calcium is usually presented as calcium borogluconate.
This amount of calcium will elevate the blood levels for ~4hrs after which the cow needs to have re-established her homeostatic mechanisms to deal with the ongoing demands. It can be provided by a single 40% Ca bottle (green top) or through a pair of 20% bottles (blue tops), in terms of the physiology either preparation should work equally well – however in terms of logistics giving two bottles is slower than one (therefore more likely to take 12mins) and if given under the skin is more isotonic (closer to the concentration of the cow's blood naturally) and therefore will be absorbed quicker – therefore the preparation of choice would appear to be a pair of 20% bottles rather than a single 40%!
We also discussed giving calcium under the skin, I was taught at university that giving calcium under the skin as well as intravenously increased the chances of a relapse in milk fever (slowed the cow's need to organise her own homeostatic mechanisms) – however I have been unable to find any scientific evidence for this! So I'm not sure whether to believe it or not!
An alternative to giving calcium under the skin (as an adjunct to intravenous) might be to give a DCAD paste (a paste containing a high concentration of salts that immediately induces a metabolic acidosis allowing the cow maintain her own calcium levels better) which essentially acts in the same way as

feeding DCAD salts during the transition period. I'm not sure if the practice carries any of these and hopefully we will stop the milk fevers shortly so this is likely to be academic! If you want further details then please ask.

Body Condition Scoring (BCS)

During the last meeting with Mike we decided to push on and organise BCS monitoring monthly as an additional method of monitoring energy status.
(Thinking ahead, when we come to summarise the data we collect it is important to remember that using a mean or median is not an appropriate method using threshold summaries as we are only concerned with the proportion of individuals over- or under-conditioned.)

The number of cows which require BCS on an individual farm is largely related to the number of cows on the unit. Table 4 summarises the number of cows requiring scoring at each of the monitoring stages, the target BCS (Mike may wish to comment here), the number of cows usually present at that time at that stage (based on the last six months) and the number of cows we need to score to detect a 10% prevalence (of over- or under-conditioned animals) with a confidence of 90%. (In a study of 10,000 dairy cows 9% of cows between 6-12wks post-partum were below condition score 2 (Ward, Murray et al. 1995)).

Table 4 - Summary of sample sizes for BCS

Stage/DIM	Target BCS (Husband 2006)	Number of cows currently in milk at this stage (averaged from the last 6mths)	Number of cows needing to be scored to be statistically significant
Calving 0-20	3.0	34	18
6-8wks 42-56	2.0-2.25	23	16
Late lactation 250-300	3.0	57	18

I have supplied Alan with charts to refresh him on the 1-5 scoring system.

Lameness Review

In preparation for Nick Bell's visit (Friday 8th January 2010) I have been reviewing the last 12mths of foot trimmer records and we have also instigated monthly mobility scoring.
Much of the data I am not in a position to comment on (we need to wait for Nick for this) however I have added a brief summary and discussion below together with a the rest of the data for interest.

Summary

Foot trimming records of clinical lameness

The results of the last 12mths of foot trimmer records are summarised in Figure 11 and together with the published figures for the UK in Table 5.
****** appears to have too much lameness across the board when compared to the published figures (however care needs to be taken over these figures as the more I work with them the more I realise they are likely to be much too low) – Nick may be better placed to provide more typical UK values and help us know how we fit in.

Table 5 - Previous 12mths of foot trimmer records (Dec 08-Nov 09)

	Laminitis	Slurry heel	Sole ulcer	Digital dermatitis	White line disease	Sole separation	Fibroma
Total no. of cases	257	40	335	80	69	98	27
Proportion	28.4%	4.4%	37.0%	8.8%	7.6%	10.8%	3.0%
Incidence cases/100cows/year	42.83	6.67	55.83	13.33	11.50	16.33	4.50
Published incidence values (Barker 2007)	*N/A*	*N/A*	*7.20*	*1.70*	*5.60*	*6.20*	*N/A*

