The Importance of BCS Management to Cow Welfare, Performance and Fertility

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Take Home Messages

- Cows attempt to regulate their body energy reserves to a target BCS during early lactation; thus, cows with greater BCS at calving will lose more BCS in early lactation.
- Increasing BCS at calving exacerbates negative energy balance in early lactation rather than preventing it.
- Genetic selection for milk production has decreased the target BCS of cows.
- Extreme negative energy balance and loss of BCS in early lactation may avoidable.
- For high producing Holstein cows in North America, BCS at calving should not be greater than 3.0.

Introduction

Dairy cows, like all mammals, store surplus energy not immediately needed in the form of fat (triglycerides) in various adipose tissues throughout the body (Friggens, 2003). The physiological regulation of pregnancy and lactation results in cyclic changes in body fat reserves, as fat is mobilized in early lactation to meet energy demands of increasing milk production and then replenished in mid- to late lactation in anticipation of the next calving and lactation.

Management of body fat content is critical to achieving the sometimes antagonistic goals of good fertility, high milk production, and health. At

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present, the best on-farm tool for long-term management of body energy reserves is body condition scoring. Assessment of body condition scores (BCS) in late lactation, at dry-off, at calving, and at initiation of breeding can be helpful in determining whether the nutritional program and other management practices are adequate. Where problems in health, fertility or production are present, evaluation of BCS can help troubleshoot the cause or causes.

The topic of BCS is not new and has been addressed by a number of authors in previous years of this series. Several good scientific reviews are available for the interested reader to find more information (Garnsworthy, 2007; Roche et al., 2009). My objective is to review well-established principles of biology related to BCS, as well as to address some newer aspects of the relationships between BCS and health, fertility and production. In many cases, managers and their advisors overestimate what an optimal BCS at calving should be.

▪ Optimal BCS From the Cow's Perspective: The "Target BCS"

Although BCS is assigned according to different scales around the world, the scale used in Canada and the rest of North America ranges from 1 (emaciated) to 5 (obese). Scorers today usually attempt to assign scores with quarter-point increments. By definition, it would seem that the midpoint (BCS = 3.0) of the scale should be the desired score at the start of the lactation cycle (calving).

There is strong evidence to indicate that the degree of body fatness is regulated to a certain optimum within individual cows. This optimum appears to represent a "target BCS" that cows attempt to reach somewhere between 10 and 20 wk of lactation (Garnsworthy, 2007). The cow's target score is a genetically determined "set point", which allows the cow to produce milk, reproduce and remain healthy. The cow's target BCS should not be confused with management recommendations for optimal BCS based on data or perceptions of managers.

The target BCS for most high-producing Holstein cows is now in the range of 2.0 to 2.5, which has continued to decrease with genetic selection for high milk yield and high yields of milk components (Garnsworthy, 2007). Where management pushes cows away from their optimum score, either too fat or too thin, cows will respond by repartitioning dietary nutrients to restore body fatness to the optimum target BCS. This means that cows that are thin relative to their target score at calving will gain BCS after calving, and cows with excessive BCS will mobilize body fat during early lactation (Figure 1). Such responses have been observed in other studies too, including our own (Douglas et al., 2006).

▪ BCS and Welfare: Associations with Health

As with other mammals, including humans, both excessively thin and excessively fat cows may represent a welfare problem (Friggens, 2003). In most herds in confinement systems and fed TMR, it is rare to see cows in excessively thin BCS unless as a result of illness or lameness. Occasionally, widespread drought or crippling economic conditions might lead to herds being too thin, but usually not to the point of semi-starvation. In grazing systems, such as those in New Zealand and Ireland, declining grass abundance and quality as cows move into winter can result in the herd being too thin for optimum reproduction and production in the next lactation. Thin cows may be more susceptible to infectious disease.

