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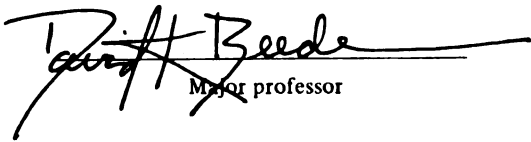
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PERIPARTUM RESPONSES OF LATE PREGNANT DAIRY COWS TO
VARYING DIETARY CORN GRAIN CONTENT OR LENGTH OF
FEEDING PERIOD PREPARTUM

presented by

Douglas G. Mashek

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of the requirements for

M.S. degree in Animal Science


Major professor

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**PERIPARTUM RESPONSES OF LATE PREGNANT DAIRY COWS TO
VARYING DIETARY CORN GRAIN CONTENT OR LENGTH OF FEEDING
PERIOD PREPARTUM**

By

Douglas G. Mashek

A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

MASTER OF SCIENCE

Department of Animal Science

1999

ABSTRACT

PERIPARTUM RESPONSES OF LATE PREGNANT DAIRY COWS TO VARYING DIETARY CORN GRAIN CONTENT OR LENGTH OF FEEDING PERIOD PREPARTUM

By

Douglas G. Mashek

The objective of this research was to study nutritional management strategies prepartum that may improve periparturient energy status, health, milk yield and composition, and reproduction in dairy cows. In the first experiment, 189 cows were assigned randomly to a late dry period diet with supplemental corn grain (SC) or without supplemental corn grain (NC) during the last 17 d prepartum. Cows fed SC had lower plasma β -hydroxybutyrate and tended to have increased insulin concentrations prepartum. Treatment had no effect on milk yield or composition, health, or reproduction, but several interactions involving parity showed that SC was more beneficial to parity 3+ cows. In the second experiment, 189 cows in two farms were assigned randomly to the late dry period for < 26 d (S), or $\geq$ 26 d (L). Cows in L gained more body condition prepartum, tended to have lower plasma non-esterified fatty acid and higher insulin concentrations postpartum, and lost less body condition during the first 3 wk postpartum. Cows in L had lower milk yields, increased incidences of metritis, increased days open and somatic cell count in one farm, but not in the other. Correlation analysis of dependent variables of the combined datasets grouped by parity showed that relationships between blood variables were similar for parity 1 and 3+ cows, and relationships involving BCS and BCS changes were similar for cows in parities 2 and 3+.

DEDICATION

To Mara, for making me the luckiest man alive.

ACKNOWLEDGEMENTS

I would like to thank Dr. Beede for his guidance, advice, and friendship throughout Master's program. I thank Dr. Allen, Dr. Bucholtz, and Dr. Herdt for serving on my graduate committee. I also appreciated the use of Dr. Tucker's and Dr. Orth's laboratories for analysis of blood samples. I appreciated the help from Larry Chapin and Dr. Chris McMahon while working in Dr. Tucker's laboratory.

I would like to thank Jim Leisman for his help with the blood assays and answering numerous SAS questions, and Dr. Rob Templeman for enduring hours of endless statistical questions.

I extend my thanks to Laura Bolinger for her help with data collection, and Katie Gould and Irene Choi for their help in the laboratory. I thank Tom Pilbeam for his help with sample collection, and his friendship and advice. I would like to thank Jill Davidson, Sara Scheurer and Luis Rodriquez for their friendships and support.

I would like to thank the Michigan Corn Growers Association and Mr. Keith Muxlow for partially financing these studies.

Thanks to Webster Ridge and Nobis Dairy Farms for participating in this research.

Finally, thanks to Mara for her help with every aspect of my research and for her love and support.

TABLE OF CONTENTS

LIST OF TABLES.....	vii
LIST OF FIGURES.....	x
LIST OF ABBREVIATIONS.....	xii
CHAPTER 1	
INTRODUCTION	
CHAPTER 2	
REVIEW OF LITERATURE.....	3
Aspects of Feeding High Concentrations of Grain Prepartum.....	3
Effects of Grain Supplementation Prepartum on Milk Production.....	4
Effects of Grain Supplementation Prepartum on Indicators of Energy Status....	7
Effects of Grain Supplementation Prepartum on Health Disorders.....	9
Effects of Grain Supplementation Prepartum on Reproduction.....	10
Altering Length of the Late Dry Period.....	11
Relationships Between Body Condition Score and Milk Production.....	12
Relationships Between Body Condition Score and Dry Matter Intake.....	13
Relationships Between Body Condition Score and Health Disorders.....	14
Relationships Between Body Condition Score and Reproduction.....	16
CHAPTER 3	
Peripartum responses of dairy cows to partial substitution of corn silage with corn grain in diets fed during the late dry period	
ABSTRACT.....	17
INTRODUCTION.....	18
MATERIALS AND METHODS.....	19
Cows and Treatments	
Sample Collection and Analysis	
Statistical Analysis	
RESULTS AND DISCUSSION.....	23
Diet Composition	
Body Condition and Udder Edema	
Metabolic Variables	
Health Disorders	
Milk Yield, Composition, and Somatic Cell Count	
Reproductive Measurements	

CONCLUSIONS.....	28
TABLES AND FIGURES.....	30
CHAPTER 4	
Peripartum responses of dairy cows to altering length of the late dry period	
ABSTRACT.....	48
INTRODUCTION.....	49
MATERIALS AND METHODS.....	50
Cows and Treatments	
Sample Collection and Analysis	
Statistical Analysis	
RESULTS AND DISCUSSION.....	55
Diet Composition	
Body Condition and Udder Edema	
Metabolic Variables	
Health Disorders	
Milk Yield, Composition, and Somatic Cell Count	
Reproductive Measurements	
CONCLUSIONS.....	64
TABLES AND FIGURES.....	65
CHAPTER 5	
Associations among body condition score, body condition score changes, blood variables, and milk yield and composition in periparturient dairy cows	
ABSTRACT.....	92
INTRODUCTION.....	93
MATERIALS AND METHODS.....	94
RESULTS AND DISCUSSION.....	97
All Parities	
Differences Among Parities	
CONCLUSIONS.....	103
TABLES AND FIGURES.....	105
CHAPTER 6	
Discussion and Implications.....	145
LITERATURE CITED.....	152

LIST OF TABLES

CHAPTER 3

Table 1. Formulated ingredient and analyzed chemical composition, and particle size distribution of experimental diets fed in the late dry period.....	30
Table 2. Formulated ingredient and analyzed chemical composition, and particle size distribution of experimental diets fed postpartum.....	31
Table 3. Formulate ingredient and chemical composition of diets fed in the early dry period.....	32
Table 4. Least squares means and statistical significance of body condition scores (BCS) during the periparturient period.....	33
Table 5. Least squares means and statistical significance of body condition score changes during the periparturient period.....	34
Table 6. Least squares means and statistical significance of plasma NEFA concentrations during prepartum, postpartum or periparturient periods.....	35
Table 7. Least squares means and statistical significance of plasma BHBA concentrations during prepartum, postpartum or periparturient periods.....	36
Table 8. Least squares means and statistical significance of plasma insulin concentrations during prepartum, postpartum or periparturient periods.....	37
Table 9. Incidence rates and statistical significance of health disorders, and abnormal rectal temperatures.....	38
Table 10. Least squares means and statistical significance of milk yield and composition through 60 DIM.....	39
Table 11. Least squares means and statistical significance of milk yield and composition through 150 DIM.....	40
Table 12. Least squares means and significance of energy-corrected milk (ECM) and SCC through 60 or 150 DIM.....	41
Table 13. Least squares means and statistical significance of reproductive measurements.....	42

CHAPTER 4

Table 1. Days spent in the late dry period and allocation of cows to treatments.....	65
Table 2. Formulated ingredient and analyzed chemical composition, and particle size distribution of diets fed in the late dry period.....	66
Table 3. Formulated ingredient and analyzed chemical composition, and particle size distribution of diets fed postpartum in Farm 1.....	67
Table 4. Formulated ingredient and analyzed chemical composition, and particle size of diets fed postpartum in Farm 2.....	68
Table 5. Formulated ingredient composition of diets fed in the early dry period.....	69
Table 6. Least squares means and statistical significance of body condition scores during the periparturient period.....	70
Table 7. Least squares means and statistical significance of body condition score changes during the periparturient period.....	71
Table 8. Least squares means of plasma NEFA concentrations for prepartum, postpartum, and periparturient periods.....	72
Table 9. Least squares means of plasma BHBA concentrations for prepartum, postpartum, and periparturient periods.....	73
Table 10. Least squares means of plasma insulin concentrations for prepartum, postpartum, and periparturient periods.....	74
Table 11. Incidence rates and statistical significance of health disorders and abnormal rectal temperatures.....	75
Table 12. Least squares means and statistical significance of milk yield and composition through 60 DIM.....	76
Table 13. Least squares means and statistical significance of milk yield and composition through 150 DIM.....	77
Table 14. Least squares means and statistical significance of daily milk yields in Farm 1.....	78

Table 15. Least squares means and statistical significance of energy-corrected milk (ECM) yield and SCC through 60 DIM.....79

Table 16. Least squares means and statistical significance of energy-corrected milk (ECM) yield and SCC through 150 DIM.....80

Table 17. Least squares means and significance of reproductive measurements.....81

CHAPTER 5

Table 1. Arithmetic means, standard deviations, and ranges for all variables.....105

Table 2. Correlations, P-values and n amongst variables pooled across parities.....109

Table 3. Correlations, P-values and n amongst variables in parity 1 cows.....118

Table 4. Correlations, P-values and n amongst variables in parity 2 cows.....127

Table 5. Correlations, P-values and n amongst variables in parity 3+ cows.....136

LIST OF FIGURES

CHAPTER 3

Figure 1. Least squares means and 95% confidence intervals for the interaction of treatment by time ($P = 0.02$) on plasma BHBA concentrations in the periparturient period.....	43
Figure 2. Least squares means and SEM for the interaction of treatment by parity by time ($P = 0.10$) for milk production during the first 60 DIM.....	44
Figure 3. Least squares means and SEM for the interaction of treatment by parity by time ($P = 0.13$) for protein yield through 150 DIM.....	45
Figure 4. Least squares means and 95% confidence intervals for the interaction of treatment by parity ($P = 0.08$) for SCC through 60 DIM.....	46
Figure 5. Least squares means and SEM for the interaction of treatment by parity ($P = 0.08$) for days open.....	47

CHAPTER 4

Figure 1. Least squares means and 95% confidence intervals for the interaction of treatment by time ($P = 0.03$) for plasma insulin in the periparturient period.....	82
Figure 2. Least squares means and 95% confidence intervals for the interaction of treatment by parity by farm ($P = 0.13$) for plasma insulin concentrations postpartum.....	83
Figure 3. Least squares means and 95% confidence intervals for the interaction of treatment by parity by time ($P = 0.12$) on plasma insulin concentrations postpartum.....	84
Figure 4. Least squares means and 95% confidence intervals for the interaction ($P = 0.06$) for milk yield through 150 DIM.....	85
Figure 5. Least squares means and SEM for the treatment by parity interaction ($P = 0.07$) of milk protein content through 60 DIM.....	86
Figure 6. Least squares means and SEM for the treatment by time interaction ($P < 0.01$) of milk protein content through 60 DIM.....	87

Figure 7. Least squares means for the interaction of parity by treatment
by time ($P = 0.03$) for milk protein content through 150 DIM.....88

Figure 8. Least squares means and SEM for the interaction of treatment
by time ($P < 0.01$) for milk protein yield through 150 DIM.....89

Figure 9. Least squares means and 95% confidence intervals for the
interaction of treatment by farm ($P < 0.01$) for SCC through 150 DIM.....90

Figure 10. Least squares means and SEM for the interaction of
treatment by farm ($P = 0.06$) on days to first service.....91

LIST OF ABBREVIATIONS

ADF	Acid detergent fiber
BCS	Body condition score
BHBA	Betahydroxybutric acid
BW	Body weight
CP	Crude protein
DIM	Days in milk
DMI	Dry matter intake
EE	Ether extract
ECM	Energy-corrected milk
FCM	Fat-corrected milk
NDF	Neutral detergent fiber
NEFA	Non-esterified fatty acids
NFC	Non-fiber carbohydrate
NRC	National Research Council
SCC	Somatic cell count
TG	Triglyceride
TMR	Total mixed ration
VFA	Volatile fatty acids
VLDL	Very low density lipoprotein

CHAPTER 1

INTRODUCTION

Dairy cows undergo a host of physiological and metabolic changes during late gestation. The growing gravid uterus metabolizes increasing amounts of nutrients and energy throughout the dry period (Bell, 1995), and lactogenesis requires sufficient amounts of substrates during the last week of gestation (Davis et al., 1979). Additionally, a series of endocrine changes may be responsible partially for an accelerated decline in feed intake during the last 2 wk before parturition (Bertics et al., 1992). Together, these changes can result in negative energy status and mobilization of body tissue reserves in late gestation and early lactation. Excessive mobilization of body reserves prepartum may predispose a variety of metabolic disorders (Dyk et al., 1995).

The late dry period is defined as the time prior to parturition in which dry cows are fed and managed differently than dry cows earlier in gestation. The energy and nutrient composition of diets fed during the late dry period can have profound effects on energy status of dairy cows. Increasing the amount of dietary corn grain increases the energy concentration of the diet and may improve energy status of cows prepartum. Feeding increased concentrations of corn grain may adapt ruminal microbes to a more fermentable diet and improve rumen health in early lactation when this type of diet is typically fed (Mackie et al., 1979). In addition, fermentation of corn grain produces the desired volatile fatty acids to stimulate growth of ruminal papillae (Dirksen et al., 1985, Xu and Allen, 1998). Proper adaptation of rumen microbes and growth of ruminal papillae may improve energy status and rumen function in early lactation. Propionate produced from fermentation of corn grain results in increased blood glucose and insulin concentrations. Both insulin and glucose are antilipolytic and may minimize mobilization of body stores prepartum (Grummer, 1995). Therefore, proper nutritional

management of prepartum dairy cows is critical to improve energy status and minimize health problems that may lead to improved milk production and reproduction in early lactation.

Therefore, the working hypotheses in this research were: 1) that partial substitution of corn silage with corn grain in diets fed to dairy cows during the late dry period will improve energy status, health, reproduction, and milk production, and 2) that increasing length of the late dry period will increase body condition and improve energy status, health, reproduction, and milk production of dairy cows.

The specific objectives were: 1) to compare the effects of partial substitution of corn silage with corn grain in diets fed to dairy cows during the late dry period on body condition, energy status, health, reproduction, and milk production, and 2) to compare effects of altering length of the late dry period on body condition, energy status, health, reproduction, and milk production of dairy cows.

CHAPTER 2

REVIEW OF LITERATURE

Aspects of Feeding High Concentrations of Grain Prepartum

Increasing the concentration of grain in diets prepartum increases the energy density of diets and may help minimize negative energy status. The idea of supplementing concentrate feeds in diets of late gestation cows was introduced early in the century (Boutflour, 1928). The objectives were to build up body reserves and accustom the rumen to rations similar in energy concentration and fermentability to those fed typically in early lactation. Today, we still hold these beliefs to be true and hypothesize that increasing grain concentrations in diets prepartum may better prepare cows for the subsequent lactation by: 1) adapting rumen microbial populations to highly fermentable diets; 2) stimulating ruminal papillae development; and 3) supplying cows with additional energy during a period of negative energy status.

Ruminal microbial adaptation. It takes 3 wk approximately for the ruminal microbial population to adapt from a high forage to a high concentrate diet (Mackie et al., 1979). If large amounts of readily fermentable carbohydrate are introduced in the rumen without proper microbial adaptation (i.e., early lactation), the carbohydrates may be metabolized to lactic acid, which lowers ruminal pH (Goff and Horst, 1997). Dramatic decreases in ruminal pH can cause ruminal acidosis and has many detrimental effects on health and performance of dairy cows (Allen and Beede, 1996).

Ruminal papillae development. The concentration and type of volatile fatty acid (VFA) produced in the rumen is important to stimulate ruminal papillae development (Dirksen et al., 1985). High concentrations of butyrate and, to a lesser extent, propionate increase papillae size and absorptive surface area within the rumen (Sakata and Tamate, 1978). Ruminal fermentation of grain increases propionate production; however, an economically feasible feedstuff that yields butyrate upon fermentation has yet to be found. Increasing absorptive surface area is important for removing VFA from the rumen. Improved absorption of VFA helps prevent ruminal acidosis and increases energy uptake which is critical when cows are in negative energy status (i.e., during late gestation and early lactation [Allen and Beede, 1996]).

Negative energy status. Typically, cows experience approximately a 30% reduction in dry matter intake (DMI) during the last 1 to 2 wk of gestation (Bertics et al., 1992). They become energy deficient when energy intake fails to meet the needs of maintenance, and increasing requirements for pregnancy and lactogenesis (Bell, 1995). Feeding a diet with higher energy density in late gestation may improve energy status and minimize glycogenolysis and lipolysis (Grummer, 1995). Additionally, improving energy status around parturition also may influence immune function (Kimura et al., 1997) and incidence of health disorders (Dyk et al., 1995).

Effects on Grain Supplementation Prepartum on Milk Production

In early studies, effects of energy intake prepartum on milk production were variable. Cows fed hay and corn silage plus 2.3 kg/d of concentrate starting 6 wk prepartum and increased gradually to 5.5 kg/d at parturition had similar milk yields as those fed only hay and corn silage (Greenhalgh and Gardner, 1958). Schmidt and Schuldtz (1959) found no additional milk production by supplementing corn silage and hay with 5.8 kg/d of concentrate during the final 8 wk of gestation. Similarly, supplementing ad libitum hay and pasture with concentrates did not alter milk production

(Davenport and Rakes, 1969). The above mentioned studies did not record BCS which can influence DMI, fed diet components separately, and reported low milk production. Therefore, it is difficult to extrapolate these results to modern high producing herds fed a total mixed ration (TMR).

Nocek et al. (1983) randomly assigned 289 cows to prepartum diets of: 1) all hay fed ad libitum; 2) 50% hay and 50% corn silage fed adlibitum; and, 3) corn silage fed at 1% of body weight (BW) supplemented with 1.1 kg/d of a concentrates. Cows fed diet 1 produced 1.5 kg/d more milk during the first 9 wk of lactation than cows fed diets 2 and 3 ($P < 0.05$). However, there were no differences in fat-corrected milk (FCM) yield due to low milk fat concentrations of cows fed diet 1. It could be theorized that the lack of adaptation of the ruminal microbes and papillae was responsible for some degree of acidosis and reduced fiber digestion postpartum that caused the decrease in milk fat concentration. Daily NE_L intakes prepartum were 9.9, 11.8 and 9.6 Mcal per cow for groups 1, 2 and 3, respectively. Cows fed diet 3 lost body weight during the dry period because of the low energy intakes, making it difficult to compare these cows to those fed ad libitum.

Several studies showed positive responses to increasing the amount of concentrate in the diet prepartum. Addition of concentrates, primarily corn grain, to ad libitum hay consumption prepartum increased milk yields (Swanson and Hinton, 1962; Emery et al., 1969). Recent research utilizing higher producing cows showed benefits of increasing NE_L intake prepartum. Johnson and Combs (1991) fed cows either 1.5 or 1.68 Mcal NE_L /kg of dietary dry matter from 70 to 10 d prepartum. The lower energy diet consisted of 59% alfalfa silage and 41% corn silage; ground corn and soybean meal replaced 43% of the silage mixture in the higher energy diet, dry basis. All cows were fed a medium-energy diet (1.61 Mcal NE_L /kg) the last 10 d prepartum. Cows fed the high-energy diet produced 6 kg/d more FCM ($P = 0.06$), but only 6 cows were used in this study due to other experimental objectives. Minor et al. (1996) fed cows a standard diet with 1.34

Mcal NE_L/kg and 23.5% non-fiber carbohydrate (NFC; NFC = 100 – neutral detergent fiber – crude protein - ether extract – ash, dry basis) or a high diet of 1.63 Mcal NE_L/kg and 43.8% NFC for the last 19 d of gestation. Partially substituting corn grain and starch for alfalfa silage, corn silage and straw altered the NFC of the diets. Cows fed the high diet produced 2.8 kg/d more milk ($P < 0.05$) that was higher in protein percentage ($P < 0.01$), but tended to be lower in fat percentage ($P < 0.10$) during the first 40 wk of lactation.

In addition to total dietary energy, the proportion of concentrates relative to forage may also be important. Two unique experiments compared isocaloric diets varying in forage-to-concentrate ratio (Olsson et al., 1997). The diets were fed in different amounts to compensate for the changes in energy value of varying the forage-to-concentrate ratio. Grass silage and hay were the forage sources and the concentrate fraction comprised oats and barley. Concentrate feeding started at 4 wk prepartum and gradually increased to meet the assigned ratio in both experiments at 3 d prepartum. The forage-to-concentrate ratios for the three diets in experiment 1 were 95:5, 70:30, and 40:60 for diets 1, 2 and 3, respectively. Cows fed diet 3 produced 3.8 kg/d more FCM during wk 2 through 4 of lactation than cows fed diet 2 despite having lower DMI prepartum because of energy restriction ($P < 0.05$). Although numerically higher for cows fed diet 3, no significant differences in milk yield were detected between the groups during the first 2 wk or after 4 wk postpartum. In experiment 2, forage-to-concentrate ratios of 60:40 (diet 1) and 40:60 (diet 2) were fed. Cows fed diet 2 produced more milk than those fed diet 1 during wk 5 to 14 of lactation ($P < 0.05$), but no differences were seen before and after this period or in FCM yield. Based on these studies, the type of energy source (grain vs. fiber) may be important in determining the effectiveness of the diet prepartum. These results support the theory that perhaps the proper fermentation end-products (propionate and butyrate vs. acetate) are essential to prepare the rumen by adapting microbes and stimulating ruminal papillae development.

Effects of Grain Supplementation Prepartum on Indicators of Energy Status

Insulin. Increasing the amount of grain in diets increases propionate and glucose production and subsequent insulin secretion. It is well documented that increased concentrate feeding prepartum increases plasma insulin concentrations (Fronk et al., 1980; Holtenuis et al., 1993; Kunz et al., 1985; Olsson et al., 1997). Increased plasma insulin concentrations prepartum have been suggested to cause insulin resistance postpartum; thereby, reducing the antilipolytic effects of insulin (Holtenuis, 1993). However, high circulating concentrations of insulin also have been hypothesized to increase hepatic glucose production and spare glycogen usage (Grummer, 1995). This could reduce hepatic triglyceride (TG) deposition and incidences of ketosis and fatty liver syndrome (Grummer, 1993). The significance of insulin concentration prepartum on insulin receptor sensitivity postpartum and hepatic lipid metabolism has not been elucidated.

Non-esterified fatty acids (NEFA). NEFA are a measure of lipolysis and are indicators of energy status. NEFA are produced in the greatest quantities during periods of negative energy status such as late gestation and early lactation when adipose tissue is mobilized to meet additional energy demands. The liver removes 7 to 25% of circulating NEFA (Emery et al., 1992), but NEFA concentrations in blood regulate uptake into the liver (Bell et al., 1980). Once in the liver, NEFA can be oxidized completely to CO₂ and H₂O, oxidized partially to ketones, or re-esterified to TG (Bruss, 1993). The bovine liver has a decreased ability to export TG as very low-density lipoprotein (VLDL); therefore, increased uptake of NEFA may predispose TG deposition in hepatic tissue and may lead to fatty liver syndrome (Grummer, 1993). Additionally, high NEFA concentrations in the liver can exceed the mitochondrial and peroxisomal oxidative capacity and result in ketone body production and subsequent ketosis.

The effects of concentrate feeding prepartum on NEFA concentrations in blood

vary among studies. Cows fed to meet maintenance energy requirements had higher serum NEFA concentrations from 70 to 5 d prepartum than cows fed the same diet supplemented with concentrates throughout the dry period (Kunz et al., 1985). However, NEFA concentrations postpartum were higher for the cows supplemented with concentrates prepartum. Higher DMI postpartum for the maintenance-fed group may account for the lower NEFA concentrations. Several studies reported no effects of prepartum energy intake on NEFA concentrations (Boisclair et al., 1986; Holtenuis, 1989; Jones and Garnsworthy, 1989), but others reported increased NEFA for cows fed higher energy diets prepartum (Fronk et al., 1980; Nachtomi et al., 1986). Discrepancies in the results may have arisen from the wide ranges of DMI, BCS, and milk production among studies.

Recent research suggests that increasing concentrates prepartum may improve energy status and decrease NEFA concentrations. Holtenuis et al. (1996) fed cows rations in which concentrates were increased gradually into the diet starting at 4 wk prepartum to reach levels of 10 and 50 % concentrate at 3 d prepartum. Cows fed 50% concentrate had lower NEFA concentrations from 1 wk prepartum through 1 wk postpartum than cows fed 10% concentrate at calving ($P < 0.01$). No changes were observed before or after this 2 wk period. Similarly, Minor et al. (1998) found numerically lower NEFA concentrations from 7 d prepartum through 60 d postpartum in cows fed 43.8% NFC diets compared with cows fed 23.5% NFC during the last 3 wk of gestation. Cows fed 1.68 Mcal NE_L/kg of dry matter (DM) during the last 26 d of gestation had lower NEFA concentrations in the last 7 d prepartum than cows fed 1.30 Mcal NE_L/kg of DM (VandeHaar et al., 1995). Unfortunately, higher concentrations of protein in the high energy diet confounded clear determination of the effect of energy in this study. Future research should address the role of grain supplementation and other contributing factors such as BCS and DMI on adipose tissue mobilization.

Ketone Bodies. Incomplete oxidation of NEFA in hepatic tissue results in

production of ketone bodies (beta-hydroxybutyrate, acetoacetate, and acetone). Excess acetyl CoA is converted to acetoacetate in hepatic mitochondria through a series of metabolic reactions. Acetoacetate can be reduced to beta-hydroxybutyrate if the reduced-to-oxidized ratio of nicotinamide adenine dinucleotide is sufficient. Acetone comprises a small percentage of total ketones and is formed from a spontaneous decarboxylation of acetoacetate. High circulating concentrations of ketones combined with low blood glucose may predispose cows to ketosis.

Several studies reported no effect of increasing grain supplementation in diets fed prepartum on blood ketone concentrations postpartum (Boisclair et al, 1986; Olsson et al., 1997; Schmidt and Schultz, 1959). Gardner (1969) observed increased blood ketone body concentrations during the first 2 wk postpartum for cows fed increased energy (20.52 vs. 14.88 Mcal NE_L/d) prepartum. Energy concentrations were adjusted by feeding varying amounts of alfalfa hay. Minor et al. (1998) found decreased blood ketone concentrations during early lactation in cows fed diets with high NFC concentrations prepartum. Additionally, cows in that study also had lower plasma NEFA concentrations and liver TG during the periparturient period. Diets high in grain may reduce ketone synthesis by increasing propionate and subsequent insulin production which are both antiketogenic (Grummer, 1993).

Effects of Grain Supplementation Prepartum on Health Disorders

Because large numbers of animals are needed, few studies have been able to detect statistically significant differences in effects of prepartum diet on incidences of health disorders. Much early research focused on the effects of concentrate feeding prepartum on udder edema. Generally, primiparous cows have more udder edema than multiparous cows (Greenhalgh and Gardner, 1958; Zamet et al., 1979). Emery et al. (1969) reported increased udder edema of primiparous cows fed up to 7 kg/d of grain

during the last 3 wk of gestation compared with cows not receiving supplemental grain. The effects of concentrate feeding prepartum on udder edema in multiparous cows are less well documented. Cows fed 12 or 46.5% of dietary DM as high moisture corn for 30 d prepartum had more edema than cows fed all hay (Johnson and Otterby, 1981). However, most studies showed no effect of concentrate feeding prepartum on udder edema regardless of parity (Greenhalgh and Gardner, 1958; Schmidt and Schultz, 1959; Fountaine et al., 1949; Hathaway et al., 1957). Other factors such as dietary mineral element concentrations may influence udder edema more than prepartum concentrate feeding.

Incidences of other health disorders to varying amounts of grain fed prepartum are not consistent across studies. Many studies with small numbers of cows report no changes on incidence of disorders (Boisclair et al., 1987; Johnson and Otterby, 1981; Schmidt and Schultz, 1959). However, Emery et al. (1969) observed significant increases in the incidence of mastitis and milk fever of 148 cows fed up to 7 kg/d of grain compared with cows fed ad libitum hay prepartum. However, increasing energy requirements above NRC (1989) recommendations during the last 3 wk of gestation decreased the incidence of health disorders in 1374 cows in 31 herds (Curtis et al., 1985).

Effects of Grain Supplementation Prepartum on Reproduction

Similar to health disorders, reproductive measurements require large numbers of cows to detect significant differences. Not only did many studies fail to measure reproductive traits, but those that did had insufficient replications. Typically, the variables measured to indicate reproductive efficiency are days open, days to first estrus, days to first artificial insemination, conception rate and percent of cows pregnant by a given date. Several studies utilizing only small numbers of cows ($n < 30$) reported no differences in any of the above mentioned measurements (Holter et al., 1990; Keys et al., 1984; Olsson et al., 1997). Nocek et al. (1983) reported the only study with reproductive

responses and large sample sizes ($n > 90$). Cows were fed all hay (9.9 Mcal NE_L/d), 50% hay and 50% corn silage (11.8 Mcal NE_L/d), or corn silage at 1% of BW plus 1.1 kg/d of concentrates (9.6 Mcal NE_L/d). Cows fed all hay had more days open than cows fed the corn silage and hay mix ($P < 0.05$). As previously mentioned, the study is confounded by the limited energy intakes of less than 10 Mcal NE_L/d.

Altering Length of the Late Dry Period

There could be two potential benefits of feeding a diet with higher grain content for longer than the traditional 2 to 3 wk prepartum. The first is to promote body condition gain of dry cows. The current recommendation is for dry cows to maintain BCS during the dry period (NRC, 1989). This recommendation is based partially on the contention that cows deposit energy more efficiently during lactation than while dry (Moe and Tyrell, 1972). However, replenishment of body reserves of high producing cows during late lactation has become increasingly difficult and cows may need to replenish body reserves during the dry period to reach an optimal BCS at parturition. Indeed, higher producing cows dry-off with lower BCS than less productive cows (Wildman et al., 1982).

Generally, cows can not gain sufficient amounts of body reserves during the last 2 to 3 wk prepartum when higher energy diets are more typically fed. In order to increase BCS during the dry period, cows may need to be fed higher energy diets for longer than 3 wk. No studies have evaluated effects of altering the length of time of feeding a higher energy diet prepartum.

Secondly, feeding a high grain diet for a longer period of time prepartum may be an effective method of promoting ruminal papillae development. As previously mentioned, ruminal papillae size influences absorptive capacity of the rumen. However, papillae may require 4 to 6 wk of increasing grain concentration to reach maximum absorptive capacity (Dirksen et al., 1985). In support of this concept, recent research

from Michigan State University showed increased papillae development of non-pregnant, non-lactating dairy cows fed 43.5% ground corn (Xu and Allen, 1998). Interestingly, the surface area of ruminal papillae increased proportionally to time on treatment (28 d). Therefore, cows may benefit from higher grain diets for longer than the traditional 2 to 3 wk prepartum to achieve maximum ruminal papillae size and absorptive surface capacity at parturition.

Feeding higher grain diets for longer than 3 wk prepartum may improve papillae development, VFA absorption and energy status as well as increase BCS during late gestation. Future research should determine the length of time a more fermentable diet needs to be fed prepartum for proper ruminal adaptation and maximal papillae growth. Additionally, future research should investigate how BCS changes during the dry period and BCS at parturition can influence postpartum performance.

Relationship Between BCS and Milk Production

Several field studies used regression analysis to evaluate relationships between milk production and BCS changes during the dry period. Dimecq et al. (1997) showed an increase of 545 kg of milk in the first 120 d of lactation for cows increasing BCS by 1 unit during the dry period. The BCS cows in this study averaged 2.77 (1.0 to 5.0 scale) at the beginning of the dry period and produced an average of 38 kg/d of milk through 120 d in milk (DIM).

The effects of BCS change during the dry period in controlled studies is not well documented. Boisclair et al. (1986) randomly assigned cows to one of the following dietary treatment groups: 1) all forage diet (54% alfalfa silage, 44% corn silage; 1.44 Mcal NE_L/kg) during the last 90 d of lactation and fed to requirements (1.5 Mcal NE_L/kg) during the dry period; 2) all forage diet during the last 90 d of lactation and ad libitum feeding of a high energy TMR (1.64 Mcal NE_L/kg) during the dry period; 3) ad libitum feeding of a high energy TMR (1.64 Mcal NE_L/kg) during the last 90 d of lactation and

restricted energy intake (70% of energy requirement) during the dry period; and 4) ad libitum high energy diet (1.64 Mcal NE_L/kg) during late lactation and the dry period. High moisture corn replaced alfalfa silage and corn silage in the TMR to increase energy density of diets. BCS changes during the dry period for the four groups were -.24, .45, -.58 and .22 for dietary treatments 1, 2, 3 and 4, respectively. BCS at parturition were 3.19, 3.95, 3.26 and 3.99 (1.0 to 5.0 scale) for dietary treatments 1, 2, 3 and 4, respectively. Feeding diets 2 and 4 increased BCS and subsequent milk production compared with cows fed diets 1 and 3 (35.9 and 35.3 vs. 33.1 and 32.7 kg/d; $P < 0.05$). A series of studies examined feeding cows starting at 12 wk prepartum to adjust BCS to either thin (2.0 to 2.3) or fat (3.2 to 3.5) on a 1.0 to 4.0 scale (Garnsworthy and Huggett, 1992; Garnsworthy and Jones, 1987; Jones and Garnsworthy, 1989). There were no changes in DMI, or milk composition and yield. Although BCS were recorded at parturition, initial BCS and BW were not measured. Therefore, BW or BCS change during the dry period could not be calculated. Increasing BCS from 3.0 to 3.8 (1.0 to 5.0 scale) during the dry period by liberal grain feeding with corn silage resulted in similar milk yields (Fronk et al., 1980). Other studies have tried to change BCS substantially throughout the dry period, but failed due to inadequate dietary energy concentrations or because cows were fed higher energy diets for only a short period of time (Gardner, 1969; Grum et al., 1996; Nocek et al., 1983; Nocek et al., 1986). Overall, it appears that cows entering the dry period with inadequate BCS may benefit from replenishment of body reserves.