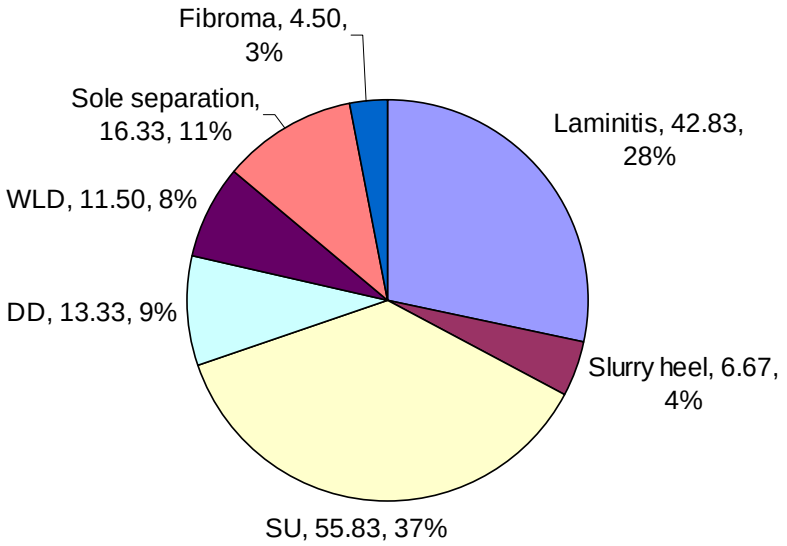


Figure 11 - Breakdown of cases by lesion

Mobility scoring

******* has kindly undertaken a single session of mobility scoring on the 25th November. The results have been favourable and apparently they have improved since it was last undertaken by the foot trimmer.

The results are summarised in Figure 12 – it will be interesting how they fair as we gather more data.