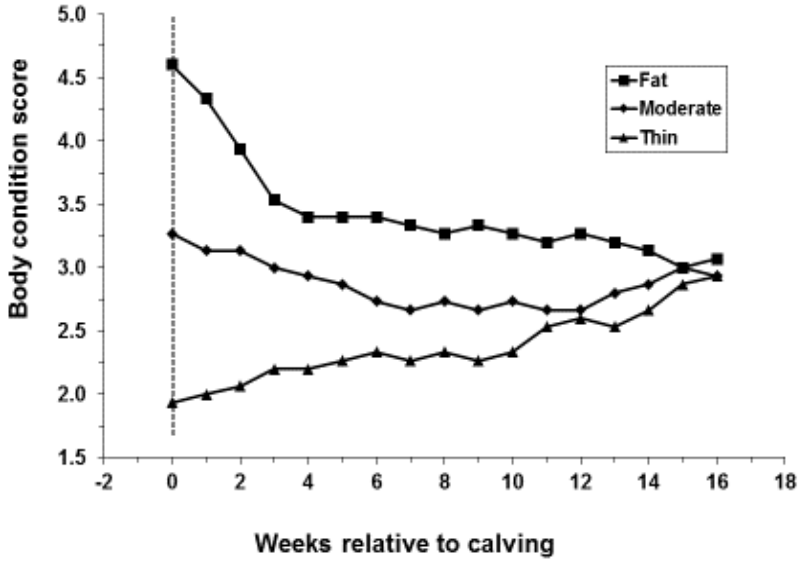

Figure 1. Cows were fed during mid- to late lactation to be fat, moderate, or thin BCS at calving (vertical dotted line). All cows were fed the same lactation ration after calving for ad libitum intake. By 15–16 weeks into lactation all cows had converged at the same BCS. Thin cows produced more milk and consumed more DM than fat cows, with cows of moderate BCS being intermediate. Redrawn from Garnsworthy (2007).

On the other hand, excessive BCS can be common in confinement systems. Improperly balanced diets, poor forage quality that leads to more grain feeding, and poor fertility often lead to cows becoming overconditioned by the next calving. While uninformed consumers may see the thin cow as the most obvious indication of poor welfare, from the standpoint of our common management, the fat cow is generally the greater welfare risk.

The belief that essentially all high producing dairy cows enter negative energy balance (NEB) after calving is deeply engrained in those who work with dairy production. However, as will be shown in a later section, we have known that this is not necessarily the case. Cows that calve with BCS greater than their target will mobilize that BCS in early lactation. The mobilized fat circulates in blood as nonesterified fatty acids (NEFA), which can be used as a diagnostic tool for adequacy of management during the transition period (Ospina et al., 2010). While NEFA mobilization provides fatty acids to make milk fat and may provide metabolizable energy in addition to what the cow consumes for fueling greater milk production, the resulting NEB carries a greater risk of health disorders and is a major cause of poor reproductive success (Butler, 2003; Garnsworthy et al., 2008).

Rapid loss of body fat after calving and into early lactation directly increases the risks of fatty liver and ketosis (Drackley et al., 2005). The liver takes approximately one-third of the mobilized NEFA. During NEB, most of the NEFA are either converted back into triglycerides that accumulate and cause fatty liver or are converted into the ketone bodies such as beta-hydroxybutyrate (BHBA). Recent studies have shown that subclinical ketosis may occur in more than 40% of cows after calving, with the greatest incidence during the first 2 wk after calving (McArt et al., 2012). The NEB represented by high NEFA and BHBA concentrations in cows is associated with greater occurrence of displaced abomasum and ketosis, loss of milk production, and decreased fertility (Chapinal et al., 2012; Ospina et al., 2010). Negative secondary effects of ketosis are more severe if ketosis occurs within the first week post-calving than in the second or later weeks (McArt et al., 2012).

