Effect of a Whey Protein Concentrate Used as a Colostrum Substitute or Supplement on Calf Immunity, Weight Gain, and Health

JOHN F. MEE,*,[1] KEVIN J. O'FARRELL,* PIETER REITSMA,† and RAJ MEHRA‡
*Teagasc, Moorepark Research Centre, Fermoy, County Cork, Ireland
†Agricultural University, Zodiac, Marijkeweg 40, Wageningen, The Netherlands
‡Teagasc, National Dairy Products Research Centre, Fermoy, County Cork, Ireland

ABSTRACT

The efficacy of a whey protein concentrate was evaluated as a colostrum substitute or supplement in two experiments using four groups of 29 calves. In Experiment 1, calves were fed either 2 L of pooled colostrum (group 1) or 500 g of whey protein concentrate (group 2). A mean total of 123.6 and 17.7 g of Ig was fed to calves in groups 1 and 2, respectively. Mean serum IgG, total protein, and globulin concentrations and Ig antibody activities to *Escherichia coli* K99 and rotavirus were significantly higher for calves in group 1 at 24 to 36 h and at 3 wk of age. Weight gain from birth to 3 wk of age was significantly lower for calves in group 2. The incidence of diarrhea was high but not different between treatments. The mortality rate (0 to 3 wk) was significantly higher for calves in group 2 (27.6%) than for calves in group 1 (3.4%). In Experiment 2, calves were fed either 2 L of pooled colostrum (group 3) or a solution of 1 L of pooled colostrum plus 500 g of whey protein concentrate (group 4). A mean total of 117.2 and 69.1 g of Ig was fed to calves in groups 3 and 4, respectively. Absorption rate of IgG was significantly lower for calves in group 4.

Mean serum IgG, total protein, and globulin concentrations and Ig antibody activities to *E. coli* K99 and rotavirus were significantly higher for calves in group 3 at 24 to 36 h and at 3 wk of age. Mortality rate, BW gain, and incidence of diarrhea did not differ significantly between groups.

(**Key words**: whey protein concentrate, calf immunity, weight gain, diarrhea)

Abbreviation key: **RV** = rotavirus, **TP** = total protein, **WPC** = whey protein concentrate.

INTRODUCTION

The epitheliochorial placentation of ruminants prevents in utero passage of Ig to the bovine fetus, which is born agammaglobulinemic. Hence, the newborn calf is dependent upon passive immunity from colostrum to prevent neonatal morbidity and mortality. Passive transfer of immunity may fail if an insufficient volume of colostrum or colostrum of inadequate quality (low in Ig content) is ingested or if colostrum is fed after cessation of intestinal absorption of macromolecules. Recent advances in milk processing technologies have led to the development and commercial production of colostrum substitutes and supplements (10, 12). These products are derived from ultrafiltration of bovine whey, dried colostrum, or blood serum. Despite their commercial availability, concerns have been expressed about their efficacy (29). Recent experiments with either dried colostrum powder (6, 28, 30) or a blend of lyophilized bovine colostrum and dried whey (1) have failed to demonstrate a significant increase in serum Ig concentrations or average daily gain or a significant decrease in calf morbidity or mortality compared with those for calves fed colostrum.

The whey protein concentrate (**WPC**) evaluated in this study (Colostrx™; Fisons' Animal Health, Leicestershire, England) is derived from ultrafiltrated and freeze-dried cheese whey collected from thousands of cows in North America. According to the manufacturer, Colostrx™ can be used as a supplement to poor quality colostrum or as a replacement for colostrum. Each dose (500 g) contains 30 g of Ig with the following profile: 72% IgG_1, 7% IgG_2, 10% IgA, and 11% IgM. The manufacturers of Colostrx™ claim that it contains specific antibodies against *Escherichia coli* K99, *Salmonella dublin*, *Clostridium perfringens*, *Brucella abortus*, *Haemophilus somnus*, infectious bovine rhinotracheitis virus, and rotavirus (**RV**). Published data from one trial (4) have shown that calves treated with Colostrx™ had better BW gain than and health status similar to calves fed good quality colostrum. In another study (20), in which the product was tested both as a replacer and as a

Received May 17, 1995.
Accepted January 9, 1996.
[1]Present address: Department of Agriculture, Food, and Forestry, Veterinary Research Laboratory, Abbotstown, Castleknock, Dublin 15, Ireland.

supplement, Colostrx™ could only be recommended in an emergency as a replacer, but could be of some help as a supplement to poor quality colostrum. However, in that study, the clinical history was only recorded for 5 d after birth, but diarrhea is one of the major causes of calf mortality during the first 2 wk of life (21). The objectives of this study were to determine the efficacy of this WPC when used as a colostrum substitute (Experiment 1) or a colostrum supplement (Experiment 2) when compared with pooled colostrum from unvaccinated cows in terms of preventing calf morbidity and mortality. Particular emphasis was placed on diarrhea of neonatal calves during the first 3 wk of life.