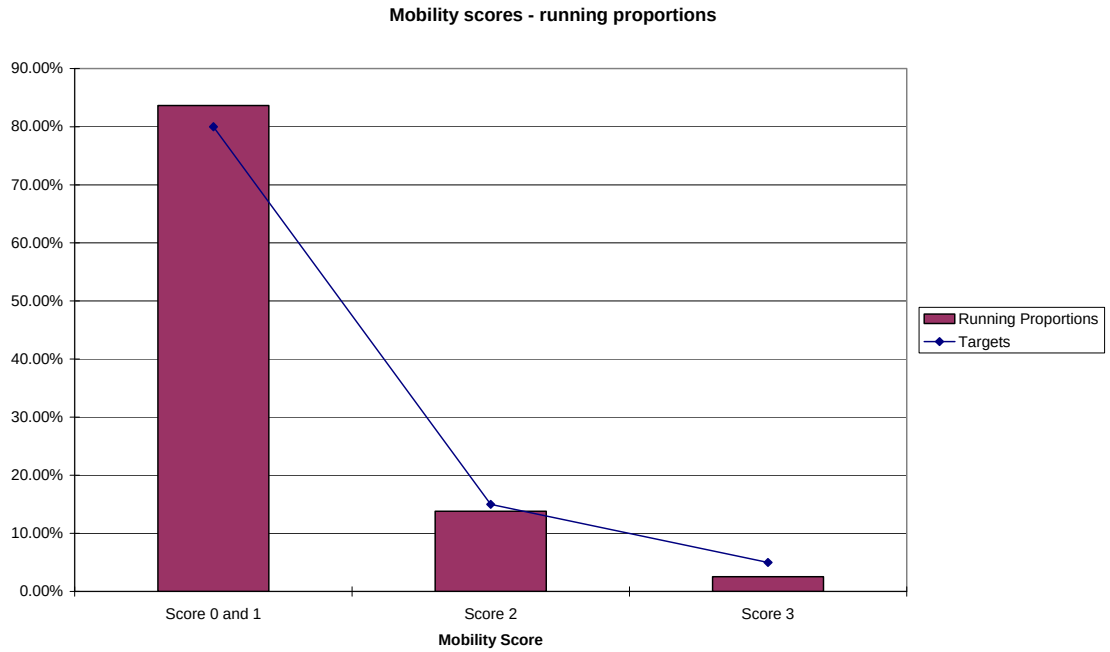
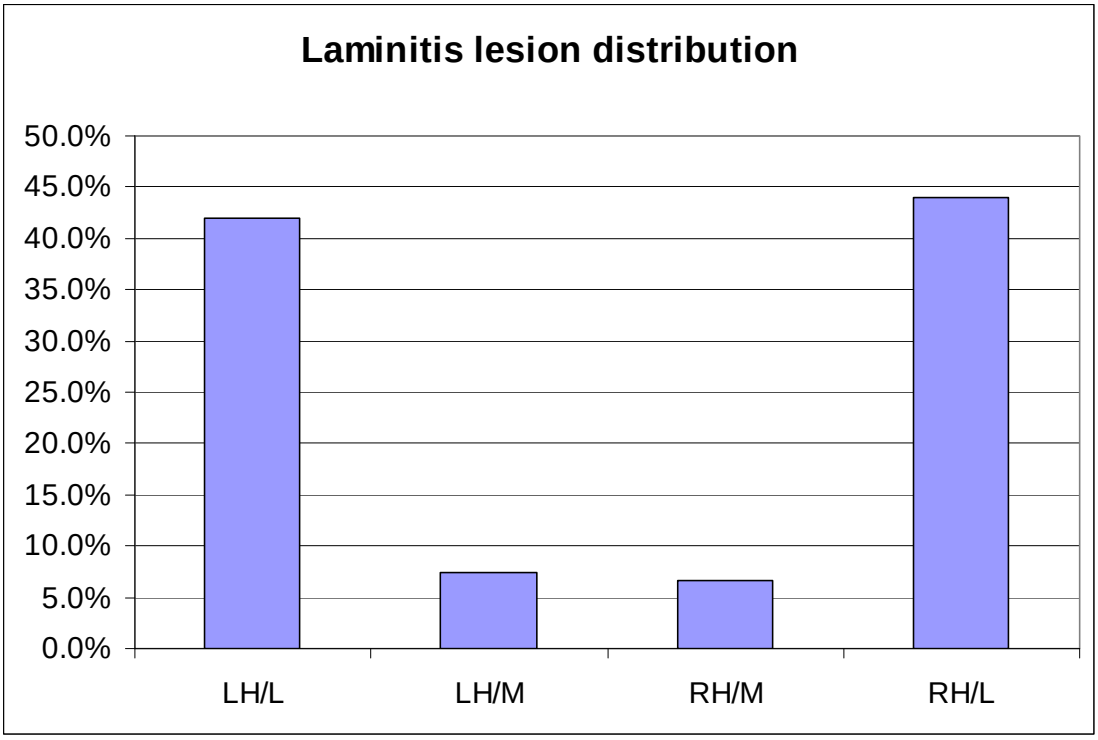
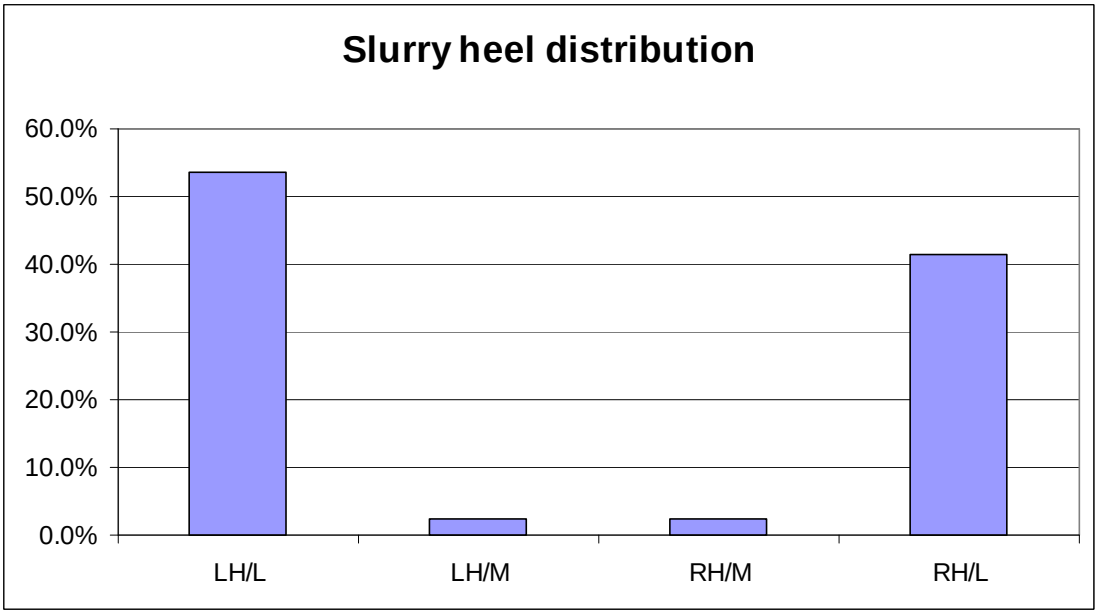