High BCS at calving, and the NEB and rapid loss of BCS that follow after calving, are also associated with increased occurrence of dystocia, retained placenta, metritis, hypocalcemia and milk fever, mastitis, and lameness (Garnsworthy, 2007; Roche et al., 2009). The “fat cow syndrome” is well known to result in a complex of metabolic disorders and infectious disease problems, many of which may be exceptionally difficult to treat and resolve. Evidence indicates that NEB impairs function of cells of the immune system (Lacetara et al., 2005), which likely explains the greater incidence of infectious diseases like metritis and mastitis. Some of this impairment may result from changes in energy metabolites in blood; high NEFA and high BHBA have been shown to negatively affect immune cells, especially when blood glucose is low (Contreras and Sordillo, 2011). Another factor involved may be the increase in oxidative stress caused by the fat mobilization (Bernabucci et al., 2005).

Cows that calve with excessive BCS have poor appetites and lower DMI than their thinner counterparts (Grummer et al., 2004). This may be a result of the cows’ biological drive to return to their target BCS. Mechanistically, recent research has shown that high NEFA mobilization may decrease DMI through

increasing the rate of ATP production within the liver, which is part of the "hepatic oxidation theory" established in cows by Michigan State University researchers (Allen et al., 2009). According to this theory, cows that mobilize BCS will have lower DMI; this can result in greater NEB that in turn increases NEFA mobilization and so on. Cows can enter a "death spiral" of decreasing intake and increasing fat mobilization, contributing to the complex of health problems and perhaps accounting for the greater death loss in confinement TMR systems.

▪ BCS and Fertility

Like health issues, both low and high BCS at calving can negatively affect reproductive efficiency. Cows that are thinner than their target BCS may have prolonged periods of postpartum anestrus (Roche et al., 2009). High BCS and the resulting NEB after calving clearly decrease fertility in cows. Although studies have demonstrated a weak and variable relationship between the degree of NEB and impaired fertility, the time to the lowest NEB and the rate of change in NEB are more strongly related (Butler, 2003; Garnsworthy et al., 2008). Detrimental effects of NEB on reproduction include 1) delayed resumption of ovarian cyclicity, 2) impacts on oocyte or corpus luteum "quality", viability, or function (sometimes referred to as "follicular memory"), and 3) development of fatty liver (Drackley and Cardoso, 2014).

In general, reproductive success is better in cows that ovulate sooner after calving (Butler, 2003). In NEB after calving, the pulse frequency of LH release, the size and development rate of follicles, concentrations of estrogen and progesterone, and size of the corpus luteum all are decreased (Garnsworthy et al., 2008). Successful ovulation depends on estrogen production by the dominant follicle, restoration of pulsatile luteinizing hormone (LH) secretion, and responsiveness of the ovary to LH. The state of NEB is associated negatively with reproductive performance in part because it interrupts these 3 factors (Butler, 2003).

Insulin concentrations generally reflect energy status and dietary adequacy. Insulin links the metabolic and reproductive systems by its necessity to increase synthesis of insulin-like growth factor 1 (IGF-1) in the liver in response to elevated concentrations of growth hormone, to increase estrodiol production by the dominant follicle and to increase LH receptors for ovulation and corpus luteum development (Lucy, 2000; Garnsworthy et al., 2008). Lower insulin and IGF-1 during NEB thus may be related to eventual increases in days to first ovulation, first estrus and conception, and decreased rates of conception and pregnancy.

Extreme NEB also may negatively impact oocyte or corpus luteum quality or viability due to reduced concentrations of progesterone and IGF-1. The decrease in these compounds may be a result of increased uptake of NEFA

and BHBA by the ovary and its follicles, particularly when glucose concentrations are low (Drackley and Cardoso, 2014).

Fatty liver is negatively associated with fertility (Drackley et al., 2005), which may be an indirect effect of the extreme NEB in these cows. However, direct negative effects of fat infiltration on reproduction cannot be discounted. Blood flow through the liver may be altered by fat accumulation expanding cell volume and compressing the circulation between cells. Fat accumulation also may decrease the normal ability of liver cells to metabolize or clear reproductive and metabolic hormones (Drackley and Cardoso, 2014), thus altering the normal signaling to reproductive tissues and pituitary.

