RESEARCH UPDATE: STARCH LEVEL AND RUMENSIN IN FRESH COW RATIONS

M. M. McCarthy,[1] T. Yasui,[1] C. M. Ryan,[1] G. D. Mechor,[2] and T. R. Overton[1]
[1]Cornell University Department of Animal Science
[2]Elanco Animal Health, Greenfield, IN

INTRODUCTION

Many studies have evaluated the effect of prepartum nutrition on postpartum lactation performance and its associated metabolic changes. Surprisingly there have been relatively few studies that have evaluated the effects of postpartum nutrition on these metabolic adaptations and their effects on production performance.

After parturition the nutrients required for milk synthesis utilize a large portion of maternal nutrients (Bauman and Currie, 1980). In the immediate postpartum period dry matter intake (DMI) is insufficient to support the high milk production of early lactation and results in increased mobilization of adipose tissue and the oxidation of non-esterified fatty acids (NEFA) by the liver. Higher DMI generally results in lower circulating NEFA and has been associated with improved health and performance (Ingvartsen and Andersen, 2000).

Optimizing DMI during this postpartum period is especially important to provide sufficient energy to support milk production. Due to the increased glucose demand for milk lactose synthesis, liver glucose production nearly doubles within 11 days of calving compared to prepartum glucose output (Reynolds et al., 2003). Propionate that is produced via fermentation of starch in the rumen is the main precursor for liver glucose production. Rumensin also has been shown to increase ruminal propionate production (Armentano and Young, 1983). While there is a large increase in the liver's utilization of lactate, glycerol, and the glucogenic amino acids postpartum, propionate is still quantitatively the greatest contributor to liver gluconeogenesis at about 60% of precursor supply (Reynolds et al., 2003). Because of this increased demand for glucose postpartum, the liver should have the capacity to direct any additional propionate supply towards glucose synthesis during this early postpartum period (Drackley et al., 2001).

Allen et al. (2009) proposed that liver energy status is a major regulator of DMI in dairy cows. The premise is that when oxidative fuel metabolism (mainly propionate, but also NEFA) by the liver exceeds energy requirements, the brain is signaled to reduce DMI. This hepatic oxidation theory would suggest that feeding diets that would increase propionate supply (e.g. greater amounts or fermentability of starch, addition of Rumensin) during early lactation would decrease DMI via this liver signaling mechanism. If the hepatic oxidation theory applies in this manner to the early lactation period, then reducing the dietary starch content during this period would likely increase DMI by reducing propionate production in the rumen and decreasing the hypophagic effect from propionate oxidation (Allen et al., 2009). However, because liver energy requirements increase dramatically at the onset of lactation (Reynolds et al., 2003) and adipose mobilization is increased (Vernon, 2005), we believe that NEFA are likely to be