Relationship Between BCS and DMI

The negative relationship between BCS at parturition and DMI is becoming more evident with more recent research. Generally, it is accepted in the field that cows calving with excessive BCS have compromised DMI. However, past research does not fully support this claim. Two often cited studies reported lower peak DMI in cows with high

BCS, although no significant differences were reported (Garnsworthy and Topps, 1982; Treacher et al., 1986). Additionally, many studies reported no changes in DMI as it relates to BCS at parturition (Boisclair et al., 1986; Erb et al., 1982; Holter et al., 1990; Smith et al., 1997). In contrast, several studies have reported effects of BCS on DMI. One study involving three groups of eight cows varying in BCS showed a longer interval to peak DMI in the group with the highest BCS (Garnsworthy and Topps, 1982). Perkins (1982) showed that both cows with high BCS at calving and cows that had accelerated BCS loss during the first 2 wk postpartum had depressed DMI. Roseler et al. (1997) showed that BCS accounted for approximately 6% of the variation in a linear model used to predict DMI. It should be noted that of the 241 cows used in this study none with a BCS of greater than 4.0 (1.0 to 5.0 scale) were included in the model. BCS was a significant factor when predicting DMI of transition cows (Hayirli et al., 1998). This study utilized 299 cows in various research projects from three universities. The authors also noted that BCS affected the shape of the DMI curve around parturition. The prepartum DMI depression in thin cows (BCS < 3.0) began later, but rate of depression was much more severe during the last 3 d prior to parturition. This area requires further research to elucidate the effects of BCS on DMI in the periparturient period. Research should investigate the effects of different diets postpartum as well as BCS at parturition on the potential depression of DMI and time required for cows to reach peak DMI in early lactation.

Relationship Between BCS at Parturition on Health Disorders

The effects of BCS at parturition on incidences of health disorders postpartum are not well understood. Treacher et al. (1986) showed higher total incidences of health disorders for cows that were over-conditioned at parturition, but only 18 cows were included in this study. Others suggested increased problems postpartum, but differences were not statistically significant (Fronk et al., 1980; Keys et al., 1983; Perkins, 1982).

However, several studies utilizing cows at different milk production levels showed no relationship between BCS at parturition on health disorders in early lactation (Boisclair et al., 1986; Garnsworthy and Topps, 1982; Gearheart et al., 1990; Ruegg and Milton, 1995). Morrow (1976) suggested that feeding high amounts of corn silage during the dry period and thus increasing the energy of the diet may predispose cows to health disorders postpartum. However, often BCS of cows at dry-off were not considered. Morrow et al. (1979) investigated a herd with a high incidences of fatty liver syndrome. Cows were fed diets high in corn silage during the dry period and throughout lactation. Reducing corn silage consumption during the dry period decreased the incidence of fatty liver syndrome and other health disorders associated with it. Although BCS were not reported, the authors stated that cows reached dry-off with excessive body condition. Feeding high amounts of corn silage promoted further, unneeded weight gain during the dry period resulting in obese cows at parturition. This study does not represent modern, high-producing cows which may have more difficulty replacing body condition than becoming over-conditioned during late lactation. Benefits from increasing grain supplementation during late gestation may be compromised if cows are gaining to achieve BCS in excess of those recommended (3.5 to 3.75) (Michigan State University Dairy Programs Group, 1995). Recent research involving 1556 cows in 95 Michigan dairy farms showed cows calving with BCS greater than 4.0 had an increased risk of ketosis and abomasal displacement (Dyk, 1995).

Decreasing BCS during the dry period in an attempt to reach the recommended BCS at parturition is not recommended. Lowering BCS during the dry period caused increased incidences of health disorders in early lactation (Gearheart et al., 1990; Zamet et al., 1979). Other factors such as diet composition, DMI, and rate of acceleration to peak DMI, immune function and parity should be considered when evaluating the role of BCS at parturition on health disorders.

Relationship Between BCS at Parturition on Reproduction

Several studies have evaluated the effects of BCS at parturition to reproductive performance. Research shows that reproductive variables are not affected by BCS at parturition (Garnsworthy and Topps, 1982; Gearheart et al., 1990; Pedron et al., 1993; Reugg and Milton, 1995; Treacher et al., 1986; Waltner et al., 1993). The severity of negative energy status in early lactation appears to be a major determinant of reproductive function (Butler and Smith, 1989; Nebel and McGilliard, 1993; Staples et al., 1990). Cows that lost BCS rapidly in early lactation had more days open and days to first estrus (Perkins, 1982). Additionally, cows with a BCS of 4.2 at parturition lost 57% more BW during the first 4 d of lactation compared with cows with a BCS of 3.5 (Smith et al., 1997). These findings suggest that obese cows that lose more BCS in early lactation may have reduced reproductive efficiency compared with cows that have moderate BCS. Future research efforts should investigate the effects BCS at parturition on severity and extent of BCS loss in early lactation, and how BCS changes in early lactation influence reproductive performance.

CHAPTER 3

PERIPARTUM RESPONSES OF DAIRY COWS TO PARTIAL SUBSTITUTION OF CORN SILAGE WITH CORN GRAIN IN DIETS FED DURING THE LATE DRY PERIOD

ABSTRACT

One hundred eighty-nine cows in a commercial dairy farm were assigned randomly to either a diet with supplemental corn grain (SC) or without supplemental corn grain (NC) approximately 17 d before parturition. Diets were formulated to be similar with the exception that dry ground corn replaced 21% of the corn silage in the SC diet, dry basis. The SC diet reduced plasma betahydroxybutyrate and tended to increase plasma insulin concentrations prepartum compared with the NC diet. Effects of treatment on production responses were highly dependent upon parity as indicated by parity by treatment by time interactions for milk and protein yields. Primiparous cows fed the SC diet had reduced milk protein yield, increased somatic cell count and days open compared with cows of the same parity fed the NC diet. The SC diet resulted in lower milk yields in early lactation and increased somatic cell count and days open for cows in their second parity. However, cows in their third parity or greater fed the SC diet yielded more milk and protein in early lactation, and had decreased somatic cell count and days open. Increasing the corn grain concentration of diets fed prepartum was advantageous to third and greater parity cows in this experiment, but showed no benefits during lactation for cows in first or second parities.

INTRODUCTION

Dairy cows undergo a host of physiological and metabolic changes during late gestation. The growing gravid uterus metabolizes increasing amounts of nutrients throughout the dry period (Bell, 1995) and lactogenesis requires sufficient amounts of substrates during the last week of gestation (Davis et al., 1979). In addition, a series of endocrine changes may be responsible partially for an accelerated decline in feed intake during the last 2 wk before parturition (Bertics et al., 1992). Together these changes can result in negative energy status and mobilization of body tissue reserves in late gestation and early lactation. Excess mobilization of body reserves prepartum may predispose a variety of health disorders (Dyk et al., 1995).

The late dry period is the time prior to calving when often dairy cows are fed and managed differently than cows earlier in the dry period. Feeding increased amounts of corn grain to cows in the late dry period may be advantageous for several reasons. Increasing corn grain may acclimatize ruminal microbes to higher energy diets fed typically in early lactation (Mackie et al., 1979). Increased propionate production from ruminal fermentation of corn grain may stimulate ruminal papillae development (Dirksen et al., 1985, Xu and Allen, 1998). The additional energy supplied by the corn grain may help offset the negative energy status prior to parturition. In addition, fermentation of corn grain results in increased concentrations of propionate and subsequently glucose (Olsson et al., 1997). Improving the carbohydrate status of periparturient cows may promote glycogenesis and improve hepatic function (Grummer, 1995).

Both propionate and glucose promote secretion of insulin, an antilipolytic

hormone. Therefore, increased corn grain potentially could improve lipid metabolism and energy status of periparturient dairy cows by reducing plasma NEFA concentrations and improving hepatic NEFA metabolism.

The objective of this experiment was to determine the effects of partially replacing corn silage with corn grain in a diet fed during the late dry period on body condition, health, energy status, milk production, and reproduction of dairy cows.

MATERIALS AND METHODS

Cows and Treatments

One hundred eighty-nine cows in a commercial dairy farm were completely randomized and assigned to be fed a diet with supplemental corn grain (SC) or without supplemental corn grain (NC) approximately 3 wk before their expected calving date (Table 1). There were 50 parity 1, 58 parity 2, and 81 parity 3+ cows in the experiment. Cows were co-mingled and fed the same diet during the first 2 to 3 wk postpartum (early lactation diet; Table 2). Subsequently, primi- or multiparous cows were grouped separately, and fed different diets (Table 2).

Sample Collection and Analysis

Silage dry matter content was determined weekly using a Koster Tester (Koster Crop Tester, Inc., Medina, OH) and adjustments were made to maintain the same dietary composition on a dry basis. TMR were sampled weekly prepartum through 150 d postpartum and dried at 55° C for 72 h for future analysis. Particle size distribution of TMR samples, as-fed basis, was determined using the Penn State Particle Separator

(Nasco, Fort Atkinson, WI; Lammers et al., 1996). Feed samples were ground through a Wiley mill (1 mm screen, Authur H. Thomas, Philadelphia , PA) and composited monthly. Samples were analyzed for NDF, ADF, CP, ether extract (EE), ash, ammonia, and minerals (Northeast DHI Forage Laboratory, Ithaca, NY).

One evaluator scored cows for BCS [five-point scale where 1 = thin to 5 = fat; (Wildman et al., 1982)] weekly prepartum, at calving, and 3 and 6 wk postpartum. Additionally, one evaluator assigned udders edema scores (0 = none , 1 = mild, 2 = moderate, and 3 = severe) within 3 d following parturition.

Blood samples were collected in evacuated test tubes containing sodium heparin (Vacutainer; Becton Dickson Vacutainer Systems USA, Rutherford, NJ) from the coccygeal vessels twice weekly prior to parturition, within 3 d following parturition, and 1 and 2 wk postpartum. Blood samples were collected approximately 7 to 8 h after feeding. Samples were stored on ice during transport to the laboratory. Upon arrival they were centrifuged, and plasma was harvested and stored at -4 C until later analysis of NEFA (NEFA-C kit, Waco Chemicals USA, Richmond, VA) with modifications (Johnson and Peters, 1993), insulin (Coat-A-Count, Diagnostic Products Corporation, Los Angeles, CA), and BHBA (310-A, Sigma Diagnostics, St. Louis, MO). All reagents in the BHBA assay except beta-hydroxybutyrate dehydrogenase were reduced proportionally to fit into 350 μ l wells of cell culture plates. Beta-hydroxybutyrate dehydrogenase was added at twice the reduced dose to shorten the incubation time. Inter-assay and intra-assay coefficients of variation for BHBA, NEFA and insulin were 5.6 and 7.0, 6.9 and 8.0, and 4.3 and 6.6, respectively.

Herdspersons and veterinarians were responsible for recording incidences of

health disorders and reproductive performance data throughout the experiment. Health disorders are defined as the following: displaced abomasum was an abnormal location of the abomasum as diagnosed by percussion that required corrective surgery; ketosis was a positive urine ketone test of moderate or greater (Ketostix; Bayer Corp., Elkhart, IN); mastitis was abnormal stripping or inflammation that required treatment; and, retained placenta was fetal membranes retained longer than 24 h after parturition.

The farm that participated in this study had an intensive milk fever prevention protocol. All third parity cows received a bottle of CMPK (10.8g Ca, 75g dextrose; Jice Pharmaceuticals Co., Lowell, MI) orally immediately following parturition. All fourth or greater parity cows, or any cows having twins, received a bottle of CMPK orally plus 500 ml of Calnate (10.7g Ca; The Butler Co., Columbus, OH) subcutaneously. In addition, rectal temperatures of all cows were monitored daily through 12 DIM. For analysis, cows having a rectal temperature above 39.4°C for at least 1 d during the first 12 DIM were considered abnormal. Individual milk weights were recorded every 2 wk and samples were analyzed for fat, protein and SCC monthly (Michigan DHIA). Energy-corrected milk (ECM) was calculated by the equation $ECM (lb) = 0.3246 \times \text{milk yield} (lb) + 12.86 \times \text{fat yield} (lb) + 7.04 \times \text{protein yield} (lb)$ (Dairy Herd Improvement Glossary, Fact sheet A-4, 1999). Over 60% of the cows in each treatment received bST injections (Monsanto Co., St. Louis, MO) during the sampling period. The voluntary waiting period for bST administration was 63 and 90 d for multiparous and primiparous cows, respectively. Cows received bST unless BCS was less than 2.5 or milk yield was greater than 40 kg/d for primiparous cows and 50 kg/d for multiparous cows.

Days open, days to first service, pregnancy rate and percentage of cows pregnant

by 150 DIM were recorded. All analyses of reproductive variables were based on cows that were confirmed pregnant by 200 DIM. The Ovsynch[®] protocol (Pursley et al., 1996) was used to synchronize breeding for all cows at 70 DIM.

Statistical Analysis

All blood measurements and SCC data were log transformed to correct for heterogeneity of variance. Milk production, BCS and blood measurements were analyzed as repeated measures using the PROC MIXED procedure of SAS [version 6.1; SAS, (1989)]. The statistical model included the fixed effects of treatment, parity, time, two- and three-way interaction terms of all fixed effects, the random effect of cow nested within treatment, and residual error. Blood measurements were analyzed and reported for the prepartum and postpartum periods separately, or across both periods (periparturient period). For all models, non-significant interaction terms ($P > 0.15$) were removed in a backwards stepwise manner. Udder edema and reproduction data were analyzed using PROC MIXED with main effects of farm, parity and treatment, and all two- and three-way interaction terms. Incidences of health disorders and percentage of cows pregnant by 150 DIM were analyzed using the PROC GENMOD procedure of SAS [version 6.1; SAS, (1989)] and differences were determined by Chi-Square tests. Differences between treatments were determined by F-test. Least squares means and standard error of the means are reported for all data except blood and SCC measurements. Because of the transformations to remove heterogeneity, 95% confidence intervals are reported instead of standard error of the means for blood and SCC data. Statistical significance was declared at $P < 0.05$, and tendency towards significance at $P > 0.05$ to $P < 0.15$.

RESULTS AND DISCUSSION

Diet Composition

Cows were fed treatment diets for 17 ± 6 d prepartum (mean $\pm$ standard deviation). Ingredient and chemical compositions of the treatment diets are presented in Table 1. The only differences in ingredient compositions between the diets were the concentration of corn grain and corn silage. As expected, diets were similar in CP, EE and mineral concentrations. The SC diet had higher DM content and smaller particle size because of the replacement of corn silage with dry ground corn. The NFC concentrations of the diet were more similar than expected. Diets fed from parturition to 150 DIM are shown in Table 2. Ingredient composition of diets differed slightly, but chemical compositions among diets were similar. Additionally, formulated ingredient and chemical composition of the diet fed during the early dry period prior to the treatment diets is shown in Table 3.

Body Condition and Udder Edema

Body condition scores of cows throughout the periparturient period are shown in Table 4. Treatment had no effect on BCS when analyzed as repeated measures across time ($P = 0.16$). BCS changes during the periparturient period are shown in Table 5. Effect of treatment on BCS changes tended to vary among cows of different parities (treatment by parity interaction; $P = 0.09$; Table 5). Cows of parity 1 fed the NC diet gained BCS in the late dry period, but cows of the same parity fed the SC diet lost BCS in

the late dry period. However, cows of parity 2 fed the NC diet lost BCS, whereas parity 2 cows fed the SC diet gained BCS in the late dry period. Both treatments promoted BCS gain of parity 3+ cows. It is doubtful that the small changes observed in BCS changes prepartum among parities and treatment have biological significance. There were no effects of treatment on BCS changes from parturition to 3 or 6 wk postpartum, or from 3 to 6 wk postpartum (Table 5). Therefore, treatment had no effect on rate or extent of BCS loss in early lactation.

Udder edema scores were not affected by treatment ($P = 0.24$; data not shown), but were influenced by parity ($P < 0.01$). Primiparous cows had higher udder edema scores compared with multiparous cows (1.9, 1.3, and 1.4, respectively). Feeding higher amounts of concentrates prior to parturition caused increased udder edema in some studies (Emery et al., 1969; Johnson and Otterby, 1981), but not in others (Greenlaugh and Gardner, 1958; Hathaway et al, 1957; Schmidt and Schultz, 1959). It is common for primiparous cows to have more udder edema at calving compared with multiparous cows (Greenhalgh and Gardner, 1958; Zamet et al., 1979).

Metabolic Variables

Plasma NEFA concentrations for the prepartum, postpartum and periparturient periods (both pre- and postpartum) are shown in Table 6. Treatment did not affect plasma NEFA concentrations during any period. Plasma NEFA increased as parturition approached and peaked at 7 d postpartum. The similar plasma NEFA concentrations in cows between treatments suggest that both treatment groups were in similar energy status in the periparturient period.

Cows fed the SC diet had lower plasma BHBA concentrations prepartum compared with cows fed the NC diet ($P < 0.01$; Table 7). There were no differences observed in the postpartum period, but the SC diet tended to reduce plasma BHBA across the entire periparturient period ($P = 0.07$). There was a significant treatment by time interaction for plasma BHBA in the periparturient period ($P = 0.02$). Plasma BHBA concentrations were higher prepartum for cows fed the NC diet, but were lower postpartum compared with cows fed the SC diet (Figure 1). Contrary to these results, feeding a higher energy diet prepartum decreased plasma NEFA concentrations (Holtenius et al., 1996; Minor et al, 1998; VandeHaar et al., 1995), but had no effect on ketone production (Boisclair et al., 1986; Minor et al, 1998; Olsson et al., 1997; Schmidt and Schultz, 1959) prior to parturition.

Plasma insulin concentrations are shown in Table 8. Cows fed the SC diet tended to have higher plasma insulin concentrations prior to parturition compared with cows fed the NC diet (14.17 vs. 12.63 $\mu\text{IU/ml}$; $P = 0.07$). Treatment had no effect on plasma insulin concentrations postpartum or during the periparturient period. Insulin has both antilipolytic and antiketogenic properties (Grummer, 1993; Holtenhuis, 1993). A reduction in lipolysis elicited by insulin reduces the substrates available for ketogenesis. Increasing the amount of corn grain in the diet may promote propionate and glucose production and subsequently, insulin secretion. Indeed, cows fed the SC diet tended to have higher insulin concentrations prepartum, probably a result of increased propionate and glucose production. However, no reduction in lipolysis, as measured by plasma NEFA concentration, was observed in this study. A metabolite of propionate, succinyl-CoA, directly inhibits ketogenesis (Lowe and Tubbs, 1985). Therefore, improved

carbohydrate supply and subsequently reduced ketogenesis in cows fed the SC diet may have caused the decrease in plasma BHBA as opposed to a decrease in NEFA concentrations.

Health Disorders

Incidence rates of health disorders and the occurrence of rectal temperatures above 39.4°C are shown in Table 9. Incidence rates of mastitis are not reported because of the low incidence rate (1.6%). There were no significant effects of treatment on incidence rates of any health disorders. However, incidence rates of displaced abomasum and ketosis were numerically higher for cows fed the SC diet compared with cows fed the NC diet. Several studies and reviews have reported that feeding higher energy diets prior to parturition may lead to an increased risk of displaced abomasum (Cameron et al., 1998; Shaver, 1997). Contrary to these findings, Curtis et al, (1985) reported that decreased incidences of left displaced abomasum and dystocia were associated with higher than average dietary energy intake during the last 3 wk prepartum.

Milk Yield, Composition and Somatic Cell Count

There were no differences in milk yields through 60 or 150 DIM between treatments (Tables 10 and 11). During the first 60 DIM there was a tendency towards a treatment by parity by time interaction ($P = 0.10$; Figure 2). The interaction was evident only in parity 2 and 3+ cows. Parity 2 cows fed the SC diet had lower milk yields during the first 15 d of lactation compared with parity 2 cows fed the NC diet (33.7 vs. 38.0 kg/d). In contrast, cows of parity 3+ fed the SC diet yielded more milk during the first 15

d of lactation compared with cows of the same parity fed the NC diet (44.2 vs. 37.4 kg/d). Additionally, cows of parity 3+ fed the SC diet tended to have slightly higher milk yields throughout the first 60 DIM compared with parity 3+ cows fed the NC diet (45.2 vs. 42.6 kg/d). Primiparous cows fed the SC diet had consistently lower milk yields through 60 DIM although the differences were small. No differences were observed in milk fat yield or content through 60 or 150 DIM (Tables 10 and 11). The differences in milk yield may have been a result of differences in DMI in early lactation. Because of experimental conditions, DMI was not measured in this study.

Treatment had no effect on milk protein content through either 60 or 150 DIM (Table 10 and 11). No treatment differences were observed for protein yield through 60 DIM, but there was a tendency for a treatment by parity by time interaction through 150 DIM ($P = 0.13$; Figure 3). Additionally, parity 3+ cows fed the SC diet had higher protein yields during the first 15 DIM compared with parity 3+ cows fed the NC diet. Cows of parity 1 fed the NC had higher protein yields through 150 DIM compared with parity 1 cows fed the SC diet (0.98 vs. 0.91 kg/d). The higher milk protein yield in parity 3+ cows during the first 15 d postpartum follows the trend in milk production. However, it is unclear why primiparous cows fed the NC diet tended to have consistently higher protein yields. Feeding more energy-dense diets to primiparous cows for the last 170 d of gestation had no effects on milk yield or composition (Grummer et al., 1995). Research evaluating the effects of prepartum diets on primiparous cows is scanty and deserves further attention.

There were no effects of treatment on ECM yields through 60 or 150 DIM (Table 12). There was no main effect of treatment on SCC, but there was a treatment by parity

interaction through 60 d ($P = 0.01$; Table 12 and Figure 4) and 150 d ($P = 0.08$; Table 12). The SCC of cows fed the SC diet decreased, and the SCC of cows fed the NC diet increased as parity increased. High concentrations of somatic cells in the mammary gland do not necessarily lead to increased incidences of mastitis (Erskine et al., 1988). Therefore, it is difficult to interpret these findings.

Reproduction

Several cows were selected not to re-breed ($n=12$), or were culled ($n=26$) and not included in analysis of reproductive measurements. Reproductive measurements are shown in Table 13. Treatment had no effect on days to first service or pregnancy rate. As mentioned, all cows were synchronized using Ovsynch[®] and targeted to breed at 70 DIM. Therefore, the similarity in days to first service is not surprising. There was no main effect of treatment on days open, but a treatment by parity interaction tended towards significance ($P = 0.08$; Figure 5). Cows in their first and second parities fed the SC diet had increased days open compared with cows of the same parities fed the NC diet. Contrary to this, parity 3+ cows fed the SC diet had decreased days open compared with parity 3+ cows fed the NC diet. It is unknown why treatment affected days open differently between parities. However, the number of replications used to compare interactions in reproductive measurements may be too low to draw conclusions.

CONCLUSIONS

With the exception of plasma concentrations of BHBA and insulin prepartum, the effect of treatment was dependent upon parity. The SC diet tended to benefit cows in

their third or greater parity, but had no or negative effects on production responses of first or second parity cows compared with the NC diet. Based on this study, parity should be considered when recommending feeding programs for late gestation dairy cows.

However, interactions between parity and diets fed prepartum require further research before specific recommendations can be derived.

TABLES AND FIGURES

Table 1. Formulated ingredient and analyzed chemical composition, and particle size distribution of experimental diets fed in the late dry period.

Item	NC ¹	SC ¹
Ingredient, % of DM		
Corn silage	45.5	24.2
Alfalfa-grass mixed hay	16.6	16.6
Beet pulp, dehydrated	13.4	13.4
Corn, dry ground	-	21.2
Custom mix ²	15.4	15.4
Soybean meal	9.1	9.2
Chemical composition, %		
DM	49.3	61.1
CP	15.8	16.2
ADF	26.2	22.6
NDF	39.3	34.9
EE	2.9	3.0
Ash	9.32	9.73
CP equivalent from ammonia	2.43	2.15
NFC ³	35.2	38.3
Ca	1.43	1.66
P	0.36	0.41
Mg	0.40	0.42
K	1.42	1.35
Na	0.18	0.22
Particle size⁴ (%)		
> 19.0 mm	7.1±4.1	6.4±3.6
8.0 to 19.0 mm	47.6±5.7	32.5±4.9
< 8.0 mm	45.3±3.9	61.1±3.9

¹Treatments: NC = no supplemental corn grain, SC = supplemental corn grain.

²Contained 19.9% CP, 2.8% fat, 7.21% Ca, 0.54% P, 1.4% Mg, 0.82% K, 0.67% Na, 5.9% Cl, 2.6% S, 3.6% Se, 53 KIU/kg of Vitamin A, 11 KIU/kg of Vitamin D, and 528 IU/kg of Vitamin E, dry basis; mix included wheat middlings and soyhulls as carriers.

³NFC = 100 - % NDF - % CP + % CP equivalent from ammonia - % EE - % ash.

⁴Particle size determined by the Penn State Particle Separator (Lammers et al., 1996), as-fed basis; mean ± SD.

Table 2. Formulated ingredient and analyzed chemical composition, and particle size distribution of diets fed postpartum.

Item	Early lactation ¹	Primiparous	Multiparous
Corn silage	23.9	27.6	29.4
Alfalfa silage	8.9	9.6	9.6
Corn, high moisture	18.3	20.0	17.9
Corn grain	8.8	10.9	9.2
Corn distillers dried grains	5.9	11.3	11.0
Soybean meal	12.7	9.6	10.6
Custom mix ²	5.6	5.7	5.2
Beet pulp, dehydrated	4.8	3.5	5.6
Alfalfa-grass mixed hay	8.8	-	-
Wheat straw	2.3	1.9	1.6
Chemical composition, %			
DM	56.7	52.4	51.6
CP	18.7	18.0	18.2
ADF	19.7	18.4	19.1
NDF	30.4	29.3	28.9
EE	3.4	3.9	4.0
Ash	8.1	8.5	8.0
CP equivalent from ammonia	0.93	1.05	1.06
NFC ³	40.3	41.4	42.0
Ca	1.15	1.22	1.13
P	0.56	0.61	0.59
Mg	0.37	0.38	0.37
K	1.46	1.43	1.40
Na	0.65	0.66	0.56
Particle size ⁴ (%)			
> 19.0 mm	5.1±2.7	3.4±1.9	3.0±1.4
8.0 to 19.0 mm	36.5±3.1	39.0±3.4	41.3±3.9
< 8.0 mm	58.4±2.6	57.6±3.2	55.7±3.5

¹Cows of all parities were fed this diet for the first 17 DIM, approximately.

²Contained 23.3% CP, 0.5% fat, 12.4% Ca, 2.9% P, 2.8% Mg, 0.4% K, 8.9% Na, 11.0% Cl, 1.7% S, 3 ppm of Co, 254 ppm of Cu, 1270 ppm of Fe, 13 ppm of I, 1016 ppm of Mn, 6 ppm Se, 152 KIU/kg of Vitamin A, 25 KIU/kg of Vitamin D, and 484 IU/kg of Vitamin E.

³NFC = 100 - % NDF - % CP + % CP equivalent from ammonia - % EE - % ash.

⁴Particle size determined by the Penn State Particle Separator (Lammers et al., 1996), as fed basis; mean ± SD.

Table 3. Formulated ingredient and chemical composition of the diet fed in the early dry period.

Ingredient	% of DM
Corn silage	35.0
Alfalfa silage	41.9
Beet pulp, dehydrated	20.7
Mineral mix ¹	2.4
Chemical composition, %	
DM	40.9
CP	15.6
ADF	28.7
NDF	43.2
EE	2.8
Ash	6.8
NFC ²	31.7
Ca	1.03
P	0.32
Mg	0.34
K	1.52
Na	0.11

¹Contained 15.0% CP, 2.0% EE, 2.0% Ca, 0.2% P, 1.4 ppm of Se, 14 KIU/kg of Vitamin A, 3.1 KIU/kg of Vitamin D, and 195 KIU/kg of Vitamin E.

²NFC = 100 - % NDF - % CP - % EE - % ash.

Table 4. Least squares means and statistical significance of body condition scores (BCS) during the periparturient period.

Variable	BCS	SEM
NC ¹	3.13	0.05
SC ¹	3.22	0.05
Parity 1	3.27	0.07
Parity 2	3.11	0.06
Parity 3+	3.14	0.05
Time, d relative to Parturition		
-14	3.56	0.04
-7	3.55	0.04
2	3.38	0.04
21	2.69	0.04
42	2.69	0.04
P-value		
Treatment	0.16	
Parity	0.18	
Time	< 0.01	
Parity*time	< 0.01	

¹Treatments: NC = no supplemental corn grain, SC = supplemental corn grain.

Table 5. Least squares means and statistical significance of body condition score changes during the periparturient period.

Variable	Prepartum	SEM	Weeks postpartum					
			0 to 6	SEM	0 to 3	SEM	3 to 6	SEM
NC ¹	0.03	0.03	-0.84	0.06	-0.84	0.05	0.01	0.04
SC ¹	0.00	0.03	-0.88	0.06	-0.90	0.05	0.03	0.04
Parity 1	-0.01	0.04	-0.92	0.08	-0.90	0.08	-0.02	0.06
Parity 2	-0.01	0.03	-0.63	0.07	-0.74	0.07	0.14	0.05
Parity 3+	0.06	0.03	-1.03	0.06	-0.97	0.06	-0.06	0.04
NC*parity 1	0.06	0.05						
NC*parity 2	-0.05	0.05						
NC*parity 3+	0.08	0.04						
SC*parity 1	-0.08	0.06						
SC*parity 2	0.03	0.05						
SC*parity 3+	0.03	0.04						
P-value ²								
Treatment (Trt)	0.40		0.60		0.38		0.85	
Parity	0.22		< 0.01		0.04		0.01	
Trt*parity	0.09		RM ²		RM		RM	

¹Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

²Independent variables were removed (RM) from the statistical model if $P > 0.15$.

Table 6. Least squares means and statistical significance of plasma NEFA concentrations during prepartum, postpartum or periparturient periods.

Variable	NEFA ($\mu\text{Eq/L}$)			95% confidence intervals (lower limit/upper limit)		
	Prepartum	Postpartum	Periparturient ¹	Prepartum	Postpartum	Periparturient
NC ²	124.7	346.5	177.8	109.8/141.6	315.9/380.2	162.9/194.1
SC ²	121.7	376.8	188.3	107.5/137.8	342.1/415.1	172.5/205.6
Parity 1	185.7	466.6	255.9	156.4/220.4	409.0/532.4	226.9/288.7
Parity 2	83.6	259.4	127.4	70.3/99.4	230.8/291.6	112.9/143.9
Parity 3+	120.4	389.8	187.8	105.3/137.7	352.7/430.8	170.8/206.6
Time, d						
relative to						
parturition						
-14	86.2		85.6	71.5/103.5		68.7/106.4
-11	104.5		99.5	90.4/120.7		84.8/116.7
-8	115.5		115.6	102.5/130.1		101.5/130.3
-5	140.3		139.8	126.3/155.8		126.5/155.9
-2	194.2		190.6	175.6/215.1		172.4/210.6
2		428.9	431.3		391.1/470.4	391.5/473.0
7		411.5	407.5		375.5/451.1	376.2/450.3
14		267.2	267.7		243.2/293.8	244.7/294.4
P-value ³						
Treatment	0.77	0.21	0.33			
Parity	<0.01	<0.01	<0.01			
Time	<0.01	<0.01	<0.01			
Parity*time	0.13	0.03	0.06			

¹Periparturient period = combined pre- and postpartum periods.

²Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

³All 2- and 3-way interaction terms not listed were removed from the statistical model ($P > 0.15$).

Table 7. Least squares means and statistical significance of plasma BHBA concentrations for prepartum, postpartum or periparturient periods.

Variable	BHBA (mg/dl)			95% confidence intervals (lower limit/upper limit)		
	Prepartum	Postpartum	Periparturient ¹	Prepartum	Postpartum	Periparturient
NC ²	5.79	7.99	6.53	5.39/6.21	7.35/8.68	6.08/7.02
SC ²	4.97	8.28	5.97	4.64/5.33	7.59/9.03	5.58/6.39
Parity 1	5.19	9.61	6.32	4.72/5.70	8.54/10.82	5.77/6.92
Parity 2	5.14	6.54	5.80	4.70/5.62	5.89/7.26	5.29/6.36
Parity 3+	5.79	8.56	6.65	5.38/6.23	7.82/9.36	6.19/7.15
Time, d relative to parturition						
-14	5.36		5.41	4.83/5.94		4.56/6.42
-11	5.27		5.12	4.84/5.73		4.55/5.76
-8	5.26		5.24	4.91/5.63		4.78/5.74
-5	5.25		5.26	4.94/5.58		4.87/5.68
-2	5.69		5.63	5.37/6.03		5.23/6.06
2		7.24	7.26		6.70/7.84	6.78/7.78
7		10.05	10.05		9.30/10.87	9.38/10.75
14		7.38	7.40		6.81/7.99	6.90/7.93
P-value ³						
Treatment (Trt)	< 0.01	0.55	0.07			
Parity	0.06	< 0.01	0.07			
Time	0.06	< 0.01	< 0.01			
Trt*time	RM ⁴	RM	0.02 ⁵			
Parity*time	RM	< 0.01	< 0.01			

¹Periparturient period = combined pre- and postpartum periods. ²Treatments: NC = no supplemental corn grain; SC = supplemental corn grain. ³All 2- and 3-way interaction terms not listed were removed from the statistical model ($P > 0.15$). ⁴Independent variables were removed (RM) from the statistical model if $P > 0.15$. ⁵See Figure 1.

Table 8. Least squares means and statistical significance of plasma insulin concentrations for prepartum, postpartum or periparturient periods.

Variable	Insulin (μ U/dl)				95% confidence intervals (lower limit/upper limit)	
	Prepartum	Postpartum	Periparturient ¹	Prepartum	Postpartum	Periparturient
NC ²	12.63	9.42	11.49	11.47/13.92	8.80/10.08	10.72/12.31
SC ²	14.17	9.60	11.99	12.90/15.56	8.94/10.30	11.18/12.85
Parity 1	11.94	9.75	11.01	10.47/13.61	8.85/10.74	10.01/12.11
Parity 2	15.91	10.32	13.57	13.91/18.20	9.48/11.24	12.36/14.90
Parity 3+	12.61	8.54	10.81	11.39/13.96	7.93/9.19	10.03/11.65
Time, d relative to parturition						
-14	18.92		18.56	16.09/22.24		15.87/21.71
-11	15.56		15.55	13.79/17.57		13.86/17.44
-8	13.07		13.06	11.87/14.4		11.94/14.28
-5	11.12		11.13	10.23/12.1		10.31/12.01
-2	10.00		10.10	9.24/10.84		9.39/10.87
2		10.08	10.00		9.47/10.75	9.34/10.71
7		8.64	8.64		8.11/9.20	8.07/9.25
14		9.85	9.83		9.24/10.51	9.16/10.52
P-value ³						
Treatment	0.07	0.70	0.37			
Parity	< 0.01	< 0.01	< 0.01			
Time	< 0.01	< 0.01	< 0.01			
Parity*time	0.13	0.01	0.01			

¹Periparturient period = combined pre- and postpartum periods.

²Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

³All 2- and 3-way interaction terms not listed were removed from the statistical model ($P > 0.15$).

Table 9. Incidence rates and statistical significance of health disorders, and abnormal rectal temperatures.

Variable	n	Incidence rates (%)			
		Displaced abomasum	Ketosis	Retained placenta	Rectal temperature ¹
NC ²	93	7.5	6.5	15.0	44.0
SC ²	97	10.3	11.3	12.4	48.0
Parity 1	50	16.0	22.0	18.0	60.0
Parity 2	58	5.2	1.7	13.8	42.0
Parity 3+	81	6.2	4.9	9.9	38.3
P-value ³					
Treatment		0.60	0.27	0.53	0.82
Parity		0.07	<0.01	0.20	0.02

¹Rectal temperature > 39.4°C for at least 1 d during the first 12 d of lactation.

²Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

³Treatment by parity interactions were removed from all statistical models ($P > 0.15$).

Table 10. Least squares means and statistical significance of milk yield and composition through 60 DIM.

Variable	Milk			Fat			Protein			
	yield (kg/d)	SEM	Fat (%)	SEM	yield (kg/d)	SEM	Protein (%)	SEM	yield (kg/d)	SEM
NC ¹	37.4	0.8	4.68	0.12	1.77	0.07	3.07	0.03	1.12	0.02
SC ¹	37.4	0.8	4.79	0.12	1.82	0.07	3.02	0.03	1.12	0.02
Parity 1	27.3	1.2	4.36	0.16	1.21	0.11	3.01	0.05	0.82	0.03
Parity 2	40.9	1.0	4.89	0.14	2.01	0.09	3.11	0.04	1.26	0.03
Parity 3+	43.9	0.8	4.95	0.12	2.17	0.08	3.01	0.04	1.27	0.03
Time, d relative to parturition										
15	32.9	0.8	5.10	0.15	1.66	0.09	3.35	0.05	1.07	0.03
30	37.7	0.7	4.83	0.11	1.86	0.07	3.12	0.03	1.17	0.02
45	39.4	0.7	4.55	0.10	1.86	0.06	2.87	0.03	1.12	0.02
60	39.5	0.7	4.46	0.10	1.81	0.06	2.85	0.03	1.11	0.02
P-value										
Treatment (Trt)	0.95		0.49		0.60		0.28		0.96	
Parity	<0.01		0.10		<0.01		0.13		<0.01	
Time	<0.01		<0.01		0.12		<0.01		<0.01	
Trt*parity	0.21		RM ²		RM		RM		RM	
Trt*time	0.33		RM		RM		RM		RM	
Parity*time	0.03		RM		RM		0.07		0.03	
Trt*parity*time	0.10 ³		RM		RM		RM		RM	

¹Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

²Independent variables were removed (RM) from the statistical model if $P > 0.15$.

³See Figure 2.

Table 11. Least squares means and statistical significance of milk yield and composition through 150 DIM.

Variable	Milk			Fat			Protein			
	yield (kg/d)	SEM	Fat (%)	SEM	yield (kg/d)	SEM	Protein (%)	SEM	yield (kg/d)	SEM
NC ¹	38.3	0.7	4.46	0.08	1.73	0.05	3.09	0.02	1.17	0.02
SC ¹	38.0	0.7	4.51	0.08	1.74	0.05	3.06	0.02	1.15	0.02
Parity 1	31.0	1.0	4.27	0.11	1.33	0.07	3.08	0.03	0.95	0.03
Parity 2	40.1	0.8	4.48	0.09	1.82	0.06	3.15	0.03	1.25	0.02
Parity 3+	43.3	0.7	4.71	0.08	2.06	0.05	3.00	0.02	1.28	0.02
Time ²										
P-value										
Treatment (Trt)	0.76		0.63		0.82		0.31		0.50	
Parity	<0.01		<0.01		<0.01		<0.01		<0.01	
Time	<0.01		<0.01		<0.01		<0.01		<0.01	
Trt*time	RM ³		RM		RM		RM		0.29	
Trt*parity	RM		RM		RM		RM		0.29	
Parity*time	<0.01		0.03		<0.01		0.06		<0.01	
Trt*parity*time	RM		RM		RM		RM		0.13 ⁴	

¹Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

²Time not shown.

³Independent variables were removed (RM) from the statistical model if $P > 0.15$.

⁴See Figure 3.

Table 12. Least squares means and significance of energy-corrected milk (ECM) and SCC through 60 or 150 DIM.

	ECM yield (kg/d)	SEM	SCC (cells/ml)	95% confidence interval	
Variable				Lower	Upper
0 to 60 DIM					
NC ¹	42.6	1.3	104,708	79,674	137,613
SC ¹	43.6	1.3	93,517	70,983	123,204
Parity 1	30.5	1.8	96,929	65,736	142,929
Parity 2	47.9	1.6	93,591	67,501	129,768
Parity 3+	50.9	1.4	106,810	80,330	142,017
Time, d relative to parturition					
15	39.4	1.6	184,702	131,268	259,886
30	44.6	1.2	99,983	73,109	136,735
45	44.5	1.1	72,558	53,982	97,529
60	43.8	1.1	71,557	52,607	97,334
P-value ²					
Treatment (Trt)	0.59		0.57		
Parity	< 0.01		0.82		
Time	< 0.01		< 0.01		
Trt*parity	RM ³		0.01 ⁴		
0 to 150 DIM					
NC	42.8	0.9	79,348	63,380	99,340
SC	42.9	0.9	80,465	64,094	101,023
Parity 1	33.8	1.4	76,672	55,843	105,272
Parity 2	45.2	1.2	77,304	59,101	101,114
Parity 3+	49.5	1.0	86,074	68,125	108,749
Time ⁵					
P-value					
Trt	0.96		0.93		
Parity	< 0.01		0.78		
Time	< 0.01		< 0.01		
Trt*parity	RM		0.08		
Parity*time	< 0.01		RM		

¹Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

²All 2- and 3-way interactions not shown were removed from the statistical model ($P > 0.15$).

³Independent variables were removed (RM) from the statistical model if $P > 0.15$.

⁴See Figure 4.

⁵Time not show

Table 13. Least squares means and statistical significance of reproductive measurements.

Variable	Days to first service	SEM	Days open	SEM	Pregnancy rate, %	SEM	% bred by 150 d
NC ¹	72	0.4	98	4.1	67.2	4.0	79.5
SC ¹	71	0.4	101	4.0	64.9	3.9	80.7
Parity 1	72	0.6	97	5.5	64.0	5.4	86.1
Parity 2	71	0.5	107	5.0	63.2	4.9	74.5
Parity 3+	72	0.5	94	4.3	71.0	4.2	81.3
P-values ²							
Treatment (Trt)	0.60		0.57		0.67		0.88
Parity	0.62		0.15		0.40		0.53
Trt*parity	RM ³		0.08 ³		RM		RM

¹Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

²Independent variables were removed (RM) from the statistical model if $P > 0.15$.

³See Figure 5.

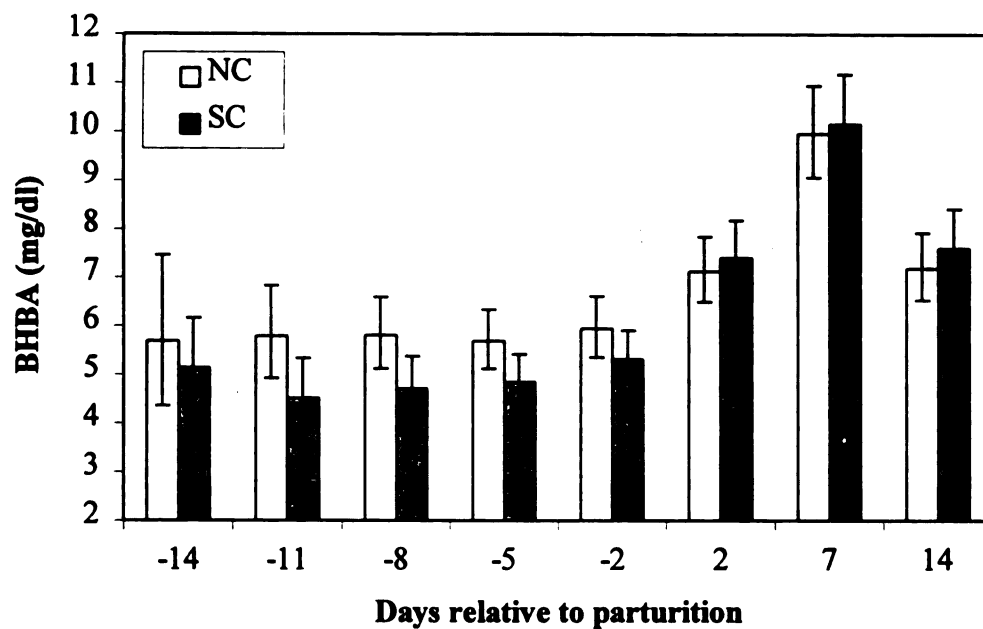


Figure 1. Least squares means and 95% confidence intervals for the interaction of treatment by time ($P = 0.02$) on plasma BHBA concentrations in the periparturient period. Other significant variables in the model included: treatment ($P = 0.07$), parity ($P = 0.07$), time ($P < 0.01$), and parity by time ($P < 0.01$). Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

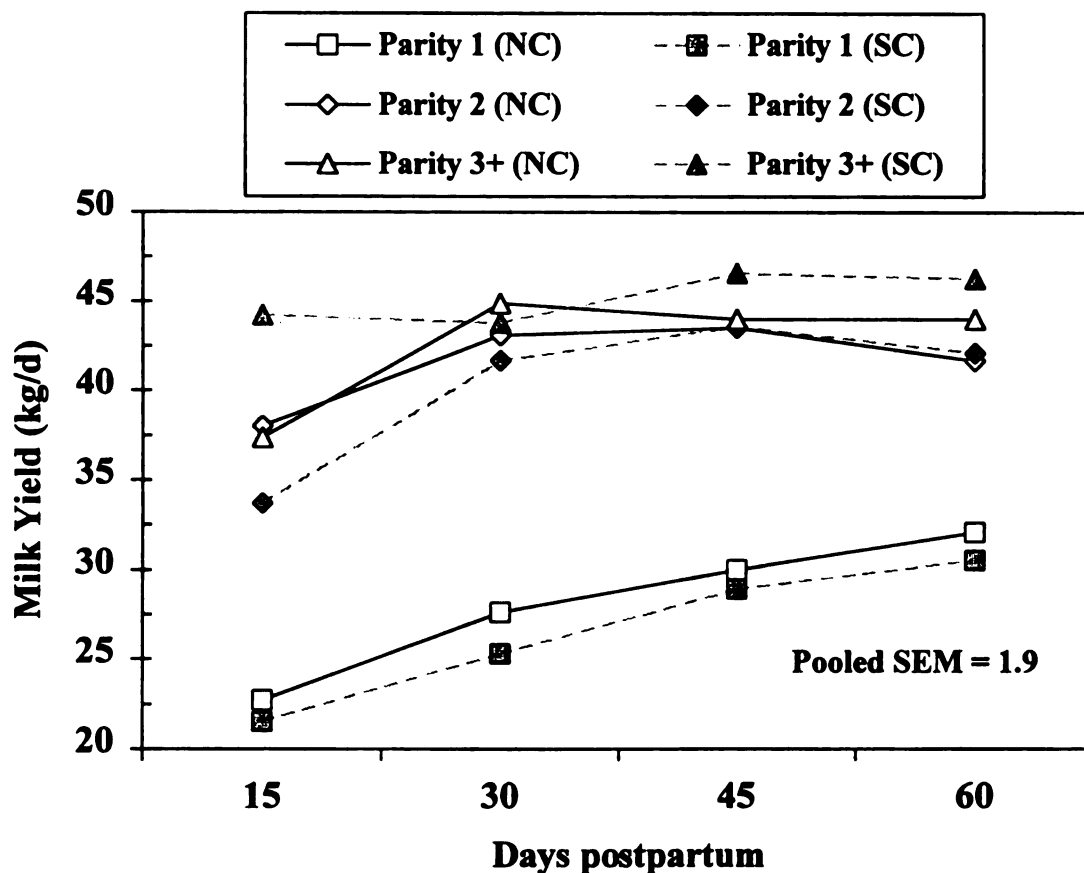


Figure 2. Least squares means and standard error of the means for the interaction of treatment by parity by time ($P = 0.10$) for milk production during the first 60 DIM. Other significant variables in the model included: Parity ($P < 0.01$), time ($P < 0.01$), and parity by time ($P = 0.03$). Days postpartum on the x-axis represent an average of the previous 15 d. Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

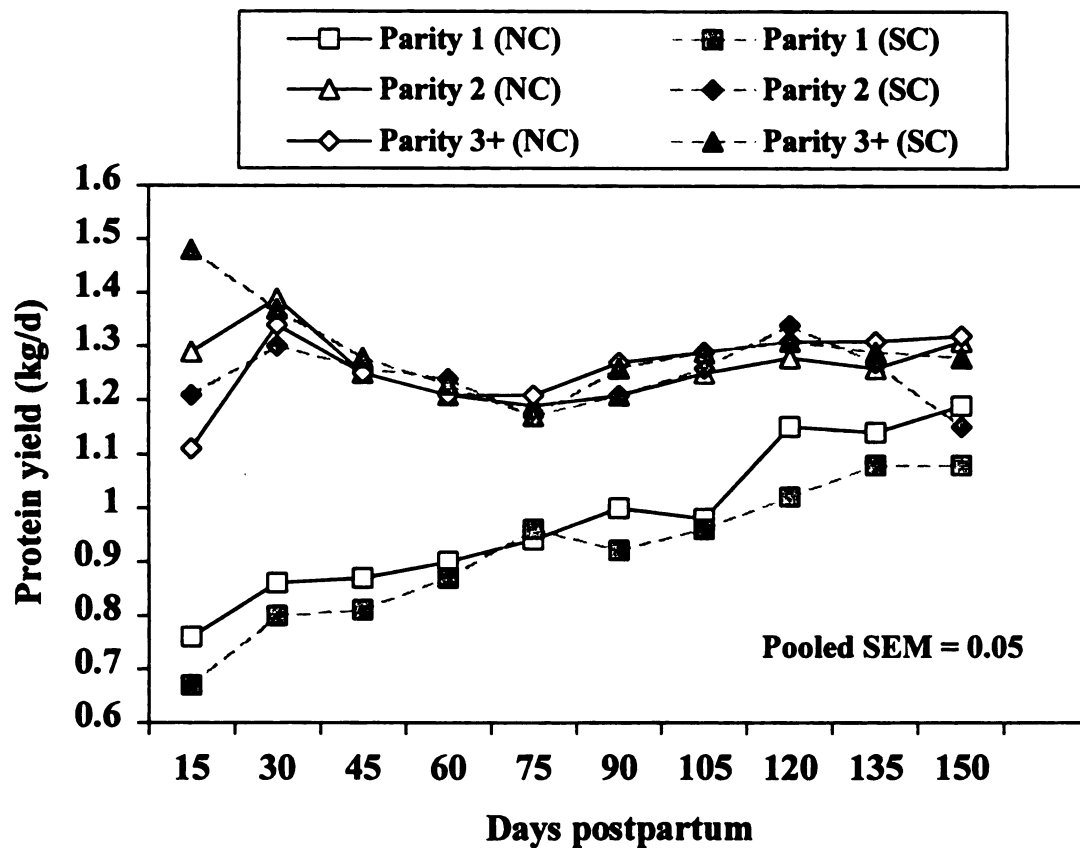


Figure 3. Least squares means and standard error of the means for the interaction of treatment by parity by time ($P = 0.13$) for protein yield through 150 DIM. Other significant variables in the model included: parity ($P < 0.01$), time ($P < 0.01$), and parity by time ($P < 0.01$). Days postpartum on the x-axis represent an average of the previous 15 d. Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

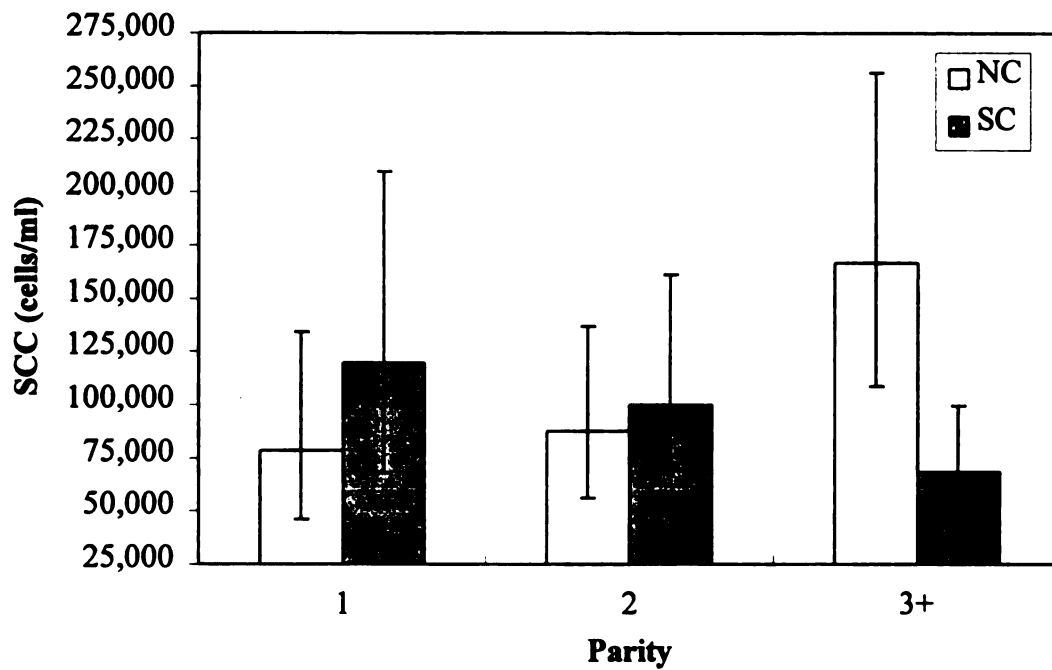


Figure 4. Least squares means and 95% confidence intervals for the interaction of treatment by parity ($P = 0.01$) for somatic cell count through 60 DIM. The only other significant variable in the model was time ($P < 0.01$). Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

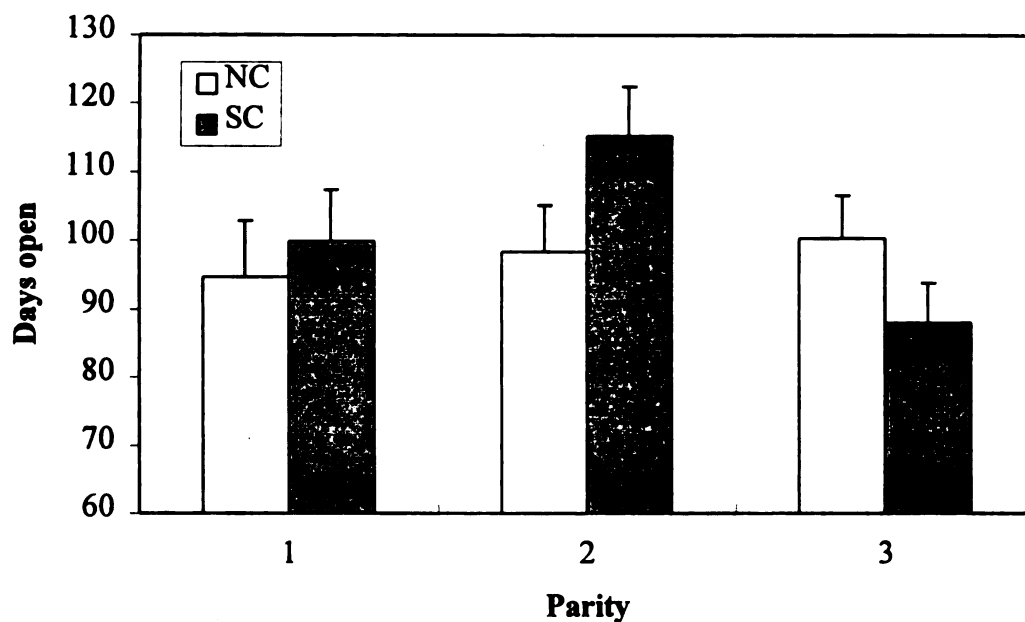


Figure 5. Least squares means and standard error of the means for interaction of treatment by parity ($P = 0.08$) on days open. There were no other significant effects in the model. Treatments: NC = no supplemental corn grain; SC = supplemental corn grain.

CHAPTER 4

PERIPARTUM RESPONSES OF DAIRY COWS TO ALTERING LENGTH OF THE LATE DRY PERIOD

ABSTRACT

One hundred eighty-nine cows in two commercial dairy farms were assigned randomly to enter the late dry period at either 3 or 6 wk prepartum. During this time, cows were fed diets with increased nutrient and energy densities compared with diets fed earlier in the dry period. Cows in the late dry period ≤ 26 d were designated short late dry period (S) and those in the late dry period > 26 d were the long late dry period (L). Cows in L tended to gain more body condition during the late dry period. Treatment L improved energy status of cows during the first 2 wk postpartum as indicated by a trend towards lower plasma non-esterified fatty acid and higher insulin concentrations postpartum, and reduced BCS loss during the first 3 wk postpartum. Cows in L had higher milk protein content through 60 DIM, but tended to have lower milk fat content and yield through 150 DIM. In Farm 1, cows in L lost more body condition from 3 to 6 wk postpartum, had a higher incidence rate of metritis and a longer interval to first service. Additionally, cows in L in Farm 1 produced less milk and had higher somatic cell counts through 150 DIM. Increasing the length of time cows were in the late dry group elicited profound changes in Farm 1, but had little effect in Farm 2. Based on these results, the L treatment may improve energy status immediately postpartum, but long-term effects varied between farms, likely due to management differences.

INTRODUCTION

The late dry period is the time prior to calving when often dairy cows are fed and managed differently than cows earlier in the dry period. It is common for cows to be fed diets with increased concentrations of energy, protein, and certain vitamins and minerals during the late dry period. The purpose of feeding dry cows differently during the last few weeks of gestation is to help meet the physiological changes occurring at this time. During the last few weeks of gestation, an accelerated decrease in DMI (Bertics et al., 1992) coincides with increasing energy requirements for conceptus growth (Bell, 1995) and lactogenesis (Davis et al., 1979). Cows may mobilize body stores during the late dry period in an attempt to compensate for the inadequate energy intake. Excess mobilization of body stores prepartum may predispose cows to several health disorders (Dyk et al., 1995).

The practice of partially substituting concentrates for lower energy forages is a method to increase energy density of diets and help minimize declining or negative energy status of dairy cows in the late dry period. Additionally, increased amounts of concentrates help ruminal microbial populations adapt to a more fermentable diet that is typically fed in early lactation, and promotes ruminal papillae development (Goff and Horst, 1998). Dirksen et al. (1985) showed that feeding increasing amounts of grain to cows increased ruminal papillae growth and VFA absorption. However, ruminal papillae did not reach maximum size until 5 to 6 wk after initiation of the high energy diet. In support of this concept, recent research showed that feeding a diet containing 43.5% corn grain to non-pregnant, non-lactating dairy cows caused ruminal papillae to grow (Xu and Allen, 1998). The size of ruminal papillae increased linearly over the 28 d

cows were sampled. Therefore, cows may benefit from a higher energy diet for longer than the traditional 2 to 3 wk late dry period to achieve maximal papillae size and to improve energy status at parturition and early lactation.

Feeding a higher energy diet to dry cows for a longer period of time also may promote body condition gain. Higher producing cows have more difficulty replenishing body reserves in late gestation than lower producing cows (Wildman et al., 1982). If body condition is inadequate at dry-off, increasing body condition to recommendations during the dry period may improve postpartum performance. Increasing body condition during the dry period has resulted in improved milk yields (Boisclair et al., 1986). Yet, other studies showed no benefits of promoting body condition gain during the dry period (Jones and Garnsworthy, 1989; Garnsworthy and Huggett, 1992; Garnsworthy and Jones, 1987). However, these experiments evaluated the effects of over-conditioning dry cows. No studies have evaluated the optimal length of time the late dry diet should be fed.

The objective of this experiment was to determine the effects of altering the length of the late dry period on body condition, periparturient health and energy status, and postpartum production and reproduction of dairy cows.

MATERIALS AND METHODS

Cows and Treatments

One hundred eighty-nine cows in two commercial dairy farms were completely randomized and assigned to enter the late dry period at either 3 or 6 wk prepartum. Retrospectively, cows were assigned to treatment based on how many days they spent in the late dry period (Table 1). Cows in the late dry period ≤ 26 d were designated short

late dry period (S) and cows in the late dry group longer than 26 d were designated long late dry period (L). Means and standard deviations for days spent in the late dry group were calculated for each treatment group and seven cows lying outside two standard deviations were removed from the study. There were 43 first parity, 63 second parity, and 83 third or greater parity cows in the experiment (Table 1). All cows within farms were housed together and were fed the same diets prepartum (Table 2) and were grouped as primiparous or multiparous after calving and fed accordingly (Tables 3 and 4). Additionally, cows in Farm 2 were grouped and fed separately during the first 2 to 3 wk of lactation (early lactation group; Table 4).

Sample Collection and Analysis

Silage dry matter was determined weekly using a Koster Tester (Koster Crop Tester, Inc., Medina, OH) and adjustments were made to maintain the same diet composition, dry basis. TMR were sampled weekly through 150 DIM and dried at 55° C for 72 h for future analysis. Particle size distribution of TMR samples, as-fed basis, was determined using the Penn State Particle Separator (Nasco, Fort Atkinson, WI; Lammers et al., 1996). Feed samples were ground through a Wiley mill (1 mm screen, Authur H. Thomas, Philadelphia , PA) and composited monthly. Samples were analyzed for NDF, ADF, CP, ether extract (EE), ash, ammonia and minerals (Northeast DHI Forage Laboratory, Ithaca, NY).

One evaluator scored cows for BCS [five-point scale where 1 = thin to 5 = fat; (Wildman et al., 1982)] weekly prepartum, at calving, and 3 and 6 wk postpartum.

Additionally, one evaluator assigned udder edema scores (0 = none , 1 = mild, 2 = moderate, and 3 = severe) within 3 d following parturition.

Blood samples were collected in evacuated test tubes containing sodium heparin (Vacutainer; Becton Dickson Vacutainer Systems USA, Rutherford, NJ) from the coccygeal vessels twice weekly prior to parturition, within 3 d following parturition, and 1 and 2 wk postpartum. Blood samples were collected at approximately 20 h after feeding in Farm 1 and about 7 h after feeding in Farm 2. Samples were stored on ice during transport to the laboratory. Upon arrival they were centrifuged and plasma was stored at -4°C until later analysis of NEFA (NEFA-C kit, Waco Chemicals USA, Richmond, VA) with modifications (Johnson and Peters, 1993), insulin (Coat-A-Count, Diagnostic Products Corporation, Los Angeles, CA), and BHBA (310-A, Sigma Diagnostics, St. Louis, MO). All reagents in the BHBA assay except beta-hydroxybutyrate dehydrogenase were reduced proportionally to fit into 350 µl wells of cell culture plates. Beta-hydroxybutyrate dehydrogenase was added at twice the reduced dose to shorten the incubation time. Inter-assay and intra-assay coefficients of variation for BHBA, NEFA and insulin were 5.9 and 7.1, 6.9 and 8.4, and 4.4 and 7.0, respectively.

Herdspersons and veterinarians were responsible for recording incidences of health disorders and reproductive measurements throughout the experiment. Health disorders are defined as the following: displaced abomasum was an abnormal location of the abomasum diagnosed by percussion that required corrective surgery; ketosis was a positive urine ketone body test of moderate or greater (Ketostix; Bayer Corp., Elkhart, IN); mastitis was an abnormal stripping or inflammation of the udder

which required treatment; retained placenta was fetal membranes retained for greater than 24 h after parturition; and, metritis was diagnosed as an uterine infection which required treatment.

Farm 2 had an intensive milk fever prevention protocol. All third parity cows received an oral bottle of CMPK (10.8 g Ca, 75 g dextrose; Jice Pharmaceuticals Co., Lowell, MI) immediately following parturition. All fourth parity cows, or any cows having twins, received a bottle of CMPK orally plus 500 ml of Calnate (10.7 g Ca; The Butler Co., Columbus, OH) subcutaneously. In addition, Farm 2 measured rectal temperatures on all cows daily through 12 DIM. For analysis, cows having a rectal temperature above 39.4° C for at least 1 d during the first 12 DIM were considered abnormal. In Farm 1, milk weights were collected monthly and were analyzed for fat and protein content and SCC every three months (Michigan DHIA). Daily milk weights through 150 DIM also were recorded on Farm 1. In Farm 2, individual milk weights were recorded every 2 wk and samples were analyzed for fat and protein content and SCC monthly (Michigan DHIA). Energy-corrected milk was calculated from the equation $ECM\ (lb) = 0.3246 \times \text{milk yield}\ (lb) + 12.86 \times \text{fat yield}\ (lb) + 7.04 \times \text{protein yield}\ (lb)$ (Dairy Herd Improvement Glossary, Fact Sheet A-4, 1999). Both farms used bST (Monsanto Co., St. Louis, MO) during the experiment. All cows received bST at 100 DIM in Farm 1 and remained on bST throughout the sampling periods. The voluntary waiting period for bST administration on Farm 2 was 63 and 90 d for multiparous and primiparous cows, respectively. Cows received bST unless BCS was less than 2.5 or milk yield was greater than 40 kg/d for primiparous cows and 50 kg/d for

multiparous cows. Over 60% of the cows in each treatment in Farm 2 received bST during the sampling periods.

Days open, days to first service, pregnancy rate and percentage of cows pregnant by 200 DIM were recorded in each farm. All analyses of reproductive variables were based on cows that had been confirmed pregnant by 200 DIM. The voluntary waiting period was 55 d on Farm 1 and 70 days on Farm 2. Farm 2 used the Ovsynch® protocol (Pursley et al., 1996) to synchronize breeding for all cows on the experiment. Several cows in Farm 1 were exposed to a bull for natural service and were removed from analysis of reproductive measurements.

Statistical Analysis

All blood and SCC data were log transformed to correct for heterogeneity of variance. Milk production data from DHIA in both farms were reduced to monthly means for analysis. Additionally, in Farm 1, daily milk weights were available for analysis. These data were reduced to weekly means for subsequent analysis. Milk production and blood measurements were analyzed as repeated measures using the PROC MIXED procedure of SAS [version 6.1; SAS (1989)]. The statistical model for milk and blood data included the fixed effects of treatment, parity, farm, time, two- and three-way interaction terms of all fixed effects, the random effect of cow nested within treatment and farm, and the residual error. Blood measurements were analyzed and reported for the prepartum or postpartum periods separately, or across both periods (periparturient period). Udder edema, BCS changes, days open, days to first service, and pregnancy rate were analyzed using PROC MIXED procedure of SAS [version 6.1; SAS (1989)] with

farm, parity, treatment and all two- and three-way interaction terms, and the residual error. Incidences of health disorders and percentage of cows pregnant by 200 DIM were analyzed using the PROC GENMOD procedure of SAS [version 6.1; SAS (1989)] and differences were determined by chi-square tests. For all models, non-significant variables ($P > 0.15$) were removed in a backwards stepwise manner. Least squares means could not be generated for BCS if treatment was in the model because treatment was confounded in time. For cows in L, BCS were recorded before 3 wk prepartum. However, cows in S only had BCS data during the last 3 wk prepartum and had no values prior to this time. Therefore, a separate model was used for analysis each treatment to generate least squares means. The model included the fixed main effects of farm, parity, time, the random effect of cow, and the residual error. Differences between treatments for all models were determined by F-test. Least squares means and standard error of the means are reported for all data except blood and SCC measurements. Because of the transformations to remove heterogeneity, 95% confidence intervals are reported instead of standard error of the means for blood and SCC data. Statistical significance was declared at $P < 0.05$, and tendency towards significance at $P > 0.05$ to $P < 0.15$.

RESULTS AND DISCUSSION

Diet Composition

Chemical compositions of diets fed pre- and postpartum in both farms are presented in Tables 2, 3, and 4. The diets fed prepartum varied in ingredient composition between farms, but the analyzed chemical compositions were similar. Overall, particle size was larger in the diet fed prepartum in Farm 1 because of a larger inclusion of hay

and the corn fed in Farm 2 was finely ground. Chemical composition of diets fed postpartum was similar between primi- and multiparous groups within and between farms (Tables 3 and 4). The exception is the inclusion of whole cottonseed that increased the EE concentration of diets in Farm 1. Additionally, the ingredient composition of the diets fed during the early dry period prior to the treatment diets is shown in Table 5. Diets fed during this time could potentially affect papillae development and ruminal microbial adaptation. However, diets fed during the early dry period were similar in chemical composition.

Body Condition and Udder Edema

Cows entered the late dry group with similar BCS regardless of treatment (Table 6). Cows in S and L gained 0.08 and 0.14 BCS units, respectively in the late dry group (Table 7). An objective of this study was to determine if lengthening the late dry period would increase BCS. The difference of body condition gain in the late dry group between treatments of 0.06 BCS units tended towards significance ($P = 0.14$). Parity had a strong influence on body condition gain (Table 7). Cows entering their second lactation gained more body condition during the late dry period than cows of parity 1 or 3+. Cows entering their third or greater parity did not gain substantial BCS during the late dry period (0.01 and 0.02 BCS units in Farm 1 and 2, respectively). Cows of parity 2 entered the late dry period with the lowest BCS of any parity (3.70, 3.40, 3.60 for parity 1, 2 and 3+, respectively; data not shown). A review of several studies measuring DMI of periparturient cows reported increased DMI of cows with lower BCS (Hayirli, 1998). This may partially explain the differences in BCS gain observed in the late dry period.

There were no significant differences between treatments in total BCS loss during the first 6 wk of lactation (Table 7). However, cows in S lost more BCS during the first 3 wk postpartum compared with cows in L (-1.15 vs. -0.95; $P < 0.01$). In contrast, cows in L tended to lose more BCS from 3 to 6 wk postpartum compared with cows in S (-0.13 vs. -0.27; $P = 0.06$). A tendency towards a farm by treatment interaction ($P = 0.13$) for BCS change from 3 to 6 wk was observed. In Farm 1, cows in L lost more BCS from 3 to 6 wk postpartum than cows in S (-0.33 vs. -0.08). Treatment had no effect on BCS change in Farm 2 (-0.18 vs. -0.21 for S and L, respectively). Based on BCS changes, cows in S appeared to be in poorer energy status during the first 3 wk postpartum in both farms. At 6 wk postpartum, cows in S appeared to be closer to positive energy status compared with cows in L in Farm 1 or in similar energy status with L in Farm 2 as indicated by BCS changes from 3 to 6 wk (Table 7). Several studies have shown increased BCS losses in early lactation if BCS is high at parturition (Garnsworthy and Jones, 1987; Garnsworthy and Topps, 1982; Pedron et al., 1993; Smith et al., 1997; Treacher et al., 1986).

There were no treatment effects on udder edema scores ($P = 0.70$; data not shown). Parity did influence the severity of udder edema. Cows in their first parity had higher udder edema scores (1.6; $P = 0.05$) than cows in their second parity (1.4) or third and greater parities (1.3).

Metabolic Variables

Plasma NEFA concentrations for the prepartum, postpartum and periparturient (both pre- and postpartum) periods are shown in Table 8. There were no treatment

differences in plasma NEFA concentrations during the prepartum or periparturient periods. Plasma NEFA concentrations in the postpartum period tended to be higher for cows in S compared with cows in L (542.4 vs. 487.1 $\mu\text{Eq/L}$; $P = 0.15$). Plasma NEFA concentrations coincide with BCS data that showed a reduced BCS loss of cows in L during the first 3 wk postpartum. One hypothesis of this experiment was that increasing the time spent in the late dry group may improve energy status in early lactation. This hypothesis is supported partially by the lower BCS loss and tendency toward lower plasma NEFA of cows in L during the first 3 wk of lactation compared with cows in S. For both treatments, plasma NEFA concentrations showed an accelerated increase as parturition approached, peaked at 2 d (629.4 $\mu\text{Eq/L}$) and slowly declined at 7 and 14 d postpartum.

There was no effect of treatment on plasma BHBA concentrations during the prepartum, postpartum or periparturient periods (Table 9). Concentrations of BHBA increased gradually to peak values (9.24 mg/dl) at 7 d postpartum and declined by 14 d. Similar plasma BHBA concentrations between treatments suggest that treatment had no effect on hepatic lipid metabolism during the periparturient period.

Plasma insulin concentrations are shown in Table 10. There was a significant treatment by time interaction ($P = 0.03$) on plasma insulin across the periparturient period (Figure 1). Insulin concentrations of cows in L were lower prepartum and higher postpartum compared with cows in S. Although plasma insulin concentrations were numerically different between S and L (11.66 vs. 10.48 $\mu\text{IU/ml}$, respectively) in the prepartum period they were not significantly different ($P = 0.28$). Large variations in the insulin concentrations prepartum may have precluded detecting a statistical difference.

However, there was a tendency for a treatment by parity by farm interaction during the prepartum period ($P = 0.13$; Figure 2). In parity 1, L increased plasma insulin concentrations in farm 1, but decreased insulin in farm 2. L decreased insulin in cows of parities 2 and 3+ in a similar manner compared with S. Cows in L tended to have higher plasma insulin concentrations postpartum compared with cows in S (7.19 vs. 6.46 $\mu\text{IU/ml}$; $P = 0.12$). Additionally, a parity by treatment by time interaction tended toward significance ($P = 0.07$) in the postpartum period (Figure 3). Plasma insulin concentrations of primiparous cows in S decreased over the first 2 wk postpartum, but plasma insulin of primiparous cows in L increased during the same period. Plasma insulin concentrations of cows in parities 2 and 3+ followed similar patterns during the first 2 wk postpartum for both treatments. Lower plasma insulin concentrations postpartum for cows in S coincide with increased BCS losses during the first 3 wk and the tendency for higher plasma NEFA during the first 2 wk after parturition compared with cows in L. Feeding high concentrations of energy prepartum has been hypothesized to induce insulin resistance postpartum, thereby elevating plasma insulin (Holtenius, 1993). Feeding a higher energy diet for a longer period of time prepartum could further exacerbate insulin resistance. Cows in L were fed a higher energy diet for an additional 17 d on average compared with cows in S and potentially could be at an increased risk of insulin resistance. Yet, adipose tissue of cows in L may have been more sensitive to insulin as noted by the reduction in plasma NEFA concentrations.

Health Disorders

Incidence rates of health disorders are shown in Table 11. The incidence rate of milk fever was recorded, but not reported because of the low incidence rate. Farm 1 reported two cases and Farm 2 had no clinical cases of milk fever. Cases of metritis were not recorded in Farm 2. In addition, Farm 1 did not measure rectal temperatures. The only significant effect of treatment was on metritis incidence in Farm 1. Cows in L had an increased incidence rate (12.9 vs. 6.3; $P = 0.06$) of metritis compared with cows in S. Ten cases of metritis were reported for L and only two for S. It is surprising that cows in apparently more negative energy status (i.e., treatment S) during the first few weeks postpartum had a lower incidence rate of metritis. Several studies have reported non-significant increases in the incidence of health disorders when cows gained appreciable BCS during the dry period (Fronk et al., 1980; Keys et al., 1983; Treacher et al., 1986). However, these studies reported effects of gaining body condition well in excess of the present study.

Milk Yield, Composition and Somatic Cell Count

Milk yield and composition for 60 and 150 DIM are shown in Tables 12 and 13, respectively. Treatment had no effect on milk production during the first 60 DIM. Through 150 DIM, cows in the S tended to produce more milk compared with cows in L (41.4 vs. 39.2 kg/d; $P = 0.06$). However, effects of treatment are only evident in Farm 1 as indicated by the tendency towards a farm by treatment interaction ($P = 0.06$; Figure 4). Cows in Farm 1 in S produced 43.2 kg/d, whereas cows in L produced only 38.8 kg/d. In Farm 2, there was no difference in milk yield between treatments with both groups producing 39.5 kg/d through 150 DIM. The daily milk yields reported in Farm 1 also

support the effect of treatment on milk production (Table 14). Cows in S in Farm 1 tended to have greater milk yields through 56 DIM compared with cows in L ($P = 0.12$). Through 150 DIM, milk yields were greater for cows in S in Farm 1 compared with cows in L (42.0 vs. 36.6 kg/d; $P = 0.03$). Differences in management practices between farms may have influenced effects of treatment on milk production.

Treatment had no significant effect on milk fat content through 60 d ($P = 0.47$; Table 12), but milk fat content of cows in S tended to be higher compared with cows in L through 150 DIM ($P = 0.15$; Table 13). Because of higher milk production and numerically higher milk fat content, cows in S tended to have higher fat yields through 150 DIM than cows in L (1.62 vs. 1.52 kg/d; $P = 0.11$). No differences in fat yield between treatments were detected during the first 60 DIM. NEFA can be used as substrates for endogenous fatty acids synthesis in the mammary gland. Cows in S had higher plasma NEFA concentrations during the first 2 wk of lactation compared with cows in L. NEFA were measured only during the first 2 wk postpartum, but higher plasma NEFA concentrations may have persisted into lactation for cows in S and caused the higher milk fat content and yield.

Cows in L had higher protein content compared with cows in S through 60 DIM ($P < 0.01$), but not through 150 DIM. Parity by treatment ($P = 0.07$) and treatment by time ($P < 0.01$) interactions for the analysis for milk protein content through 60 DIM are shown in Figures 5 and 6, respectively. These interactions show that L increased milk protein content the most in cows of parities 1 and 2, and in the first month of lactation. Additionally, there was a parity by treatment by time interaction on milk protein percentage through 150 DIM ($P = 0.03$; Figure 7). Cows of all parities in S followed

similar patterns of milk protein percentages through 150 DIM. In the first test month, protein percentages were higher for all parities in L compared with those in S. Within the L treatment, cows in parity 3+ had lower protein content compared with parity 1 and 2 cows through 5 mo of lactation with the exception of month 4. Although protein percentage was lower for all cows in month 4, the decline was less severe for cows of parity 3+. There was a significant interaction of treatment by time on milk protein yield through 150 DIM ($P < 0.01$; Figure 8). The pattern of milk protein yield over time mirrored that of milk protein percentage. Cows in L had appreciably higher protein yields during the first month and lower yields during mo 4 than cows in S. Interestingly, cows in L had higher protein content during the first month of lactation compared with cows in S. In addition, the tendency towards higher insulin and lower NEFA in early lactation for cows in L indicates improved energy status. Improved energy status may reduce the amount of amino acids used for gluconeogenesis thereby allowing more substrate to be available for milk protein synthesis in the first month postpartum (McGuire et al., 1995). Furthermore, consuming a higher energy diet for a longer time may have better prepared the rumen environment for the early lactation ration and thus, maximized microbial protein yield.

There were no significant effects of treatment on ECM yield through 60 or 150 DIM (Tables 15 and 16). Treatment had no effect on SCC through 60 DIM, but cows in S had lower SCC (49,258 vs. 80,505 cells/ml; $P = 0.04$) compared with cows in L through 150 DIM (Table 16). Figure 9 illustrates the treatment by farm interaction ($P < 0.01$) for SCC analyzed through 150 DIM. In Farm 1, cows in S had greatly reduced SCC compared with cows in L (29,790 vs. 96,586 cells/ml). In Farm 2, cows in S had

slightly higher SCC than cows in L (81,479 vs. 67,119 cells/ml). Although the number of mastitis incidences were low and not significantly different, 5 cases of mastitis were reported for cows in L in Farm 1 compared with only 2 cases for cows in S in Farm 1.

Reproduction

Several cows were sold or died ($n = 26$), selected not to rebreed ($n = 10$) or were exposed to a bull ($n = 21$; Farm 1 only) and therefore were removed from the analysis of reproductive measurements. There were 99 cows remaining for analysis plus and additional 33 which were not pregnant by 200 DIM.

All reproductive measurements are shown in Table 17. Cows in S had fewer days to first service than cows in L (73 vs. 66; $P = 0.04$). However, the trend towards an interaction of farm by treatment indicated that the effect of treatment was only in Farm 1 ($P = 0.06$; Figure 10). In Farm 1, cows in L had longer days to first service than cows in S (74 vs. 61 d). No differences due to treatment were detected in Farm 2 (71 vs. 71 d). It is not surprising that no changes in days to first service were found in Farm 2 because all cows were synchronized and bred using the Ovsynch[®] protocol. Again, DMI could be postulated to affect days to first service in Farm 1. Cows in more negative energy status or cows in negative energy status for a longer time have delayed return to ovarian activity and estrous cycles (Butler and Smith, 1989; Staples et al., 1990). The shorter days to first service for cows in S in Farm 1 would agree with the theory that reduced BCS losses from 3 to 6 wk postpartum and increased milk yields are a result of improved DMI. There were no significant effects of treatment on days open, pregnancy rate, or percentage of cows pregnant by 200 DIM.

CONCLUSIONS

Extending the time spent in the late dry group slightly improved BCS of late gestation dairy cows, but did not promote the large changes observed in previous research. Cows may have gained BCS more readily if BCS at dry-off were lower than the current study. In agreement with the hypothesis, cows in L apparently had improved energy status postpartum as indicated by a tendency towards lower plasma NEFA and higher insulin concentrations and a less severe BCS loss from parturition to 3 wk postpartum. However, effects on production, health, and reproduction were dependent upon farm. Cows in Farm 1 had lower milk yields, poorer health and reproduction, but treatment had no effect on the same variables measured in Farm 2. Differences in management between farms, or nutrition at other stages of the production cycle need to be considered when evaluating effects of diets fed in the late dry period.

TABLES AND FIGURES

Table 1. Days spent in the late dry group and allocation of cows to treatments.

	S ¹	L ¹
Days		
Mean	17.5	36.6
SD	4.1	6.9
Minimum	6	27
Maximum	26	60
n		
Farm 1	23	54
Parity 1	9	9
Parity 2	6	19
Parity 3+	8	26
Farm 2	70	42
Parity 1	15	10
Parity 2	26	12
Parity 3+	29	20
Total	93	96

¹Treatments: S = short late dry period; L = long late dry period.

Table 2. Formulated ingredient and analyzed chemical composition, and particle size distribution of diets fed in the late dry period.

Item	Farm 1	Farm 2
Ingredient, % of DM		
Corn silage	22.1	32.5
Alfalfa silage	8.5	-
Alfalfa-grass mixed hay	14.9	9.3
Beet pulp, dehydrated	-	13.4
Corn, high moisture	21.3	-
Corn, dry ground	-	18.6
Corn distillers grains	2.2	-
Cottonseed, whole	6.2	-
Soybean meal	-	9.8
Mineral mix	24.8 ¹	16.4 ²
Chemical composition, %		
DM	50.6	55.2
CP	17.4	16.7
ADF	18.8	20.4
NDF	29.3	32.0
EE	4.8	2.6
Ash	7.9	8.6
CP equivalent from ammonia	1.7	1.9
NFC ³	42.4	42.0
Ca	1.13	1.60
P	0.47	0.37
Mg	0.36	0.45
K	1.17	1.14
Na	0.13	0.21
Particle size ⁴ (%)		
> 19.0 mm	8.0±6.6	3.1±1.7
8.0 to 19.0 mm	47.1±8.1	36.8±4.0
< 8.0 mm	44.9±12.5	60.8±4.3

¹Contained 27.0% CP, 3.7% lipid, 2.47% Ca, 0.51% P, 0.79% Mg, 0.75% K, 0.26% Na, 1.8% Cl, 1.07% S, 1 ppm Co, 144 ppm Cu, 449 ppm Fe, 5 ppm I, 360 ppm Mn, 3 ppm Se, 360 ppm Zn, 38 KIU/kg of Vitamin A, 7 KIU/kg of Vitamin D, and 308 IU/kg of Vitamin E, dry basis.

²Contained 13.7% CP, 2.6% lipid, 3.31% Ca, 0.51% P, 0.40% Mg, 0.69% K, 0.51% Na, .31% Cl, 2.4% S, 1 ppm Co, 40 ppm Cu, 201 ppm Fe, 2 ppm I, 161 ppm Mn, 2 ppm Se, 161 ppm Zn, 10 KIU/kg of Vitamin A, 2 KIU/kg of Vitamin D, and 110 IU/kg of Vitamin E, dry basis.

³NFC = 100 - % NDF - % CP + % CP equivalent from ammonia - % EE - % ash.

⁴Particle size determined by the Penn State Particle Separator (Lammers et al., 1996), as-fed basis; mean ± SD.

Table 3. Formulated ingredient and analyzed chemical composition, and particle size distribution of diets fed postpartum in Farm 1.

Item	Primiparous	Multiparous
Ingredient, % of DM		
Corn silage	22.0	23.7
Alfalfa silage	5.9	4.8
Corn, high moisture	29.0	28.9
Corn distillers grains, dry	10.9	10.6
Custom mix ¹	10.6	10.3
Alfalfa hay	6.6	5.4
Soybean meal	5.5	6.3
Cottonseed, whole	7.8	7.8
Chemical composition, %		
DM	54.3	54.4
CP	18.8	18.8
ADF	18.8	18.4
NDF	30.8	29.2
EE	6.0	5.5
Ash	7.7	7.4
CP equivalent from ammonia	0.87	0.88
NFC ²	37.6	40.0
Ca	1.11	1.10
P	0.56	0.58
Mg	0.30	0.31
K	1.33	1.30
Na	0.42	0.40
Particle size³ (%)		
> 19.0 mm	8.3±4.2	7.1±2.5
8.0 to 19.0 mm	42.3±3.1	44.0±2.9
< 8.0 mm	49.3±3.8	48.9±3.3

¹Contained 36.2% CP, 10.2% lipid, 5.60% Ca, 1.48% P, 0.89% Mg, 1.21% K, 3.06% Na, 1.55% Cl, 0.60% S, 1.76 ppm Co, 97 ppm Cu, 809 ppm Fe, 5.6 ppm I, 398 ppm Mn, 3.5 ppm Se, 444 ppm Zn, 25 KIU/kg of Vitamin A, 5 KIU/kg of Vitamin D, and 161 IU/kg of Vitamin E, dry basis.

²NFC = 100 - % NDF - % CP + % CP equivalent from ammonia - % EE - % ash.

³Particle size determined by the Penn State Particle Separator (Lammers et al., 1996), as-fed basis; means ± SD.

Table 4. Formulated ingredient and analyzed nutrient composition, and particle size of diets fed postpartum in Farm 2.

Item	Early lactation ¹	Primiparous	Multiparous
Ingredient, % of DM			
Corn silage	23.9	27.6	29.4
Alfalfa silage	8.9	9.6	9.6
Alfalfa-grass mixed hay	8.8	-	-
Corn, high moisture	18.3	20.0	17.9
Corn, dry ground	8.8	10.9	9.2
Corn distillers grains	5.9	11.3	11.0
Soybean meal	12.7	9.6	10.6
Custom mix ²	5.6	5.7	5.5
Beet pulp, dehydrated	4.8	3.5	5.6
Wheat straw	2.3	1.9	1.6
Chemical composition, %			
DM	55.7	49.2	50.9
CP	18.7	18.0	18.2
ADF	19.7	18.4	19.1
NDF	30.4	29.3	28.9
EE	3.4	3.9	4.0
Ash	8.1	8.5	8.0
CP equivalent from ammonia	0.93	1.05	1.06
NFC ³	40.3	41.4	42.0
Ca	1.15	1.22	1.13
P	0.56	0.61	0.59
Mg	0.37	0.38	0.37
K	1.46	1.43	1.40
Na	0.58	0.66	0.56
Particle size ⁴ (%)			
> 19.0 mm	7.0±4.3	3.0±1.8	3.1±1.9
8.0 to 19.0 mm	34.4±4.7	39.7±4.0	41.6±4.0
< 8.0 mm	58.6±3.3	57.3±4.2	55.4±4.0

¹Cows of all parities were fed this diet for the first 17 DIM approximately.

²Contained 23.3% CP, 0.5% fat, 12.4% Ca, 2.9% P, 2.8% Mg, 0.4% K, 8.9% Na, 11.0% Cl, 1.7% S, 3 ppm of Co, 254 ppm of Cu, 1270 ppm of Fe, 13 ppm of I, 1016 ppm of Mn, 6 ppm Se, 152 KIU/kg of Vitamin A, 25 KIU/kg of Vitamin D, and 484 IU/kg of Vitamin E.

³NFC = 100 - % NDF - % CP + % CP equivalent from ammonia - % EE - % ash.

⁴Particle size determined by the Penn State Particle Separator (Lammers et al., 1996), as-fed basis; means±SD.

Table 5. Formulated ingredient composition of diets fed in the early dry period.

Item	Farm 1	Farm 2
Ingredient, % of DM		
Corn silage	72.0	35.0
Alfalfa silage	18.5	41.9
Beet pulp, dehydrated	-	20.7
Soybean meal	7.3	-
Mineral mix	2.2 ¹	2.4 ²
Chemical composition, %		
DM	34.4	40.9
CP	15.0	15.6
ADF	25.1	28.7
NDF	42.7	43.2
EE	2.9	2.8
Ash	6.9	6.8
NFC ³	32.4	31.7
Ca	0.65	1.03
P	0.36	0.32
Mg	0.29	0.34
K	1.30	1.52
Na	0.05	0.11

¹Contained 2.5 KIU/kg of Vitamin E and 2 ppm of Se.

²Contained 15.0% CP, 2.0% EE, 2.0% Ca, 0.2% P, 1.4 ppm of Se, 14 KIU/kg of Vitamin A, 3.1 KIU/kg of Vitamin D, and 195 KIU/kg of Vitamin E.

³NFC = 100 - % NDF - % CP - % EE - % ash.

Table 6. Least squares means and statistical significance of body condition scores during the periparturient period.

Variable	S ¹	SEM	L ¹	SEM
Farm 1	3.11	0.04	3.27	0.05
Farm 2	3.27	0.03	3.50	0.05
Parity 1	3.29	0.04	3.58	0.06
Parity 2	3.11	0.04	3.31	0.05
Parity 3+	3.17	0.04	3.27	0.05
Time, d relative to parturition				
-42	-	-	3.52	0.11
-35	-	-	3.52	0.07
-28	-	-	3.65	0.05
-21	3.49	0.12	3.65	0.05
-14	3.52	0.05	3.69	0.05
-7	3.60	0.05	3.68	0.05
2	3.30	0.05	3.41	0.05
21	2.50	0.05	2.69	0.05
42	2.36	0.05	2.43	0.05
P-value				
Parity	< 0.01	-	< 0.01	-
Farm	< 0.01	-	< 0.01	-
Time	< 0.01	-	< 0.01	-

¹Treatments: S = short late dry period; L = long late dry period.

Table 7. Least squares means and statistical significance of body condition score changes during the periparturient period.

Variable	Prepartum	SEM	Weeks postpartum					
			0 to 6	SEM	0 to 3	SEM	3 to 6	SEM
S ¹	0.08	0.03	-1.30	0.06	-1.15	0.06	-0.13	0.06
L ¹	0.14	0.03	-1.21	0.06	-0.95	0.05	-0.27	0.05
Parity 1	0.11	0.04	-1.08	0.08	-0.99	0.07	-0.07	0.07
Parity 2	0.20	0.03	-1.21	0.07	-0.96	0.06	-0.23	0.06
Parity 3+	0.01	0.03	-1.46	0.06	-1.21	0.06	-0.31	0.06
Farm 1	0.16	0.03	-1.35	0.07	-1.14	0.06	-0.19	0.06
Farm 2	0.05	0.03	-1.15	0.05	-0.96	0.05	-0.21	0.06
S*farm 1							-0.08	0.10
L*farm 1							-0.33	0.07
S*farm 2							-0.18	0.05
L*farm 2							-0.21	0.07
P-value ²								
Treatment (Trt)	0.14		0.30		< 0.01		0.06	
Parity	< 0.01		< 0.01		< 0.01		0.03	
Farm	< 0.01		0.02		0.03		0.83	
Trt*farm	RM		RM		RM		0.13	
Parity*farm	0.08		RM ³		RM		0.14	

¹Treatments: S = short late dry period; L = long late dry period.

²All 2- and 3-way interaction terms not listed were removed from the statistical model because $P > 0.15$.

³Independent variables were removed (RM) from the statistical model if $P > 0.15$.

Table 8. Least squares means of plasma NEFA concentrations for prepartum, postpartum, and periparturient periods.

Variable	NEFA (μ Eq/L)			95% confidence intervals (lower limit/upper limit)		
	Pre-partum	Post-partum	Peripart-urient ¹	Pre-partum	Post-partum	Peripart-urient
S ²	171.7	542.4	270.2	150.2/196.4	484.7/607.0	240.8/303.1
L ²	178.9	487.1	260.7	157.9/202.7	444.4/533.9	236.6/287.2
Parity 1	211.7	480.5	298.8	176.8/253.6	421.0/548.5	260.1/343.2
Parity 2	120.4	469.8	204.0	103.9/139.5	418.4/527.6	181.1/229.8
Parity 3+	211.3	601.5	306.6	185.5/240.6	539.3/670.8	276.3/340.3
Farm 1	208.7	543.7	305.3	180.6/241.1	483.2/611.8	270.8/344.3
Farm 2	147.2	485.9	230.7	131.0/165.4	447.0/528.3	210.8/252.4
Time, d relative to parturition						
-14	129.6		135.0	107.8/155.7		109.6/166.4
-11	133.5		142.3	115.5/14.3		123.1/164.5
-8	151.5		149.2	133.6/171.7		132.0/168.8
-5	199.8		198.8	177.8/224.6		177.3/222.9
-2	316.0		313.6	282.5/353.5		282.0/348.8
2		628.1	629.4		573.5/687.9	569.7/695.3
7		550.8	554.0		501.1/605.3	499.7/614.2
14		392.6	394.6		356.7/431.9	355.6/438.0
P-value ³						
Trt	0.66	0.15	0.65			
Parity	< 0.01	< 0.01	< 0.01			
Farm	< 0.01	0.13	< 0.01			
Time	< 0.01	< 0.01	< 0.01			
Trt*parity	RM ⁴	0.32	0.28			
Trt*farm	RM	0.86	0.86			
Trt*time	RM	0.37	0.16			
Parity*farm	0.06	< 0.01	< 0.01			
Parity*time	RM	0.08	< 0.01			
Farm*time	RM	0.14	0.03			
Parity*farm *time	RM	0.07	0.15			

¹Periparturient period = combined pre- and postpartum periods.

²Treatments: S = short late dry period; L = long late dry period.

³All 2- and 3-way interaction terms not listed were removed from the statistical model ($P > 0.15$).

⁴Independent variables were removed (RM) from the statistical model if $P > 0.15$.

Table 9. Least squares means and statistical significance of plasma beta-hydroxybutyric acid (BHBA) concentrations for prepartum, postpartum, and periparturient periods.

Variable	BHBA (mg/dl)			95% confidence intervals (lower limit/upper limit)		
	Pre-Partu	Post-partum	Peripart-urient ¹	Pre-partum	Post-partum	Peripart-urient
S ²	4.15	7.42	5.17	3.85/4.48	6.66/8.27	4.80/5.57
L ²	4.27	7.41	5.20	3.98/4.59	6.72/8.17	4.86/5.57
Parity 1	4.12	7.07	4.95	3.70/4.59	6.12/8.16	4.47/5.50
Parity 2	4.04	6.69	4.94	3.70/4.41	5.93/7.54	4.54/5.38
Parity 3	4.48	8.63	5.70	4.16/4.83	7.73/9.63	5.30/6.14
Farm 1	3.92	6.92	4.83	3.61/4.26	6.15/7.79	4.44/5.24
Farm 2	4.52	7.94	5.58	4.22/4.84	7.27/8.69	5.22/5.96
Time, d relative to						
-14	3.93		3.83	3.52/4.39		3.31/4.42
-11	3.82		3.86	3.50/4.17		3.45/4.32
-8	3.94		3.94	3.66/4.24		3.58/4.34
-5	4.29		4.26	4.00/4.60		3.90/4.65
-2	5.22		5.23	4.89/5.56		4.82/5.68
2		6.36	6.39	5.81/6.96	5.81/6.96	5.92/6.89
7		9.31	9.24	8.49/10.22	8.49/10.22	8.54/10.0
14		6.86	6.86	6.27/7.56	6.27/7.56	6.33/7.42
P-value ³						
Trt	0.58	0.98	0.91			
Parity	0.16	0.01	0.02			
Farm	< 0.01	0.07	< 0.01			
Time	< 0.01	< 0.01	< 0.01			
Parity*farm	0.02	0.09	0.05			
Parity*time	0.11	0.05	0.02			
Farm*time	< 0.01	0.15	0.05			

¹Periparturient period = combined pre- and postpartum periods.

²Treatments: S = short late dry period; L = long late dry period.

³All 2- and 3-way interaction terms not listed were removed ($P > 0.15$).

Table 10. Least squares means and statistical significance of plasma insulin concentrations for prepartum, postpartum and periparturient periods.

Variable	Insulin (μ IU/ml)			95% confidence intervals (lower limit/upper limit)		
	Pre-partum	Post-partum	Peripart-urient ¹	Pre-partum	Post-partum	Peripart-urient
S ²	11.66	6.46	9.21	10.0/13.5	5.5/7.2	8.3/10.3
L ²	10.48	7.19	8.96	9.2/11.8	6.6/7.8	8.2/9.8
Parity 1	12.05	9.29	11.05	10.1/14.4	8.2/10.5	9.7/12.6
Parity 2	12.42	6.56	9.53	10.5/14.7	5.9/7.3	8.5/10.7
Parity 3+	9.02	5.19	7.12	7.8/10.4	4.7/5.7	6.5/7.8
Farm 1	9.83	6.68	8.25	8.4/11.2	6.0/7.5	7.4/9.2
Farm 2	12.43	6.95	10.01	11.1/14.0	6.4/7.5	9.2/10.9
Time, d relative to parturition						
-14	13.21		13.28	11.2/15.6		11.4/15.5
-11	12.50		12.45	11.0/14.3		11.0/14.0
-8	11.18		10.68	10.0/12.6		9.6/11.9
-5	10.37		10.16	9.3/11.6		9.2/11.2
-2	8.60		8.44	7.7/9.6		7.7/9.3
2		6.72	6.71		6.2/7.3	6.2/7.3
7		6.64	6.55		6.1/7.2	6.0/7.2
14		7.08	6.98		6.5/7.7	6.4/7.7
P-value ³						
Trt	0.28	0.12	0.70			
Parity	< 0.01	< 0.01	< 0.01			
Farm	0.01	0.57	< 0.01			
Time	< 0.01	0.29	< 0.01			
Trt*parity	0.40	0.87	RM ⁴			
Trt*farm	0.24	0.24	0.13			
Trt*time	0.87	0.59	0.03 ⁵			
Parity*farm	0.02	0.15	< 0.01			
Parity*time	0.01	0.20	< 0.01			
Farm*time	0.85	0.71	0.03			
Trt*parity*farm	0.13 ⁶	RM	RM			
Trt*parity*time	RM	0.07 ⁷	RM			

¹Periparturient period = combined pre- and postpartum periods.

²Treatments: S = short late dry period; L = long late dry period.

³All 2-, 3-way interaction terms not listed were removed from the statistical model ($P > 0.15$).

⁴Independent variables were removed (RM) from the statistical model if $P > 0.15$.

⁵See Figure 1.

⁶See Figure 2.

⁷See Figure 3.

Table 11. Incidence rates and statistical significance of health disorders, and abnormal rectal temperatures.

Variable	N	Incidence rates (%)					
		Displaced abomasum	Ketosis	Mastitis	Metritis ¹	Retained placenta	Rectal temperature ^{2,3}
S ⁴	93	4.3	4.3	2.2	2.2	12.9	30.0
L ⁴	96	8.3	2.1	7.3	10.4	6.3	42.9
Parity 1	43	9.3	9.3	4.7	14.0	16.3	52.0
Parity 2	63	4.8	1.6	1.6	6.3	4.8	31.6
Parity 3+	83	6.0	1.2	7.2	2.4	9.6	28.6
Farm 1	77	9.1	1.3	9.1	15.6	5.2	-
Farm 2	112	4.5	4.5	1.8	-	12.5	25.7
P-value ⁵							
Trt	-	0.38	0.64	0.31	0.06	0.27	0.17
Parity	-	0.44	0.06	0.68	< 0.01	0.30	0.05
Farm	-	0.36	0.27	0.08	-	0.17	-

¹Includes Farm 1 only. No incidences of metritis were reported on Farm 2.

²Includes Farm 2 only. Farm 1 did not record daily rectal temperatures.

³Rectal temperature > 39.4°C for at least 1 d during the first 12 d of lactation.

⁴Treatments: S = short late dry period; L = long late dry period.

⁵All 2- and 3-way interaction terms not listed were removed from the statistical model because $P > 0.15$.

Table 12. Least squares means and statistical significance of milk production and composition through 60 DIM.

Variable	Milk			Fat			Protein		
	yield (kg/d)	SEM	Fat (%)	SEM	yield (kg)	Fat (%)	SEM	yield (kg)	SEM
S ¹	37.4	1.5	4.29	0.15	1.63	3.08	0.10	1.13	0.04
L ¹	37.3	1.4	4.19	0.13	1.60	3.24	0.09	1.18	0.04
Parity 1	24.6	2.9	4.06	0.19	1.06	3.30	0.13	0.80	0.09
Parity 2	44.9	1.5	4.34	0.14	1.86	3.11	0.10	1.37	0.05
Parity 3+	42.6	1.6	4.32	0.14	1.93	3.06	0.10	1.30	0.05
Farm 1	36.5	2.3	3.53	0.23	1.29	3.00	0.06	1.03	0.07
Farm 2	38.2	0.8	4.95	0.08	1.94	3.31	0.15	1.28	0.02
Time, mo									
postpartum									
1	35.3	1.3	4.56	0.14	1.67	3.22	0.09	1.14	0.04
2	39.5	1.2	3.92	0.13	1.56	3.09	0.08	1.17	0.04
P-value ²									
Treatment (Trt)	0.96		0.47		0.84	<0.01		0.33	
Parity	<0.01		0.30		<0.01	0.05		<0.01	
Farm	0.49		<0.01		<0.01	<0.01		<0.01	
Time	<0.01		<0.01		0.07	<0.01		0.16	
Trt*parity	RM ³		RM		RM	0.07 ⁴		RM	
Trt*time	RM		RM		RM	<0.01 ⁵		RM	
Parity*farm	0.06		RM		RM	0.08		0.07	
Parity*time	0.13		RM		0.01	RM		RM	

¹Treatments: S = short late dry period; L = long late dry period.

²All 2- and 3-way interaction terms not listed were removed from the statistical model because $P > 0.15$.

³Independent variables were removed (RM) from the statistical model if $P > 0.15$.

⁴See Figure 5.

⁵See Figure 6.

Table 13. Least squares means and statistical significance of milk yield and composition through 150 DIM.

Variable	Milk yield		Fat		Fat yield		Protein		Protein yield	
	(kg/d)	SEM	(%)	SEM	(kg)	SEM	(%)	SEM	(kg)	SEM
S ¹	41.4	0.9	4.02	0.08	1.62	0.05	3.05	0.03	1.23	0.03
L ¹	39.2	0.8	3.87	0.08	1.52	0.05	3.08	0.03	1.20	0.03
Parity 1	33.2	1.0	3.73	0.10	1.15	0.07	3.08	0.04	0.98	0.03
Parity 2	42.8	0.9	4.05	0.08	1.72	0.06	3.10	0.03	1.32	0.03
Parity 3+	44.8	0.9	4.05	0.08	1.83	0.05	3.01	0.03	1.35	0.03
Farm 1	41.0	1.0	3.26	0.11	1.30	0.07	2.95	0.04	1.18	0.04
Farm 2	39.5	0.6	4.62	0.06	1.84	0.04	3.18	0.02	1.25	0.02
Time, mo										
postpartum										
1	36.5	1.2	4.57	0.09	1.71	0.06	3.18	0.05	1.14	0.04
2	41.9	0.9	3.94	0.09	1.61	0.06	3.04	0.04	1.22	0.04
3	41.2	0.7	3.74	0.08	1.54	0.05	3.12	0.03	1.23	0.02
4	41.3	0.7	3.82	0.09	1.57	0.06	2.93	0.06	1.19	0.05
5	40.3	0.7	3.64	0.09	1.42	0.05	3.06	0.04	1.24	0.03
P-value										
Treatment (Trt)	0.06		0.15		0.11		0.40		0.33	
Parity	< 0.01		0.02		< 0.01		0.09		< 0.01	
Farm	0.24		< 0.01		< 0.01		< 0.01		0.05	
Time	< 0.01		< 0.01		< 0.01		< 0.01		0.02	
Trt*parity	RM ²		RM		RM		0.50		RM	
Trt*farm	0.06 ³		RM		RM		0.26		RM	
Trt*time	RM		RM		RM		< 0.01 ⁴		< 0.01 ⁴	
Parity*farm	RM		RM		RM		0.26		RM	
Parity*time	RM		RM		0.01		0.49		< 0.01	
Farm*time	0.12		RM		RM		< 0.01		< 0.01	
Parity*trt*time	RM		RM		RM		0.03 ⁵		RM	

¹Treatments: S = short late dry period; L = long late dry period. ²Independent variables were removed (RM) from the statistical model if $P > 0.15$. ³See Figure 4. ⁴See Figure 8. ⁵See Figure 7.