▪ BCS and Production

Across systems, countries, and climates, the available evidence indicates that milk production is maximized when the calving BCS is approximately 3.5 (Roche et al., 2009). However, in these same studies there was little additional milk response when BCS greater than 3.0. Thus, it appears that a calving BCS of 0.5 to 0.75 BCS unit greater than the proposed cow's target BCS during early lactation (2.0 to 2.5) is adequate for maximal lactation response. Thinner cows have greater DMI, which in turn will support high milk yields as well as restore body fat reserves (Garnsworthy, 2007).

Cows with high BCS at calving will produce milk with greater fat content, which is a result of the mobilized NEFA being directly incorporated into milk fat (Roche et al., 2009). If dietary energy, particularly glucogenic energy, intake is limited, milk protein may be decreased.

▪ Relationships with Dry Period and Transition Management

Research by our group over the last two decades has shown that allowing dry cows to consume a marked excess of energy relative to their requirements results in many changes typical of excessive BCS, even if cows do not appear to be overconditioned (Drackley and Cardoso, 2014). In these studies, cows averaged about 3.0 to 3.25 at calving. Cows fed a high fibre, controlled energy diet to limit intake to near requirements showed a better metabolic profile after calving than cows fed higher energy close-up diets (Beever, 2006; Janovick and Drackley, 2010; Janovick et al., 2011). Recent studies have uncovered evidence that differences in internal fat deposition may be responsible (Drackley et al., 2014).

Dairy cattle accumulate relatively more fat in the internal adipose depots (omental, mesenteric, and perirenal) and less subcutaneously compared with

beef cattle. The BCS systems rely mainly on assessment of these subcutaneous fat stores. Nevertheless, in general the correlations among different adipose depots in dairy cows are high, indicating that observed BCS will adequately reflect the non-visible adipose sites and overall body fatness (Roche et al., 2009).

In humans, there is wide variation in the site of fat accumulation, resulting in the so-called “apple” and “pear” shapes. Visceral fat accumulation is linked more strongly with risk for chronic health problems that make up the complex called the “metabolic syndrome”. We wondered whether this might be the case in cows during the dry period; might some individuals be more likely to accumulate fat in the internal depots than others, and is internal fat deposition more likely with excessive energy intake (particularly from the starch in corn) during the dry period? Assessment of individual variation is so far impractical due to the lack of economic ways to measure internal fat deposition in cows, but we were able to address the second question in our research.

We randomized non-lactating and non-pregnant cows into two groups with equal starting BCS (Drackley et al., 2014). The groups were fed either a controlled energy, high fibre diet or a higher energy close-up type diet for 8 weeks to mimic a typical dry period. Then, the cows were killed and dissected to determine body composition (Table 1). Surprisingly, despite the huge difference in dietary energy intake the final BCS was not different between groups, although both groups gained BCS during the 8-wk period. However, the masses of internal adipose tissue were greatly increased in cows fed the higher energy diet. Although BCS may provide a very useful indicator of general nutritional adequacy and fat reserves, it may not be sensitive enough to detect potentially important differences in internal fat reserves that develop over the relatively short timeframe of the dry period.

The omental and mesenteric fat depots are located around the digestive tract, and blood that circulates through these tissues drains directly to the liver before reaching the rest of the body. So, large increases in fat mass would mean that more NEFA directly reach the liver during NEB. Furthermore, cytokines and other adipokines produced by adipose tissue also would be increased, which could negatively impact the liver and other tissues. Such changes might help to explain what we have observed in our feeding studies.

We recently completed a second trial with a similar design, except that the controlled energy diet was made even lower in energy density to prevent body weight gain in the non-pregnant cows. Results were very similar, with little difference in BCS but substantial increases in the internal fat depots.