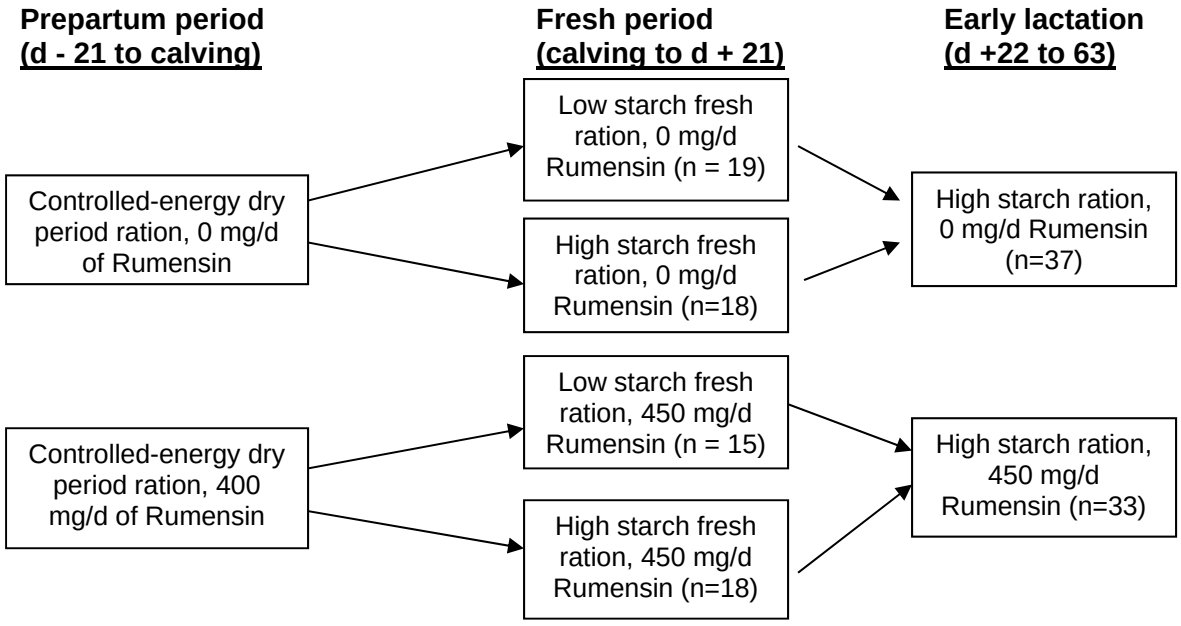
the predominant oxidative fuel for the liver. Recent work from the Allen lab with early lactation animals has shown propionate infusion to be more hypophagic in animals with higher liver acetyl CoA concentrations, which is indicative of higher NEFA mobilization (Stocks and Allen, 2012; 2013). We believe any hypophagic effect of propionate is likely to be reduced immediately after calving because of the large increases in liver energy demands postpartum (Overton, 2011), although, the effect of propionate load in early lactation is still in debate.

The objectives of this research were to further investigate these effects of propiogenic diets on intake, production, energy metabolism, and liver propionate metabolism in early lactation dairy cows.

EXPERIMENTAL APPROACH

A total of 70 Holstein cows (n= 49 multiparous and n=21 primiparous) were enrolled in this experiment at 21 d before expected calving and fed a controlled energy diet based upon corn silage, wheat straw, and a concentrate mix and topdressed daily with a pellet containing either 0 or 400 mg/d of Rumensin. After calving, cows were further randomized within Rumensin treatment and fed either a 26.2% starch (HS) or 21.5% starch (LS) fresh cow diet with Rumensin administration continuing at either 0 or 450 mg/d by daily topdress pellet (Figure 1). From d 22 through 63 all cows were fed the HS diet with their assigned daily Rumensin topdress.

Figure 1. Experimental design treatment schematic.



The postpartum diets were formulated on the basis of a lactation diet in which BMR corn silage was the predominant forage, with smaller amounts of wheat straw and haylage (Table 1). The concentrate portion of the HS diet was based on ground corn grain (20.1% of diet DM). For the LS diet corn grain (9.8% of diet DM) was partially

replaced with citrus pulp (6.5% of diet DM) and soy hulls (3.4% of diet DM). The HS and LS diets were formulated to contain 28.0 and 21.0% starch, respectively, although, the analyzed starch content of the HS and LS diets were 26.2 and 21.5%. The analyzed starch content of the HS diet was lower than expected; however, the difference between the two diets is still large enough that it makes for a meaningful comparison.

Table 1. Ingredient and nutrient content of experimental diets (DM basis)

Item	Prepartum	Postpartum	
		High Starch	Low Starch
Ingredient, % of DM			
Corn silage	39.5	–	–
BMR corn silage	–	37.0	37.0
Haylage	–	9.3	9.3
Wheat straw	20.5	11.1	11.1
Corn grain	3.8	20.1	9.8
Corn germ meal	–	2.3	5.4
Citrus pulp	6.6	0.8	6.5
Soy hulls	6.6	–	3.4
Soybean meal	5.0	5.5	3.6
Canola meal	4.3	2.5	2.0
Blood meal	1.0	1.9	1.9
Minerals and vitamins[1]	6.6	5.3	5.8
Topdress	6.1	4.2	4.2
Chemical Analaysis			
DM, %	50.7 ± 2.4	48.3 ± 2.7	48.0 ± 3.2
CP, %	13.0 ± 0.8	15.5 ± 1.2	15.4 ± 0.8
ADF, %	28.2 ± 1.2	22.7 ± 1.2	25.2 ± 1.2
NDF, %	42.9 ± 2.0	34.3 ± 1.5	36.9 ± 1.5
30 h NDFD, % of NDF	–	55.1 ± 2.0	56.1 ± 1.4
Sugar, %	4.9 ± 0.8	3.5 ± 0.6	4.5 ± 0.4
Starch, %	17.4 ± 1.2	26.2 ± 1.2	21.5 ± 1.0
Fat, %	2.6 ± 0.2	4.0 ± 0.2	2.2 ± 0.6
Calcium, %	1.28 ± 0.16	0.94 ± 0.09	1.01 ± 0.04
Phosphorous, %	0.30 ± 0.02	0.34 ± 0.02	0.34 ± 0.02
Magnesium, %	0.41 ± 0.04	0.28 ± 0.02	0.3 ± 0.03
Potassium, %	1.12 ± 0.13	1.12 ± 0.09	1.18 ± 0.08
Sulfur, %	0.37 ± 0.04	0.21 ± 0.09	0.22 ±0.01
Sodium, %	0.12 ± 0.02	0.47 ±0.08	0.46 ± 0.05
Chloride, %	0.37 ± 0.01	0.44 ± 0.04	0.44 ± 0.03