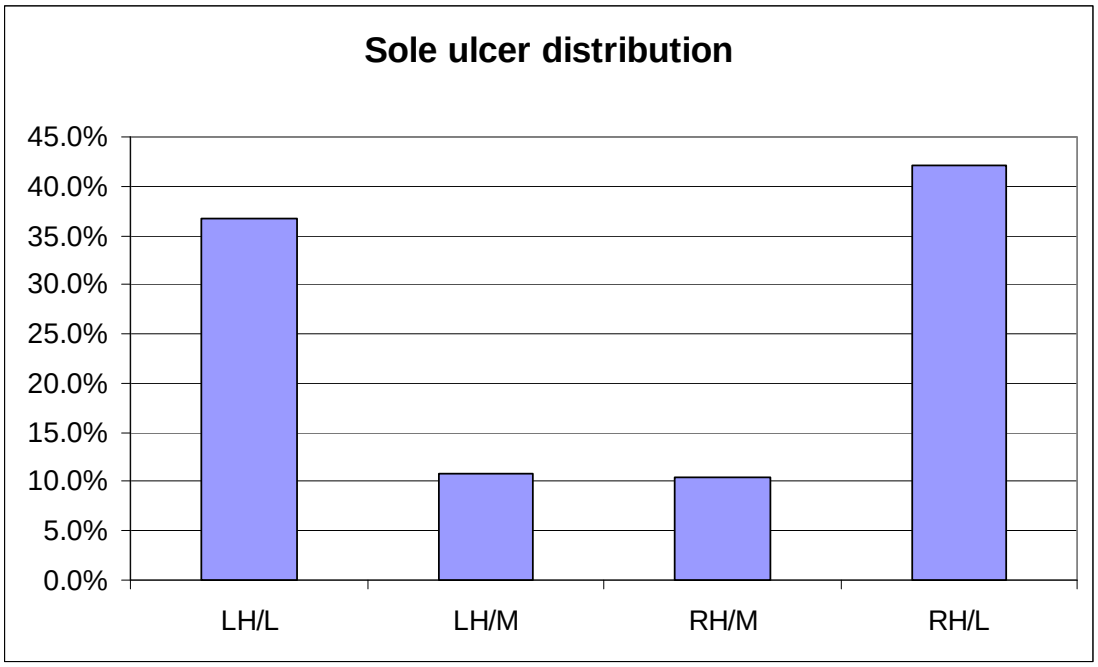
Figure 12 - Mobility score results for 25/11/09

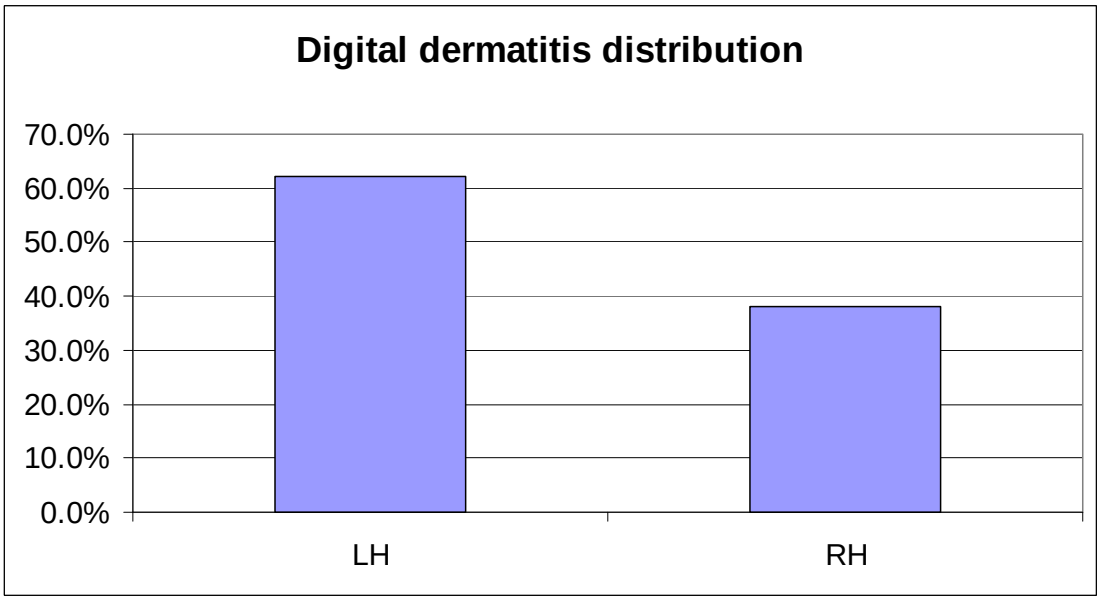
More clinical lameness data

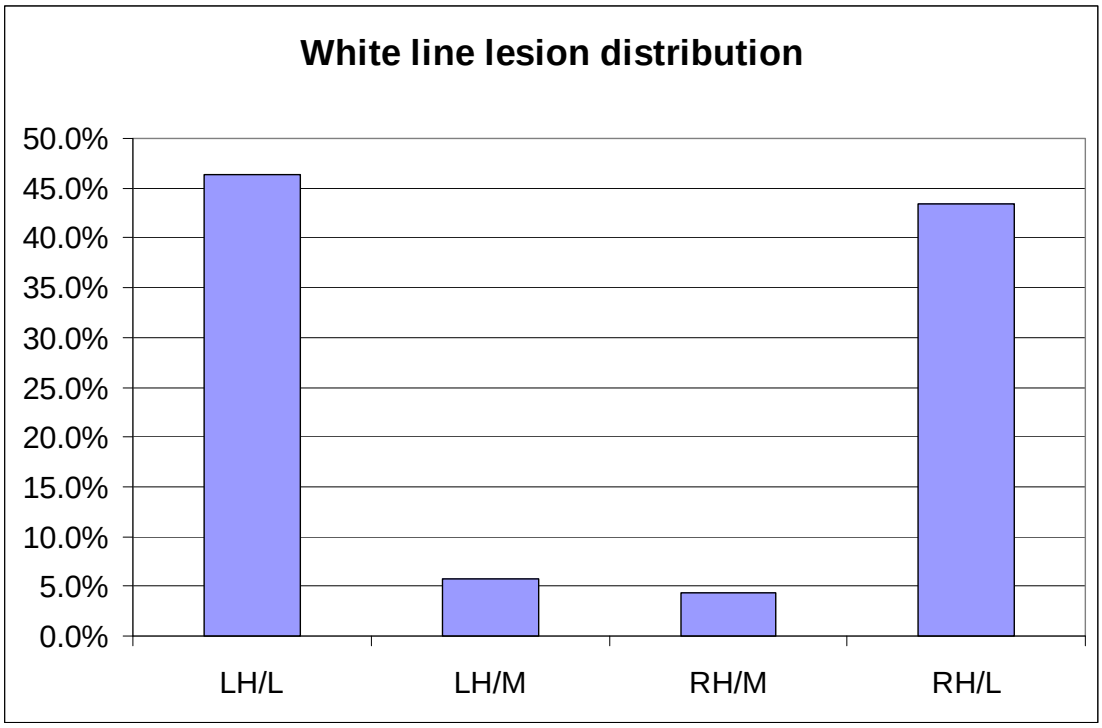
As mentioned earlier most of this data I'm not in a position to comment on. However there has been some discussion regarding the shearing forces present in the foot as cows turn on and off the rotary parlour, looking through the results below this seems unlikely. If the parlour were creating differential shearing forces you would expect a high incidence of sole separation, laminitis and while line lesions on the outer claw of one of the hind feet (I can't comment which as really I haven't spent enough time watching cows enter and exit the parlour), this does not appear to be the case in the results below (admittedly I haven't run any stats to check for significance, but by 'eye-ball' it seems unlikely).

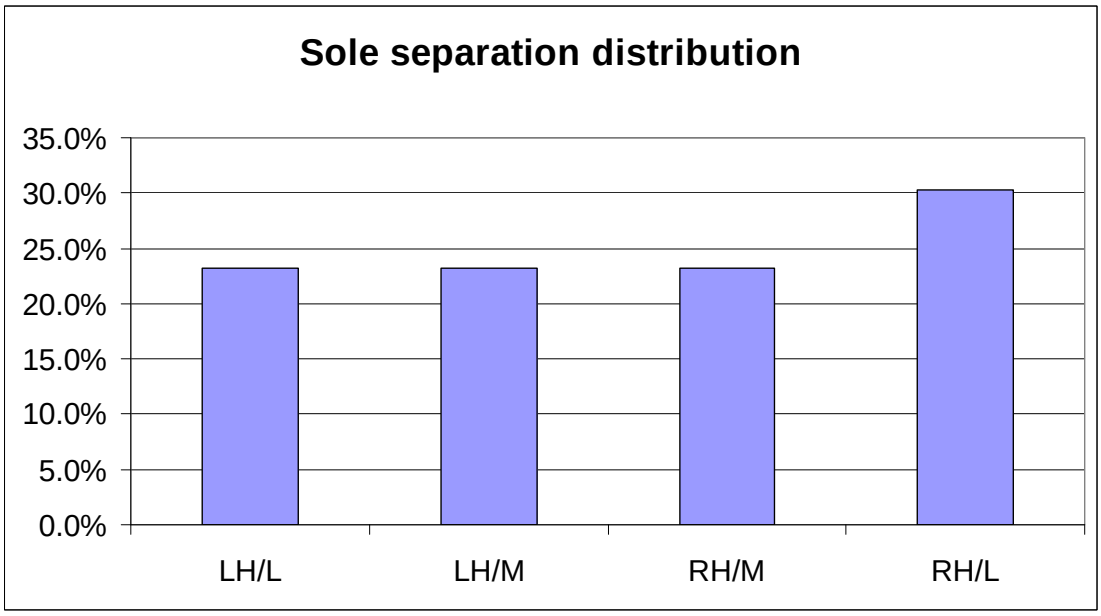


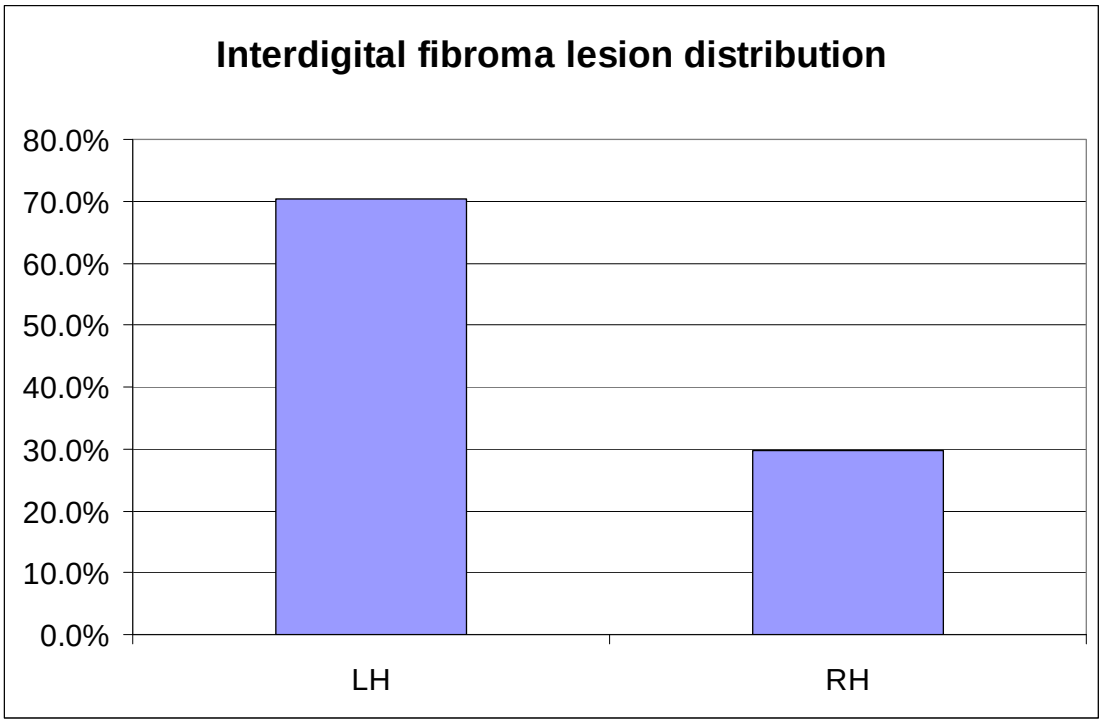



Sole ulcer distribution
45.0%
40.0%
35.0%
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
LH/L
LH/M
RH/M
RH/L


Digital dermatitis distribution
70.0%
60.0%
50.0%
40.0%
30.0%
20.0%
10.0%
0.0%
LH
RH


White line lesion distribution
50.0%
45.0%
40.0%
35.0%
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
LH/L
LH/M
RH/M
RH/L


Sole separation distribution
35.0%
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
LH/L
LH/M
RH/M
RH/L



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