Cardoso et al. (2013) completed a pooled statistical summary of the dry period feeding studies conducted by our group. With over 200 cows per group of controlled energy versus overfed cows, median days to pregnancy was 10

days shorter in cows fed the controlled energy diets. Such a difference might be related to the changes in body fatness even though BCS were not greatly different between groups.

Table 1. Visceral and internal adipose tissues in nonlactating cows fed low energy (LE) or high energy (HE) diets for 8 weeks.

	Diet		
Variable	LE	HE	SEM
Initial BCS	3.00	3.08	0.25
Final BCS	3.55	3.62	0.11
BW, kg	710	722	33
Adipose tissue site			
Omental, kg	17.5	28.1**	1.3
Mesenteric, kg	12.1	22.0**	2.4
Perirenal, kg	6	9.9*	1.2

n = 9 per diet
** *P* < 0.01; * *P* < 0.05 (Drackley et al., 2014)

▪ So What Should *Our* Target BCS Be?

From the standpoint of the cow's biology, the concept of the target BCS argues strongly that a thinner cow (but not undernourished and unhealthy) will be more likely to meet the combined goals of health, production and reproduction. It is to some degree a different question to ask what the optimal BCS at calving should be for best management outcomes.

Until the last decade or so, many experts recommended a higher BCS (3.5 to 4.0) at calving. The rationale was that cows became thin at peak lactation, perhaps having difficulty in conceiving and maintaining a subsequent pregnancy. A higher BCS at calving was thought necessary to provide a "reserve" to let cows "milk off their backs" to avoid this scenario. As we know now, however, striving for a higher BCS at calving actually promotes this scenario rather than preventing it. As Garnsworthy's (2007) research clearly shows, cows with higher BCS lose more BCS after calving. Over time the normal BCS curve (essentially the inverse of the lactation curve) becomes distorted, with higher maximums and lower minimums, all with struggles of transition health problems, poor fertility, disappointing milk yield, and decreased herd life.

The optimal BCS for maximum milk yield may vary across productions systems, as compared by Roche et al. (2009). For example, cows in grazing

systems are more likely to be too thin going into dry-off. Outcomes from differing BCS also are dependent on the genetic potential for milk within those systems. This is shown conceptually in Figure 2. If cows of high genetic merit calve with high BCS they will lose BCS, whereas if they calve in thin BCS they will maintain BCS. In contrast, low-merit cows that calve with high BCS will maintain BCS, but low-merit cows calving in thin condition will gain BCS. All of these outcomes can be predicted from the concept that increasing genetic merit for milk also means that we are selecting for a thinner cow with a lower target BCS. Garnsworthy (2007) estimated that the target BCS for high-merit Holsteins in the UK had decreased from about 2.49 to 2.10 in approximately 20 years. A calving BCS of approximately one-half score unit above the target seems reasonable, which means that BCS at calving should be around 2.75.

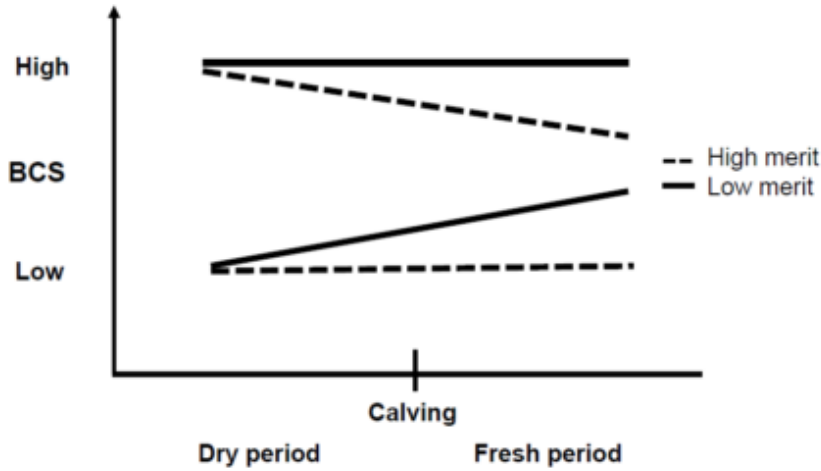

Figure 2. Conceptual depiction of the effect of high or low BCS at calving on BCS change during early lactation in cows of high or low genetic merit for milk production. Based on studies by Garnsworthy (2007) and McNamara (1991).

The concepts demonstrated so eloquently by Garnsworthy's research can be seen in modern large-scale production systems. Carvalho et al. (2014) studied 2 large commercial dairy herds in Wisconsin with the same owners and general management. As shown in Table 2, responses to timed-AI protocols were affected by the BCS change from calving to 21 days in milk.

Pregnancy percentage at either 40 days or 70 days of lactation was markedly greater for cows that gained BCS in early lactation than for cows that maintained or lost BCS, with no difference in energy-corrected milk yield.

These findings are not surprising in themselves and are consistent with long-known relationships between BCS status and fertility. What was surprising, however, was that nearly 60% of the cows in the 2 herds either maintained or gained BCS early postpartum. This evidence contradicts the long-held dogma that nearly all cows are in NEB after calving such that they lose BCS. Cows were thinner on average at calving (BCS = 2.9) than many experts' recommendations. Of interest is that both of these herds used a controlled-energy dry cow program, with a management aim to minimize change in BCS during the dry period and minimize health problems after calving. Anecdotal evidence from many consultants working with high-producing dairy herds in the US confirms that in well-managed herds it is not inevitable that fresh cows must lose BCS.

Table 2. Reproductive and productive responses of Holstein cows (n = 1,887) in two commercial herds in Wisconsin that lost, maintained or gained BCS from calving to 3 wk postpartum.

	BCS change category			
Item	Lost	Maintained	Gained	*P*
% of cows	41.8	35.8	22.4	
Pregnant to AI at 40 d (%)	25.1	38.2	83.5	< 0.01
Pregnant to AI at 70 d (%)	22.8	36.0	78.3	< 0.01
Pregnancy loss (%)	9.1	5.8	6.2	0.34
BCS at calving	2.93	2.89	2.85	< 0.01
BCS at 21 DIM	2.64	2.89	3.10	< 0.01
Energy-corrected milk[a] (kg/d)	30.9	31.5	28.7	0.30

[a] Mean from calving to d 21 postcalving
From Carvalho et al., 2014

▪ Conclusions

Use of BCS to monitor body energy reserves across the lactation cycle remains a valuable tool for dairy producers and their advisors. Cows have a target BCS that they will attempt to reach, all other things being equal. This target BCS has decreased with time and genetic selection for high milk yield, and likely now is in the range of 2.0–2.5 depending on genetic merit for milk yield. If cows calve with BCS considerably greater than that, they will lose BCS during early lactation and be in substantial negative energy balance. Loss of BCS is associated with greater risk for metabolic and infectious health problems, as well as reduced fertility. Consideration of what makes an optimal BCS score at calving must factor in the welfare, fertility and production implications. Although it may appear a paradox to many producers (and perhaps consumers), healthy cows with relatively thin BCS may have improved welfare and longer productive lives than heavier cows. For most North American Holstein cows, BCS at calving should not be greater than 3.0.

▪ References

Allen, M.S., B.J. Bradford, and M. Oba. 2009. Board invited review: The hepatic oxidation theory of the control of feed intake and its application to ruminants. J. Anim. Sci. 87:3317-3334.

Beever, D.E. 2006. The impact of controlled nutrition during the dry period on dairy cow health, fertility and performance. Animal Repro. Sci. 96:212-226.

Bernabucci, U., B. Ronchi, N. Lacetara, and A. Nardone. 2005. Influence of body condition score on relationships between metabolic status and oxidative stress in periparturient dairy cows. J. Dairy Sci. 88:2017-2026.