[1]Contained 30,317 mg/kg of Cu, 136,466 mg/kg of Mn, 3,393 mg/kg of Cot, 3,040 mg/kg of I, and 153,916 mg/kg of Zn, 30,464 IU/kg of Vitamin A, 5,862 IU/kg of Vitamin D, and 93,784 IU/kg of Vitamin E, 510,750 IU/kg of Vitamin E.

Samples of all TMR and ration ingredients were obtained weekly and composited at 4 wk intervals for analysis of chemical composition using wet chemistry techniques. All cows were weighed once weekly and body condition scores were assigned for all

cows weekly by 2 technicians using the 5 point system (Wildman et al., 1982). All cows were milked 2 times daily for the 63 d of the lactation phase of the trial and daily milk yield was measured electronically. Milk samples were collected weekly from 2 consecutive milkings obtained over a 24-h period. Samples were analyzed for milk fat, protein, and lactose.. Liver biopsies were taken on d 7 postpartum and used for an in vitro gluconeogenesis experiment.

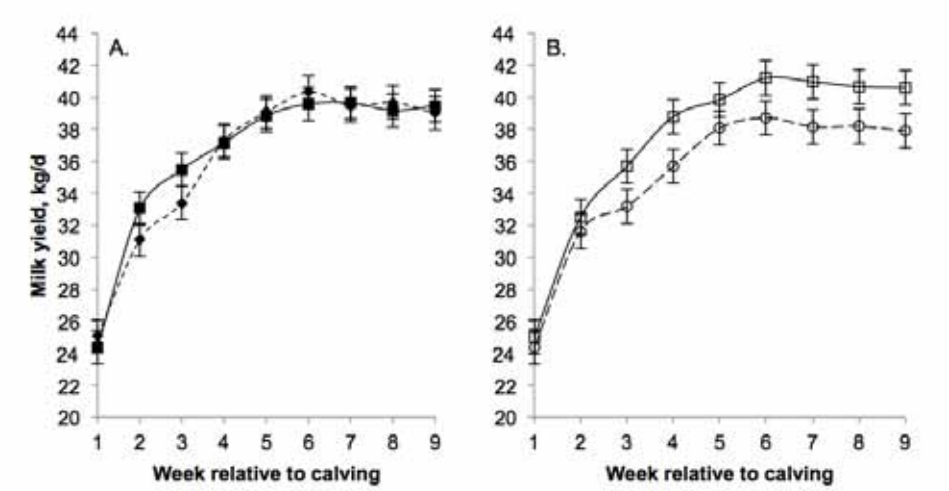
Statistical computations were performed using SAS software (version 9.2; SAS Institute Inc., Cary, NC). Postpartum data were analyzed as a completely randomized design with a 2 × 2 factorial arrangement of treatments. Fixed effects included starch level, Rumensin treatment, parity, time (wk or d), and all 2-way interactions. The random effect was cow nested within starch and Rumensin treatment. Postpartum data were analyzed separately as wk 1 to 3 and wk 1 to 9. Data measured over time were subjected to ANOVA by using the REPEATED statement in the MIXED procedure of SAS (Littell et al., 1996). For variables with measurements repeated over time, three covariance structures were tested: compound symmetry, autoregressive order 1, and unstructured covariance. The covariance structure that resulted in the Akaike information criterion closest to zero was used (Littell et al., 1996). Data not analyzed over time were subjected to ANOVA using the MIXED procedure of SAS (Littell et al., 1996).

Degrees of freedom were estimated by using the Kenward-Roger option in the model statement. Least squares means for treatment effects were separated by use of the PDIFF statement when the overall F-test was $P < 0.05$ and trends were declared at $0.05 < P < 0.10$. Because a subset of animals were used for the liver biopsy data trends for these data were declared at $0.05 < P < 0.15$. There was no interaction of starch × Rumensin treatment so all results will be presented as main effects of either starch or Rumensin.

DO PROPIOGENIC FRESH RATIONS AFFECT PERFORMANCE?

Milk yield data for the fresh period treatments are presented in Figure 2. The overall effect of starch level in the fresh period diet on milk yield from wk 1 through 9 was not significant ($P = 0.81$; average 30.4 kg/d); however, cows fed the HS diet had a faster increases in milk in wk 2 and 3 postpartum compared to cows fed the LS diet ($P = 0.0003$). Further evaluation of the patterns of milk yield during wk 1 to 3 using daily milk yield data suggested that cows fed the HS diet tended ($P = 0.10$) to have higher overall milk yield compared to cows fed the LS diet (31.1 vs. 29.2 kg/d). Cows fed Rumensin during the transition period produced 2.2 kg/d ($P = 0.05$) more milk than control cows when evaluated from wk 1 to 9 postpartum. Trends for Rumensin × week interactions during wk 1 to 3 for both milk yield ($P = 0.07$) and lactose yield ($P = 0.06$) suggested that yields of each were increased by wk 3 of lactation compared to control cows.

Figure 2. Least squares means and standard errors for milk yield for starch and Rumensin dietary treatments. Panel A depicts milk yields for cows fed either high (-■-) or low starch (--◆--) fresh diets. Panel B depicts milk yields for cows fed Rumensin at 0 mg/d (–O–) or 450 mg/d (-□-) during the transition period.

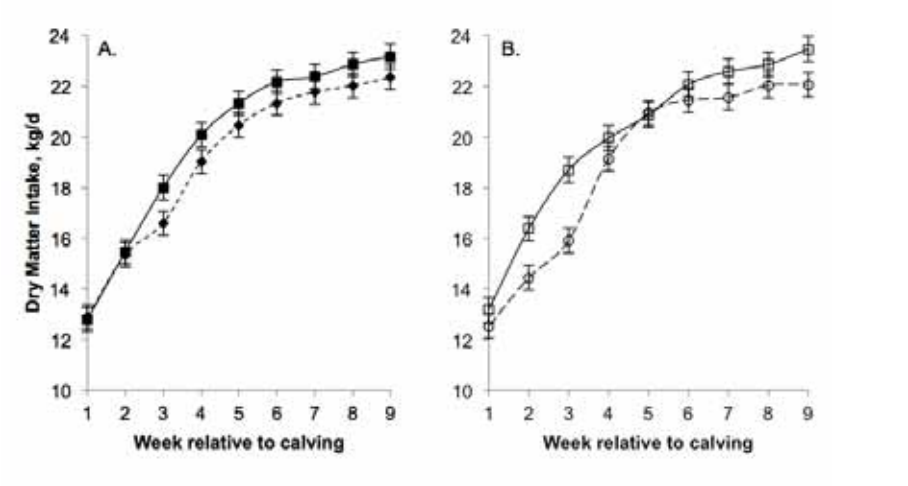


Cows fed HS fresh diets had lower percentages of milk fat (4.38 vs. 5.01%; *P* = 0.01) and true protein (3.31 vs. 3.84%; *P* = 0.05) than cows fed LS diets during wk 1 to 3 postpartum, however, when evaluated over the 9 wk postpartum period these effects were not significant. Percentages of lactose (4.60 vs. 4.83%; *P* = 0.05), and total solids (13.31 vs. 14.76%; *P* = 0.009) also were decreased during wk 1 to 3 in cows fed the HS fresh diets compared to those fed LS diets; these effects also were significant when evaluated over the 9 wk postpartum period (*P* = 0.03 for both variables). Despite the differences in milk component percentages, the effects of starch level on overall yields of milk fat, true protein, lactose, total solids, and ECM were not significant when evaluated for either wk 1 to 3 or 1 to 9 (Table 2). For cows fed the LS diet, component yields were generally higher during wk 1 compared to cows fed the LS diet, but both groups had similar component yields during wk 2 and 3. LS cows likely had greater component yields in wk 1 postpartum because they had greater adipose tissue mobilization. There was no effect of Rumensin treatment on percentages of milk true protein and total solids or yields of fat, true protein, lactose, total solids, and ECM during wk 1 to 3. During wk 1 to 9, cows fed Rumensin had lower percentages of milk lactose (4.82 vs. 4.93%; *P* = 0.03); however, there was no difference in lactose yield (average 1.76 kg/d) and percentages and yields of other milk components and ECM were not affected by treatment. Cows fed Rumensin had higher MUN during both wk 1 to 3 and wk 1 to 9 (*P* = 0.007 and *P* = 0.02, respectively).

Dry matter intake data for the fresh period treatments are presented in Figure 3. Cows fed the HS fresh diet had similar overall DMI compared to cows on the LS diet but increased (*P* = 0.006 DMI when expressed as a percentage of BW (2.67 vs. 2.41%;) during wk 1 to 3. Significant interactions of starch level and week for DMI suggested that cows fed HS had a faster increase in intake (*P* = 0.04). Cows fed Rumensin had higher DMI than controls during both wk 1 to 3 (16.1 vs. 14.3 kg/d; *P* = 0.004) and wk 1

to 9 (20.0 vs. 18.9 kg/d; $P = 0.02$). There was an interaction of Rumensin × week for both wk 1 to 3 ($P = 0.009$) and wk 1 to 9 ($P < 0.0001$) such that cows fed Rumensin had greater DMI during wk 2 and 3 postcalving.

Figure 3. Least squares means and standard errors for dry matter intake (DMI) for starch and Rumensin dietary treatments. Panel A depicts DMI for cows fed either high (-■-) or low starch (--◆--) fresh diets. Panel B depicts DMI for fed Rumensin at 0 mg/d (–O–) or 450 mg/d (-□-) during the transition period.



There was no effect of dietary starch level on postpartum BW, BW change, or average BCS; however, heifers fed the HS fresh diet lost less BCS during the first 3 wk postcalving than animals in the other treatment groups (starch × parity interaction; $P = 0.01$). Milk production efficiency during both wk 1 to 3 and wk 1 to 9, calculated either as milk yield per unit of DMI or ECM yield per unit of DMI, was increased in cows fed LS fresh diets (Table 2). This increased milk efficiency is likely because cows fed the LS diet had decreased DMI and mobilized more adipose tissue during wk 1 to 3. Although overall effects of Rumensin treatment on postpartum BCS and BCS change during both wk 1 to 3 and 1 to 9 were not significant, an interaction of Rumensin and parity existed during wk 1 to 9 such that heifers fed Rumensin lost slightly less BCS and cows fed Rumensin lost slightly more BCS ($P = 0.006$). Overall effects of Rumensin treatment on feed efficiency, expressed as units of milk per unit of DMI, were not significant during either wk 1 to 3 or wk 1 to 9. However, when milk production efficiency was expressed as units of ECM per unit of DMI, cows fed Rumensin had slightly lower efficiency during wk 1 to 3 ($P = 0.05$), likely contributed to by the higher DMI for cows fed Rumensin.

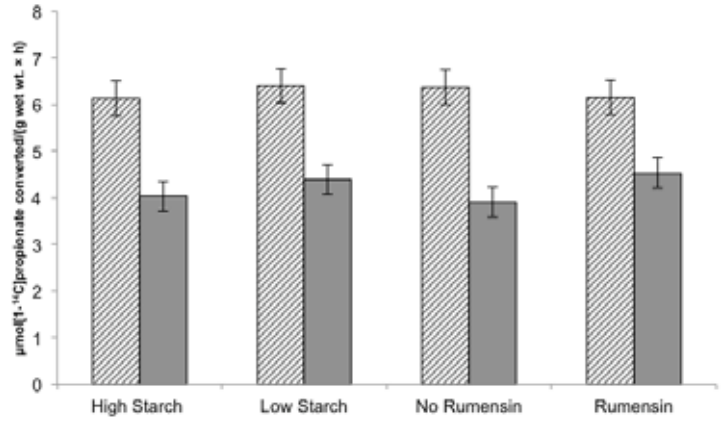
DO PROPIOGENIC DIETS AFFECT LIVER GLUCONEOGENIC CAPACITY?

Overall, animals fed diets with greater propiogenic capacity had faster increases in milk production and DMI, which would indicate that increased propionate supply in early lactation allows animals a better start. One of our main questions in conducting this study was how the liver handles propionate load in early lactation, which we evaluated

using an in vitro system by incubating liver tissue slices obtained on d 7 postpartum with a [1-^{14}C]propionate and measuring label incorporation into glucose and CO_2.

There was no effect of starch or Rumensin treatment on liver capacity to oxidize [1-^{14}C]propionate to CO_2 and no effects of starch on liver capacity to convert propionate to glucose (Figure 4). Cows that were fed Rumensin tended (P = 0.14) to have greater capacity to convert [1-^{14}C]propionate to glucose than control cows. Heifers had greater capacity to both oxidize [1-^{14}C]propionate to CO_2 and convert [1-^{14}C]propionate to glucose than did multiparous animals (P = 0.04 and P = 0.01, respectively).

Figure 4. Conversion rates of [1-^{14}C]propionate to CO_2 (striped bars) and glucose (solid bars) at d 7 postpartum for all cows (treatment indicated on the x axis).



In the TCA cycle the [1-^{14}C]propionate label randomizes such that every mole of [1-^{14}C]propionate directed toward phosphoenylpyruvate and gluconeogenesis would yield 0.5 moles of radiolabeled CO_2 and 0.5 moles of radiolabeled glucose (Knapp et al., 1992). Therefore, the ratio of rates of conversion of radiolabeled propionate to glucose and CO_2 provides an index of the efficiency of propionate utilization for gluconeogenesis. Rumensin administration increased the ratio of glucose to CO_2 (Figure 3; P = 0.05), which indicates that cows fed Rumensin have a greater propensity to convert propionate to glucose (Figure 5).

IMPLICATIONS AND CONCLUSIONS

In early lactation the liver has the ability to preferentially use additional propionate supply for gluconeogenesis. Increasing the ruminal propionate supply that is available to the liver for glucose synthesis after calving provides the cow with better energy status and allows a better start to lactation. This is indicated by improvements in DMI, increased milk yield, as well as faster increases in milk yield postpartum. In cows fed more propiogenic diets the increased liver glucose output provides the animal with better energy status and less dependence on adipose mobilization. Based on the data from this study it would appear that DMI in fresh cows is not limited by propionate oxidation at the liver, and feeding diets with higher starch levels and containing Rumensin in fresh rations results in improved milk production and energetic status.

Figure 5. Ratio of glucose to CO_2 for control cows (striped bar) and for Rumensin treated cows (solid bar) at d 7 postpartum.

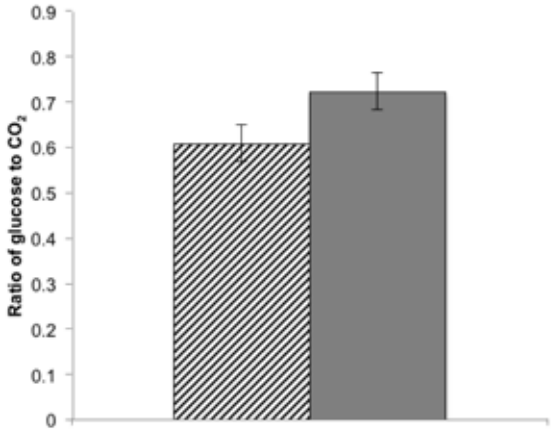


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Table 2. Milk yield, intake, and feed efficiency for cows fed varying levels of starch and Rumensin in the fresh period

	Diet[1]		SEM	Topdress[2]		SEM	*P*-values[3]							
Item	HS	LS		Con	Rum		S	Rum	P	S×Rum	S×Wk	Rum×Wk	S×P	Rum×P
Milk yield, kg/d														
wk 1 to 3	31.0	29.8	0.9	29.8	31.0	0.9	0.35	0.32	<0.001	0.45	0.002	0.07	0.57	0.63
wk 1 to 9	36.3	36.0	0.8	35.1	37.3	0.8	0.81	0.05	<0.001	0.43	0.0003	0.19	0.33	0.71
ECM, kg/d[5]														
wk 1 to 3	34.7	36.7	1.2	35.9	35.4	1.2	0.24	0.80	<0.001	0.97	0.01	0.93	0.45	0.93
wk 1 to 9	36.9	37.6	1.0	36.8	37.8	1.0	0.59	0.47	<0.001	0.51	0.19	0.48	0.44	0.55
Fat, kg														
wk 1 to 3	1.32	1.44	0.06	1.41	1.34	0.06	0.14	0.34	<0.001	0.91	0.11	0.97	0.67	0.86
wk 1 to 9	1.33	1.37	0.04	1.35	1.35	0.05	0.48	0.93	<0.001	0.95	0.20	0.61	0.56	0.96
True protein, kg														
wk 1 to 3	0.99	1.07	0.05	1.02	1.04	0.05	0.25	0.85	<0.001	0.79	0.09	0.44	0.35	0.81
wk 1 to 9	1.03	1.06	0.03	1.02	1.06	0.04	0.53	0.33	<0.001	0.76	0.15	0.64	0.62	0.33
Lactose, kg														
wk 1 to 3	1.43	1.44	0.05	1.42	1.44	0.05	0.89	0.77	<0.001	0.58	0.04	0.06	0.23	0.90
wk 1 to 9	1.75	1.78	0.04	1.73	1.79	0.04	0.62	0.32	<0.001	0.35	0.09	0.36	0.08	0.70
Total Solids, kg														
wk 1 to 3	4.04	4.27	0.15	4.17	4.13	0.15	0.26	0.84	<0.001	0.90	0.02	0.69	0.39	0.94
wk 1 to 9	4.44	4.55	0.11	4.44	4.55	0.12	0.49	0.51	<0.001	0.44	0.21	0.63	0.26	0.49
MUN, mg/dL														
wk 1 to 3	11.6	10.9	0.4	10.5	11.9	0.4	0.16	0.007	0.05	0.86	0.44	0.46	0.89	0.59
wk 1 to 9	11.7	11.5	0.4	11.0	12.2	0.4	0.59	0.02	0.23	0.42	0.21	0.37	0.98	0.49
DMI, kg/d														
wk 1 to 3	15.6	14.8	0.4	14.3	16.1	0.4	0.21	0.004	<0.001	0.77	0.04	0.009	0.24	0.74
wk 1 to 9	19.8	19.1	0.3	18.9	20.0	0.3	0.13	0.02	<0.001	0.66	0.32	<0.001	0.99	0.67
DMI, % of BW														
wk 1 to 3	2.67	2.41	0.06	2.48	2.60	0.06	0.006	0.20	<0.001	0.71	0.01	0.29	0.84	0.25
wk 1 to 9	3.37	3.22	0.05	3.24	3.35	0.05	0.03	0.11	<0.001	0.47	<0.001	0.66	0.98	0.99
Feed efficiency														
Milk/DMI														
wk 1 to 3	1.95	2.10	0.05	2.05	2.03	0.06	0.04	0.59	0.84	0.48	0.51	0.95	0.30	0.04
wk 1 to 9	1.84	1.95	0.03	1.88	1.90	0.03	0.04	0.67	0.97	0.17	0.53	0.95	0.28	0.21
ECM/DMI														
wk 1 to 3	2.20	2.50	0.07	2.45	2.26	0.07	0.002	0.05	0.47	0.98	0.66	0.76	0.45	0.07
wk 1 to 9	1.90	2.05	0.04	1.99	1.97	0.04	0.006	0.69	0.14	0.26	0.13	0.58	0.28	0.65

[1]Postpartum diets HS = High starch, LS = Low starch.

[2]Con = control topdress, contained 0 g/metric ton Rumensin, and Rum = Rumensin topdress, contained 441 g/metric ton Rumensin.

[3]S = starch level, and P = parity.

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