MATERIALS AND METHODS

Experimental Design

In Experiment 1, 58 healthy dairy male and female calves were assigned randomly to treatment groups according to parity of the dam, calf sire breed, sex, and calving date. This experiment was conducted using calves born during January and February on a dairy research farm with spring calving. Calves were drenched by esophageal tube within 30 min of calving as follows: group 1 (n = 29), 2 L of warm, pooled colostrum collected at the first milking postcalving from at least 4 dams; and group 2 (n = 29), 500 g of WPC (Colostrx™) mixed with 1.2 L of lukewarm water.

In Experiment 2, 58 healthy dairy male and female calves were randomly assigned to treatment groups by parity of the dam, calf sire breed, sex, and calving date. This experiment was conducted using calves born during February and March. Calves were fed by esophageal tube within 30 min of calving as follows: group 3 (n = 29), 2 L of warm, pooled colostrum; and group 4 (n = 29), 500 g of WPC (Colostrx™) in 1 L of lukewarm water mixed with 1 L of pooled colostrum.

Calf Management

All calvings were monitored using a closed circuit television system (model 12TX 3412; Philips Electrical Ltd., Newstead, Clonskeagh, Dublin, Ireland). Cows calved in tie stalls or loose pens. Tincture of iodine was used as an umbilical antiseptic immediately after calving. After feeding, a muzzle was placed on each calf to prevent suckling, and the calf was left with the dam for at least 3 h. Calves were then moved into pens in the calf house; the maximum was 4 calves per pen. Pens had slatted, sloped floors and straw bedding; an equal number of control and treated calves were mixed in each pen. After the initial drenching, calves were fed 2 L of fresh whole milk twice daily from a bucket. This process continued until the calves were 2 wk of age at which stage they were moved into a larger pen containing 8 to 10 calves. Calves were then fed through an automatic feeder (Associated Steel Engineering Ltd., Liscarroll, Mallow, County Cork, Ireland), which gave them ad libitum access to warm, acidified whole milk. The acidifier used was a solution of citric acid; vitamins A, E, and D; and flavophospholipol (Milk Shake; In-Form Nutrition Ltd., Whites Cross, County Cork, Ireland). Calves were weighed at birth and 3 wk later. Calves were monitored several times daily for signs of illness. Diarrhea was diagnosed as defined by Greene and Bakheit (9).

Analysis

A sample was collected from each first feeding. Concentration of IgG in whey was measured by single radial immunodiffusion (8). The anti-*E. coli* K99 and anti-RV Ig activities in whey were determined using a competitive solid-phase ELISA (17). Immunoglobulin activity was expressed as a percentage of that activity in a reference standard (whey from a lactating cow vaccinated with Rotavec K99; Mallinckaedt Veterinary, Dublin, Ireland) that was set at 100%. Blood samples were collected from the jugular vein of each calf before first feeding, between 24 and 36 h later ($\bar{X}$ ± SEM = 26 ± 0.3 h), and at 21 d of age. Serum IgG concentration and anti-*E. coli* K99 and anti-RV Ig activities were determined as outlined previously. Serum total protein (**TP**) and albumin concentrations were measured using kits (total protein and albumin kits; Ciba-Corning Diagnostics Ltd., Essex, England) on a random access analyzer (Cobas MIRA; Hoffmann-La Roche, Basel, Switzerland). Serum globulin concentration was calculated by subtracting the albumin from the TP concentration. Blood samples that were collected from calves fed WPC at 1 (n = 38), 2 (n = 31), 3 (n = 26), 4 (n = 19), and 5 wk (n = 12) after calving were examined for the presence of Ig to *B. abortus* by the serum agglutination test, complement fixation test, and Rose-Bengal test (2). Fecal samples (n = 45) that were collected from calves with diarrhea were examined for the presence of RV using a latex agglutination test (Rotascreen M80; Mercia Diagnostics Ltd., Salford Surrey, England) and electron microscopy, for cryptosporidial oocysts by a modified Ziehl-Neelsen stain technique (11), for bacterial pathogens by direct culture and enrichment techniques (7), and, in the case of *E. coli*, using the slide

agglutination test and serotyping. Twenty-nine isolates were examined by the plate sensitivity test to determine their in vitro sensitivity to 16 commonly used antimicrobial agents. A complete necropsy examination was carried out for each calf (n = 14) that died during both experiments within 36 h of death (7.5 ± 2.3 h).

Statistical Analysis

Incidence was defined as the number of calves developing a particular disorder or dying during an experimental period divided by the number of calves at risk during that period. Prevalence was calculated by multiplying incidence by duration (16). Because the prevalence and duration of diarrhea were not normally distributed, the Mann-Whitney Wilcoxon U test was used. The frequency distribution for morbidity and mortality and the results of fecal analyses were analyzed using the chi-square test. The IgG absorption rate at 24 to 36 h after calving was calculated using the formula developed by Kim and Schmidt (13).

An ANOVA was used to compare BW gains, feed and serum Ig content and activity, serum TP, albumin, and globulin content. Analyses were conducted using the general linear models procedure of SAS (22). Differences between treatment means were determined using the F test; differences were considered to be significant at $P < 0.05$. All statistical comparisons were made within, not between, experiments. Data are presented as the mean and the standard error of the mean, where appropriate, in text and tables.

RESULTS

Mortality rate for calves in group 2 was significantly higher than for calves in group 1, but no significant difference in the mortality rate existed between groups in Experiment 2 (Table 1). During Experiment 1, 9 calves died, 1 in group 1 and 8 in group 2. The calf in group 1 died at 8 d of age from severe diarrhea and dehydration; no significant pathogens were isolated. Seven calves in group 2 died from severe diarrhea and dehydration between 4 and 10 d of age. Rotavirus was detected in fecal samples from 6 of these calves. The 8th calf in group 2 died from polyserositis, arthritis, and meningitis at 7 d of age; no significant pathogens were isolated. Five calves died in Experiment 2, 4 in group 3, and 1 in group 4. One calf in group 3 died from abomasal dilation and volvulus at 10 d of age, 1 died from polyserositis and partial atelectasis at 1 d of age, and 1 died from severe diarrhea and dehydration at 7 d of age. No pathological diagnosis was made on the fourth calf in group 3 that died at 9 d of age; RV was detected in a fecal sample from that calf. The single calf that died in group 4 died at 7 d of age from severe diarrhea and dehydration. Twelve of the 14 calves that died had diarrhea.

The incidence of diarrhea between groups within either experiment was not significantly different (Table 2). The mean duration of days that calves were affected by diarrhea between group 1 (2.0 ± 0.2 d) and group 2 (3.6 ± 0.5 d) or between group 3 (3.7 ± 0.3 d) and group 4 (4.1 ± 0.3 d) was not significantly different. The prevalence (calf-days) of diarrhea was greater for group 2 (93.9 d) than for group 1 (42.9 d) and was greater for group 4 (119.0 d) than for group 3 (103.9 d). The percentage of calves requiring intravenous electrolyte therapy was not significantly different between group 1 (10.3%) and group 2 (24.1%) or between group 3 (10.3%) and group 4 (0%). The percentage of fecal samples that were positive for RV or *Cryptosporidium* between group 1 (RV, 62.5%; *Cryptosporidium*, 12.5%) and group 2 (RV, 68.2%; *Cryptosporidium*, 4.5%) and between group 3 (RV, 37.5%; *Cryptosporidium*, 12.5%) and group 4 (RV, 42.9%; *Cryptosporidium*, 14.3%) was not significantly different. No pathogenic bacteria were detected in any of the 45 fecal samples examined.

Concentrations of serum albumin prefeeding were similar for both groups in Experiment 1 (23.3 ± 0.4 vs. 23.3 ± 0.4 mg/ml), but in Experiment 2, group 4 had higher ($P < 0.01$) concentrations than did group 3 (23.2 ± 0.5 vs. 21.3 ± 0.5 mg/ml). However, in both experiments, concentrations of serum albumin postfeeding were not significantly different between calves fed WPC and those fed colostrum at either 24 to 36 h (Experiment 1: 24.4 ± 0.4 vs. 22.1 ± 0.4 mg/ml; Experiment 2: 21.0 ± 0.5 vs. 21.3 ± 0.5 mg/ml) or 3 wk of age (Experiment 1: 25.3 ± 0.4 vs. 25.3 ± 0.5 mg/ml; Experiment 2: 24.5 ± 0.4 vs. 24.3 ± 0.4 mg/ml). Although there was no consistent trend in concentrations of serum albumin between birth and 24 to

TABLE 1. Mortality rate (0 to 3 wk of age).

Experimental group	Calves		P
	(no.)	(%)	
Experiment 1			
Group 1	1	3.5	
Group 2	8	27.6	*
Experiment 2			
Group 3	4	13.8	
Group 4	1	3.5	NS

*$P \leq 0.05$.

TABLE 2. Incidence of diarrhea.

Experimental group	Diarrhea incidence				P
	0 to 3 d		4 d to 3 wk		
	(no.)	(%)[1]	(no.)	(%)	
Experiment 1					
Group 1	2	6.9	20	69.0	
Group 2	2	6.9	24	83.0	NS[2]
Experiment 2					
Group 3	3	10.3	26	89.7	
Group 4	2	6.9	27	93.1	NS

[1]Percentage of the total calves in the group.

[2]$P > 0.05$.

36 h of age, concentrations at 3 wk of age were greater than those at birth.

Concentrations of serum TP prefeeding were similar for both groups in Experiment 1, but, in Experiment 2, group 4 had higher ($P < 0.01$) concentrations than did group 3. However, in both experiments, groups 2 and 4 had significantly lower concentrations of serum TP than did groups 1 and 3 between 24 to 36 h postfeeding and at 3 wk of age (Table 3).

Serum globulin, IgG concentration and anti-*E. coli* K99 and anti-RV Ig activities prefeeding did not differ between groups within either experiment (Tables 4, 5, 6, and 7). However, at 24 to 36 h and at 3 wk of age, these measurements in calves fed WPC were significantly lower than those in calves fed colostrum only in both experiments (Tables 4, 5, 6, and 7). The concentrations of serum IgG of calves in groups 1, 2, 3, and 4 at 24 to 36 h after calving represent, on average, 28.8, 20.4, 31.4, and 27.6% of the respective average IgG concentration of the first feeding (absorption efficiency) (Tables 5 and 8). Anti-*B. abortus* Ig were not detected in any of the 126 sera that were examined by the three serological assays.

In both experiments, the feeds containing WPC had significantly lower IgG concentrations and anti-*E. coli* K99 and anti-RV Ig activity than those of the colostrum only (Table 8). In Experiment 1, the mean absorption rate of IgG was similar for both groups of calves (17.3 ± 2.3% vs. 11.9 ± 2.3%), but, in Experiment 2, group 4 had a lower ($P < 0.01$) mean absorption rate (14.2 ± 1.2% vs. 19.2 ± 1.2%).

Mean BW gain from birth to 3 wk of age was significantly higher for calves in group 1 (5.6 ± 1.0 kg) than for those in group 2 (2.1 ± 1.2 kg). There was no significant difference in BW gain between group 3 (3.8 ± 1.1 kg) and group 4 (2.3 ± 1.0 kg) in Experiment 2.

DISCUSSION

Calves fed the WPC alone had a higher mortality rate and prevalence of diarrhea and a lower concen-

TABLE 3. Serum total protein concentration prefeeding (0 h), between 24 to 36 h, and at 3 wk of age.

Experimental group	0 h		24 to 36 h		3 wk	
	(mg/ml)					
	$\bar{X}$	SEM	$\bar{X}$	SEM	$\bar{X}$	SEM
Experiment 1						
Group 1	42.9	0.5	53.72	1.0	52.4	0.7
Group 2	43.3	0.5	44.0	1.0	49.3	0.9
P	NS[1]		***		**	
Experiment 2						
Group 3	39.4	0.8	55.0	1.5	49.4	0.7
Group 4	42.6	0.8	49.3	1.5	46.4	0.7
P	**		**		**	

[1]$P > 0.05$.

**$P \leq 0.01$.

***$P \leq 0.001$.

TABLE 4. Serum globulin concentration prefeeding (0 h), between 24 to 36 h, and at 3 wk of age.

Experimental group	0 h		24 to 36 h		3 wk	
	(mg/ml)					
	$\bar{X}$	SEM	$\bar{X}$	SEM	$\bar{X}$	SEM
Experiment 1						
Group 1	19.8	0.3	32.3	0.9	27.1	0.6
Group 2	20.0	0.3	21.9	0.9	24.0	0.6
P	NS[1]		***		***	
Experiment 2						
Group 3	18.1	0.6	34.0	1.4	24.6	0.7
Group 4	19.5	0.6	28.0	1.4	22.1	0.6
P	NS		**		**	

[1]$P > 0.05$.

**$P \leq 0.01$.

***$P \leq 0.001$.

TABLE 5. Serum IgG concentration prefeeding (0 h), between 24 to 36 h, and at 3 wk of age.

Experimental group	0 h		24 to 36 h		3 wk	
	(mg/ml)					
	$\bar{X}$	SEM	$\bar{X}$	SEM	$\bar{X}$	SEM
Experiment 1						
Group 1	0.4	0.1	17.8	0.5	8.0	0.4
Group 2	0.3	0.1	3.0	0.5	4.3	0.4
P	NS[1]		***		***	
Experiment 2						
Group 3	0.4	0.2	18.4	1.0	9.1	0.7
Group 4	0.6	0.2	9.5	1.0	6.5	0.7
P	NS		***		**	

[1]$P > 0.05$.

**$P \leq 0.01$.

***$P \leq 0.001$.

TABLE 6. Serum anti-*Escherichia coli* K99 Ig activity prefeeding (0 h), between 24 to 36 h, and at 3 wk of age.

Experimental group	0 h		24 to 36 h		3 wk	
	(%)[1]					
	$\bar{X}$	SEM	$\bar{X}$	SEM	$\bar{X}$	SEM
Experiment 1						
Group 1	0.1	0.1	11.7	1.9	39.7	5.4
Group 2	0.2	0.1	3.2	1.9	1.3	6.2
P	NS[2]		**		***	
Experiment 2						
Group 3	0.1	0.1	159.8	22.8	81.2	12.4
Group 4	0	0	43.6	22.8	18.6	11.7
P	NS		***		***	

[1]Reference standard for Ig activity set at 100%.

[2]$P > 0.05$.

**$P \leq 0.01$.

***$P \leq 0.001$.

TABLE 7. Serum anti-rotavirus Ig activity prefeeding (0 h), between 24 to 36 h, and at 3 wk of age.

Experimental group	0 h		24 to 36 h		3 wk	
	(%)[1]					
	$\bar{X}$	SEM	$\bar{X}$	SEM	$\bar{X}$	SEM
Experiment 1						
Group 1	2.8	1.2	1625	173.1	770	154
Group 2	0.7	1.2	103	173.1	285	178
P	NS[2]		***		*	
Experiment 2						
Group 3	0	0	870	103	609	78
Group 4	0.1	0.1	366	103	221	75
P	NS		**		***	

[1]Reference standard for Ig activity set at 100%.

[2]$P > 0.05$.

*$P \leq 0.05$.

**$P \leq 0.01$.

***$P \leq 0.001$.

tration of serum IgG postfeeding than did calves fed colostrum (Table 1). In one of the other published reports (23) on this product during a long-term (46 d) experiment, differences in calf health were not found between calves fed WPC and calves fed colostrum. However, this apparent discrepancy might have been caused by pooled colostrum in our study. Colostrum in the previous study (23) was collected from one dam. Also, in our study, calves received colostrum for only one feeding, but, in the study of Seymour et al. (23), calves received colostrum through d 4. Additionally, the absence of calf mortality in either group in their study (23) suggests very low infectious challenge. Pickel et al. (20) reported lower concentrations of serum IgG postfeeding but no difference in the health of calves fed this WPC compared with calves fed colostrum. However, the 25 calves were only monitored over 5 d, and no deaths were reported. In the present study, the majority of calf deaths occurred after d 5.

In Experiment 2, despite the significant differences in concentrations of serum IgG between treatments postfeeding (Table 5), there was no significant difference in calf health. Similarly, Bambauer (4) reported no difference in calf health over 31 d for calves fed either good quality colostrum (>50 mg of Ig/ml) or poor quality colostrum (0.5 L; <50 mg of Ig/ml) plus Colostrx™ (2 L; 500 g). Calf health was poorer for calves fed poor quality colostrum (0.5 L; <50 mg Ig/ml) than for calves fed good quality colostrum.

TABLE 8. The IgG concentration and anti-*Escherichia coli* K99 and anti-rotavirus Ig activities of the first feeding.

Experiment	IgG		Anti-*E. coli* K99 Ig activity		Anti-rotavirus Ig activity	
	(mg/ml)		(%)[1]			
	$\bar{X}$	SEM	$\bar{X}$	SEM	$\bar{X}$	SEM
Experiment 1						
Group 1	61.8	1.5	1331	265	12,421	978
Group 2	14.7	1.5	2	220	714	890
P	***		***		***	
Experiment 2						
Group 3	58.6	2.1	4397	724	24,799	2030
Group 4	34.6	2.1	2038	656	15,046	1799
P	***		*		***	

[1]Reference standards for Ig activities set at 100%.

*$P \leq 0.05$.

*** $P \leq 0.001$.

Although significant differences in calf health were not detected in Experiment 2, the significantly higher concentration of serum IgG postfeeding for calves fed colostrum suggests better systemic immunity of these calves.

The efficiency of absorption of Ig from colostrum is a function of the mass of Ig fed, the age at colostrum feeding, and the birth weight of the calf. In these experiments, the age at colostrum feeding (<30 min after calving) was standardized, and calf birth weight, which did not differ between groups within each experiment, was included in the equation that was used to calculate absorption rate of Ig (13). The lower absorption rate and absorption efficiency of Ig from WPC feeds compared with colostrum only might be explained by the manufacturer's hypothesis that a new IgG_1 complex or aggregate existed in this WPC, which was not absorbed but remained in the intestinal lumen (12). The mean absorption rates of IgG from colostrum reported here (group 1, 17.3%; group 3, 19.2%) are greater than those reported by Kim and Schmidt (13) for beef calves (12.7%). The mean absorption efficiencies of IgG from colostrum (29 to 31%) are within the ranges reported by Lopez et al. (15) (2 to 46%).

Measurement of blood TP is one of the most convenient methods of indirectly evaluating the humoral immune status of calves because significant correlations exist among blood TP content, blood IgG concentration (19), and the risk of neonatal disease. The significantly higher serum TP content of calves fed colostrum only reflects both the higher TP content of colostrum compared with WPC and the lower absorption rate of IgG from the latter. Researchers (18, 19) have suggested that threshold serum TP concentrations (42 to 55 mg/ml) assist in the diagnosis of failure of passive transfer of immunity and are predictors of disease risk. The results of these experiments (Table 3) suggest the higher cut-off value (55 mg/ml) might be more relevant under the present study conditions.

In this study, the BW gain from birth to 3 wk of age for calves in group 1 was higher than for calves in group 2. This result differs from the findings of Seymour et al. (23), who observed no difference in BW gain between the two groups. This difference may be explained by the different feeding regimens following the first feeding. In the investigation of Seymour et al. (23), calves were given either the colostrum from the dam (range, 12 to 137 mg of Ig/L) or WPC (Colostrx™) at first feeding. All calves received colostrum from the second feeding through d 4 of life. Continued feeding of colostrum during this period probably conferred enteric immunity on both groups of calves; hence, differences between treatments in average daily gain would not be expected. This feeding regimen might have overcome or diluted the individual effect of WPC at the first feeding. Bambauer (4) found higher BW gain in calves fed WPC plus colostrum than in calves fed colostrum only. This result was also contrary to our finding that BW gain did not differ between calves in groups 3 and 4. Bambauer's report (4) is not clear as to the quantity of colostrum fed to the calves that received colostrum only or as to what was fed to the calves after the first feeding. Hence, the difference in results between the two studies cannot be reasonably explained.

Diarrhea and severe dehydration were the primary causes of calf mortality and accounted for the significant difference between treatments in mortality rate in Experiment 1. All diarrheic calves died between 4 to 10 d after calving, the peak period of loss from RV infection (14). This period is consistent with a history of inadequate passive immunity, the short viral incubation period (18 to 96 h), and rapid onset of clinical signs with RV infection (2 to 3 h) (14).

As expected from the results of the feed IgG concentration and activities and the absorption rate calculations, mean serum globulin, IgG concentration, and anti-*E. coli* K99 and anti-RV Ig activities were greater in calves fed colostrum only than in calves fed WPC (Table 8). The mean concentrations of serum IgG postfeeding found in calves fed colostrum only were similar to the 14.9 mg/ml reported by Stott et al. (25) for dairy calves that were artificially fed 2 L of colostrum immediately after birth. The concentrations of serum IgG of groups 1 and 3 at 24 to 36 h after calving reached 80 to 90% of the maximum IgG absorption achievable (26) for the amount of colostrum fed. Mean concentration of serum IgG of calves in group 2 at 24 to 36 h after calving (3.0 mg/ml) was well below the maximum acceptable concentration required to achieve adequate performance of the immune system of the calf (24). Similar results for serum IgG concentrations before feeding and at 24 and 48 h of age were reported by Pickel et al. (20). The serum anti-*E. coli* K99 and anti-RV activities of groups 1, 3, and 4 at 24 to 36 h after calving were much higher than those of group 2. The class of Ig responsible for the activity detected against *E. coli* K99 and RV was not determined in this study. Hence, the absorption pattern of IgG did not necessarily explain the absorption pattern of the pathogen-specific Ig. However, this pattern might be due to the presence of undetected pathogen-specific IgM activity. More than 50% of colostral Ig to *E. coli* K99 in some

cows have been shown to be the IgM class (27). Although the WPC fed here contained anti-*B. abortus* Ig (3) and although these could affect interpretation of brucellosis serology, no Ig to *B. abortus* were detected in the sera from calves fed WPC.

The IgG concentration measured during the first feeding of calves fed WPC (Table 8) was within the broad range of values (6.0 to 16.0 mg/ml) published for this product in other studies (3, 5, 10, 12). The amount of total IgG given to calves fed WPC (17.7 g) was about 65% of the amount of Ig (27 g) that appears to represent a therapeutically effective dosage; such a dosage would be able to achieve immune system performance comparable with that attainable through use of natural colostrum (24). Comparison of the anti-*E. coli* K99 and anti-RV activities (specific antibodies to the stated pathogens) of WPC and colostrum feeds demonstrated that the WPC feed possessed insignificant pathogen-specific antibodies in relation to colostrum. No such data are available for comparison in other reported studies. Overall, the WPC feed alone appeared to have inadequate concentrations of Ig and specific antipathogen activities that are essential for its beneficial effect as a colostrum substitute as claimed by the manufacturer.

CONCLUSIONS

The results of this study have shown that the efficacy of WPC, used as a colostrum substitute and administered as a single feeding, was poor in preventing neonatal morbidity and mortality compared with a single feeding of pooled colostrum. Calves fed colostrum had significantly higher serum Ig concentration, pathogen-specific activities, and average daily gain and significantly lower mortality between birth and 3 wk of age. When WPC was used as a colostrum supplement, calves had similar BW gains and mortality rates, but their immune status was significantly lower than for calves fed colostrum alone. This result suggests a potentially greater risk of disease for calves supplemented with WPC.

ACKNOWLEDGMENTS

The authors acknowledge the technical assistance of T. Condon for field work, C. Jones and A. Madden for laboratory work, and D. Harrington for statistical work. J. F. Mee was financed through the Dairy Levy Fund by Irish farmers. The financial assistance provided by Fisons' Animal Health, Loughborough, Leicestershire, England, is gratefully acknowledged.

REFERENCES

1 Abel Francisco, S. F., and J. D. Quigley, III. 1993. Serum immunoglobulin concentrations after feeding maternal colostrum or maternal colostrum plus colostral supplement to dairy calves. Am. J. Vet. Res. 54:1051.
2 Alton, G. G., L. M. Jones, and D. E. Pietz. 1975. Laboratory Techniques in Brucellosis. 2nd ed. World Health Org. Monogr. Series No. 55. World Health Org., Geneva, Switzerland.
3 Andrews, A. H. 1990. Colostrum. Part of nature's survival kit. Feed Compounder 10:22.
4 Bambauer, C. 1988. Zum Einsatz des Spezialfuttermittels Colostrx® bei neugeborener Kalbern. Tierarztl. Hochsch., Diss. Ludwig-Maximilians-Universitat, Munich, Germany.
5 Beyer, C. 1988. Gesundheitszustand und Immunstatus neugeborener Kalber nach Gabe des Molkeneiweißpulvers Colostrx® Tierarztl. Hochsch., Diss. Hannover, Germany.
6 Chelack, B. J., P. S. Morley, and D. M. Haines. 1993. Evaluation of methods for dehydration of bovine colostrum for total replacement of normal colostrum in calves. Can. Vet. J. 34:407.
7 Fagan, J. G., P. J. Dwyer, and J. F. Quinlan. 1994. The diagnosis and occurrence of enteropathogens associated with calf diarrhoea in Ireland. Ir. Vet. J. 47:313.
8 Fahey, F. L., and E. M. McKelvey. 1965. Quantitative determination of serum immunoglobulins in antibody-agar plates. J. Immunol. 94:84.
9 Greene, H. J., and H. A. Bakheit. 1984. A study of the aetiology, epidemiology and control of diarrhoea in Ireland. Ir. Vet. J. 38: 63.
10 Haines, D. M., B. J. Chelack, and J. M. Naylor. 1990. Immunoglobulin concentrations in commercially available colostrum supplements for calves. Can. Vet. J. 31:36.
11 Henriksen, S. A., and J.F.L. Pohlenz. 1981. Staining of cryptosporidia by a modified Ziehl-Neelsen technique. Acta Vet. Scand. 22:594.
12 Hunt, E., S. D. Van Camp, and S. Fleming. 1988. Developing technologies for prevention of bovine failure of passive transfer of antibody (FPTA). Bovine Pract. 23:131.
13 Kim, J. W., and F. W. Schmidt. 1983. Zur Frage der Absorption von Kolostralen Immunglobulinen durch das Kalb. Dtsch. Tierärztl. Wochenschr. 90:283.
14 Lester, T. L. 1993. Bovine rotavirus. Page 439 *in* Current Veterinary Therapy. J. L. Howard, ed. W. B. Saunders Co., Philadelphia, PA.
15 Lopez, J. W., S. D. Allen, J. Mitchell, and M. Quinn. 1988. Rotavirus and cryptosporidium shedding in dairy calf feces and its relationship to colostrum immune transfer. J. Dairy Sci. 71: 1288.
16 Martin, S. W., A. H. Meek, and P. Willberg. 1987. Veterinary Epidemiology. Principles and Methods. Iowa State Univ. Press, Ames.
17 McEnroe, M.A.M. 1989. Studies on antimicrobial proteins in bovine whey. M.S. Thesis. Natl. Univ. Dublin, Ireland.
18 Naylor, J. M., D. S. Kronfeld, S. Bech-Nielsen, and R. C. Bartholomew. 1977. Plasma total protein measurement for prediction of disease and mortality in calves. JAVMA (J. Am. Vet. Med. Assoc.) 171:635.
19 Perino, L. J., R. L. Sutherland, and N. E. Woollen. 1993. Serum γ-glutamyltransferase activity and protein concentration at birth and after suckling in calves with adequate and inadequate passive transfer of immunoglobulin G. Am. J. Vet. Res. 54:56.
20 Pickel, M., C. Beyer, E. Grunert, and G. Trautwein. 1991. Gesundheitszustand und Immunoglobulinstatus beim neugeborenen Kalb nach Verabreichung des Molkeneiweißpulvers Colostrx®. Tieraerztl. Umsch. 46:210.
21 Roy, J.H.B. 1990. Management of health. Page 53 *in* The Calf. Vol. 1, 5th ed. Butterworths, London, England.
22 SAS® User's Guide: Statistics, Version 5 Edition. 1985. SAS Inst., Inc., Cary, NC.
23 Seymour, W. H., J. E. Nocek, J. E. English, and J. Siciliano-Jones. 1989. Effect of a whey protein concentrate (Colostrx®)

and brewer's yeast-35 on health and performance of Holstein calves. J. Dairy Sci. 72(Suppl. 1):414.(Abstr.)
24 Stott, G. H., and D. O. Lucas, inventors. 1987. Immunologically active whey fraction and recovery process. Eur. Pat. Appl. Cl. No. A23 J 1/20.
25 Stott, G. M., D. B. Marx, B. E. Menefee, and G. T. Nightengale. 1979. Colostral immunoglobulin transfer in calves. III. Amount of absorption. J. Dairy Sci. 62:1902.
26 Stott, G. M., D. B. Marx, B. E. Menefee, and G. T. Nightengale. 1979. Colostral immunoglobulin transfer in calves. IV. Effect of suckling. J. Dairy Sci. 62:1908.
27 Trainin, Z, and H. Ungar-Waron. 1981. IgM in the bovine immune system and its relevance to disease. Page 169 *in* The Ruminant Immune System. J. E. Butler, K. Nielsen, and J. R. Duncan, ed. Plenum Press, New York, NY.
28 White, D. G. 1993. Colostral supplementation in ruminants. Compend. Contin. Educ. Pract. Vet. 15:335.
29 White, D. G., and A. J. Wagstaff. 1990. Colostrum supplements and substitutes. Vet. Rec. 127:483.
30 Zaremba, W., W. M. Guterbock, and C. A. Holmberg. 1993. Efficacy of a dried colostrum powder in the prevention of disease in neonatal Holstein calves. J. Dairy Sci. 76:831.