Butler, W.R. 2003. Energy balance relationships with follicular development, ovulation and fertility in postpartum dairy cows. Livest. Prod. Sci. 83:211-218.

Cardoso, F.C., S.J. LeBlanc, M.R. Murphy and J.K. Drackley. 2013. Prepartum nutritional strategy affects reproductive performance in dairy cows. J. Dairy Sci. 96:5859-5871.

Carvalho, P.D., A.H. Souza, M.C. Amundson, K.S. Hackbart, M.J. Fuenzalida, M.M. Herlihy, H. Ayres, A.R. Dresch, L.M. Vieira, J.N. Guenther, R.R. Grummer, P.M. Fricke, R.D. Shaver, and M.C. Wiltbank. 2014. Relationships between fertility and postpartum changes in body condition and body weight in lactating dairy cows. J. Dairy Sci. 97:3666-3683.

Chapinal, N., S.J. Leblanc, M.E. Carson, K.E. Leslie, S. Godden, M. Capel, J.E. Santos, M.W. Overton and T.F. Duffield. 2012. Herd-level associations of serum metabolites in the transition period with disease, milk production, and early lactation reproductive performance. J. Dairy Sci. 95:5676-5682.

Contreras, G.A. and L.M. Sordillo. 2011. Lipid mobilization and inflammatory responses during the transition period of dairy cows. Comp. Immun. Microb. Infect. Dis. 34:281-289.

Douglas, G.N., T.R. Overton, H.G. Bateman, H.M. Dann and J.K. Drackley. 2006. Prepartal plane of nutrition, regardless of dietary energy source, affects periparturient metabolism and dry matter intake in Holstein cows. J. Dairy Sci. 89:2141-2157.

Drackley, J.K. and F.C. Cardoso. 2014. Prepartum and postpartum nutritional management to optimize fertility in high-yielding dairy cows in confined TMR systems. Animal. 8 Suppl 1:5-14.

Drackley, J.K., H.M. Dann, G.N. Douglas, N.A. Janovick Guretzky, N.B. Litherland, J.P. Underwood and J.J. Loor. 2005. Physiological and pathological adaptations in dairy cows that may increase susceptibility to periparturient diseases and disorders. Italian J. Anim. Sci. 4:323-344.

Drackley, J.K., R.L. Wallace, D. Graugnard, J. Vasquez, B.F. Richards, and J.J. Loor. 2014. Visceral adipose tissue mass in non-lactating dairy cows fed diets differing in energy density. J. Dairy Sci. 97:3420-3430.

Friggens, N.C. 2003. Body lipid reserves and reproductive cycle: towards a better understanding. Lives. Prod. Sci. 83:219-236.

Garnsworthy, P.C. 2007. Body condition score in dairy cows: targets for production and fertility. In Recent advances in animal nutrition (ed. P.C. Garnsworthy and J. Wiseman). pp. 61-86. Nottingham University Press, Nottingham, UK.

Garnsworthy, P.C., K.D. Sinclair, and R. Webb. 2008. Integration of physiological mechanisms that influence fertility in dairy cows. Animal. 2:1144-1152.

Grummer, R.R., D.G. Mashek, and A. Hayirli. 2004. Dry matter intake and energy balance in the transition period. Vet. Clinics N.A. Food Anim. Pract. 20:447-470.

Janovick, N.A. and J.K. Drackley. 2010. Prepartum dietary management of energy intake affects postpartum intake and lactation performance by primiparous and multiparous Holstein cows. J. Dairy Sci. 93:3086–3102.

Janovick, N.A., Y.R. Boisclair, and J.K. Drackley. 2011. Prepartum dietary energy intake affects metabolism and health during the periparturient period in primiparous and multiparous Holstein cows. J. Dairy Sci. 94:1385-1400.

Lacetara, N., D. Scalia, U. Bernabucci, B. Ronchi, D. Pirazzi, and A. