Effect of Feeding Colostrum of High Immunoglubulin Content on Calf Growth Rates and Disease

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Introduction

Undifferentiated pre-weaning diarrhoea in calves is a common clinical problem in New Zealand, and internationally. It may lead to significant morbidity and mortality in some herds, with long term effects on growth rates and survival of the infected calves. Reduction in weaning weights can affect the long term productivity and survival of cattle (Everitt and Jury, 1977).

Calf diarrhoea may be caused by viruses (rotavirus, coronavirus), bacteria (*E. coli*, *Salmonella* sp.), protozoa (*Cryptosporidium* sp., coccidia.) or dietary factors (Blood and Radostits, 1989). These pathogens can survive in the environment for extended periods of time, and result in recurring infections within herds year after year. The prevalence of these pathogens in New Zealand has not been defined clearly. Over 40% of faeces samples from calves with diarrhoea submitted for laboratory analysis are rotavirus positive (Roger Ellison, Alpha Scientific, pers. comm.) Analyses of faecal samples from calves in Victoria, Australia (a similar dairy production system) revealed that 49% of scouring calves had rotavirus, 36% had cryptosporidiosis and 5% had enterotoxigenic *E. coli* (Jerrett, 1985). Multiple infections are common, which results in more severe disease.

Insufficient intake of immunoglobulins (Ig), and other milk components which offer enhanced immunity within the first 12 to 24 hours of life, are a major predisposing cause for diarrhoea. Immunoglobulins fed within 24 hours of calving are absorbed and confer passive immunity to calves until endogenous immunoglobulins are produced (active immunity).

Feeding of colostrum based supplements beyond the initial 24 hours has advantages, as the Ig's are active within the lumen of the bowel and will bind to infectious agents, reducing infectious agent attachment to the bowel wall and, hence, disease prevalence and severity (Saif and Smith, 1985). Excretion of rotavirus antigen may be completely prevented where calves are fed colostrum from vaccinated dams, in comparison to calves fed milk who excreted viral antigen for 9 days (Saif and Smith, 1985) following viral challenge. Inclusion of colostrum (250 ml daily) derived from cows immunised prepartum with rotavirus antigens into calf feed reduces the incidence of clinical diarrhoea (Roger Blowey, pers. comm.; Blood and Radostits, 1994). Colostrum from the first 3 milkings contains approximately 50 mg/ml of Ig (Quigley et al., 1996), but declines at following milkings.

This trial aimed to test the efficacy of a low temperature dried colostrum, containing high levels of Ig, in preventing diarrhoea, and in maintaining weight gain of calves. The specific hypotheses tested were that feeding a colostrum-based supplement in conjunction with milk replacer on a daily basis would result in a

higher average daily liveweight gain pre-weaning, result in a lower proportion of calves excreting rotavirus antigens and reduce the incidence of clinical diarrhoea.

Materials and Methods

A prospective trial testing the efficacy of including dried colostrum in conjunction with the normal milk replacer fed to calves was performed to examine the effect of additional colostrum on calf growth rates, calf survival and prevalence of rotavirus antigens in the calves faeces. A total of 307 calves from 2 commercial dairy herds (Herd A, n = 110; and Herd B, n = 197) were enrolled. Calves from Herd A were born between 24th of July and the 9th September 1998, while those in Herd B were calved between the 26th of July and the 12th of September, 1998. Herd A enrolled 9 bulls and 101 heifers and the breeding included Friesian, Jersey, dairy cross-breds (Jersey x Friesian) and some beef x dairy cross (Angus x dairy, Hereford x dairy or Piedmont x dairy) calves. All animals in Herd B were Friesian bull calves. All calves remained with the dams for 12 to 24 hours before being removed to a rearing shed. Calves were randomly assigned to treatment by 4 days after birth. The calves were weighed within 3 days of entering the facility and then every 2 weeks until weaning at greater than 80 or 100 kg liveweight, depending on the herd. All calves were fed with a commercially available calf replacement milk (Ancalf, New Zealand Dairy Ingredients Limited) ad-lib twice daily until approximately 2 week of age, and then once daily until weaning. Half the calves were also fed 20 grams of a colostrum based supplement twice daily from day 4 until weaning. The supplement was incorporated with the milk replacer. The other group was fed a placebo (dried milk powder) of similar mass and appearance. Treatment was double blinded and the bags containing the treatment and control material were marked as "Group A" or "Group B".

Group A product was the colostrum powder, whilst Group B product was the placebo.

Faecal samples were collected fortnightly from 38 calves in Group A and 40 in Group B with 37 and 41 calves sampled from Herd A and Herd B, respectively. Samples were collected between 1 and 7 times from each calf, depending on the period of enrolment for that calf. These samples were analysed for the presence of rotavirus antigen using a commercially available and validated ELISA assay.

Calves were observed daily by herdowners for any evidence of diarrhoea. Calves with clinical diarrhoea were isolated from the main calf rearing area, milk was withheld for 48 hours and they were fed with 2 L of a dextrose/electrolyte solution (Revive) every 12 hours. Antibiotics were used to treat some calves following veterinary consultation. Faecal samples were collected from a subset of calves with clinical diarrhoea for laboratory assessment of cause of diarrhoea. Herdowners recorded the date, number and

treatment of any calf with diarrhoea. Faecal samples from calves with clinical diarrhoea were assessed for the presence of rotavirus and coronavirus antigen using commercially available and validated ELISA assays. *E. Coli* were detected using a specific K99 antigen ELISA. Faecal culture was used to detect *Salmonella* spp.. *Cryptosporidium* and coccidia were searched for by light-microscopy following Zeil-Neilsen staining of faecal smears. All laboratory work was performed by Alpha Scientific, 141 Ellis St, Hamilton.

The average liveweight gain (ALWG; kg/calf/day) was calculated as the final weight minus the initial weight divided by the number of days between these weighings. Analysis of variance was performed using ALWG as the dependant variable, with group (treatment) and herd included as fixed effects and with calving date (Julian date), initial weight, and sex of the calf initially included as covariates. Only calving date was a significant covariate and the other factors were removed from the final model.

The colour reaction of the rotavirus faecal antigen ELISA was subjectively coded (ie 0 = nil, 1 = suspicious, 2 = weak positive, 3 =positive, 4 = strongly positive). The data was analysed both as a yes/no outcome (ie nil and suspicious coded as 0 and positives as 1) by Π^2 and as the score data (0 to 4) using a Mann-Whitney Rank Sum test, as the scores were not normally distributed at each time period.
The proportion of calves that died was analysed by the Π^2 statistic.

Results

The treatment groups were balanced for the calf breed and the sex of the calf ($P > 0.5$ and $P > 0.08$, respectively). There was a herd by group interaction for the birth date of the calves, with the Herd B, Group B calves being enrolled significantly later (9 days) than Group A (Table 1; Figure 1). There was also a significant difference in initial weight between the herds ($P < 0.001$) and the initial weights of groups approached a significant difference ($P = 0.08$; Table 2; Figure 2). The average weight at first weighing was 43.5 $\pm$ 6.1 kg (mean $\pm$ st.dev). The genetics of the calf significantly effected the initial weight with Friesian calves heavier than beef cross calves, which were heavier than dairy cross calves which were in turn heavier than Jersey calves (Figure 3).

Herd B had a higher ALWG than Herd A ($P < 0.005$; Table 3, Figure 4). There was no effect of treatment group on ALWG and there was no group by herd interaction ($P > 0.2$; Table 3). The ALWG was positively related to the calving date ($p<0.001$; Table 3, Figure 6) but not related to initial weight ($P > 0.5$) or sex of the calf ($P > 0.5$; Figure 5).

The interval (days) between the initial and final weighing (weaning) did not differ among herds or groups and there was no herd by group interaction ($P > 0.4$ in all cases; Table 4). The initial weight was negatively related to the interval to weaning ($P < 0.005$), bull calves were weaned earlier than heifer calves ($P = 0.02$) and there were breed differences in the interval to weaning ($P < 0.005$).

The proportion of sampled calves that had rotaviral antigens in their faeces declined with age (Figure 7). There was a significantly higher rotavirus antigen score at week 2 for Group B than for Group A (P = 0.12 and P = 0.03 for Herds A and B, respectively, Mann-Whitney Rank sum test) and trend for a higher score at week 4 in Herd B (P = 0.09), but with no difference at weeks 0 or 6 (P>0.2). There was no difference in prevalence of rotavirus antigen positive faecal samples between the herds at week 0 and 2 (P > 0.1), but there was a tendency for Herd B to have a higher proportion than Herd A at week 4 (ie 10.8% vs 0%; P = 0.11). This sampling in Herd B occurred at the time of the second outbreak of clinical diarrhoea in Herd B (see below).

Herd A had no clinical diarrhoea and no calves died before weaning. Herd B had two outbreaks of clinical diarrhoea starting on the 28th of August and the 21st of September, respectively. During outbreak one, a total of 28 calves were treated with an oral electrolyte solution (Revive, Virbac Laboratories (NZ) Ltd.) at a rate of 2 litres twice daily for 3 days. They were also given, either 3 intramuscular injections of a parenteral trimethoprim/sulphonamide treatment (Zaquilin, Schering Plough, Upper Hutt, NZ) or an oral tablet containing 4.75 g of sulphonamides (StrinacinTablets, Merial, Auckland, NZ) followed by half a tablet twice daily for a further 2.5 days. There was no difference in the proportion of calves in each group that were diagnosed and treated with clinical diarrhoea (n = 12 (12%) vs n = 16 (16.3%) for Group A and B; respectively, P > 0.2). But significantly more Group B calves died than Group A calves (5.1% vs 0 % for Group B and A, respectively; P = 0.03; Fishers exact Π^2).

A further two calves from Group A were treated orally with sulphonamides (StrinacinTablets, Merial, Auckland, NZ) commencing on the 21st of September, and both animals survived. Faeces were submitted for laboratory examination from 10 clinically ill calves between the 28th of August and the 8th of September. Rotavirus antigen was detected in 9 of 10 samples tested, while neither *E. coli*, *Salmonella spp.*, nor cryptosporidiosis, were detected in any sample. Faecal samples were taken from 13 calves on the 7th of September, and no coccidia were found.

A further 2 calves (1 each in Group A and B) died acutely without diarrhoea while grazing on pasture, probably due to black leg (*Clostridium chauvoei*) infection in Group A, and to an abdominal crisis in Herd B.

Discussion

Treatment had no effect on the average liveweight gain of the calves despite the presence of clinical diarrhoea due to rotavirus in one herd. The impact of the clinical disease may have been insufficient to result in significant weight loss. Alternatively, a degree of compensatory weight gain may have occurred, whereby ill calves had reached liveweights not dissimilar from those of non-infected animals. Average liveweight gain was however affected by herd of origin and birth date of the calf. Differences in the genetics and gender of the calves between the herds, and/or differences in feeding and general management between the two herds may account for this difference. It is not clear why later born calves had higher liveweight gains. The calves were turned out to pasture for short periods from two or more

weeks of age, depending on the weather. It is possible that later born calves had more pasture available and/or the pasture was of higher quality than that available to the earlier born calves.

Rotavirus was found in a high proportion of calves (>50%) at the initial faecal sampling (ie < two weeks of age) in both the treated and control group, and in both herds. Subclinical rotavirus infections have been previously reported in 42% of sampled calves (McNulty and Logan, 1983). Rotavirus antigen was present in the faeces of a significant number of calves, despite daily intake of colostrum with a high Ig concentration. Presumably if the concentration of rotavirus is greater than the concentration of Ig, then a proportion of unbound viral particles will be present in the faeces, and hence react to the rotavirus antigen ELISA. Colostrums vary in their ability to prevent rotavirus antigen from appearing in the faeces. One study demonstrated that colostrum from cows vaccinated with one type of rotavirus vaccine prevented excretion of viral particles, while another vaccine did not (Saif and Smith, 1985). The decline in prevalence of rotaviral antigen faecal samples with time may reflect development of active immunity by the calves. This active immunity may produce sufficiently high IgG and IgM titres to prevent secretion of viral antigens (Saif and Smith, 1985).

Treatment significantly reduced the mortality associated with clinical diarrhoea in Herd B. The clinical outbreak of diarrhoea was most likely due to rotavirus infection as 9 of 10 sampled calves with clinical diarrhoea had rotaviral antigen present, and there was no evidence of the presence of the other common causes of calf diarrhoea including *E. coli*, salmonellosis, cryptosporidiosis or coccidiosis.

A similar proportion of Group A and B were diagnosed with clinical diarrhoea, but more calves in Group B calves with clinical diarrhoea died (5 of the 16), while none of the 12 Group A animals did so. Immunoglobulins within the diet appear not to prevent infection, but does reduce the mortality associated with clinical diarrhoea in young calves. Presence of immunoglubulins may protect the gastrointestinal tract by reducing the number of virus particles binding to the mucosa, and hence reducing the severity of the disease process. Clinical disease was only evident in Herd B, but not Herd A. Yet the rotavirus antigen prevalence in Herd B was not higher than that in Herd A at the time of the major clinical outbreak in Herd B (late August). This suggests that other managerial or environmental effects were operating in conjunction with the presence of rotavirus that resulted in diagnosis of diarrhoea in one herd and not the other. No other pathogens were isolated from the ill calves, suggesting that differences in feeding practices, or housing conditions, rather than infection by another pathogen, was the cause of the clinical disease.

Conclusions

Feeding of immunoglobulin rich colostrum was associated with reduced mortality in calves with clinical diarrhoea. There was no difference in average liveweight gain or interval from calving to weaning between calves fed an immunoglobulin rich colostrum, compared to control calves.

References

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Table 1. The calving date of enrolled calves by herd (HERD_CDE) and group (GRP_CODE).

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN + RESIDUAL	29245.40	252	116.05		
GRP_CODE	1274.62	1	1274.62	10.98	.001
HERD_CDE	812.69	1	812.69	7.00	.009
GRP_CODE BY HERD_CDE	1584.47	1	1584.47	13.65	.000
(Model)	4415.13	3	1471.71	12.68	.000
(Total)	33660.53	255	132.00		
R-Squared =	.131				
Adjusted R-Squared = .	.121				

Means and standard error of the means (Std. Err.) for the Julian calving date by herd and group

Group	Mean day	Std. Err.
Herd A group A	221.9	1.5
Herd A group B	221.4	1.5
Herd B group A	220.5	1.3
Herd B group B	230.0	1.2

Figure 1. The mean birth date by herd and breed

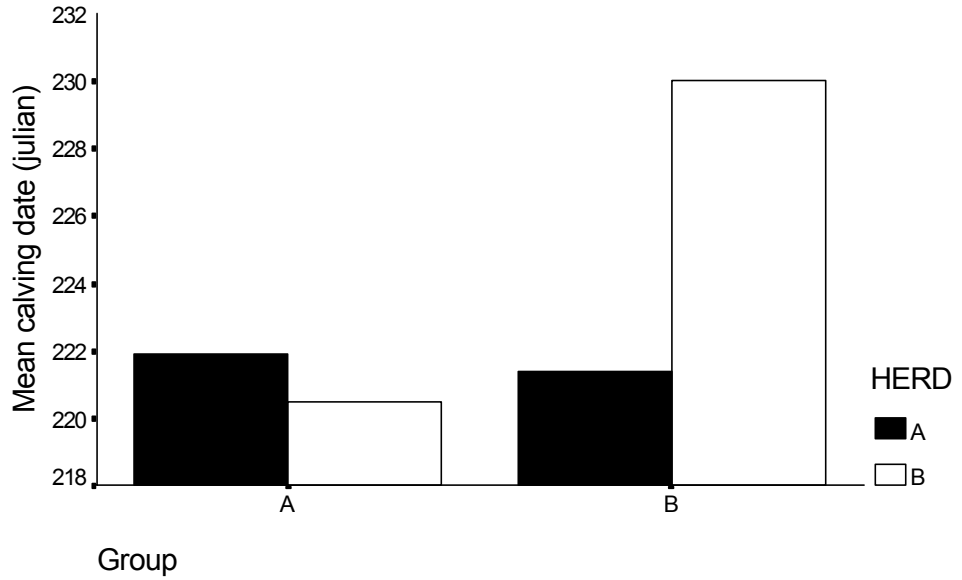

Table 2. The weight at initial weighing of enrolled calves by herd (HERD_CDE) and group (GRP_CODE)

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN + RESIDUAL	13766.29	303	45.43		
GRP_CODE	137.03	1	137.03	3.02	.083

HERD_CDE	5701.07	1	5701.07	125.48	.000
GRP_CODE * HERD_CDE	78.96	1	78.96	1.74	.188
(Model)	6017.00	3	2005.67	44.15	.000
(Total)	19783.29	306	64.65		

R-Squared	=	.304
Adjusted R-Squared	=	.297

The mean and standard error (Std. Err.) of the initial weight of enrolled calf by herd and group.

Group	**Weight 0**	**Std. Err.**
Herd A group A	40.8	0.9
Herd A group B	40.5	0.9
Herd B group A	50.9	0.7
Herd B group B	48.4	0.7

Figure 2. The initial weight of enrolled calves by herd and group

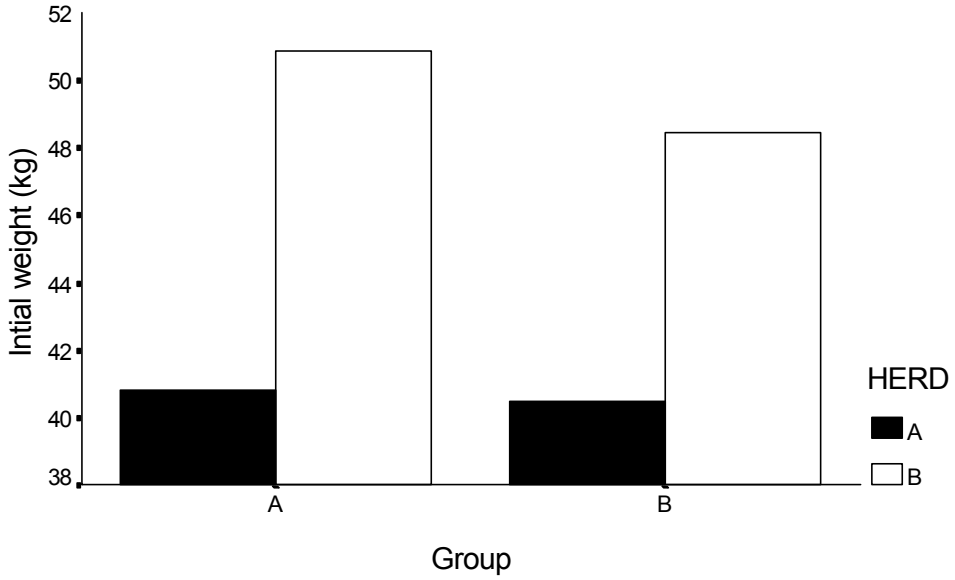

Figure 3. Boxplot of weight at enrollment by breed of calf.

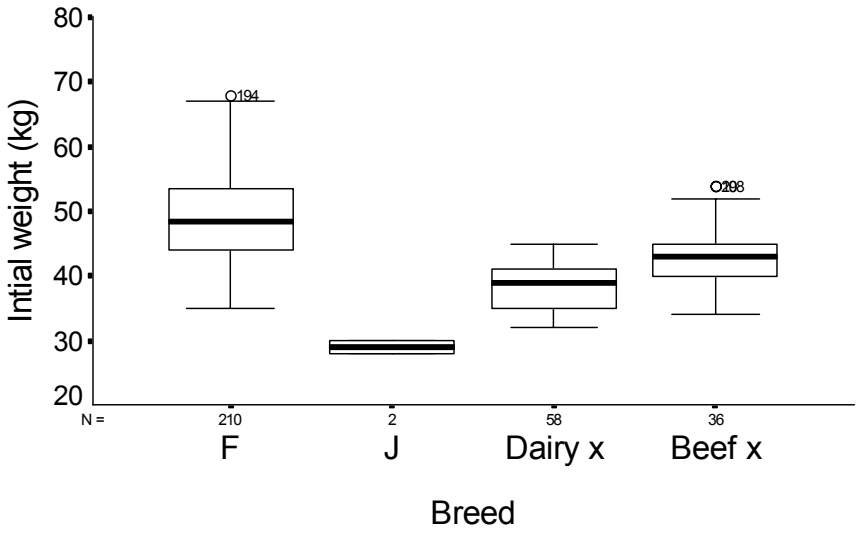

Table 3. Analysis of variance of the average liveweight gain (AVLG) with herd and group (treatment) as fixed effects and the Julian calving date as a covariate.

Source of Variation	**SS**	**DF**	**MS**	**F**	**Sig of F**
WITHIN + RESIDUAL	3.07	248	.01		
REGRESSION	.13	1	.13	10.68	.001
HERD_CDE	.93	1	.93	75.21	.000
GRP_CODE	.00	1	.00	.15	.702
HERD_CDE BY GRP_CODE	.02	1	.02	1.73	.189
(Model)	1.22	4	.31	24.71	.000
(Total)	4.29	252	.02		

R-Squared =	.285
Adjusted R-Squared =	.273

COVARIATE	B	Beta	Std. Err.	t-Value	Sig. of t
JUL_CD	.00213	.18856	.001	3.268	.001

COVARIATE	Lower -95% CL-	Upper
JUL_CD	.001	.003

Figure 4. The average liveweight gain (kg/calf/day) by herd and group.

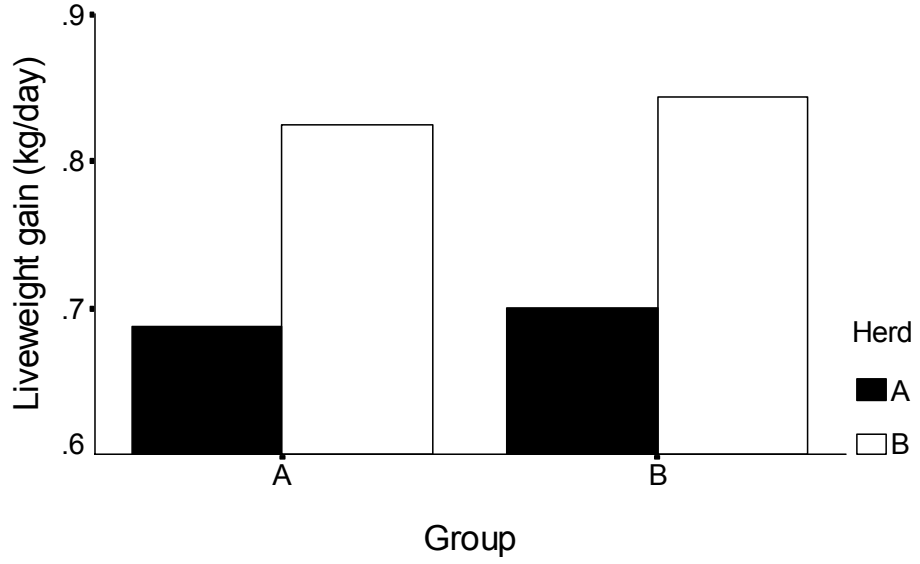

Figure 4. The average liveweight gain versus sex of the calf and its initial weight.

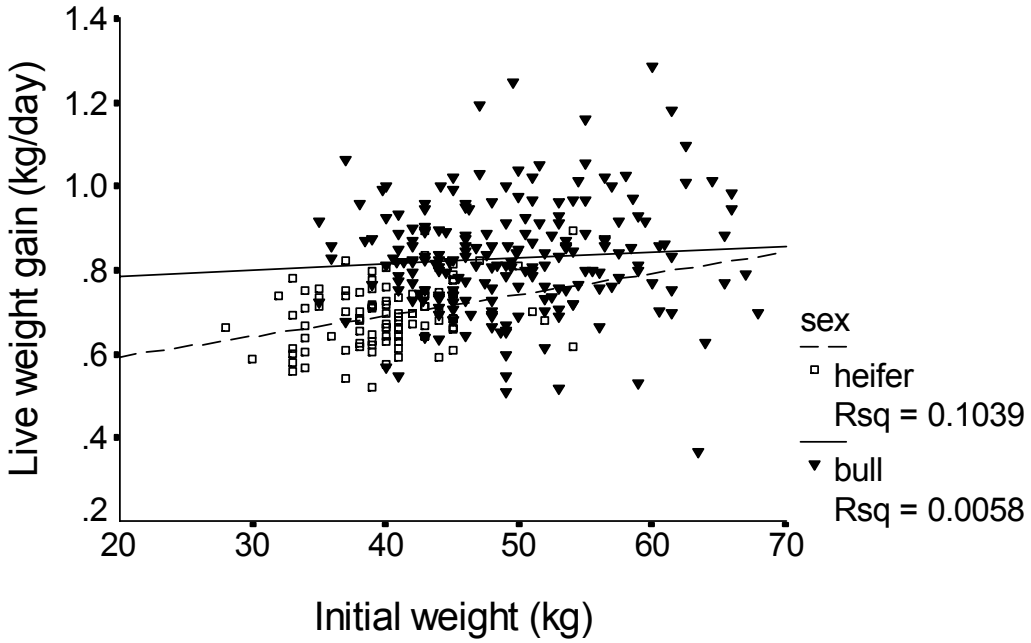

Figure 5. The relationship between calving date (Julian) and average liveweight gain.

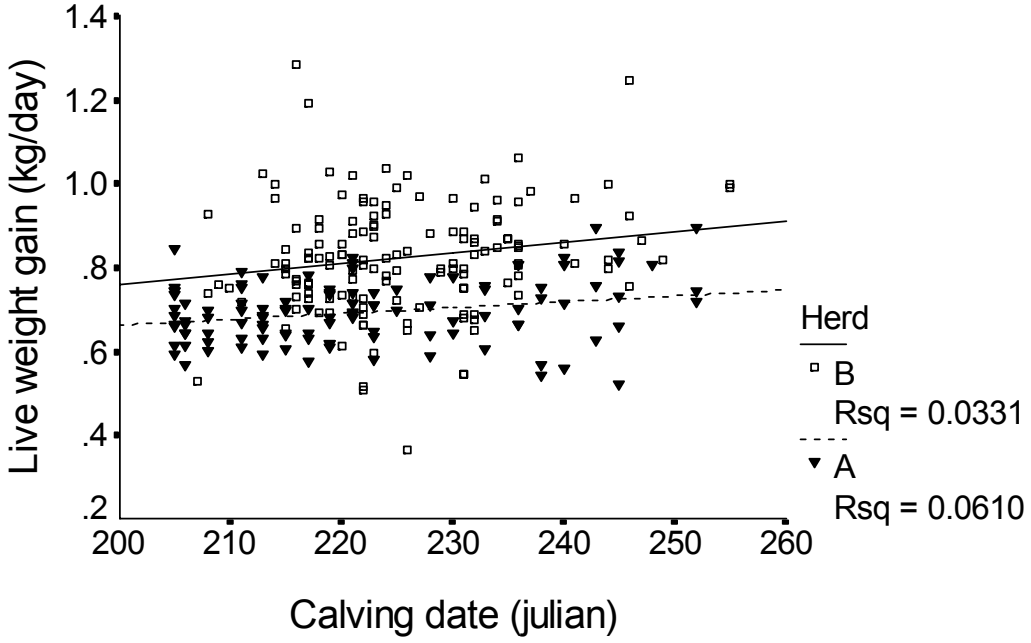

Table 6. The effect of group, herd, calving date, sex of calf, breed of calf and weight at initial weighing on the number of days from initial weighing to final weighing (weaning).

Source of Variation	SS	DF	MS	F	Sig of F
WITHIN + RESIDUAL	47820.70	247	193.61		
REGRESSION	8217.16	4	2054.29	10.61	.000
HERD_CDE	129.10	1	129.10	.67	.415
GRP_CODE	59.02	1	59.02	.30	.581
HERD_CDE BY GRP_CODE	126.68	1	126.68	.65	.419
Model)	11414.34	7	1630.62	8.42	.000
Total)	59235.04	254	233.21		
R-Squared =	.193				
Adjusted R-Squared =	170				

Correlations between Covariates and Predicted Dependent Variable
COVARIATE

VARIABLE	JUL_CD	BREED	SEX	WT0
DAYSENRL	.099	.189	.462	-.882

Squared Correlations between Covariates and Predicted Dependent Variable

VARIABLE	AVER. R-SQ
JUL_CD	.010
BREED	.036
SEX	.213
WT0	.778

Regression analysis for WITHIN + RESIDUAL error term
Individual Univariate .9500 confidence intervals
Dependent variable .. DAYSENRL

COVARIATE	B	Beta	Std. Err.	t-Value	Sig. of t
JUL_CD	.12683	.09555	.083	1.519	.130
BREED	-4.16395	-.37518	1.391	-2.993	.003
SEX	15.02681	.48220	6.315	2.380	.018
WT0	-.75760	-.35638	.152	-4.999	.000

Figure 7. Percentage of calves that had rotavirus antigens in their faeces by week of sampling (week 0 is the first sampling, ie calves 0 to 2 weeks of age).

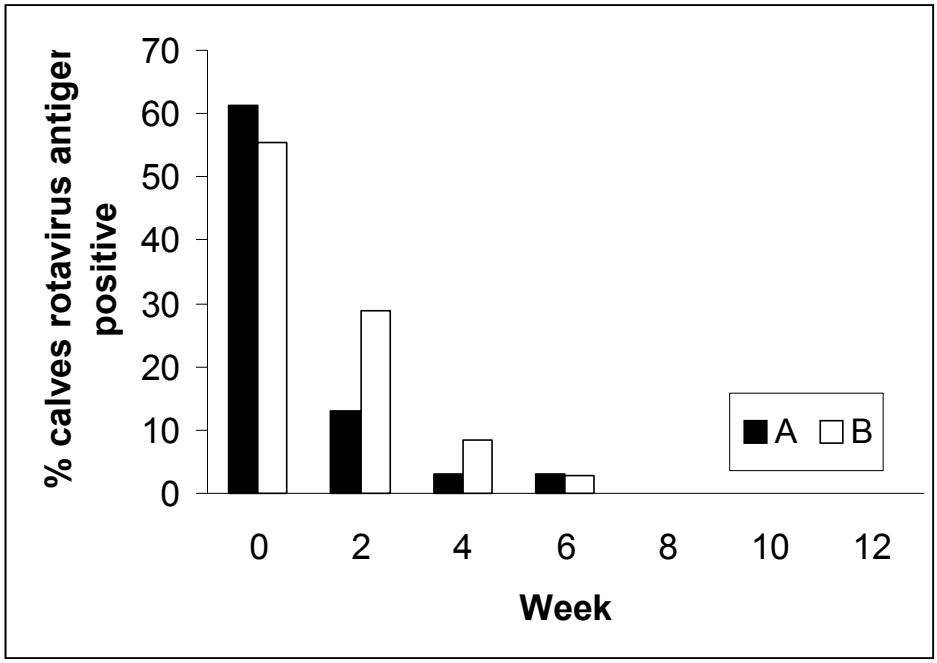

Figure 8. A boxplot of the rotavirus antigen scores by herd and group, at the second sampling after enrolment (week 2).