Table 14. Least squares means and statistical significance of daily milk yields in Farm 1.

Variable	Milk yield (kg/d)	SEM
0 to 56 DIM		
S ¹	38.4	2.2
L ¹	34.2	1.5
Parity 1	25.9	2.4
Parity 2	42.7	2.1
Parity 3+	40.4	2.1
Time, mo postpartum		
1	26.0	1.5
2	30.2	1.4
3	34.3	1.5
4	37.0	1.5
5	39.4	1.5
6	40.2	1.5
7	41.5	1.5
8	41.9	1.5
P-value ²		
Treatment (Trt)	0.12	
Parity	< 0.01	
Time	< 0.01	
0 to 150 DIM		
S	42.0	2.0
L	36.6	1.3
Parity 1	32.2	2.1
Parity 2	43.2	1.8
Parity 3+	42.4	1.9
Time ³		
P-value		
Trt	0.03	
Parity	< 0.01	
Time	< 0.01	
Parity*time	< 0.01	

¹Treatments: S = short late dry period, L = long late dry period

²All 2- and 3-way interaction terms not listed were removed from the statistical model because $P > 0.15$.

³Time not shown.

Table 15. Least squares means and statistical significance of energy-corrected milk (ECM) yield and SCC through 60 DIM.

Variable	ECM yield (kg/d)	SEM	SCC (cells/ml)	95% confidence intervals	
				Lower	Upper
S ¹	41.3	2.0	94,051	59,213	149,388
L ¹	41.5	1.7	70,597	46,831	106,425
Parity 1	28.7	2.5	126,355	71,396	223,619
Parity 2	47.0	1.9	56,846	36,450	88,654
Parity 3+	48.4	1.9	75,325	48,098	117,960
Farm 1	36.3	2.9	107,621	52,923	218,841
Farm 2	46.5	1.1	61,696	48,441	75,582
Time, mo postpartum					
1	41.4	1.6	90,744	60,018	137,201
2	41.4	1.6	73,171	48,977	109,316
P-value ²					
Treatment	0.92		0.22		
Parity	< 0.01		0.03		
Farm	< 0.01		0.15		
Time	0.96		0.20		

¹Treatments: S = short late dry period; L = long late dry period.

²All 2- and 3-way interaction terms not listed were removed from the statistical model because $P > 0.15$.

Table 16. Least squares means and statistical significance of energy-corrected milk (ECM) yield and SCC through 150 DIM.

Variable	ECM Yield (kg)	SEM	SCC (cells/ml)	95% confidence intervals	
				Lower	Upper
S ¹	42.9	1.0	49,258	34,068	71,218
L ¹	41.3	0.9	80,515	60,895	106,468
Parity 1	32.7	1.4	70,885	48,137	104,391
Parity 2	45.7	1.1	48,204	34,839	66,696
Parity 3+	47.8	1.1	73,096	53,407	100,047
Farm 1	38.8	1.4	53,630	35,355	81,349
Farm 2	45.3	0.8	73,951	60,162	90,899
Time, mo postpartum					
1	42.9	1.1	59,418	43,019	82,068
2	42.8	1.0	47,452	34,403	65,447
3	42.4	0.9	52,505	39,755	69,342
4	42.6	1.0	87,444	63,557	120,307
5	39.5	1.0	76,520	55,938	104,673
P-value ²					
Treatment (Trt)	0.22		0.04		
Parity	< 0.01		0.09		
Farm	< 0.01		0.17		
Time	< 0.01		< 0.01		
Trt*farm	RM ³		< 0.01 ⁴		
Parity*time	<0.02		0.02		

¹Treatments: S = short late dry period; L = long late dry period.

²All 2- and 3-way interaction terms not listed were removed from the statistical model because $P > 0.15$.

³Independent variables were removed (RM) from the statistical model if $P > 0.15$.

⁴See Figure 9.

Table 17. Least squares means and significance of reproductive measurements.

Variable	Days to first service	SEM	Days open	SEM	Pregnancy rate, %	SEM	Pregnant by 200 d, %
S ¹	66	2.5	113	7.2	60	5.4	74
L ¹	73	2.1	105	6.9	64	5.2	76
Parity 1	66	2.6	98	9.0	65	6.7	77
Parity 2	70	2.6	113	5.5	59	6.4	67
Parity 3+	72	2.3	115	7.7	63	5.8	80
Farm 1	68	2.8	103	9.4	64	7.1	77
Farm 2	71	1.5	114	5.3	60	4.0	70
P-values ²							
Treatment (Trt)	0.04		0.40		0.57		0.73
Parity	0.16		0.28		0.77		0.61
Farm	0.29		0.31		0.63		0.44
Trt*farm	0.06 ³		RM ⁴		RM		RM

¹Treatments: S = short late dry period; L = long late dry period.

²All 2- and 3-way interaction terms not listed were removed from the statistical model because $P > 0.15$.

³See Figure 10.

⁴Independent variables were removed (RM) from the statistical model if $P > 0.15$.

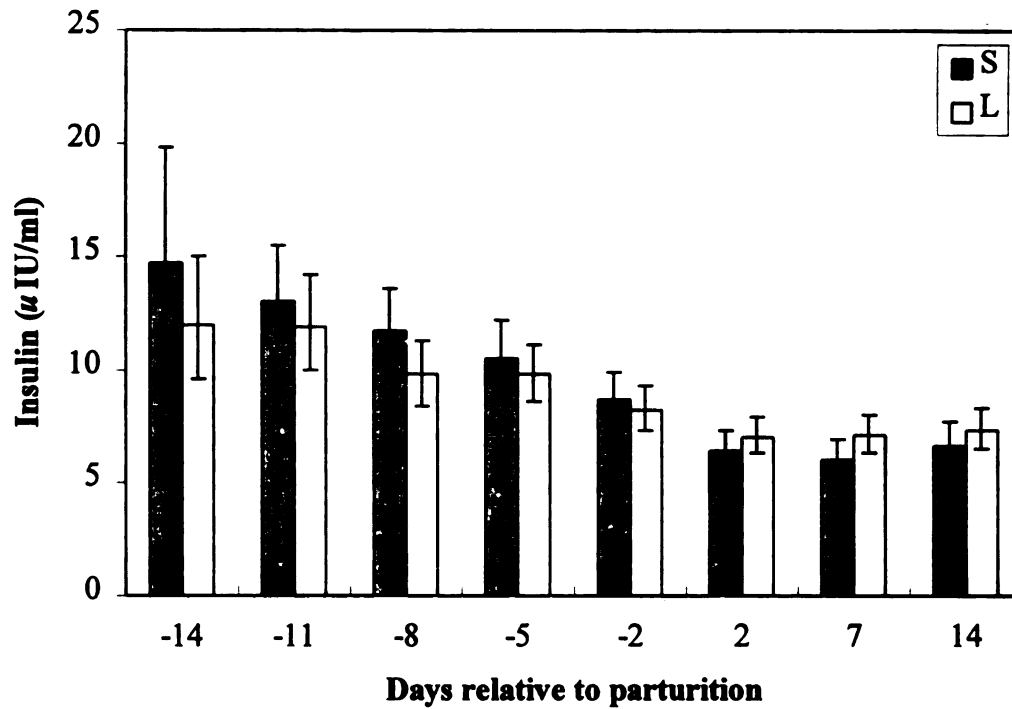


Figure 1. Least squares means and 95% confidence intervals for the interaction of treatment by time ($P = 0.03$) for plasma insulin in the periparturient period. Other significant effects in the model for the periparturient period included: parity ($P < 0.01$), farm ($P < 0.01$), time ($P < 0.01$), treatment by farm ($P = 0.13$), parity by farm ($P < 0.01$), parity by time ($P < 0.01$), farm by time ($P = 0.03$). Treatments: S = short late dry period; L = long late dry period.

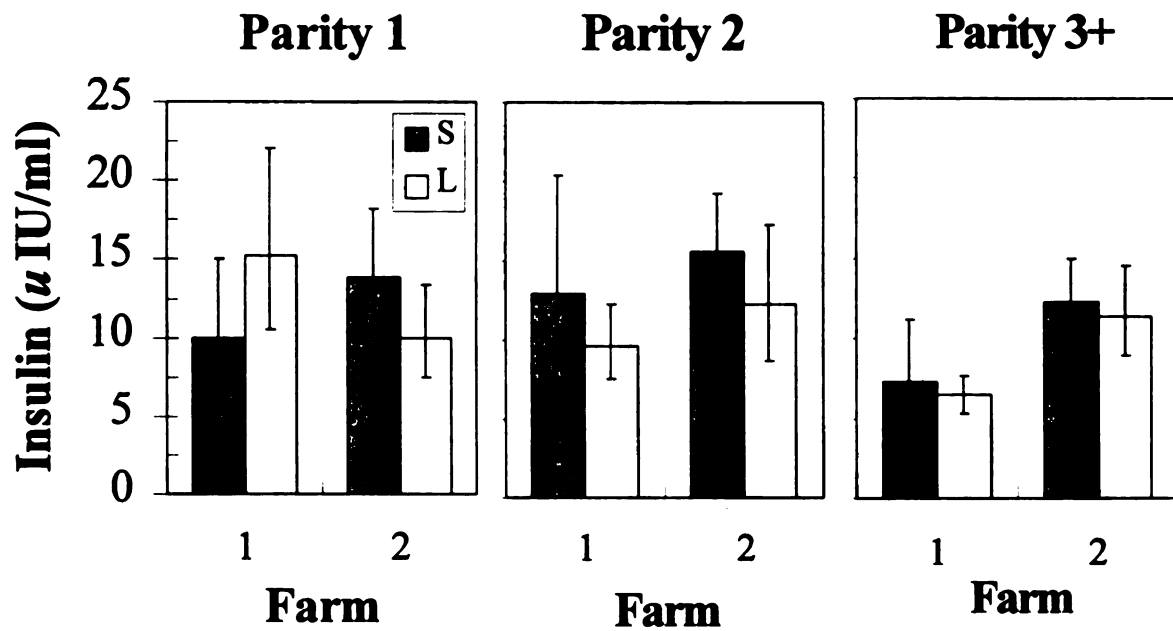


Figure 2. Least squares means and 95% confidence intervals for the interaction of treatment by parity by farm ($P = 0.13$) on plasma insulin concentrations prepartum. Other significant effects in the model included: parity ($P < 0.01$), farm ($P < 0.01$), time ($P < 0.01$), parity by farm ($P = 0.02$), and parity by time ($P = 0.01$). Treatments: S = short late dry period; L = long late dry period.

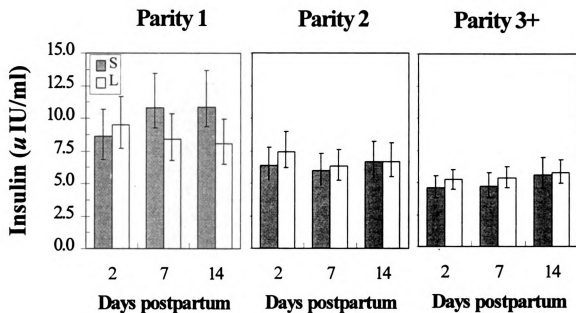


Figure 3. Least squares means and 95% confidence intervals for the interaction of treatment by parity by time ($P = 0.17$) on plasma insulin concentrations postpartum. Other significant effects in the model included: treatment ($P = 0.12$), parity ($P < 0.01$), and parity by farm ($P = 0.15$). Treatments: S = short late dry period; L = long late dry period.

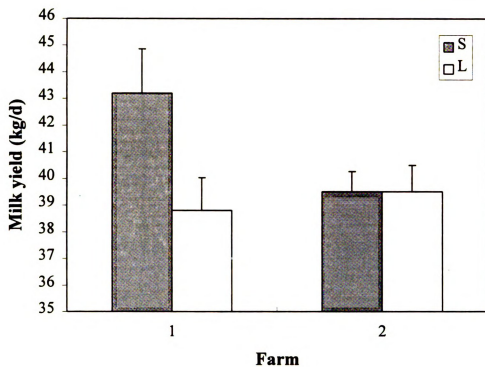


Figure 4. Least squares means and SEM for the treatment by farm interaction ($P = 0.06$) for milk yield through 150 DIM. Other significant effects in the model include: treatment ($P = 0.06$), parity ($P < 0.01$), time ($P < 0.01$), and farm by time ($P = 0.12$). Treatments: S = short late dry period; L = long late dry period.

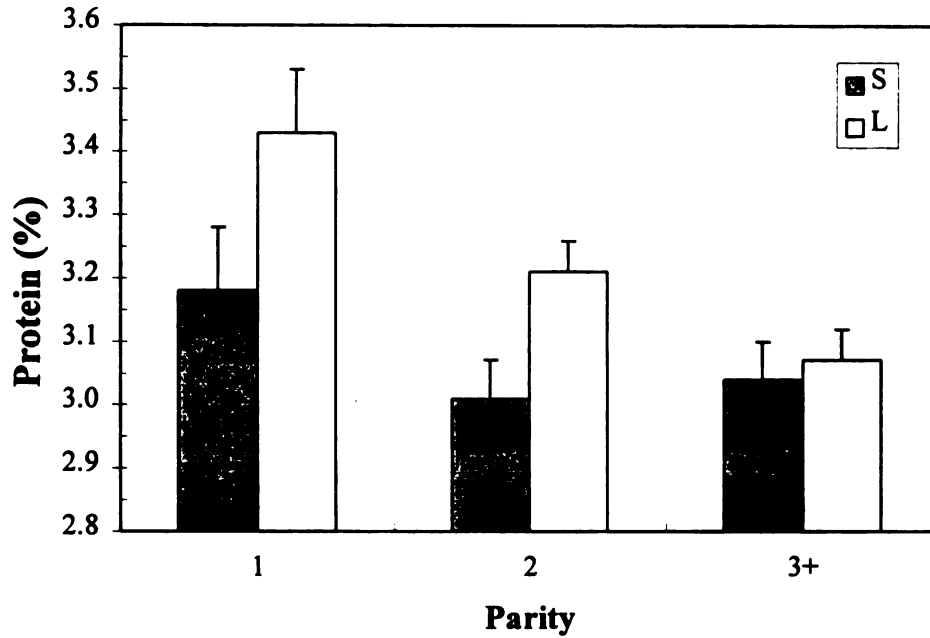


Figure 5. Least squares means and SEM for the treatment by parity interaction ($P = 0.07$) of milk protein content through 60 DIM. Other significant effects in the model include: treatment ($P < 0.01$), farm ($P < 0.01$), parity ($P < 0.05$), time ($P < 0.01$), treatment by time ($P < 0.01$), and parity by farm ($P = 0.08$). Treatments: S = short late dry period; L = long late dry period.

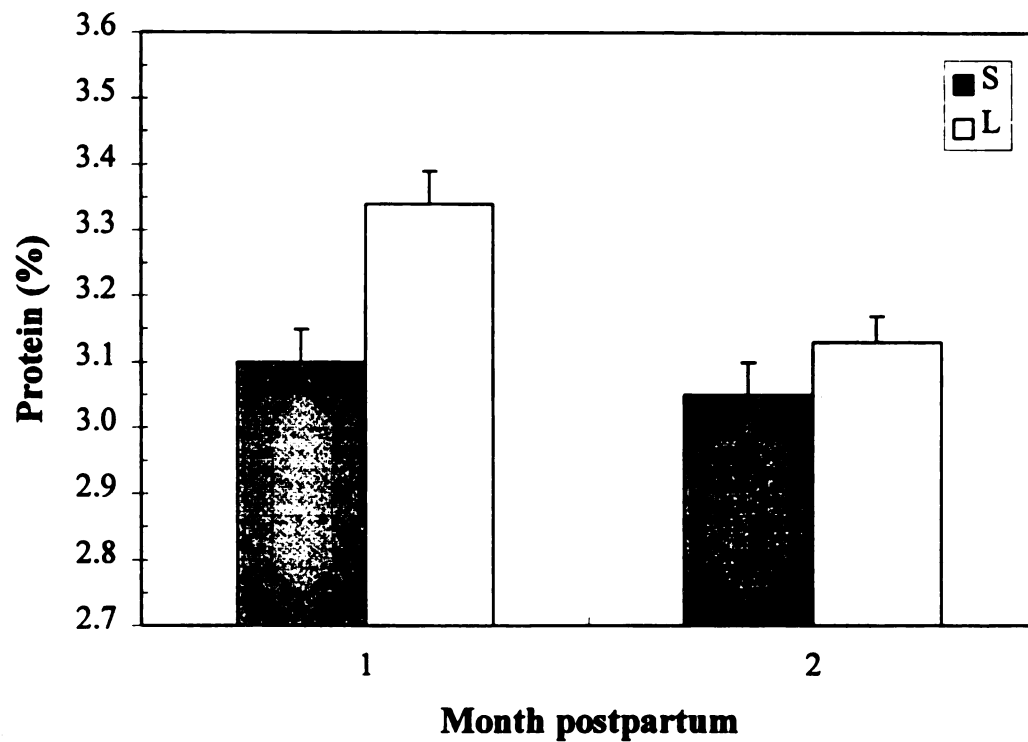


Figure 6. Least squares means and SEM for the treatment by time interaction ($P < 0.01$) of milk protein content through 60 DIM. Other significant effects in the model include: treatment ($P < 0.01$), farm ($P < 0.01$), parity ($P < 0.05$), time ($P < 0.01$), treatment by parity ($P = 0.07$), and parity by farm ($P = 0.08$). Treatments: S = short late dry period; L = long late dry period.

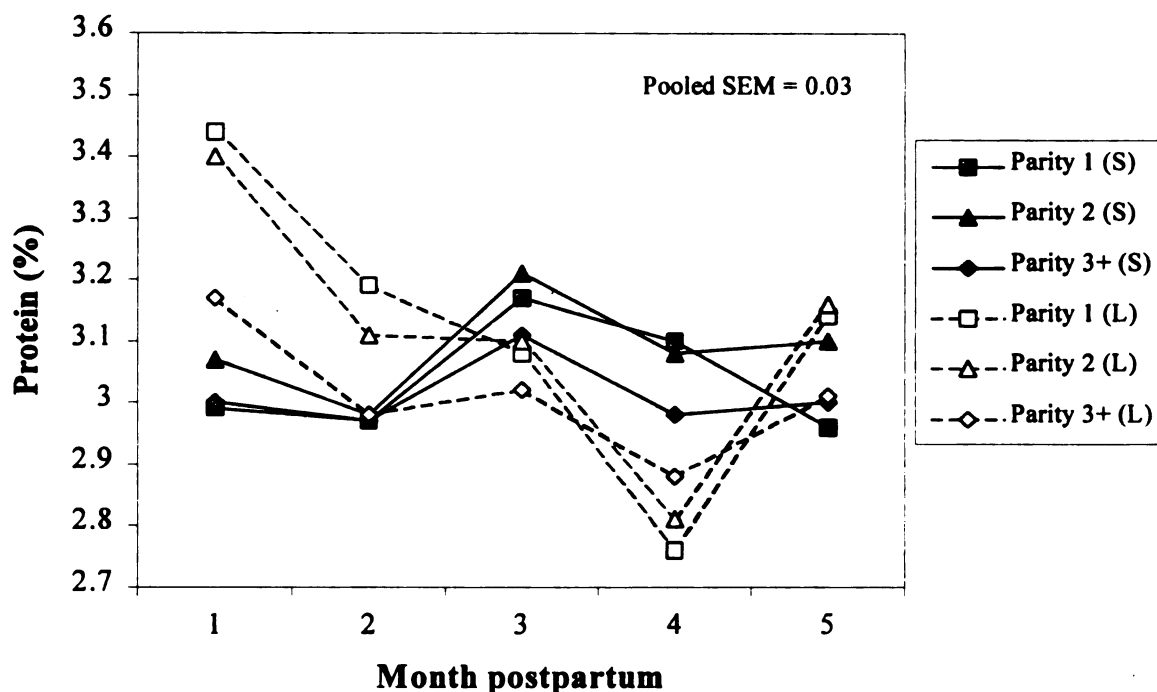


Figure 7. Least squares means for the interaction of parity by treatment by time ($P = 0.03$) on milk protein percentage through 150 DIM. Other significant effects in the model through 150 DIM were: farm ($P < 0.01$), parity ($P = 0.09$), time ($P < 0.01$), farm by time ($P < 0.01$), and treatment by time ($P < 0.01$). Significant effects of the model through 60 DIM were: farm ($P < 0.01$), parity ($P = 0.05$), treatment ($P < 0.01$), time ($P < 0.01$), farm by parity ($P = 0.08$), parity by treatment ($P = 0.07$) and treatment by time ($P < 0.01$). Treatments: S = short late dry period; L = long late dry period.

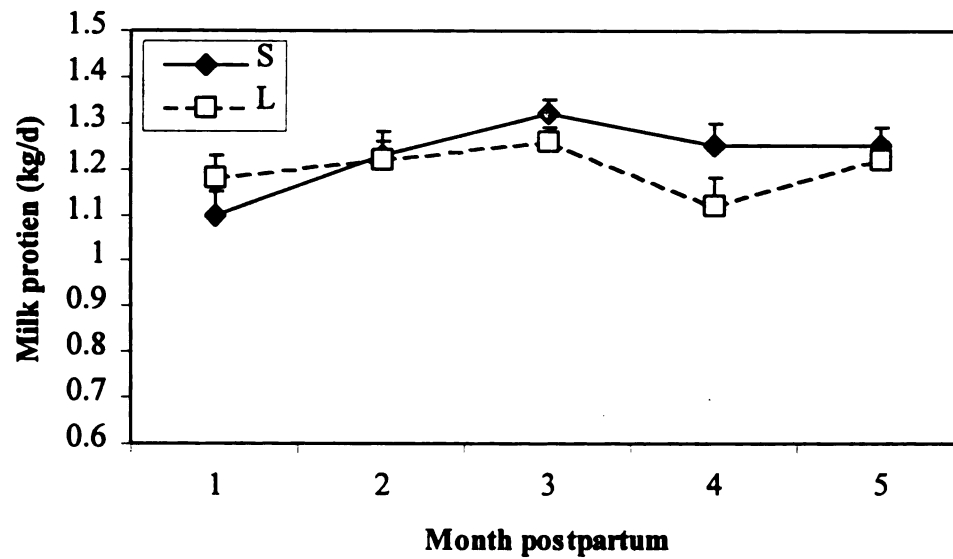


Figure 8. Least squares means and standard error of the means for the interaction of treatment by time ($P < 0.01$) on milk protein yield through 150 DIM. Other significant effects in the model include: farm ($P = 0.05$), parity ($P < 0.01$), time ($P = 0.02$), farm by time ($P < 0.01$), and parity by time ($P < 0.01$). Treatments: S = short late dry period; L = long late dry period.

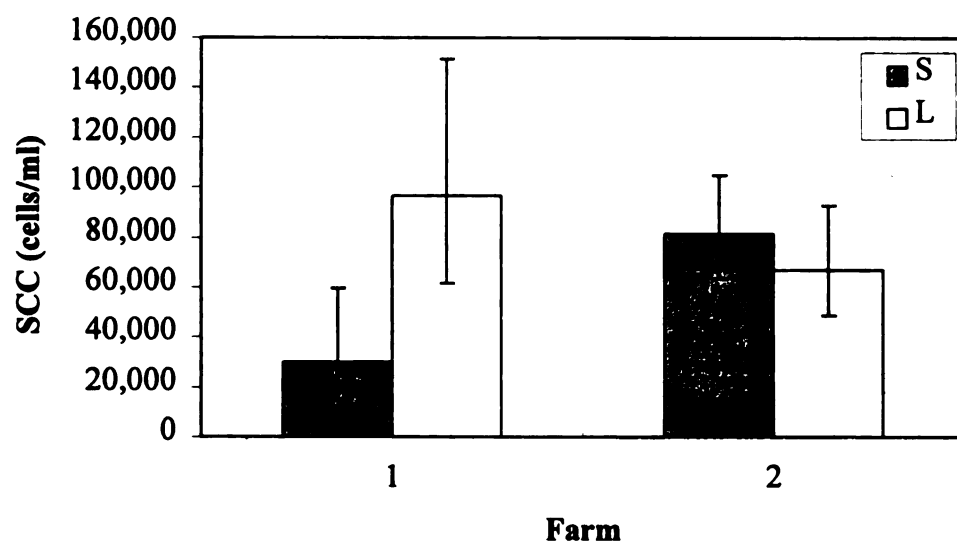


Figure 9. Least squares means and 95% confidence intervals for the interaction of treatment by farm ($P < 0.01$) for SCC through 150 DIM. Other significant variables in the model included: treatment ($P = 0.04$), parity ($P = 0.09$), time ($P < 0.01$) and parity by month ($P = 0.02$). Treatments: S = short late dry period; L = long late dry period.

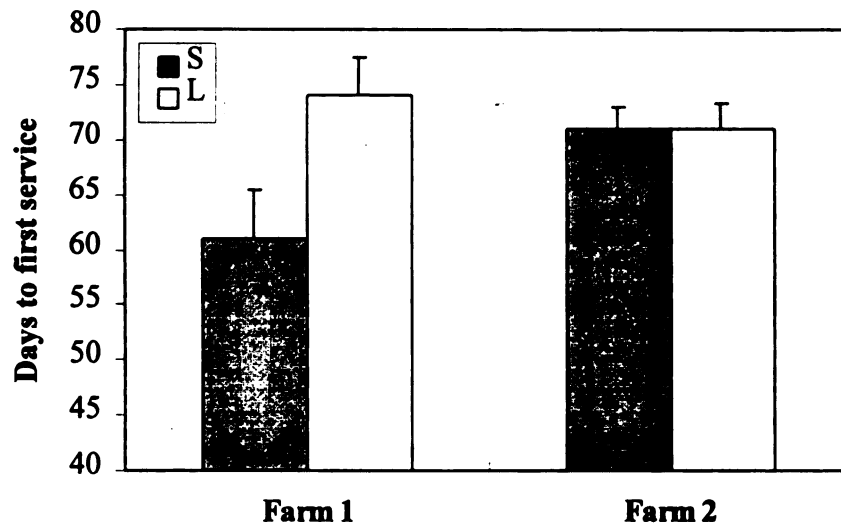


Figure 10. Least squares means and standard error of the means for the interaction of treatment by farm ($P = 0.06$) on days to first service. The only other significant variable in the model was treatment ($P = 0.04$). Treatments: S = short late dry period; L = long late dry period.

CHAPTER 5

ASSOCIATIONS AMONG BODY CONDITION SCORES, BODY CONDITION SCORE CHANGES, BLOOD VARIABLES AND MILK YIELD AND COMPOSITION IN PERIPARTURIENT DAIRY COWS

ABSTRACT

The objective of this study was to compare the relationships among body condition scores (BCS), BCS changes, and blood variables during the periparturient period, and milk yield and composition of early lactation dairy cows. Data from 378 cows from two previous research projects were pooled and correlation coefficients were generated among variables. Days spent in the late dry period consuming a higher energy diet compared with conventional early dry period diets, and BCS changes during this time were not strongly correlated with any other variables. Increased BCS at parturition were associated with increased plasma insulin concentrations prepartum, non-esterified fatty acid (NEFA) concentrations at 2 d postpartum, and BCS losses in early lactation. Cows with greater BCS losses in early lactation had higher postpartum NEFA concentrations and milk, fat, protein, and energy-corrected milk (ECM) yields. In early lactation, plasma NEFA concentrations were correlated positively with plasma beta-hydroxybutyrate (BHBA) concentrations and BCS losses, and negatively with plasma insulin concentrations. Plasma insulin concentrations were associated negatively with BHBA postpartum and with milk, fat, protein, and ECM yields. Additionally, correlation coefficients for all variables were calculated for parity categories 1, 2, and 3+, separately.

Days spent in the late dry period and BCS changes in the late dry period were correlated with BCS at parturition only in cows of parity 2. BCS at parturition were associated with increased BCS losses in early lactation in parity 2 and 3+ cows. In cows of parity 3+, BCS at parturition were correlated positively to milk, fat, and ECM yields, and fat and protein contents. Increased BCS losses in early lactation were associated with increased milk, fat, protein, and ECM yields in cows of parities 2 and 3+. In parities 1 and 3+ only, cows with higher plasma BHBA postpartum had increased plasma NEFA concentrations and BCS losses in early lactation and lower plasma insulin concentrations. Plasma insulin concentrations postpartum were correlated negatively with milk, fat, protein and ECM yields in parity 2 cows. Overall, correlations among variables differed depending upon parity. In this dataset, relationships between blood variables were similar in parity 1 and 3+ cows, and relationships involving BCS and BCS changes were similar in cows of parities 2 and 3+.

INTRODUCTION

Many factors during the periparturient period can influence milk yield and composition of dairy cows in early lactation. One such factor is the energy status of cows both pre- and postpartum. A common, practical method of measuring energy status is assigning body condition scores (BCS) to cows based on visual appraisal (Wildman et al., 1982). This method helps dairy producers monitor changes in body condition over several weeks or months. The relationships between BCS at parturition and BCS changes during lactation have been the focus of much research. Many controlled studies have

adjusted BCS at parturition by feeding a diet higher or lower in energy density during the dry period or late lactation (Boisclair et al., 1986; Fronk et al., 1980; Garnsworthy and Huggett, 1992; Holter et al., 1990; Garnsworthy and Jones, 1987; Jones and Garnsworthy, 1989; Nachtoml et al., 1986). Other studies measured BCS and BCS changes as they occurred naturally in commercial dairy farms (Domecq et al., 1997; Gallo et al., 1996; Gearheart and Curtis, 1990; Pedron et al., 1992; Rueeg et al., 1992). A more definitive method of measuring energy status may be analysis of certain blood metabolites and hormones known to reflect energy status. BCS at a point in time do not indicate the current energy status of cows. BCS need to be monitored over weeks or months to determine changes in BCS that can be related to energy status. However, certain blood variables can define current energy status more accurately compared with BCS. Few studies comparing BCS and milk yield have included analysis of blood variables. Therefore, the objective of this analysis was to evaluate the relationships among BCS, BCS changes and blood variables in the periparturient period, and milk yield and composition in early lactation.

MATERIALS AND METHODS

Three hundred and seventy-eight cows in two commercial dairy farms were studied in two previous experiments. In the first experiment, one hundred eight-nine cows in a commercial dairy farm were fed either one of two treatments that varied in corn grain concentration during the last 17 d prepartum (Chapter 3, Table 1). In the second experiment, one hundred eighty-nine cows in two commercial dairy farms were assigned

randomly to one of two treatments that varied in length of time cows were in the late dry period (**LDP**; Chapter 4, Table 2). Data from both experiments were combined for the current analysis.

One evaluator scored cows for BCS [five-point scale where 1 = thin to 5 = fat; (Wildman et al., 1982)] within the first week after cows entered the LDP, weekly prepartum, and 3 and 6 wk postpartum. BCS changes used in the analysis were defined as: LDP BCS change = -1 wk BCS (taken within 1 wk prior to parturition) – BCS taken within 1 wk after cows entered the late dry group; and, early lactation BCS change = 6 wk BCS – -1 wk BCS. Therefore, a negative value for BCS change would indicate BCS loss. Additionally, days spent in the LDP when cows are fed a more nutrient-dense diet compared with early dry period diets are presented.

Blood samples were collected in evacuated test tubes containing sodium heparin (Vacutainer; Becton Dickson Vacutainer Systems USA, Rutherford, NJ) from the coccygeal vein twice weekly prior to parturition, within 3 d following parturition, and 1 and 2 wk postpartum. Samples were stored on ice during transport to the laboratory. Upon arrival they were centrifuged and plasma was stored at -4°C until later analysis for NEFA (NEFA-C kit, Waco Chemicals USA, Richmond, VA) with modifications (Johnson and Peters, 1993), insulin (Coat-A-Count, Diagnostic Products Corporation, Los Angeles, CA), and BHBA (310-A, Sigma Diagnostics, St. Louis, MO). All reagents in the BHBA assay except beta-hydroxybutyrate dehydrogenase were reduced proportionally to fit into 350 μ l wells of cell culture plates. Beta-hydroxybutyrate dehydrogenase was added at twice the reduced dose to truncate the incubation time.

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Inter-assay and intra-assay coefficients of variation for BHBA, NEFA and insulin were 5.8 and 7.1, 6.9 and 8.2, and 4.4 and 7.8, respectively. For this analysis, blood samples were grouped into time periods of ± 1 d from the day of sampling, because all samples were not collected on the same day relative to calving for every cow. Time periods were -8, -5, -2, 2, 7, and 14 d.

In Farm 1, daily milk yield data were recorded monthly, and samples for fat and protein content, and SCC analysis were collected every 3 mo (Michigan DHIA). In Farm 2, individual milk yield data were recorded every 2 wk and samples were analyzed for fat and protein content and SCC monthly (Michigan DHIA). Energy-corrected milk (ECM) was calculated by the equation $\text{ECM (lb)} = 0.3246 \times \text{milk yield (lb)} + 12.86 \times \text{fat yield (lb)} + 7.04 \times \text{protein yield (lb)}$. (Dairy Herd Improvement Glossary, Fact Sheet A-4, 1999). Milk yield and composition data were reduced to one mean during the first 60 DIM. All data are presented as arithmetic means and standard deviations. Gross correlation coefficients were calculated by the PROC CORR procedure of SAS [version 6.1; SAS (1989)]. Correlation coefficients were generated for all cows and for cows of parities 1, 2, and 3+, separately. Correlations were declared significant at $P < 0.05$. Generally, correlations are discussed only if $r > 0.15$, $P < 0.05$, or if there is a trend for several significant correlations over time for specific blood variables. If correlation coefficients are presented and discussed for a specific blood variable over a time series, the range of low to high coefficients are presented (i.e., $r = 0.20$ to 0.30).

RESULTS AND DISCUSSION

All parities

Arithmetic means, standard deviations and ranges for all variables are in Table 1.

Correlation coefficients for the combined analysis of all parities are shown in Table 2.

Correlations among days in the LDP, -1 wk BCS, and LDP BCS change, and with blood variables and milk yield and composition. Days in LDP were not correlated with BCS changes in the LDP or -1 wk BCS. Increasing the length of time cows were fed the late dry period diet was correlated positively with plasma NEFA concentrations prepartum. Correlation coefficients were significant from -8 to 2 d and ranged from 0.11 to 0.20. Additionally, days in LDP were associated with early lactation BCS changes ($r = -0.16$). BCS at -1 wk were correlated with 3 wk BCS ($r = 0.61$), 6 wk BCS ($r = 0.47$), LDP BCS changes ($r = 0.28$), and early lactation BCS changes ($r = -0.19$). It is well documented that higher BCS at calving increases BCS loss in early lactation (Garnsworthy and Topps, 1982; Treacher et al., 1986; Ruegg et al, 1992; Pedron et al., 1993). In addition, -1 wk BCS were associated with increased plasma insulin concentrations from -8 ($r = 0.20$) to 2 d ($r = 0.12$) and plasma NEFA at 2 d ($r = 0.18$). Contrary to a previous report (Pedron et al., 1993), there was no relationship between -1 wk BCS on plasma NEFA after parturition. LDP BCS changes were not correlated with any variables.

Correlations among 3 and 6 wk BCS and early lactation BCS change, and with blood variables and milk yield and composition.

As expected, BCS at 3 and 6 wk were correlated highly with each other and with early lactation BCS changes. BCS at 3 and 6 wk were associated negatively with plasma BHBA concentrations from -2 to 14 d ($r = -0.15$ to -0.25). BCS at 3 and 6 wk were correlated negatively with NEFA ($r = -0.13$ to -0.31) and positively with insulin ($r = 0.24$ to 0.41) concentrations throughout the periparturient period,. Early lactation BCS changes were correlated negatively with plasma BHBA postpartum ($r = -0.21$ to -0.34) and NEFA concentration both pre- and postpartum ($r = -0.17$ to -0.46).

Early lactation BCS changes were positively correlated with plasma insulin throughout the periparturient period with the strongest correlation at 14 d ($r = 0.46$). Therefore, cows with higher plasma insulin concentrations pre- and postpartum had reduced BCS losses during the first 6 wk of lactation. Insulin is an antilipolytic hormone that reduces plasma NEFA (Grummer, 1995), and thereby should minimize BCS loss. Early lactation BCS changes were correlated negatively with milk yield and composition with the exception of milk protein content (correlation coefficients ranged from -0.16 to -0.22). Therefore, cows with high milk yields lost more BCS in early lactation.

Correlations among blood variables prepartum, and with milk yield and composition.

Plasma NEFA concentrations prepartum were correlated positively with NEFA concentrations postpartum ($r = 0.19$ to 0.42). Plasma NEFA concentrations were associated positively with BHBA ($r = 0.34$ to 0.56) and negatively with insulin ($r = -0.34$ to -0.50) through both pre- and postpartum periods. Cows with higher plasma NEFA

concentrations prepartum had lower milk, protein and ECM yields ($r = -0.14$ to -0.23). Several studies have reported reduced plasma NEFA concentrations prepartum by altering the diet fed during this time (Minor et al. 1998, Olsson et al., 1997). However, it not evident if the changes in milk yield and composition can be attributed solely to a reduction in prepartum NEFA concentrations.

Plasma BHBA concentrations at -8 and -5 d were not correlated with insulin concentrations prepartum or BHBA concentrations postpartum. Plasma BHBA concentrations prepartum were not associated with milk yield or composition. Pre- and postpartum concentrations of insulin were highly correlated ($r = 0.23$ to 0.38). However, plasma insulin concentrations prepartum were not associated with milk yield or composition.

Correlations among blood variables postpartum, and with milk yield and composition.

As previously mentioned, plasma concentrations of NEFA postpartum were associated positively with BHBA and negatively with insulin concentrations postpartum. NEFA at 7 d was the only postpartum sample correlated with any milk yield or composition variables. NEFA concentrations at 7 d were correlated negatively with milk ($r = -0.18$), fat ($r = -0.16$), protein ($r = -0.19$), and ECM ($r = -0.17$) yields. Plasma BHBA concentrations postpartum were correlated negatively with insulin postpartum ($r = -0.19$ to -0.31). BHBA concentrations at 7 d were correlated negatively with milk ($r = -0.30$), protein ($r = -0.31$) and ECM ($r = -0.23$) yields, and fat content ($r = -0.17$). However, the correlations were not consistent across the postpartum period for any of the milk yield or composition variables. Plasma insulin concentrations postpartum were correlated

negatively with milk yield, fat content and yield, protein yield, and ECM yield.

Correlation coefficients ranged from -0.15 to -0.29 .

Differences Among Parities

Correlations among days in the LDP, -1 wk BCS, and LDP BCS change, and with blood variables and milk yield and composition. Correlation coefficients for individual parities are shown in Tables 3, 4, and 5 for cows of parities 1, 2, and 3+, respectively. Parity 2 cows gained the most BCS during the LDP, but still calved with the lowest BCS of any parity (Table 1). Increased DMI for cows with lower BCS in the periparturient period has been reported (Hayirli et al., 1998). Therefore, increased energy intake by parity 2 cows may have accounted for the greater gain in BCS during the LDP. Days in LDP were correlated positively with -1 wk BCS ($r = 0.18$) and LDP BCS changes ($r = 0.24$) in parity 2 cows, but negatively with -1 wk BCS in parity 3+ cows ($r = -0.19$). BCS at -1 wk were associated with LDP BCS changes in all parities, but were associated negatively with early lactation BCS changes in cows of parities 2 ($r = -0.22$) and 3+ ($r = -0.20$). Therefore, multiparous cows with higher -1 wk BCS tended to lose more BCS in early lactation. In parity 1 cows, the only blood variables associated with -1 wk BCS prepartum were insulin concentrations from -8 to -5 d ($r = 0.35$ to 0.45). BCS at -1 wk were associated with NEFA in parity 2 cows from 2 to 7 d ($r = 0.29$ and 0.31 , respectively), and NEFA and BHBA at 14 d for parity 3+ cows ($r = 0.18$ for both). In parity 3+ cows only, -1 wk BCS were correlated positively with all milk yield and composition variables ($r = 0.27$ to 0.34) with the exception of protein yield.

In parity 1 cows, LDP BCS changes were correlated positively with BHBA ($r = -0.24$) and negatively with insulin at 14 d ($r = -0.22$). Parity 2 cows with higher LDP BCS changes lost more BCS postpartum ($r = -0.22$). LDP BCS changes were not related to any variables in cows of parity 3+.

Correlations among 3 and 6 wk BCS and early lactation BCS change, and with milk yield

and composition. Primiparous cows had less BCS loss through the first 6 wk of lactation than multiparous cows which is agrees with previous research (Waltner et al, 1980; Gallo et al., 1996). Early lactation BCS changes were correlated negatively with plasma NEFA and positively with insulin concentrations postpartum for all parities. However, early lactation BCS changes were only correlated with plasma BHBA concentrations postpartum in cows of parities 1 ($r = -0.34$ to -0.52) and 3+ ($r = -0.33$). In parity 1, cows with higher early lactation BCS changes yielded milk with less fat content during the first 2 mo of lactation ($r = -0.29$). Early lactation BCS changes were associated with decreased milk, fat, protein, and ECM yields in parity 2 cows ($r = -0.36, -0.22, -0.33$, and -0.29 , respectively). Parity 3+ cows had similar associations as parity 2 cows between early lactation BCS changes and milk, fat, protein and ECM yields.

Correlations among blood variables prepartum, and with milk yield and composition.

Plasma concentrations of blood variables during the periparturient period for each parity are shown in Table 1. Plasma concentrations of blood variables varied depending upon parity. However, parity 2 cows appeared to be in more positive energy status based on plasma NEFA and BHBA concentrations during the periparturient period. Plasma NEFA

concentrations were correlated positively with BHBA prepartum for all parities. In addition, plasma NEFA were correlated with insulin concentrations both pre- and postpartum for all parities. In parity 1 cows, plasma NEFA concentrations prepartum were correlated with milk protein content ($r = 0.30$ to 0.51).

Plasma BHBA concentrations at -8 and -5 d were not correlated to postpartum BHBA concentrations. Plasma BHBA concentrations prepartum were correlated positively with NEFA concentrations prepartum for cows in parities 1 and 3+ only ($r = 0.40$ to 0.63). Contrary, plasma BHBA concentrations in parity 2 cows were correlated positively with insulin at -8 and -5 d ($r = 0.26$ and 0.27 , respectively), but not with NEFA any time prepartum. Insulin concentrations at most time points prepartum were correlated with postpartum insulin concentrations for all parities. With the exception of plasma NEFA and milk protein content in parity 1 cows, there were no differences in correlations between parities in plasma concentrations of blood variables prepartum and milk yield and composition.

Correlations among blood variables postpartum, and with milk yield and composition.

Plasma NEFA concentrations postpartum were correlated positively with BHBA ($r = 0.22$ to 0.82) and negatively with insulin ($r = -0.38$ to -0.59) concentrations postpartum in all parities. Plasma NEFA concentrations postpartum were correlated with milk fat content ($r = 0.27$ and 0.36 for 7 and 14 d, respectively) in parity 1 cows. Plasma NEFA concentrations at 14 d were correlated positively with milk ($r = 0.38$), fat ($r = 0.26$), protein ($r = 0.27$), and ECM yields ($r = 0.32$) in parity 2 cows. Plasma NEFA concentrations were not associated with any variables related to milk yield or

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composition in parity 3+ cows. Increased concentrations of NEFA postpartum and coinciding BCS loss result in increased milk fat content (Holter et al., 1990). This is supported by positive correlations between plasma NEFA concentrations postpartum and milk fat content in cows of parities 1 and 2, but not 3+.

Generally, plasma BHBA concentrations were correlated negatively with insulin concentrations in the postpartum period in cows of parities 1 and 3+ ($r = -0.33$ to -0.66). Plasma BHBA were associated with milk yield at 7 d ($r = -0.28$) in parity 1 cows and at 2 d ($r = -0.26$) in parity 3+ cows. At least one postpartum measurement of BHBA concentrations were associated with fat content in all parities ($r = 0.20$ to 0.33). Plasma insulin concentrations were associated with fat content at 2 and 7 d ($r = -0.31$ and -0.33 , respectively) in cows of parity 1. In parity 2 cows, plasma insulin concentrations postpartum were associated with reduced yields of milk, fat, protein and ECM ($r = -0.22$ to -0.32). Similarly, insulin concentrations at 14 d were correlated with reduced yields of fat, protein, and ECM in parity 3+ cows ($r = -0.19$ to -0.27). Previous research has shown that increased insulin concentrations partition nutrients away from the mammary gland resulting in reduced milk fat yield and content (McGuire et al., 1995).

CONCLUSIONS

The associations among variables were influenced by parity. Relationships among blood variables were often different for cows of parity 2 compared with cows in parities 1 and 3+. Relationships with BCS and BCS changes in parity 1 cows were unique to cows of parities 2 and 3+. Relationships among variables and parity effects

may be unique to this study. Differences observed require further investigation with a dataset involving many farms. Future research should further define the associations between these variables and attempt to draw cause and effect relationships.

TABLES

Table 1. Arithmetic means, standard deviations and ranges for all variables.

Variable	Parity			Pooled across parities
	1	2	3	
Days in LDP ¹				
Mean	22	22	24	23
SD	10.4	10.0	9.5	9.9
Range	5 - 60	3 – 55	7 – 54	3 – 60
LDP BCS change ²				
Mean	0.04	0.08	0.04	0.05
SD	0.28	0.28	0.27	0.3
Range	-1.5 - 0.75	-0.5 – 1.0	-1.0 – 0.75	-1.5 – 1.0
-1 wk BCS				
Mean	3.70	3.49	3.60	3.59
SD	0.37	0.40	0.47	0.43
Range	2.25 - 4.25	2.5 – 4.50	2.0 – 4.50	2.0 – 4.50
3 wk BCS				
Mean	2.79	2.64	2.60	2.64
SD	0.56	0.58	0.67	0.62
Range	1.25 – 3.75	1.5 – 4.00	1.0 – 4.00	1.0 – 4.00
6 wk BCS				
Mean	2.75	2.60	2.41	2.54
SD	0.61	0.60	0.66	0.64
Range	1.25 – 3.75	1.25 – 4.25	1.0 – 4.00	1.0 – 4.25
Early lactation BCS change ³				
Mean	-0.98	-0.92	-1.21	-1.06
SD	0.55	0.55	0.59	0.58
Range	-2.5 – 0.25	-2.5 – 0.0	-2.3 – 0.5	-2.5 – 0.5
-8 d NEFA (μ Eq/L)				
Mean	207	106	215	178
SD	114	84	251	189
Range	66 – 486	30 – 587	27 – 1653	27 – 1653
-5 d NEFA (μ Eq/L)				
Mean	264	138	251	218
SD	153.5	119	238	196
Range	70 – 942	44 – 908	30 – 1391	30 – 1391
-2 d NEFA (μ Eq/L)				
Mean	388	207	371	321
SD	251.6	172	345	286
Range	92 – 1569	38 – 930	46 – 2181	38 – 2181

Table 1. (cont.)

Variable	Parity			Pooled across parities
	1	2	3	
2 d NEFA (μ Eq/L)				
Mean	658	488	716	629
SD	332	360	398	383
Range	158 – 2027	112 – 2074	59 – 1955	59 – 2074
7 d NEFA (μ Eq/L)				
Mean	671	458	606	573
SD	360	318	357	354
Range	133 – 1665	40 – 1662	84 – 1674	40 – 1674
14 d NEFA (μ Eq/L)				
Mean	413	345	403	386
SD	295	221	258	256
Range	84 – 1555	60 – 1148	77 – 1541	60 – 1555
-8 d BHBA (mg/dl)				
Mean	5.2	4.6	5.3	5.1
SD	5.7	1.5	2.2	3.2
Range	1.2 – 40.6	2.4 - 8.4	1.1 – 19.2	1.1 – 40.6
-5 d BHBA (mg/dl)				
Mean	5.6	4.8	5.4	5.3
SD	5.3	1.4	2.3	3.1
Range	1.8 – 44.3	1.8 – 7.8	2.0 – 18.5	1.8 – 44.3
-2 d BHBA (mg/dl)				
Mean	6.3	5.1	6.5	6.0
SD	4.3	1.3	3.8	3.4
Range	1.5 – 33.8	1.5 – 8.6	1.8 – 34.8	1.5 – 34.8
2 d BHBA (mg/dl)				
Mean	8.9	6.1	8.7	7.9
SD	6.7	2.3	5.6	5.3
Range	1.7 – 39.4	1.9 – 14.0	1.6 – 49.0	1.6 – 49.0
7 d BHBA (mg/dl)				
Mean	16.5	8.9	12.2	12.1
SD	14.2	6.4	9.0	10.2
Range	2.5 – 58.9	2.8 – 49.7	3.3 – 72.4	2.5 – 72.4
14 d BHBA (mg/dl)				
Mean	8.6	7.5	10.0	8.8
SD	6.9	5.6	8.2	7.2
Range	1.9 – 46.3	2.9 – 52.9	2.1 – 49.7	1.9 – 52.9

Table 1. (cont.)

Variable	Parity			Pooled across parities
	1	2	3	
-8 d Insulin (μIU/ml)				
Mean	12.3	15.8	13.3	13.9
SD	6.4	8.3	7.2	7.5
Range	3.1 – 28.9	2.6 – 44.4	1.3 – 34.5	1.3 – 44.4
-5 d Insulin (μIU/ml)				
Mean	12.2	13.5	11.2	12.2
SD	5.5	7.3	7.3	7.0
Range	3.5 – 28.7	2.9 – 47.5	2.1 – 42.9	2.1 – 47.5
-2 d Insulin (μIU/ml)				
Mean	11.3	12.4	9.9	11.1
SD	5.6	6.8	6.8	6.6
Range	3.3 – 30.3	1.3 – 35.4	1.3 – 42.3	1.3 – 42.3
2 d Insulin (μIU/ml)				
Mean	9.6	10.3	7.8	9.3
SD	4.7	5.7	4.4	5.4
Range	4.0 – 25.4	1.3 – 35.8	1.3 – 22.7	1.3 – 35.8
7 d Insulin (μIU/ml)				
Mean	9.6	8.8	7.4	8.4
SD	4.7	4.8	3.6	4.4
Range	4.0 – 25.4	1.3 – 41.0	1.7 – 22.3	1.3 – 41.0
14 d Insulin (μIU/ml)				
Mean	10.6	9.1	8.6	9.2
SD	5.0	4.0	4.4	4.5
Range	4.2 – 30.6	2.8 – 24.5	1.3 – 30.0	1.3 – 30.6
Milk yield (kg/d)				
Mean	28.0	42.6	44.4	40.1
SD	6.3	6.4	8.7	9.9
Range	13.3 – 41.5	27.9 – 59.7	22.5 – 66.7	13.3 – 66.7
Fat content (%)				
Mean	4.4	4.8	4.8	4.7
SD	0.8	1.0	1.1	1.0
Range	2.7 – 6.0	2.0 – 7.6	2.0 – 8.5	2.0 – 8.5
Fat yield (kg/d)				
Mean	1.3	2.1	2.2	2.0
SD	0.4	0.6	0.8	0.7
Range	0.4 – 2.2	0.9 – 4.4	0.4 – 5.1	0.4 – 5.1

Table 1. (cont.)

Variable	Parity			Pooled across parities
	1	2	3	
Protein content (%)				
Mean	3.1	3.1	3.0	3.1
SD	0.3	0.3	0.4	0.3
Range	2.3 – 3.8	2.4 – 3.7	2.1 – 4.2	2.1 – 4.2
Protein yield (kg/d)				
Mean	0.9	1.3	1.3	1.2
SD	0.2	0.2	0.3	0.3
Range	0.4 – 1.3	0.8 – 1.8	0.5 – 2.1	0.4 – 2.1
ECM yield (kg/d)				
Mean	32.1	49.9	52.4	47.0
SD	8.0	10.2	13.7	14.0
Range	13.6 – 48.0	25.2 – 88.1	13.0 – 96.2	13.0 – 96.2

¹LDP = Late dry period.²LDP BCS change = -1 wk BCS – BCS within 1 wk after moved into LDP.³Early lactation BCS change = 6 wk BCS - -1 wk BCS.

Table 2. Correlations, P-values and n amongst variables pooled across parities.

Variable	Days in LDP ¹	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS Change ²	Early lactation BCS change ³
Days in LDP						
-1 wk BCS	0.01 ⁴ 0.91 ⁵ 372 ⁶					
3 wk BCS	-0.09 0.10 345	0.61 < 0.01 344				
6 wk BCS	-0.14 < 0.01 343	0.47 < 0.01 342	0.79 < 0.01 340			
LDP BCS change	0.09 0.07 372	0.28 < 0.01 374	0.14 < 0.01 344	0.05 0.35 342	374	
Early lactation BCS change	-0.16 < 0.01 339	-0.19 < 0.01 341	0.45 < 0.01 337	0.78 < 0.01 341	-0.14 0.01 341	
-8 d BHBA	-0.07 0.34 195	-0.06 0.40 194	-0.13 0.09 181	-0.08 0.27 179	-0.08 0.25 194	-0.05 0.55 177
-5 d BHBA	-0.15 0.02 261	-0.06 0.36 260	-0.15 0.02 244	-0.07 0.29 241	-0.07 0.24 260	-0.02 0.75 239
-2 d BHBA	0.00 0.94 287	-0.08 0.17 288	-0.21 < 0.01 272	-0.15 0.01 271	-0.12 0.04 288	-0.09 0.12 269
2 d BHBA	0.00 0.94 346	0.07 0.22 345	-0.16 < 0.01 328	-0.17 < 0.01 324	-0.10 0.06 345	-0.21 < 0.01 320
7 d BHBA	-0.07 0.19 341	0.07 0.23 341	-0.18 < 0.01 329	-0.25 < 0.01 325	-0.06 0.26 341	-0.34 < 0.01 322
14 d BHBA	-0.07 0.23 327	0.11 0.04 327	-0.05 0.33 323	-0.17 < 0.01 318	-0.13 0.02 327	-0.30 < 0.01 315

Table 2. (cont.)

Variable	Days in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
-8 d NEFA	0.20	-0.14	-0.26	-0.23	-0.14	-0.17
	< 0.01	0.045	< 0.01	< 0.01	0.045	0.03
	195	194	181	179	194	177
-5 d NEFA	0.18	0.00	-0.11	-0.13	-0.04	-0.20
	< 0.01	0.99	0.09	0.04	0.49	< 0.01
	261	260	244	241	260	239
-2 d NEFA	0.15	0.10	-0.21	-0.23	-0.11	-0.33
	< 0.01	0.09	< 0.01	< 0.01	0.06	< 0.01
	287	288	272	271	288	269
2 d NEFA	0.11	0.18	-0.16	-0.26	0.03	-0.43
	0.0336	< 0.01	< 0.01	0.01	0.56	< 0.01
	346	345	328	324	345	320
7 d NEFA	0.06	0.10	-0.19	-0.31	0.02	-0.43
	0.29	0.07	< 0.01	< 0.01	0.74	< 0.01
	341	341	329	325	341	322
14 d NEFA	0.07	0.12	-0.17	-0.28	-0.04	-0.46
	0.22	0.03	< 0.01	< 0.01	0.46	< 0.01
	327	327	323	318	327	315
-8 d Insulin	-0.07	0.20	0.37	0.36	0.08	0.27
	0.32	< 0.01	< 0.01	< 0.01	0.25	< 0.01
	193	192	179	177	192	175
-5 d Insulin	-0.05	0.14	0.27	0.28	0.08	0.21
	0.41	0.02	< 0.01	< 0.01	0.23	< 0.01
	256	255	239	236	255	234
-2 d Insulin	-0.04	0.12	0.34	0.31	0.11	0.25
	0.49	0.05	< 0.01	< 0.01	0.07	< 0.01
	285	286	270	269	286	267
2 d Insulin	-0.13	0.11	0.29	0.38	0.02	0.36
	0.02	0.05	< 0.01	< 0.01	0.78	< 0.01
	340	339	323	318	339	315
7 d Insulin	-0.06	0.04	0.28	0.40	0.06	0.44
	0.27	0.49	< 0.01	< 0.01	0.28	< 0.01
	335	335	323	319	335	316
14 d Insulin	-0.13	-0.06	0.24	0.41	-0.02	0.46
	0.03	0.30	< 0.01	< 0.01	0.67	< 0.01
	317	317	315	310	317	307

Table 2. (cont.)

Variable	Days in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
Milk yield	0.02	-0.01	-0.15	-0.19	0.08	-0.20
	0.68	0.85	0.01	< 0.01	0.17	< 0.01
	294	293	290	291	293	288
Fat content	-0.09	0.11	0.01	-0.07	-0.05	-0.16
	0.13	0.06	0.91	0.22	0.42	0.01
	294	293	290	291	293	288
Fat yield	-0.04	0.07	-0.08	-0.15	0.02	-0.21
	0.48	0.26	0.18	0.01	0.73	< 0.01
	294	293	290	291	293	288
Protein content	0.06	0.03	0.05	0.06	0.03	0.04
	0.29	0.64	0.37	0.34	0.61	0.54
	294	293	290	291	293	288
Protein yield	0.05	0.01	-0.14	-0.18	0.07	-0.20
	0.36	0.88	0.02	< 0.01	0.24	< 0.01
	294	293	290	291	293	288
ECM yield	-0.02	0.05	-0.11	-0.18	0.04	-0.22
	0.76	0.44	0.06	< 0.01	0.54	< 0.01
	294	293	290	291	293	288

Table 2. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
-8 d BHBA						
-5 d BHBA	0.86 < 0.01 158					
-2 d BHBA	0.70 < 0.01 151	0.71 < 0.01 208				
2 d BHBA	0.40 < 0.01 189	0.44 < 0.01 252	0.50 < 0.01 279			
7 d BHBA	0.02 0.81 183	0.05 0.44 247	0.25 < 0.01 276	0.36 < 0.01 334		
14 d BHBA	0.02 0.82 173	0.02 0.77 237	0.10 0.09 268	0.19 < 0.01 321	0.52 < 0.01 322	
-8 d NEFA	0.34 < 0.01 196	0.30 < 0.01 158	0.22 0.01 151	0.29 < 0.01 189	0.10 0.19 183	0.07 0.39 173
-5 d NEFA	0.42 < 0.01 158	0.42 < 0.01 262	0.43 < 0.01 208	0.47 < 0.01 252	0.17 0.01 247	0.07 0.25 237
-2 d NEFA	0.15 0.07 151	0.18 0.01 208	0.47 < 0.01 289	0.42 < 0.01 279	0.31 < 0.01 276	0.26 < 0.01 268
2 d NEFA	0.07 0.34 189	0.10 0.12 252	0.18 < 0.01 279	0.36 < 0.01 348	0.36 < 0.01 334	0.14 0.01 321
7 d NEFA	-0.01 0.93 183	0.04 0.50 247	0.20 < 0.01 276	0.20 < 0.01 334	0.56 < 0.01 343	0.31 < 0.01 322
14 d NEFA	< 0.01 0.96 173	-0.02 0.72 237	0.05 0.43 268	0.15 0.01 321	0.31 < 0.01 322	0.51 < 0.01 329

Table 2. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
-8 d Insulin	-0.02	-0.16	-0.21	-0.15	-0.16	-0.04
	0.76	0.05	0.01	0.04	0.04	0.65
	194	156	150	187	181	171
-5 d Insulin	-0.12	-0.07	-0.17	-0.19	-0.18	0.03
	0.13	0.28	0.02	< 0.01	0.01	0.68
	156	257	205	247	242	233
-2 d Insulin	-0.15	-0.12	-0.18	-0.13	-0.19	-0.07
	0.06	0.09	< 0.01	0.03	< 0.01	0.24
	150	206	287	277	274	267
2 d Insulin	-0.05	-0.06	-0.11	-0.19	-0.20	-0.15
	0.51	0.34	0.08	< 0.01	< 0.01	0.01
	184	247	274	342	328	316
7 d Insulin	0.06	0.02	-0.10	-0.05	-0.31	-0.25
	0.45	0.72	0.11	0.33	< 0.01	< 0.01
	178	242	272	328	337	317
14 d Insulin	-0.04	-0.03	-0.06	-0.12	-0.16	-0.23
	0.59	0.67	0.37	0.04	< 0.01	< 0.01
	169	230	259	311	313	319
Milk yield	-0.02	-0.08	-0.15	-0.21	-0.30	-0.04
	0.85	0.24	0.02	< 0.01	< 0.01	0.46
	150	203	232	277	281	274
Fat content	-0.02	0.07	0.03	0.12	0.06	0.20
	0.84	0.34	0.66	0.05	0.34	< 0.01
	150	203	232	277	281	274
Fat yield	-0.02	0.02	-0.08	-0.08	-0.17	0.08
	0.78	0.80	0.23	0.18	< 0.01	0.16
	150	203	232	277	281	274
Protein Content	-0.10	-0.02	-0.01	0.03	-0.04	-0.05
	0.23	0.78	0.92	0.67	0.52	0.42
	150	203	232	277	281	274
Protein Yield	-0.07	-0.06	-0.14	-0.20	-0.31	-0.05
	0.38	0.38	0.03	< 0.01	< 0.01	0.41
	150	203	232	277	281	274
ECM yield	-0.03	-0.01	-0.10	-0.13	-0.23	0.05
	0.70	0.92	0.12	0.03	< 0.01	0.45
	150	203	232	277	281	274

Table 2. (cont.)

Variable	-8 d NEFA	-5 d NEFA	-2 d NEFA	2 d NEFA	7 d NEFA	14 d NEFA
-8 d NEFA						
-5 d NEFA	0.61 < 0.01 158					
-2 d NEFA	0.41 < 0.01 151	0.62 < 0.01 208				
2 d NEFA	0.38 < 0.01 189	0.37 < 0.01 252	0.42 < 0.01 279			
7 d NEFA	0.19 0.01 183	0.27 < 0.01 247	0.39 < 0.01 276	0.41 < 0.01 334		
14 d NEFA	0.26 < 0.01 173	0.23 < 0.01 237	0.28 < 0.01 268	0.30 < 0.01 321	0.41 < 0.01 322	
-8 d Insulin	-0.43 < 0.01 194	-0.40 < 0.01 156	-0.33 < 0.01 150	-0.33 < 0.01 187	-0.18 0.02 181	-0.16 0.04 171
-5 d Insulin	-0.35 < 0.01 156	-0.41 < 0.01 257	-0.36 < 0.01 205	-0.28 < 0.01 247	-0.30 < 0.01 242	-0.15 0.02 233
-2 d Insulin	-0.24 < 0.01 150	-0.30 < 0.01 206	-0.50 < 0.01 287	-0.32 < 0.01 277	-0.21 < 0.01 274	-0.11 0.08 267
2 d Insulin	-0.25 < 0.01 184	-0.25 < 0.01 247	-0.28 < 0.01 274	-0.46 < 0.01 342	-0.32 < 0.01 328	-0.27 < 0.01 316
7 d Insulin	-0.09 0.26 178	-0.12 0.07 242	-0.21 < 0.01 272	-0.29 < 0.01 328	-0.45 < 0.01 337	-0.30 < 0.01 317
14 d Insulin	-0.19 0.01 169	-0.08 0.24 230	-0.14 0.02 259	-0.24 < 0.01 311	-0.26 < 0.01 313	-0.34 < 0.01 319

Table 2. (cont.)

Variable	-8 d NEFA	-5 d NEFA	-2 d NEFA	2 d NEFA	7 d NEFA	14 d NEFA
Milk yield	-0.23	-0.16	-0.18	-0.06	-0.18	0.08
	< 0.01	0.02	0.01	0.32	< 0.01	0.16
	150	203	232	277	281	274
Fat content	-0.14	-0.07	-0.05	0.04	-0.06	0.10
	0.10	0.31	0.45	0.52	0.35	0.09
	150	203	232	277	281	274
Fat yield	-0.23	-0.14	-0.14	-0.01	-0.16	0.12
	< 0.01	0.05	0.03	0.89	0.01	0.06
	150	203	232	277	281	274
Protein Content	0.12	0.08	0.15	0.02	-0.06	-0.08
	0.16	0.23	0.02	0.76	0.29	0.18
	150	203	232	277	281	274
Protein Yield	-0.15	-0.10	-0.09	-0.03	-0.19	0.08
	0.07	0.15	0.16	0.68	< 0.01	0.17
	150	203	232	277	281	274
ECM yield	-0.23	-0.14	-0.14	-0.02	-0.17	0.12
	< 0.01	0.05	0.03	0.75	< 0.01	0.06
	150	203	232	277	281	274

Table 2. (cont.)

Variable	-8 d Insulin	-5 d Insulin	-2 d Insulin	2 d Insulin	7 d Insulin	14 d Insulin
-8 d Insulin						
-5 d Insulin	0.62 < 0.01 154					
-2 d Insulin	0.47 < 0.01 149	0.62 < 0.01 203				
2 d Insulin	0.38 < 0.01 182	0.37 < 0.01 242	0.36 < 0.01 272			
7 d Insulin	0.23 < 0.01 176	0.25 < 0.01 238	0.29 < 0.01 270	0.53 < 0.01 322		
14 d Insulin	0.26 < 0.01 167	0.23 < 0.01 226	0.27 < 0.01 258	0.48 < 0.01 306	0.50 < 0.01 308	
Milk yield	0.07 0.40 148	-0.04 0.58 199	-0.07 0.30 230	-0.17 < 0.01 272	-0.15 0.01 275	-0.25 < 0.01 268
Fat content	0.08 0.32 148	0.17 0.02 199	0.03 0.63 230	-0.09 0.15 272	-0.13 0.04 275	-0.16 0.01 268
Fat yield	0.08 0.31 148	0.09 0.20 199	-0.02 0.78 230	-0.15 0.01 272	-0.17 0.01 275	-0.23 < 0.01 268
Protein Content	0.01 0.93 148	0.21 < 0.01 199	0.06 0.35 230	< 0.01 0.94 272	0.06 0.33 275	-0.02 0.80 268
Protein Yield	0.07 0.40 148	0.07 0.34 199	-0.03 0.64 230	-0.19 < 0.01 272	-0.15 0.01 275	-0.29 < 0.01 268
ECM yield	0.08 0.31 148	0.06 0.37 199	-0.03 0.62 230	-0.17 0.01 272	-0.17 < 0.01 275	-0.26 < 0.01 268

Table 2. (cont.)

Variable	Milk yield	Fat content	Fat yield	Protein content	Protein yield	ECM yield
Milk yield						
Fat content	0.30 < 0.01 295					
Fat yield	0.80 < 0.01 295	0.78 < 0.01 295				
Protein Content	-0.28 < 0.01 295	0.13 0.02 295	-0.14 0.02 295			
Protein Yield	0.88 < 0.01 295	0.36 < 0.01 295	0.76 < 0.01 295	0.15 0.01 295		
ECM yield	0.90 < 0.01 295	0.65 < 0.01 295	0.98 < 0.01 295	-0.14 0.01 295	0.87 < 0.01 295	

¹LDP = Late dry period.²LDP BCS change = -1 wk BCS – BCS within 1 wk after moved into LDP.³Early lactation BCS change = 6 wk BCS - -1 wk BCS.⁴Correlation coefficient.⁵P-value.⁶Number of observations.

Table 3. Correlations, P-values, and n amongst variables for cows of parity 1.

Variable	Days in LDP ¹	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change ²	Early lactation BCS change ³
Days in LDP						
-1 wk BCS	0.17 ⁴ 0.11 ⁵ 91 ⁶					
3 wk BCS	-0.01 0.91 83	0.51 < 0.01 82				
6 wk BCS	-0.02 0.89 84	0.44 < 0.01 83	0.82 < 0.01 82			
LDP BCS change	0.13 0.23 91	0.36 < 0.01 91	0.11 0.34 82	0.01 0.97 83		
Early lactation BCS change	-0.11 0.32 83	-0.09 0.40 83	0.62 < 0.01 81	0.85 < 0.01 83	-0.04 0.70 83	
-8 d BHBA	-0.18 0.23 44	-0.14 0.35 44	-0.37 0.02 39	-0.24 0.13 39	-0.10 0.54 44	-0.08 0.61 39
-5 d BHBA	-0.13 0.34 60	-0.18 0.18 59	-0.35 0.01 56	-0.19 0.17 56	-0.11 0.43 59	-0.06 0.66 55
-2 d BHBA	-0.17 0.16 70	-0.17 0.15 70	-0.38 < 0.01 65	-0.20 0.12 64	-0.09 0.46 70	-0.08 0.55 64
2 d BHBA	-0.02 0.83 83	-0.11 0.31 82	-0.41 < 0.01 79	-0.39 < 0.01 79	-0.05 0.63 82	-0.34 < 0.01 78
7 d BHBA	-0.12 0.28 84	-0.19 0.09 83	-0.43 < 0.01 81	-0.52 < 0.01 81	-0.12 0.27 83	-0.48 < 0.01 80
14 d BHBA	-0.12 0.28 78	-0.14 0.23 77	-0.26 0.02 77	-0.34 < 0.01 76	-0.24 0.03 77	-0.43 < 0.01 75

Table 3. (cont.)

Variable	Day in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
-8 d NEFA	-0.13	-0.06	-0.37	-0.31	-0.08	-0.24
	0.42	0.70	0.02	0.05	0.59	0.13
	44	44	39	39	44	39
-5 d NEFA	0.17	-0.11	-0.26	-0.10	-0.25	-0.07
	0.18	0.39	0.06	0.44	0.06	0.59
	60	59	56	56	59	55
-2 d NEFA	0.02	0.01	-0.19	-0.10	-0.05	-0.16
	0.84	0.95	0.14	0.45	0.70	0.20
	70	70	65	64	70	64
2 d NEFA	0.11	0.06	-0.18	-0.37	0.06	-0.46
	0.31	0.59	0.12	< 0.01	0.58	< 0.01
	83	82	79	79	82	78
7 d NEFA	-0.17	-0.10	-0.41	-0.48	-0.19	-0.51
	0.12	0.36	< 0.01	< 0.01	0.08	< 0.01
	84	83	81	81	83	80
14 d NEFA	0.03	-0.12	-0.38	-0.43	-0.21	-0.56
	0.77	0.29	< 0.01	< 0.01	0.07	< 0.01
	78	77	77	76	77	75
-8 d Insulin	0.06	0.45	0.38	0.36	0.19	0.07
	0.72	< 0.01	0.02	0.02	0.22	0.67
	44	44	39	39	44	39
-5 d Insulin	0.10	0.35	0.20	0.26	0.06	0.04
	0.46	0.01	0.15	0.05	0.68	0.80
	58	57	54	54	57	53
-2 d Insulin	0.03	0.19	0.29	0.23	0.14	0.11
	0.82	0.11	0.02	0.07	0.25	0.38
	70	70	65	64	70	64
2 d Insulin	0.01	0.16	0.21	0.36	-0.06	0.32
	0.92	0.17	0.07	< 0.01	0.59	< 0.01
	81	80	77	77	80	76
7 d Insulin	0.14	0.09	0.31	0.37	0.13	0.37
	0.21	0.45	0.01	< 0.01	0.23	< 0.01
	82	81	79	79	81	78
14 d Insulin	-0.06	-0.14	0.13	0.41	-0.22	0.40
	0.64	0.24	0.28	< 0.01	0.06	< 0.01
	75	74	74	73	74	72

Table 3. (cont.)

Variable	Days in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
Milk yield	0.12	0.13	0.06	0.02	0.01	-0.04
	0.35	0.31	0.63	0.88	0.98	0.73
	66	65	64	65	65	64
Fat content	-0.15	-0.07	-0.21	-0.29	-0.18	-0.26
	0.24	0.59	0.10	0.02	0.16	0.04
	66	65	64	65	65	64
Fat yield	-0.02	0.05	-0.04	-0.12	-0.12	-0.14
	0.90	0.69	0.74	0.34	0.35	0.26
	66	65	64	65	65	64
Protein content	0.05	-0.01	0.10	0.18	-0.07	0.20
	0.72	0.96	0.43	0.15	0.56	0.12
	66	65	64	65	65	64
Protein yield	0.17	0.13	0.11	0.09	-0.07	0.04
	0.17	0.30	0.38	0.46	0.58	0.77
	66	65	64	65	65	64
ECM yield	0.06	0.08	0.01	-0.06	-0.09	-0.09
	0.65	0.51	0.96	0.66	0.47	0.46
	66	65	64	65	65	64

Table 3. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
-8 d BHBA						
-5 d BHBA	0.97 < 0.01 34					
-2 d BHBA	0.91 < 0.01 36	0.89 < 0.01 46				
2 d BHBA	0.56 < 0.01 42	0.61 < 0.01 56	0.69 < 0.01 67			
7 d BHBA	-0.01 0.93 40	0.03 0.80 57	0.16 0.19 66	0.40 < 0.01 81		
14 d BHBA	-0.13 0.47 35	-0.04 0.76 53	0.01 0.93 62	0.13 0.28 75	0.47 < 0.01 77	
-8 d NEFA	0.44 < 0.01 44	0.48 < 0.01 34	0.51 < 0.01 36	0.58 < 0.01 42	0.29 0.07 40	0.10 0.58 35
-5 d NEFA	0.64 < 0.01 34	0.62 < 0.01 60	0.69 < 0.01 46	0.50 < 0.01 56	0.10 0.47 57	-0.08 0.59 53
-2 d NEFA	0.12 0.50 36	0.19 0.20 46	0.40 < 0.01 70	0.32 0.01 67	0.25 0.04 66	0.04 0.77 62
2 d NEFA	0.09 0.57 42	0.12 0.39 56	0.17 0.16 67	0.57 < 0.01 83	0.44 < 0.01 81	0.14 0.23 75
7 d NEFA	0.09 0.58 40	0.09 0.50 57	0.18 0.14 66	0.25 0.02 81	0.75 < 0.01 84	0.39 < 0.01 77
14 d NEFA	-0.05 0.76 35	0.01 0.93 53	0.03 0.80 62	0.17 0.14 75	0.34 < 0.01 77	0.83 < 0.01 78

Table 3. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
Insulin -8	-0.19 0.23 44	-0.23 0.19 34	-0.29 0.09 36	-0.21 0.18 42	-0.14 0.40 40	-0.16 0.35 35
Insulin -5	-0.29 0.10 33	-0.22 0.09 58	-0.24 0.12 45	-0.32 0.02 54	-0.25 0.06 55	0.19 0.18 52
Insulin -2	-0.26 0.12 36	-0.28 0.06 46	-0.39 < 0.01 70	-0.29 0.02 67	-0.28 0.03 66	0.04 0.73 62
2 d Insulin	-0.07 0.68 40	-0.16 0.25 54	-0.12 0.33 65	-0.35 < 0.01 81	-0.28 0.01 79	-0.14 0.22 74
7 d Insulin	0.01 0.99 38	0.02 0.86 55	-0.14 0.28 65	-0.13 0.24 79	-0.51 < 0.01 82	-0.32 0.01 76
14 d Insulin	-0.12 0.48 34	-0.11 0.44 51	-0.14 0.28 59	-0.22 0.07 72	-0.17 0.14 74	-0.17 0.16 75
Milk yield	0.23 0.20 32	0.09 0.56 45	-0.16 0.27 48	-0.14 0.28 62	-0.28 0.03 64	-0.04 0.74 60
Fat content	-0.18 0.32 32	0.13 0.40 45	0.23 0.12 48	0.33 0.01 62	0.30 0.02 64	0.21 0.11 60
Fat yield	-0.02 0.90 32	0.11 0.48 45	0.03 0.86 48	0.06 0.63 62	-0.04 0.77 64	0.07 0.60 60
Protein Content	-0.26 0.15 32	0.11 0.48 45	0.22 0.13 48	0.10 0.42 62	-0.10 0.43 64	-0.29 0.02 60
Protein Yield	0.05 0.81 32	0.11 0.49 45	-0.02 0.88 48	-0.08 0.53 62	-0.30 0.02 64	-0.17 0.19 60
ECM yield	0.05 0.77 32	0.10 0.50 45	-0.02 0.91 48	0.01 0.98 62	-0.14 0.26 64	0.01 0.98 60

Table 3. (cont.)

Variable	-8 d NEFA	-5 d NEFA	-2 d NEFA	2 d NEFA	7 d NEFA	14 d NEFA
-8 d NEFA						
-5 d NEFA	0.58 < 0.01 34					
-2 d NEFA	0.46 < 0.01 36	0.58 < 0.01 46				
2 d NEFA	0.31 0.05 42	0.22 0.10 56	0.33 0.01 67			
7 d NEFA	0.27 0.09 40	0.20 0.14 57	0.30 0.01 66	0.38 < 0.01 81		
14 d NEFA	0.25 0.15 35	0.10 0.48 53	0.09 0.47 62	0.24 0.04 75	0.41 < 0.01 77	
-8 d Insulin	-0.45 < 0.01 44	-0.36 0.04 34	-0.29 0.09 36	-0.09 0.57 42	-0.14 0.39 40	-0.19 0.26 35
-5 d Insulin	-0.31 0.08 33	-0.29 0.03 58	-0.20 0.19 45	-0.15 0.26 54	-0.30 0.02 55	0.01 0.96 52
-2 d Insulin	-0.35 0.03 36	-0.41 < 0.01 46	-0.46 < 0.01 70	-0.15 0.22 67	-0.27 0.03 66	0.15 0.25 62
2 d Insulin	-0.30 0.06 40	-0.25 0.07 54	-0.36 < 0.01 65	-0.51 < 0.01 81	-0.35 < 0.01 79	-0.16 0.19 74
7 d Insulin	-0.31 0.06 38	-0.06 0.68 55	-0.14 0.28 65	-0.20 0.08 79	-0.59 < 0.01 82	-0.25 0.03 76
14 d Insulin	-0.44 0.01 34	0.10 0.51 51	0.05 0.72 59	-0.23 0.05 72	-0.20 0.09 74	-0.17 0.15 75

Table 3. (cont.)

	-8 d	-5 d	-2 d	2 d	7 d	14 d
Variable	NEFA	NEFA	NEFA	NEFA	NEFA	NEFA
Milk yield	-0.09	0.01	-0.19	-0.19	-0.13	0.01
	0.61	0.96	0.19	0.15	0.29	0.91
	32	45	48	62	64	60
Fat content	0.26	0.09	0.17	0.21	0.36	0.27
	0.16	0.55	0.25	0.10	< 0.01	0.04
	32	45	48	62	64	60
Fat yield	0.10	0.09	-0.04	0.03	0.14	0.17
	0.60	0.54	0.81	0.83	0.29	0.19
	32	45	48	62	64	60
Protein Content	0.30	0.43	0.51	-0.06	-0.04	-0.16
	0.09	< 0.01	< 0.01	0.66	0.76	0.22
	32	45	48	62	64	60
Protein Yield	0.08	0.28	0.04	-0.12	-0.08	0.01
	0.65	0.06	0.79	0.34	0.52	0.99
	32	45	48	62	64	60
ECM yield	0.06	0.12	-0.06	-0.04	0.05	0.12
	0.74	0.43	0.67	0.78	0.72	0.36
	32	45	48	62	64	60

Table 3. (cont.)

Variable	-8 d Insulin	-5 d Insulin	-2 d Insulin	2 d Insulin	7 d Insulin	14 d Insulin
-8 d Insulin						
-5 d Insulin	0.66 < 0.01 33					
-2 d Insulin	0.43 0.01 36	0.46 < 0.01 45				
2 d Insulin	0.46 < 0.01 40	0.35 0.01 52	0.41 < 0.01 65			
7 d Insulin	0.18 0.29 38	0.28 0.04 54	0.38 < 0.01 65	0.49 < 0.01 77		
14 d Insulin	0.30 0.09 34	0.12 0.40 50	0.26 0.05 59	0.42 < 0.01 71	0.51 < 0.01 73	
Milk yield	0.05 0.80 32	-0.09 0.54 44	-0.05 0.75 48	0.03 0.83 61	0.04 0.75 62	-0.25 0.06 57
Fat content	-0.09 0.64 32	-0.12 0.43 44	-0.22 0.14 48	-0.20 0.12 61	-0.31 0.01 62	-0.33 0.01 57
Fat yield	-0.03 0.88 32	-0.14 0.37 44	-0.12 0.42 48	-0.10 0.43 61	-0.14 0.27 62	-0.31 0.02 57
Protein Content	0.01 0.97 32	0.12 0.44 44	-0.15 0.30 48	-0.11 0.41 61	0.08 0.53 62	0.12 0.37 57
Protein Yield	0.08 0.65 32	-0.03 0.85 44	-0.15 0.32 48	-0.05 0.72 61	0.04 0.74 62	-0.21 0.11 57
ECM yield	0.01 0.97 32	-0.12 0.42 44	-0.12 0.44 48	-0.07 0.60 61	-0.07 0.57 62	-0.29 0.03 57

Table 3. (cont.)

Variable	Milk yield	Fat content	Fat yield	Protein content	Protein yield	ECM Yield
Milk yield						
Fat content	0.25 0.04 66					
Fat yield	0.82 < 0.01 66	0.72 < 0.01 66				
Protein Content	-0.22 0.07 66	0.16 0.19 66	-0.08 0.54 66			
Protein Yield	0.86 < 0.01 66	0.29 0.02 66	0.78 < 0.01 66	0.22 0.07 66		
ECM yield	0.91 < 0.01 66	0.56 < 0.01 66	0.97 < 0.01 66	-0.07 0.57 66	0.89 < 0.01 66	

¹LDP = Late dry period.²LDP BCS change = -1 wk BCS – BCS within 1 wk after moved into LDP.³Early lactation BCS change = 6 wk BCS - -1 wk BCS.⁴Correlation coefficient.⁵P-value.⁶Number of observations.

Table 4. Correlations, P-values and n amongst variable for parity 2 cows.

Variable	Days in LDP ¹	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change ²	Early lactation BCS change ³
Days in LDP						
-1 wk BCS	0.18 ⁴ 0.05 ⁵ 120 ⁶					
3 wk BCS	0.01 0.91 115	0.68 < 0.01 114				
6 wk BCS	-0.12 0.21 115	0.48 < 0.01 114	0.75 < 0.01 113			
LDP BCS change	0.24 0.01 120	0.32 < 0.01 120	0.14 0.14 114	0.02 0.79 114		
Early lactation BCS change	-0.24 0.01 113	-0.22 0.02 113	0.33 < 0.01 112	0.76 < 0.01 113	-0.22 0.02 113	
-8 d BHBA	-0.14 0.26 64	0.05 0.70 63	0.20 0.13 62	0.18 0.16 61	-0.34 0.01 63	0.20 0.13 60
-5 d BHBA	-0.18 0.11 83	0.10 0.37 83	0.19 0.09 80	0.11 0.33 79	-0.11 0.31 83	0.05 0.63 79
-2 d BHBA	-0.17 0.10 95	0.01 0.98 94	0.01 0.93 92	-0.05 0.63 94	-0.12 0.26 94	-0.05 0.63 92
2 d BHBA	0.02 0.86 112	0.06 0.52 111	-0.08 0.43 107	-0.11 0.28 107	-0.08 0.40 111	-0.16 0.10 105
7 d BHBA	0.17 0.08 113	0.17 0.07 112	0.01 0.90 108	-0.07 0.45 108	0.13 0.18 112	-0.22 0.02 106
14 d BHBA	0.01 1.00 110	0.12 0.23 109	0.07 0.49 108	0.02 0.83 108	-0.13 0.18 109	-0.06 0.54 106

Table 4. (cont.)

Variable	Days in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
-8 d NEFA	-0.03	-0.25	-0.22	-0.12	-0.06	0.06
	0.81	0.04	0.08	0.37	0.63	0.63
	64	63	62	61	63	60
-5 d NEFA	0.14	0.13	< 0.01	-0.18	0.06	-0.29
	0.22	0.24	0.99	0.12	0.62	0.01
	83	83	80	79	83	79
-2 d NEFA	0.29	0.13	-0.15	-0.32	0.03	-0.45
	< 0.01	0.22	0.14	< 0.01	0.75	< 0.01
	95	94	92	94	94	92
2 d NEFA	0.24	0.31	-0.03	-0.14	0.10	-0.38
	0.01	< 0.01	0.73	0.15	0.30	< 0.01
	112	111	107	107	111	105
7 d NEFA	0.20	0.29	0.01	-0.21	0.20	-0.46
	0.03	< 0.01	0.98	0.03	0.03	< 0.01
	113	112	108	108	112	106
14 d NEFA	0.20	0.16	-0.14	-0.27	0.07	-0.42
	0.03	0.11	0.16	< 0.01	0.48	< 0.01
	110	109	108	108	109	106
-8 d Insulin	-0.06	0.07	0.33	0.32	-0.03	0.31
	0.65	0.59	0.01	0.01	0.85	0.02
	63	62	61	60	62	59
-5 d Insulin	-0.05	0.10	0.26	0.26	0.04	0.24
	0.66	0.38	0.02	0.02	0.69	0.04
	82	82	79	78	82	78
-2 d Insulin	-0.09	0.06	0.32	0.25	0.02	0.23
	0.38	0.59	< 0.01	0.02	0.82	0.03
	94	93	91	93	93	91
2 d Insulin	-0.20	-0.04	0.17	0.29	-0.01	0.35
	0.04	0.67	0.07	< 0.01	0.92	< 0.01
	109	108	105	104	108	103
7 d Insulin	-0.22	-0.06	0.18	0.39	-0.03	0.48
	0.02	0.53	0.06	< 0.01	0.77	< 0.01
	113	112	108	108	112	106
14 d Insulin	-0.26	-0.02	0.22	0.38	0.01	0.44
	0.01	0.84	0.02	< 0.01	0.98	< 0.01
	108	107	106	106	107	104

Table 4. (cont.)

Variable	Days in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
Milk yield	0.10	-0.08	-0.29	-0.36	-0.06	-0.34
	0.34	0.41	< 0.01	< 0.01	0.56	< 0.01
	102	101	99	100	101	98
Fat content	-0.03	0.04	0.04	-0.06	-0.16	-0.11
	0.76	0.68	0.67	0.55	0.10	0.29
	102	101	99	100	101	98
Fat yield	0.02	0.01	-0.10	-0.22	-0.14	-0.26
	0.84	1.00	0.33	0.03	0.18	0.01
	102	101	99	100	101	98
Protein content	-0.01	0.18	0.14	0.09	0.11	-0.06
	0.89	0.08	0.18	0.39	0.26	0.55
	102	101	99	100	101	98
Protein yield	0.10	0.03	-0.21	-0.33	0.01	-0.41
	0.34	0.73	0.04	< 0.01	0.92	< 0.01
	102	101	99	100	101	98
ECM yield	0.05	-0.01	-0.16	-0.29	-0.12	-0.32
	0.63	0.90	0.11	< 0.01	0.24	< 0.01
	102	101	99	100	101	98

Table 4. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
-8 d BHBA						
-5 d BHBA	0.40 < 0.01 52					
-2 d BHBA	0.34 0.02 50	0.36 < 0.01 68				
2 d BHBA	0.27 0.04 61	0.21 0.06 80	0.27 0.01 91			
7 d BHBA	0.01 0.93 62	-0.01 0.92 80	0.18 0.08 92	0.16 0.11 109		
14 d BHBA	0.20 0.13 60	0.08 0.47 77	0.25 0.02 92	0.17 0.08 107	0.70 < 0.01 107	
-8 d NEFA	-0.05 0.70 64	0.10 0.48 52	-0.12 0.41 50	0.44 < 0.01 61	-0.03 0.81 62	-0.03 0.80 60
-5 d NEFA	-0.06 0.66 52	-0.07 0.50 83	-0.05 0.67 68	0.30 0.01 80	0.38 < 0.01 80	0.21 0.07 77
-2 d NEFA	0.32 0.02 50	0.01 0.94 68	0.01 0.94 95	0.27 0.01 91	0.23 0.03 92	0.21 0.05 92
2 d NEFA	0.05 0.69 61	-0.13 0.24 80	0.07 0.53 91	0.35 < 0.01 112	0.35 < 0.01 109	0.17 0.08 107
7 d NEFA	-0.26 0.04 62	-0.18 0.10 80	0.11 0.28 92	0.03 0.77 109	0.54 < 0.01 113	0.27 0.01 107
14 d NEFA	-0.08 0.53 60	-0.29 0.01 77	-0.01 0.94 92	0.17 0.09 107	0.26 0.01 107	0.25 0.01 110

Table 4. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
-8 d Insulin	0.26	-0.01	-0.03	-0.22	-0.07	0.12
	0.04	0.97	0.85	0.08	0.60	0.37
	63	51	50	60	61	59
-5 d Insulin	0.05	0.27	-0.03	-0.15	-0.21	-0.07
	0.70	0.01	0.81	0.19	0.07	0.56
	52	82	67	79	79	76
-2 d Insulin	-0.12	-0.05	0.08	-0.26	-0.17	-0.11
	0.40	0.69	0.45	0.02	0.10	0.29
	50	67	94	90	91	91
2 d Insulin	-0.16	0.02	-0.07	-0.05	-0.25	-0.14
	0.22	0.86	0.53	0.61	0.01	0.15
	59	78	89	109	106	104
7 d Insulin	0.25	0.21	-0.03	0.12	-0.17	-0.10
	0.05	0.07	0.81	0.20	0.07	0.30
	62	80	92	109	113	107
14 d Insulin	-0.02	0.03	-0.12	-0.11	-0.23	-0.09
	0.88	0.80	0.26	0.25	0.02	0.37
	59	76	90	105	105	108
Milk yield	-0.14	-0.22	-0.19	-0.13	-0.04	-0.04
	0.30	0.08	0.08	0.22	0.71	0.68
	53	66	83	95	97	96
Fat content	0.04	0.11	-0.13	0.03	0.07	0.20
	0.78	0.39	0.25	0.75	0.50	0.05
	53	66	83	95	97	96
Fat yield	-0.02	0.01	-0.17	-0.05	0.01	0.14
	0.87	0.98	0.12	0.66	0.92	0.19
	53	66	83	95	97	96
Protein Content	0.23	0.14	-0.02	0.03	0.02	0.10
	0.10	0.27	0.88	0.75	0.81	0.34
	53	66	83	95	97	96
Protein Yield	0.06	-0.12	-0.15	-0.13	-0.04	0.03
	0.66	0.35	0.18	0.22	0.67	0.76
	53	66	83	95	97	96
ECM yield	-0.03	-0.06	-0.18	-0.08	-0.01	0.11
	0.80	0.64	0.10	0.45	0.95	0.30
	53	66	83	95	97	96

Table 4. (cont.)

Variable	-8 d NEFA	-5 d NEFA	-2 d NEFA	2 d NEFA	7 d NEFA	14 d NEFA
-8 d NEFA						
-5 d NEFA	0.58 < 0.01 52					
-2 d NEFA	0.08 0.59 50	0.57 < 0.01 68				
2 d NEFA	0.31 0.02 61	0.46 < 0.01 80	0.47 < 0.01 91			
7 d NEFA	0.01 0.97 62	0.36 < 0.01 80	0.28 0.01 92	0.37 < 0.01 109		
14 d NEFA	-0.02 0.89 60	0.16 0.17 77	0.29 0.01 92	0.36 < 0.01 107	0.43 < 0.01 107	
-8 d Insulin	-0.36 < 0.01 63	-0.27 0.05 51	-0.10 0.49 50	-0.21 0.11 60	-0.03 0.80 61	0.02 0.86 59
-5 d Insulin	-0.29 0.03 52	-0.38 < 0.01 82	-0.26 0.04 67	-0.30 0.01 79	-0.25 0.03 79	-0.25 0.03 76
-2 d Insulin	-0.16 0.28 50	-0.29 0.02 67	-0.51 < 0.01 94	-0.35 < 0.01 90	-0.06 0.59 91	-0.22 0.03 91
2 d Insulin	-0.18 0.18 59	-0.22 0.05 78	-0.34 < 0.01 89	-0.41 < 0.01 109	-0.34 < 0.01 106	-0.34 < 0.01 104
7 d Insulin	0.04 0.73 62	-0.21 0.06 80	-0.24 0.02 92	-0.27 < 0.01 109	-0.44 < 0.01 113	-0.37 < 0.01 107
14 d Insulin	-0.09 0.50 59	-0.12 0.32 76	-0.16 0.14 90	-0.25 0.01 105	-0.28 < 0.01 105	-0.53 < 0.01 108

Table 4. (cont.)

Variable	-8 d NEFA	-5 d NEFA	-2 d NEFA	2 d NEFA	7 d NEFA	14 d NEFA
Milk yield	-0.01	0.16	0.01	-0.02	0.11	0.38
	0.94	0.19	0.95	0.87	0.28	< 0.01
	53	66	83	95	97	96
Fat content	-0.14	0.07	-0.06	0.03	-0.07	0.10
	0.32	0.59	0.57	0.78	0.51	0.33
	53	66	83	95	97	96
Fat yield	-0.18	0.10	-0.04	0.01	-0.01	0.26
	0.20	0.42	0.70	0.91	0.93	0.01
	53	66	83	95	97	96
Protein Content	0.02	0.07	0.02	0.04	-0.15	-0.19
	0.87	0.56	0.85	0.67	0.14	0.06
	53	66	83	95	97	96
Protein Yield	-0.03	0.20	0.02	-0.01	0.03	0.27
	0.83	0.10	0.86	0.95	0.78	0.01
	53	66	83	95	97	96
ECM yield	-0.16	0.14	-0.03	0.01	0.02	0.31
	0.26	0.26	0.80	0.98	0.83	< 0.01
	53	66	83	95	97	96

Table 4. (cont.)

Variable	-8 d Insulin	-5 d Insulin	-2 d Insulin	2 d Insulin	7 d Insulin	14 d Insulin
-8 d Insulin						
-5 d Insulin	0.55 < 0.01 51					
-2 d Insulin	0.59 < 0.01 50	0.55 < 0.01 66				
2 d Insulin	0.26 0.04 58	0.30 0.01 77	0.30 < 0.01 88			
7 d Insulin	0.38 < 0.01 61	0.28 0.01 79	0.20 0.05 91	0.62 < 0.01 106		
14 d Insulin	0.29 0.03 58	0.22 0.06 75	0.23 0.03 89	0.49 < 0.01 102	0.44 < 0.01 105	
Milk yield	-0.13 0.36 52	-0.18 0.15 65	-0.14 0.22 82	-0.13 0.20 92	-0.26 0.01 97	-0.25 0.02 94
Fat content	0.01 0.92 52	0.01 0.96 65	-0.08 0.47 82	-0.12 0.27 92	-0.17 0.10 97	-0.05 0.65 94
Fat yield	-0.03 0.82 52	-0.05 0.69 65	-0.11 0.35 82	-0.13 0.21 92	-0.24 0.02 97	-0.15 0.14 94
Protein Content	0.03 0.83 52	0.26 0.04 65	0.08 0.49 82	-0.02 0.84 92	0.03 0.75 97	-0.05 0.61 94
Protein Yield	-0.08 0.57 52	0.02 0.89 65	-0.04 0.75 82	-0.17 0.11 92	-0.25 0.01 97	-0.32 < 0.01 94
ECM yield	-0.06 0.67 52	-0.07 0.58 65	-0.11 0.33 82	-0.15 0.14 92	-0.27 0.01 97	-0.21 0.04 94

Table 4. (cont.)

Variable	Milk yield	Fat content	Fat yield	Protein content	Protein yield	ECM yield
Milk yield						
Fat content	0.15 0.12 102					
Fat yield	0.60 < 0.01 102	0.87 < 0.01 102				
Protein Content	-0.36 < 0.01 102	0.13 0.18 102	-0.08 0.45 102			
Protein Yield	0.80 < 0.01 102	0.23 0.02 102	0.57 < 0.01 102	0.23 0.02 102		
ECM yield	0.77 < 0.01 102	0.72 < 0.01 102	0.97 < 0.01 102	-0.11 0.27 102	0.74 < 0.01 102	

¹LDP = Late dry period.²LDP BCS change = -1 wk BCS – BCS within 1 wk after moved into LDP.³Early lactation BCS change = 6 wk BCS - -1 wk BCS.⁴Correlation coefficient.⁵P-value.⁶Number of observations.

Table 5. Correlations, P-values and n amongst variables in parity 3+ cows.

Variable	Days in LDP ¹	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
Days in LDP						
-1 wk BCS	-0.19 ² 0.02 ³ 161 ⁴					
3 wk BCS	-0.20 0.01 147	0.61 < 0.01 148				
6 wk BCS	-0.25 < 0.01 144	0.49 < 0.01 145	0.80 < 0.01 145			
LDP BCS change	-0.04 0.65 161	0.26 < 0.01 163	0.16 0.06 148	0.07 0.39 145		
Early lactation BCS change	-0.13 0.12 143	-0.20 0.02 145	0.45 < 0.01 144	0.76 < 0.01 145	-0.17 0.04 145	
-8 d BHBA	0.11 0.32 87	-0.07 0.51 87	-0.05 0.65 80	-0.06 0.59 79	0.08 0.47 87	-0.08 0.47 78
-5 d BHBA	-0.19 0.04 118	-0.04 0.66 118	-0.16 0.10 108	-0.05 0.61 106	-0.01 0.94 118	0.01 0.88 105
-2 d BHBA	0.15 0.10 122	-0.10 0.27 124	-0.19 0.04 115	-0.16 0.09 113	-0.13 0.14 124	-0.08 0.42 113
2 d BHBA	-0.01 0.92 151	0.11 0.17 152	-0.07 0.43 142	-0.07 0.44 138	-0.13 0.11 152	-0.12 0.18 137
7 d BHBA	-0.17 0.04 144	0.09 0.30 146	-0.16 0.06 140	-0.25 < 0.01 136	-0.08 0.36 146	-0.33 < 0.01 136
14 d BHBA	-0.09 0.28 139	0.18 0.04 141	-0.01 0.90 138	-0.17 0.05 134	-0.06 0.49 141	-0.33 < 0.01 134

Table 5. (cont.)

Variable	Days in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
-8 d NEFA	0.38	-0.23	-0.31	-0.29	-0.17	-0.19
	< 0.01	0.03	0.01	0.01	0.12	0.10
	87	87	80	79	87	78
-5 d NEFA	0.25	-0.05	-0.13	-0.15	0.03	-0.20
	0.01	0.60	0.17	0.11	0.72	0.04
	118	118	108	106	118	105
-2 d NEFA	0.17	0.07	-0.25	-0.26	-0.16	-0.31
	0.07	0.41	0.01	< 0.01	0.07	< 0.01
	122	124	115	113	124	113
2 d NEFA	0.01	0.12	-0.21	-0.28	0.01	-0.40
	0.90	0.13	0.01	< 0.01	0.91	< 0.01
	151	152	142	138	152	137
7 d NEFA	0.11	-0.01	-0.24	-0.35	0.06	-0.37
	0.17	0.90	< 0.01	< 0.01	0.47	< 0.01
	144	146	140	136	146	136
14 d NEFA	-0.01	0.18	-0.10	-0.22	0.02	-0.43
	0.89	0.03	0.23	0.01	0.80	< 0.01
	139	141	138	134	141	134
-8 d Insulin	-0.15	0.26	0.45	0.44	0.08	0.33
	0.18	0.02	< 0.01	< 0.01	0.45	< 0.01
	86	86	79	78	86	77
-5 d Insulin	-0.11	0.13	0.29	0.30	0.09	0.22
	0.25	0.17	< 0.01	< 0.01	0.32	0.02
	116	116	106	104	116	103
-2 d Insulin	-0.01	0.15	0.36	0.37	0.13	0.28
	0.88	0.10	< 0.01	< 0.01	0.16	< 0.01
	121	123	114	112	123	112
2 d Insulin	-0.15	0.19	0.39	0.41	0.10	0.32
	0.07	0.02	< 0.01	< 0.01	0.23	< 0.01
	150	151	141	137	151	136
7 d Insulin	-0.01	0.10	0.31	0.39	0.09	0.41
	0.87	0.25	< 0.01	< 0.01	0.30	< 0.01
	140	142	136	132	142	132
14 d Insulin	-0.06	-0.09	0.26	0.39	0.10	0.50
	0.48	0.31	< 0.01	< 0.01	0.25	< 0.01
	134	136	135	131	136	131

Table 5. (cont.)

Variable	Day in LDP	-1 wk BCS	3 wk BCS	6 wk BCS	LDP BCS change	Early lactation BCS change
Milk yield	-0.11	0.31	0.04	0.01	0.18	-0.24
	0.24	< 0.01	0.67	0.96	0.05	0.01
	126	127	127	126	127	126
Fat content	-0.13	0.27	0.10	0.05	0.06	-0.15
	0.15	< 0.01	0.25	0.59	0.50	0.09
	126	127	127	126	127	126
Fat yield	-0.15	0.33	0.08	0.02	0.11	-0.23
	0.09	< 0.01	0.38	0.83	0.21	0.01
	126	127	127	126	127	126
Protein content	0.15	-0.02	-0.03	-0.04	0.02	-0.03
	0.09	0.80	0.78	0.65	0.84	0.74
	126	127	127	126	127	126
Protein yield	-0.03	0.30	0.01	-0.03	0.13	-0.26
	0.70	< 0.01	0.93	0.73	0.15	< 0.01
	126	127	127	126	127	126
ECM yield	-0.15	0.34	0.06	0.01	0.12	-0.25
	0.10	< 0.01	0.49	0.93	0.16	0.01
	126	127	127	126	127	126

Table 5. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
-8 d BHBA						
-5 d BHBA	0.63 < 0.01 72					
-2 d BHBA	0.30 0.01 65	0.41 < 0.01 94				
2 d BHBA	0.22 0.04 86	0.26 < 0.01 116	0.36 < 0.01 121			
7 d BHBA	-0.01 0.96 81	0.02 0.85 110	0.31 < 0.01 118	0.27 < 0.01 144		
14 d BHBA	0.07 0.57 78	0.04 0.70 107	0.10 0.30 114	0.19 0.02 139	0.54 < 0.01 138	
-8 d NEFA	0.51 < 0.01 88	0.35 < 0.01 72	0.13 0.29 65	0.16 0.15 86	-0.04 0.75 81	< 0.01 0.97 78
-5 d NEFA	0.48 < 0.01 72	0.50 < 0.01 119	0.38 < 0.01 94	0.47 < 0.01 116	0.04 0.70 110	0.03 0.72 107
-2 d NEFA	0.14 0.28 65	0.25 0.01 94	0.53 < 0.01 124	0.44 < 0.01 121	0.32 < 0.01 118	0.33 < 0.01 114
2 d NEFA	0.02 0.86 86	0.16 0.09 116	0.17 0.06 121	0.22 0.01 153	0.29 < 0.01 144	0.07 0.39 139
7 d NEFA	-0.08 0.45 81	0.02 0.86 110	0.20 0.03 118	0.14 0.10 144	0.38 < 0.01 146	0.27 < 0.01 138
14 d NEFA	0.09 0.42 78	-0.03 0.74 107	0.04 0.66 114	0.10 0.23 139	0.27 < 0.01 138	0.45 < 0.01 141

Table 5. (cont.)

Variable	-8 d BHBA	-5 d BHBA	-2 d BHBA	2 d BHBA	7 d BHBA	14 d BHBA
Insulin -8	0.04 0.70 87	-0.18 0.13 71	-0.20 0.11 64	-0.04 0.69 85	-0.15 0.19 80	0.01 0.98 77
Insulin -5	-0.04 0.74 71	-0.08 0.38 117	-0.19 0.07 93	-0.15 0.12 114	-0.12 0.23 108	0.06 0.53 105
Insulin -2	-0.11 0.40 64	-0.03 0.79 93	-0.14 0.14 123	0.01 0.92 120	-0.14 0.13 117	-0.06 0.51 114
2 d Insulin	0.06 0.62 85	0.04 0.68 115	-0.09 0.35 120	-0.12 0.13 152	-0.19 0.02 143	-0.11 0.21 138
7 d Insulin	0.05 0.64 78	-0.05 0.63 107	-0.09 0.32 115	-0.06 0.48 140	-0.36 < 0.01 142	-0.30 < 0.01 134
14 d Insulin	0.03 0.82 76	0.03 0.80 103	0.01 0.98 110	-0.08 0.38 134	-0.26 < 0.01 134	-0.33 < 0.01 136
Milk yield	-0.16 0.22 65	-0.22 0.03 92	-0.19 0.06 101	-0.26 < 0.01 120	-0.18 0.05 120	-0.09 0.36 118
Fat content	-0.05 0.72 65	0.03 0.76 92	0.06 0.57 101	0.12 0.18 120	0.05 0.62 120	0.21 0.02 118
Fat yield	-0.12 0.33 65	-0.04 0.70 92	-0.08 0.43 101	-0.08 0.38 120	-0.08 0.38 120	0.08 0.41 118
Protein Content	-0.22 0.08 65	-0.09 0.39 92	-0.07 0.49 101	0.02 0.86 120	0.01 0.96 120	-0.01 0.94 118
Protein Yield	-0.31 0.01 65	-0.19 0.06 92	-0.21 0.03 101	-0.24 0.01 120	-0.17 0.07 120	-0.08 0.42 118
ECM yield	-0.17 0.18 65	-0.08 0.43 92	-0.12 0.24 101	-0.14 0.13 120	-0.11 0.24 120	0.04 0.70 118

Table 5. (cont.)

Variable	-8 d NEFA	-5 d NEFA	-2 d NEFA	2 d NEFA	7 d NEFA	14 d NEFA
-8 d NEFA						
-5 d NEFA	0.58 < 0.01 72					
-2 d NEFA	0.40 < 0.01 65	0.62 < 0.01 94				
2 d NEFA	0.41 < 0.01 86	0.34 < 0.01 116	0.38 < 0.01 121			
7 d NEFA	0.16 0.15 81	0.19 0.05 110	0.41 < 0.01 118	0.40 < 0.01 144		
14 d NEFA	0.34 < 0.01 78	0.27 < 0.01 107	0.33 < 0.01 114	0.28 < 0.01 139	0.37 < 0.01 138	
-8 d Insulin	-0.48 < 0.01 87	-0.47 < 0.01 71	-0.42 < 0.01 64	-0.48 < 0.01 85	-0.24 0.03 80	-0.24 0.04 77
-5 d Insulin	-0.40 < 0.01 71	-0.45 < 0.01 117	-0.44 < 0.01 93	-0.26 0.01 114	-0.32 < 0.01 108	-0.15 0.12 105
-2 d Insulin	-0.24 0.06 64	-0.25 0.01 93	-0.52 < 0.01 123	-0.33 < 0.01 120	-0.25 0.01 117	-0.14 0.15 114
2 d Insulin	-0.33 < 0.01 85	-0.28 < 0.01 115	-0.28 < 0.01 120	-0.46 < 0.01 152	-0.33 < 0.01 143	-0.33 < 0.01 138
7 d Insulin	-0.10 0.40 78	-0.13 0.18 107	-0.28 < 0.01 115	-0.34 < 0.01 140	-0.42 < 0.01 142	-0.32 < 0.01 134
14 d Insulin	-0.22 0.05 76	-0.18 0.07 103	-0.28 < 0.01 110	-0.26 < 0.01 134	-0.38 < 0.01 134	-0.38 < 0.01 136

Table 5. (cont.)

	-8 d	-5 d	-2 d	2 d	7 d	14 d
Variable	NEFA	NEFA	NEFA	NEFA	NEFA	NEFA
Milk yield	-0.15	-0.20	-0.13	0.04	-0.08	0.13
	0.22	0.05	0.18	0.64	0.37	0.15
	65	92	101	120	120	118
Fat content	-0.20	-0.10	-0.03	0.03	-0.13	0.08
	0.10	0.34	0.74	0.74	0.16	0.41
	65	92	101	120	120	118
Fat yield	-0.21	-0.16	-0.10	0.04	-0.14	0.13
	0.09	0.12	0.32	0.63	0.13	0.15
	65	92	101	120	120	118
Protein Content	0.17	0.05	0.13	0.08	-0.01	0.03
	0.17	0.61	0.18	0.38	0.89	0.74
	65	92	101	120	120	118
Protein Yield	0.02	-0.11	0.01	0.13	-0.07	0.20
	0.85	0.29	0.92	0.15	0.42	0.03
	65	92	101	120	120	118
ECM yield	-0.18	-0.17	-0.08	0.06	-0.12	0.16
	0.16	0.10	0.41	0.50	0.18	0.09
	65	92	101	120	120	118

Table 5. (cont.)

Variable	-8 d Insulin	-5 d Insulin	-2 d Insulin	2 d Insulin	7 d Insulin	14 d Insulin
-8 d Insulin						
-5 d Insulin	0.69 < 0.01 70					
-2 d Insulin	0.37 < 0.01 63	0.71 < 0.01 92				
2 d Insulin	0.48 < 0.01 84	0.42 < 0.01 113	0.37 < 0.01 119			
7 d Insulin	0.21 0.06 77	0.20 0.04 105	0.29 < 0.01 114	0.38 < 0.01 139		
14 d Insulin	0.34 < 0.01 75	0.29 < 0.01 101	0.32 < 0.01 110	0.49 < 0.01 133	0.54 < 0.01 130	
Milk yield	0.06 0.66 64	0.03 0.76 90	-0.07 0.46 100	-0.13 0.17 119	-0.06 0.53 116	-0.18 0.06 117
Fat content	0.17 0.18 64	0.35 < 0.01 90	0.16 0.11 100	0.05 0.60 119	0.03 0.73 116	-0.13 0.17 117
Fat yield	0.10 0.41 64	0.25 0.02 90	0.05 0.65 100	-0.04 0.68 119	-0.03 0.72 116	-0.19 0.04 117
Protein Content	-0.06 0.65 64	0.17 0.11 90	0.08 0.41 100	0.01 1.00 119	0.03 0.78 116	-0.09 0.35 117
Protein Yield	-0.01 0.92 64	0.15 0.17 90	-0.04 0.71 100	-0.15 0.11 119	-0.08 0.39 116	-0.27 < 0.01 117
ECM yield	0.08 0.52 64	0.22 0.04 90	0.01 0.92 100	-0.07 0.42 119	-0.06 0.55 116	-0.22 0.02 117

Table 5. (cont.)

Variable	Milk yield	Fat content	Fat yield	Protein content	Protein yield	ECM yield
Milk yield						
Fat content	0.31 < 0.01 127					
Fat yield	0.77 < 0.01 127	0.82 < 0.01 127				
Protein Content	-0.38 < 0.01 127	0.14 0.12 127	-0.18 0.04 127			
Protein Yield	0.78 < 0.01 127	0.39 < 0.01 127	0.69 < 0.01 127	0.21 0.02 127		
ECM yield	0.86 < 0.01 127	0.70 < 0.01 127	0.98 < 0.01 127	-0.19 0.03 127	0.81 < 0.01 127	

¹LDP = Late dry period.²LDP BCS change = -1 wk BCS – BCS within 1 wk after moved into LDP.³Early lactation BCS change = 6 wk BCS - -1 wk BCS.⁴Correlation coefficient.⁵P-value.⁶Number of observations.

CHAPTER 6

DISCUSSION AND IMPLICATIONS

Corn Grain Supplementation in the Late Dry Period

The main hypothesis of partially replacing corn silage with corn grain was that it would improve energy status of periparturient dairy cows. The SC diet reduced plasma BHBA concentrations and tended to increase insulin concentrations prepartum. However, there was no change in plasma NEFA concentrations during the same time. Plasma NEFA are substrates for ketone body synthesis. NEFA are extracted from the blood by the liver at a rate proportional to plasma NEFA concentrations (Bruss et al. 1993). If ketone body synthesis was less in cows fed the SC diet, what happened to the additional NEFA in cows fed the SC diet? In hepatic tissue, NEFA can be oxidized completely to CO_2 and H_2O , partially to ketones or re-esterified as TG (Bruss, 1993). TG can either be exported as VLDL or stored in hepatic tissue. Excess storage of TG in hepatic tissue can result in fatty liver and reduced hepatic function (Strang et al., 1998). The other possible alternative is that plasma NEFA were not extracted from blood by the liver at equal rates between treatments. However, no research supports this possibility. If complete oxidization accounted for the remaining NEFA in cows fed the SC diet, then the decrease in ketones may have been beneficial to cows. However, if remaining NEFA were re-esterified to TG and stored in the liver, then the reduction in ketone production may have been detrimental to hepatic function.

This study identified several interactions involving parity. In general, it appeared that parity 3+ cows benefited from the SC diet, whereas the SC diet had no or negative effects on parity 1 and 2 cows. It is possible that parity 3+ cows benefited the most from the SC diet because they had the largest metabolic demands in early lactation. In general and in this experiment, cows of parity 3+ yielded more milk than those of parities 1 and 2 and therefore, likely had increased energy requirements. Generally, DMI increases to help satisfy nutritional demands, but DMI in early lactation may be compromised. Therefore, improving energy status of more mature, higher yielding cows may show more benefits than for less mature, lower yielding cows. However, the data does not fully support this interpretation. If plasma NEFA concentrations postpartum are used to indicate metabolic demands and energy status, then parity 1 cows were in more negative energy status in early lactation. Dyk (1995) also reported that parity 1 cows had higher plasma NEFA concentrations prepartum compared with any other parity. Interestingly, NEFA concentrations in cows of parity 2 and 3+ peaked at 2 d, whereas plasma NEFA of parity 1 cows peaked at 7 d. Furthermore, parity 1 cows had increased BHBA concentrations in early lactation compared with parities 2 and 3+. Although liver tryglyceride were not measured, it would have been interesting to know how parities differed in TG accumulation in hepatic tissue. The cause of differences observed in treatment effects on parity is not evident, but cows of different parities appear to be in different physiological states during the periparturient period.

The results of this experiment do not fully support the original hypothesis of improving energy status by supplementing corn grain in the late dry period. One reason could be the smaller than expected differences observed in chemical composition of diets.

Diets were formulated to be substantially different in NFC and energy contents, but analysis of TMR samples taken weekly throughout the experiment showed that diets were more similar than originally formulated. Perhaps, treatment effects would have been more evident if corn grain replaced a larger percentage of corn silage in the SC diet. Results also showed that the effects of treatment varied depending upon parity. Most nutritional studies involving late pregnant dry cows have not considered or addressed differences in parities. Future research should characterize these differences and the possible mechanisms causing the differences.

Implications of this research are that increasing the dietary corn grain content during the late dry period will benefit cows of parity 3+. However, increasing the corn grain content fed to cows of parity 1 and 2 during the late dry period may not be advantageous. However, differing responses of parity to treatment may only apply to cows in the farm studied.

Altering Length of the Late Dry Period

One hypothesis for increasing the length of the late dry period was that cows would gain more body condition during this time. Cows in L tended to have greater gains in BCS in the late dry period compared with cows in S. However, the differences observed in BCS changes prepartum were small (0.08 and 0.14 for S and L, respectively). It is unlikely that the small BCS changes observed between treatments had any physiological impact on postpartum energy status, health, or production. It was surprising that feeding a diet with higher energy and nutrient densities for longer than 26 d prepartum did not result in greater BCS changes prepartum. Several studies have

reported large increases in BCS changes prepartum when cows are fed a higher energy diet during the entire dry period (Boisclair et al., 1986, Fronk et al., 1980). Perhaps cows needed to be fed the higher energy diet during the entire dry period to promote appreciable body condition change. Additionally, cows with lower BCS upon entering the late dry period may have gained BCS more readily. BCS prepartum averaged 3.80, 3.57, and 3.63 in cows of parity 1, 2 and 3+, respectively. BCS gains in the late dry period were 0.11, 0.20, and 0.01 in cows of parity 1, 2, and 3+, respectively. Indeed, parity 2 cows gained the most BCS prepartum, but parity 1 cows had greater increases in BCS compared with parity 3+ cows.

A second hypothesis was that increasing length of the late dry period would improve energy status in the periparturient period. DMI was not measured and therefore, energy intake and balance could not be calculated in this study. However, the combination of blood variables, as proxies for energy status, and BCS change postpartum can be used as indicators of energy status. Cows in L tended to have lower plasma NEFA and higher insulin concentrations postpartum, and reduced BCS loss from parturition to 3 wk postpartum compared with cows in S. This suggests that lipolysis was reduced (as measured by plasma NEFA) in cows in L, perhaps a result of increased plasma insulin concentrations. Ruminal papillae may have been more developed and allowed for increased VFA absorption in early lactation and improved energy status. As mentioned in Chapter 3, improvements in energy status in early lactation may spare amino acids needed for gluconeogenesis, thereby supplying the mammary gland with additional substrates for protein synthesis. Increased VFA removal from the rumen also may have improved yields of microbial protein that could have contributed to amino acid

availability to the mammary gland. Indeed, cows in L had higher milk protein content during the first 60 DIM compared with cows in S.

The differences observed between farms in milk production, health, and reproduction are likely caused by the differences in management between farms. Although just speculation, differences in DMI both pre- and postpartum could have contributed to the observed discrepancies between farms. The diets fed pre- and postpartum were similar in ingredient composition and chemical analysis, but of course these values do not account for feed bunk management and DMI which may have varied between farms. Additionally, the diets fed during the early dry period or during late lactation could have influenced our findings. However, the diets fed during the early dry period (i.e., prior to treatment diets) were similar in chemical composition. Therefore, it is unlikely that diets fed during the early dry period biased the results of this study.

Further research should measure DMI and investigate changes in hepatic metabolism of energy substrates. Changing the length of time cows are fed a higher energy diet could influence enzymes and receptors in both hepatic and adipose tissue. More basic research should try to identify these mechanisms and the time it takes to modify or regulate the specific enzymes or receptors.

The implications of this research are that feeding more nutrient-dense diets during the late dry period may improve energy balance in early lactation. However, several studies have shown detrimental effects of excessive BCS at parturition. Therefore, BCS should be considered before feeding to promote gains in body condition during the dry period. For example, cows entering the dry period with BCS below 3.25 may benefit from body condition gain during the dry period. The negative effects of lengthening the

late dry period observed in Farm 1 are probably a result of management and are unlikely to occur on all farms; similar effects were not detected in Farm 2. Manipulation of management or nutritional strategies prepartum will elicit effects that should be most evident in early lactation. Therefore, the present study showed that increasing the length of the late dry period was more advantageous than late dry periods of the traditional 2 to 3 wk in length.

Correlations Among Variables

Increased BCS at parturition was correlated positively with milk yield and composition in parity 3+ cows only. This suggests that cows of parity 3+ rely more heavily upon adipose tissue reserves as an energy source in early lactation compared with cows of parities 1 and 2. Indeed, parity 3+ cows had higher plasma NEFA concentrations postpartum compared with parity 2 cows, but had lower NEFA concentrations compared with parity 1 cows. The underlying question is what happened to the NEFA in parity 1 cows if they were not being used for milk or milk fat production? Future research should answer this question and identify these mechanisms.

The other surprising correlation was with plasma BHBA in parity 2 cows. Unlike parity 1 and 3+ cows, plasma BHBA concentrations prepartum were not correlated with NEFA concentrations prepartum. In addition, plasma concentrations of insulin and BHBA postpartum were not correlated in cows of parity 2, but were correlated negatively in parity 1 and 3+ cows. NEFA can be used as substrates for ketone synthesis. Therefore, increased NEFA concentrations should result in higher plasma BHBA concentrations. Indeed, plasma NEFA concentrations were correlated positively with

BHBA concentrations throughout the periparturient period in parity 1 and 3+ cows.

Perhaps, parity 2 cows had increased hepatic capabilities to oxidize NEFA or export them as VLDL. This might explain the lower plasma BHBA concentrations postpartum in cows of parity 2. However, parity 2 cows also had lower plasma NEFA concentrations during the periparturient period. Maybe the plasma NEFA concentrations were low enough so that the liver and extra-hepatic tissue could metabolize them without resorting to ketone synthesis. This also may explain why plasma insulin concentrations had no antagonistic effects on plasma BHBA concentrations postpartum. Reducing plasma NEFA concentrations via insulin would not cause reductions in BHBA if a substantial amount of NEFA are not being metabolized to BHBA. Future research needs to identify the exact mechanisms causing the differences in energy metabolism among parities.

The implications of this analysis are that cows of different parities are in different metabolic states during the periparturient period, and need to be managed and fed accordingly. Unfortunately, the exact optimal ways of feeding and managing cows based on differences in metabolism have not been elucidated fully.

LITERATURE CITED

- Allen, M. S., and D. K. Beede. 1996. Causes, detection and prevention of ruminal acidosis in dairy cattle. Tri-States Dairy Nutr. Conf. p. 55-72.
- Bell, A. W. 1980. Lipid metabolism in the liver and selected tissues and the whole body of ruminant animals. Prog. Lipid Res. 18:117-164.
- Bell, A. 1995. Regulation of organic nutrient metabolism during transition from late pregnancy to early lactation. J. Anim. Sci. 73:2804-2819.
- Bertics, J. S., R. R. Grummer, C. Cadorniga-Valino, and E. E. Stoddard. 1992. Effect of prepartum dry matter intake on liver triglyceride concentration and early lactation. J. Dairy Sci. 75:1914-1922.
- Boisclair, Y., D. G. Grieve, J. B. Stone, O. B. Allen, and G. K. Macleod. 1986. The effect of prepartum energy intake, body condition and sodium bicarbonate on production in early lactation. J. Dairy Sci. 69:2636-2647.
- Boisclair, Y., D. G. Grieve, O. B. Allen, and R. A. Curtis. 1987. Effect of prepartum energy, body condition, and sodium bicarbonate on health and blood metabolites of Holstein cows in early lactation. J. Dairy Sci. 70:2280-2290.
- Boutflour, R. B. 1928. Limiting factors in the feeding and management of milk cows. Rep. World's Dairy Congress. p.15.
- Bruss, M. L. 1993. Metabolic fatty liver in ruminants. Adv. Vet. Sci. Comp. Med. 37:417-449.
- Butler, W. R., and R. D. Smith. 1989. Interrelationships between energy balance and postpartum reproductive function in dairy cattle. J. Dairy Sci. 72:767-783.
- Cameron, R. E. B., P. B. Dyk, T. H. Herdt, J. B. Kaneene, R. Miller, H. F. Bucholtz, J. S. Liesman, M. J. VandeHaar, and R. S. Emery. 1998. Dry cow diet, management, and energy balance as risk factors for displaced abomasum in high producing dairy herds. J. Dairy Sci. 81:132-139.
- Curtis, C. R., H. N. Erb, C. J. Sniffen, R. D. Smith, and D. S. Kronfeld. 1985. Path analysis of dry period nutrition, postpartum metabolic and reproductive disorders, and mastitis in Holstein cows. J. Dairy Sci. 68:2347-2360.
- Davenport, D. G., and A. H. Rakes. 1969. Effects of prepartum feeding level and body condition on the postpartum performance of dairy cows. J. Dairy Sci. 52:1037-1043.
- Davis, A. J., O. R. Fleet, J. A. Goode, M. H. Hamon, F. M. Maule Walker, and M.

- Peaker. 1979. Changes in mammary function at the onset of lactation in the goat: correlation with hormonal changes. *J. Physiol.* 288:33-44.
- Dairy Records Management Systems. DHI Glossary. 1999. Fact sheet: A-4:9. DRMS, 313 Chapanoke Rd. Suite 100, Raleigh, NC 27603.
- Dirksen, G. U., H. G. Liebich, and E. Mayer. 1985. Adaptive changes of the ruminal mucosa and their functional and clinical significance. *Bov. Pract.* 20:116-120.
- Domecq, J. J., A. L. Skidmore, J. W. Lloyd, and J. B. Kaneene. 1997. Relationship between body condition scores and milk yield in a large dairy herd of high yielding Holstein cows. *J. Dairy Sci.* 80:101-112.
- Dyk, P. B., R. S. Emery, J. L. Liesman, H. F. Bucholtz, and M. J. VandeHaar. 1995. Prepartum non-esterified fatty acids in plasma are higher in cows developing periparturient health problems. *J. Dairy Sci.* 78(Suppl. 1):337.
- Dyk, P. B. 1995. The association of prepartum non-esterified fatty acids and body condition with peripartum health problems on 95 Michigan dairy farms. M. S. Thesis. Michigan State University.
- Emery, R. S., H. D. Hafs, D. Armstrong, and W. W. Snyder. 1969. Prepartum grain feeding effects on milk production, mammary edema, and incidence of diseases. *J. Dairy Sci.* 52:345-351.
- Emery, R. S., J. S. Liesman, and T. H. Herdt. 1992. Metabolism of long chain fatty acids by ruminant liver. *J. Nutr.* 122:832-837.
- Erb, R. E., P. E. Malven, T. S. Stewart, C. N. Zamet, and B. P. Chew. 1982. Relationship of hormones, temperature, photoperiod, and other factors to involuntary intake of dry matter in pregnant dairy cows prior to parturition. *J. Dairy Sci.* 65:937.
- Erskine, R. J., R. J. Eberhart, L. J. Huthcinson, S. B. Spencer, and M. A. Campbell. 1988. Incidence and types of clinical mastitis in dairy herds with high and low somatic cell counts. *J. Am. Vet. Med. Assoc.* 192:761-765.
- Fountaine, F. C., D. B. Parrish, and F. W. Atkeson. 1949. Comparison of the incidence and severity of mammary edema of cows fed roughages alone or roughages plus grain during the dry period. *J. Dairy Sci.* 32:721-726.
- Fronk, T. J., L. H. Schultz, and A. R. Hardie. 1980. Effect of dry period over conditioning on subsequent metabolic disorders and performance of dairy cows. *J. Dairy Sci.* 63:1080-1090.
- Gardner, R. W. 1969. Interactions of energy levels offered to Holstein cows prepartum and postpartum. I. Production responses and blood composition changes. *J. Dairy Sci.*

52:1973-1984.

Garnsworthy, P. C., and J. H. Topps. 1982. The effect of body condition of dairy cows at calving on their food intake and performance when given complete diets. *Anim. Prod.* 35:113-119.

Garnsworthy, P. C., and C. D. Huggett. 1992. The influence of the fat concentration of the diet on the response by dairy cows to body condition at calving. *Anim. Prod.* 54:7-13.

Garnsworthy, P. C., and G. P. Jones. 1987. The influence of body condition at calving and dietary protein supply on voluntary food intake and performance in dairy cows. *Anim. Prod.* 44:347-353.

Gaynor, P. J., D. R. Waldo, A. V. Capuco, R. R. Erdman, L. W. Douglass, and B. B. Teter. 1995. Milk fat depression, the glucogenic theory, and trans-C18:1 fatty acids. *J. Dairy Sci.* 78:2008-2015.

Gearhart, M. A., C. R. Curtis, H. N. Erb, R. D. Smith, C. J. Sniffen, L. E. Chase, and M. D. Cooper. 1990. Relationship of changes in condition score to cow health in Holsteins. *J. Dairy Sci.* 73:3132-3140.

Goff, J. P., and R. L. Horst. 1997. Physiological changes at parturition and their relationship to metabolic disorders. *J. Dairy Sci.* 1260-1268.

Greenhalgh, J. F. D., and K. E. Gardner. 1958. Effects of heavy concentrate feeding before calving upon lactation and mammary gland edema. *J. Dairy Sci.* 41:822-829.

Grum, D. E., J. K. Drackley, R. S. Younker, D. W. Lacount, and J. J. Veenhuizen. 1996. Nutrition during the dry period and hepatic lipid metabolism of periparturient dairy cows. *J. Dairy Sci.* 79:1850-1864.

Grummer, R. R. 1993. Etiology of lipid-related metabolic disorders in periparturient dairy cows. *J. Dairy Sci.* 76:3882-3896.

Grummer, R. R. 1995. Impact of changes in organic nutrient metabolism on feeding the transition dairy cow. *J. Anim. Sci.* 73:2820-2833.

Hathaway, H. D., W. J. Brakel, W. J. Tyznik, and H. E. Kaeser. 1957. The effect of concentrate intake at calving time on physiological activities with special emphasis on ketosis. *J. Dairy Sci.* 40:616-623.

Hayirli, A., R. R. Grummer, E. Nordheim, P. Crump, D. K. Beede, M. J. VandeHaar and L. H. Kilmer. 1998. A mathematical model for describing dry matter intake of transition dairy cows. *J. Dairy Sci.* 81(Suppl. 1):296.

Holtenius, P. 1989. Plasma lipids in normal cows around partus and in cows with metabolic disorders with and without fatty liver. *Acta. Vet. Scand.* 30:441-445.

Holtenius, P. 1993. Hormonal regulation related to the development of fatty liver and ketosis. *Acta. Vet. Scand.* 89(Suppl. 1):55-60.

Holtenius, P., G. Olsson, and C. Bjorkman. 1993. Periparturient concentrations of insulin, glucagon and ketone bodies in dairy cows fed two different levels of nutrition and varying concentrate/roughage ratios. *J. Vet. Med. A.* 40:118-127.

Holtenius, P, G. Olsson, M. Emanuelson, and H. Wiktorsson. 1996. Effects of different energy levels, concentrate/forage ratios and lipid supplementation to the diet on the adaptation of the energy metabolism at calving in dairy cows. *J. Vet. Med. A.* 43:427-435.

Holter, J. B., M. J. Slotnick, H. H. Hayes, C. K. Bozak, W. E. Urban, Jr., and M. L. McGilliard. 1990. Effect of prepartum dietary energy on condition score, postpartum energy, nitrogen partitions, and lactation production responses. *J. Dairy Sci.* 73:3502-3511.

Johnson, D. G., and D. E. Otterby. 1981. Influence of dry period diet on early postpartum health, feed intake, milk production, and reproductive efficiency of Holstein cows. *J. Dairy Sci.* 64:290-295.

Johnson, T. R., and D. K. Combs. 1991. Effects of prepartum diet, inert rumen bulk, and dietary polyethylene glycol on dry matter intake of lactating dairy cows. *J. Dairy Sci.* 74:933-944.

Jones, G. P., and P. C. Garnsworthy. 1989. The effect of dietary energy content on the response of dairy cows to body condition at calving. *Anim. Prod.* 49:183-191.

Keys, J. E., R. E. Pearson, N. W. Hooven, H. F. Tyrrell, and G. W. Bodoh. 1983. Individual vs. group feeding of constant vs. variable forage:concentrate of total mixed rations throughout two lactations and intervening dry period. *J. Dairy Sci.* 66:1076-1083.

Keys, J. E., R. E. Pearson, and R. H. Miller. 1984. Effect of ratio of corn silage to grass-legume with high concentrate during dry period on milk production and health of dairy cows. *J. Dairy Sci.* 67:307-312.

Kimura, K., J. P. Goff, M. E. Kerhli Jr., J. A. Harp, and B. J. Nonnecke. 1997. Effect of mastectomy on phenotype and function of leukocytes in periparturient dairy cows. *J. Dairy Sci.* 80(Suppl. 1):264.

Kunz, P. L., J. W. Blum, I. C. Hart, H. Bickel, and J. Landis. 1988. Effects of different energy intakes before and after calving on food intake, performance and blood hormones

and metabolites in dairy cows. *Anim. Prod.* 40:219-231.

Lammers, B. P., D. R. Buckmaster, and A. J. Heinrichs. 1996. A simple method for the analysis of particle sizes of forage and total mixed rations. *J. Dairy Sci.* 79:922-928.

Lowe, D. M. and P. K. Tubbs. 1985. Succinylation and inactivation of 3-hydroxy-3-methylglutaryl-CoA synthase by succinyl-CoA and its possible relevance to the control of ketogenesis. *Biochem. J.* 232:37-42.

Mackie, R.I., and F. M. C. Gilchrist. 1979. Changes in lactate-producing and lactate-utilizing bacteria in relation to pH hydrogen-ion concentration in the rumen of sheep during stepwise adaptation to a high-concentrate diet. *Appl. Env. Micro.* 67:422-430.

McGuire, M. A., J. M. Griinari, D. A. Dwyer, and D. E. Bauman. 1995. Role of insulin in the regulation of mammary synthesis of fat and protein. *J. Dairy Sci.* 78:816-824.

Michigan State University Dairy Programs Group. 1996. In: *Managing the dry cow for more profit*, Michigan State University, East Lansing.

Minor, D. J., S. L. Trower, B. D. Strang, R. D. Shaver, and R. R. Grummer. 1998. Effects of nonfiber carbohydrates and niacin on periparturient metabolic status and lactation of dairy cows. *J. Dairy Sci.* 81:189-200.

Moe, P. W., and H. F. Tyrell. Metabolizable energy requirements of pregnant dairy cows. *J. Dairy Sci.* 55:480-483.

Morrow, D. A. 1976. Nutritional health program for high producing dairy herds. *Bovine Pract.* 11:16-23.

Morrow, D. A., D. Hillman, A. W. Dade, and H. Kitchen. 1979. Clinical investigation of a dairy herd with the fat cow syndrome. *J. Am. Vet. Med. Assoc.* 174:161-167.

Nachtomi, E., S. Eger, S. Amir, and H. Schindler. 1986. Postpartum nonesterified fatty acids concentration in blood plasma of dairy cows fed different energy levels prepartum. *Nutr. Rep. Int.* 34:521-527.

Nebel, R. L., and M. L. McGilliard. 1993. Interactions of high milk yield and reproductive performance in dairy cows. *J. Dairy Sci.* 76:3257-3268.

Nocek, J. E., J. E. English, and D. G. Braund. 1983. Effects of various forage feeding programs during the dry period on body condition and subsequent lactation, health, production, and reproduction. *J. Dairy Sci.* 66:1108-1118.

Nocek, J. E., R. L. Steele, and D. G. Braund. 1986. Prepartum grain feeding and subsequent lactation forage program effects on performance of dairy cows in early lactation. *J. Dairy Sci.* 69:734-744.

NRC. 1989. Nutrient Requirements of Dairy Cattle (6th Rev. Ed.). National Academy Press, Washington DC.

Olsson, G., M. Emanuelson, and H. Wiktorsson. 1997. Effects on milk production and health of dairy cows by feeding different ratios of concentrate/forage and additional fat before calving. *Acta. Agric. Scand. Sect. A. Anim Sci.* 47:91-105.

Pedron, O., F. Cheli, E. Senatore, D. Baroli, and R. Rizzi. 1993. Effect of body condition score at calving on performance, some blood parameters, and milk fatty acid composition in dairy cows. *J. Dairy Sci.* 76:2528-2535.

Perkins, B. L. 1982. Production, reproduction, health and liver function following overconditioning in dairy cattle. PhD. Thesis. Cornell University.

Pursley, J. R., M. R. Kosorok, and M. C. Wiltbank. 1997. Reproductive management of lactating dairy cows using synchronization of ovulation. *J. Dairy Sci.* 80:301-306.

Roseler, D. K., D. G. Fox, A. N. Pell, and L. E. Chase. 1997. Evaluation of alternative equations for prediction of intake for Holstein dairy cows. *J. Dairy Sci.* 80:864-877.

Ruegg, P. L., and R. L. Milton. 1995. Body condition scores of Holstein cows on Prince Edward Island, Canada: relationship with yield, reproductive performance, and disease. *J. Dairy Sci.* 78:552-564.

SAS® User's Guide: Statistics, Version 6 Edition. 1989. SAS Inst., Inc., Cary, NC.

Sakata, S., and H. Tamate. 1978. Rumen epithelial cell proliferation accelerated by rapid increase in intraruminal butyrate. *J. Dairy Sci.* 61:1109-1113.

Schmidt, G. H., and L. H. Schultz. 1959. Effect of three levels of grain feeding during the dry period on the incidence of ketosis, severity of udder edema, and subsequent milk production of dairy cows. *J. Dairy Sci.* 42:170-179.

Shaver, R. D. 1997. Nutritional risk factors in the etiology of left displaced abomasum in dairy cows: A review. *J. Dairy Sci.* 80:2449-2453.

Smith, T. R., A. R. Hippen, D. C. Beitz, and J. W. Young. 1997. Metabolic characteristics of induced ketosis in normal and obese cows. *J. Dairy Sci.* 80:1569-1581.

Staples, C. R., W. W. Thatcher, and J. H. Clark. 1990. Relationship between ovarian activity and energy status during the early postpartum period of high producing dairy cows. *J. Dairy Sci.* 73:938-947.

Swanson, E. W., and S. A. Hinton. 1962. Effects of adding concentrates to ad libitum roughage feeding in the dry period. *J. Dairy Sci.* 45:48-54.

Treacher, R. J., I. M. Reid, and C. J. Roberts. 1986. Effect of body condition at calving on the health and performance of dairy cows. *Anim. Prod.* 43:1-6.

VandeHaar, M. J., B. K. Sharma, G. Yousif, T. H. Herdt, R. S. Emery, M. S. Allen, and J. S. Liesman. 1995. Prepartum diets more nutrient-dense than recommended by NRC improve nutritional status of peripartum cows. *J. Dairy Sci.* 78(Suppl. 1):264.

Waltner, S. S., J. P. McNamara, and J. K. Hillers. 1993. Relationships of body condition score to production variables in high producing Holstein dairy cattle. *J. Dairy Sci.* 76:3410-3419.

Wildman, E. E., G. M. Jones, P. E. Wagner, R. L. Boman, H. F. Trout, and T. N. Lesch. 1982. A dairy cows body condition scoring system and its relationship to selected production characteristics. *J. Dairy Sci.* 65:495-501.

Xu, J., and M. S. Allen. 1998. Effects of dietary lactose compared with ground corn on growth rate of ruminal papillae. *J. Dairy Sci.* 81(Suppl. 1):296.

Zamet, Claudie N., V. F. Colenbrander, R. E. Erb, C. J. Callahan, B. P. Chew, and N. J. Moeller. 1979. Variables associated with peripartum traits in dairy cows. II. Effect of dietary forage and disorders on voluntary intake of feed, body weight and milk yield. *Theriogenology.* 11:229-244.

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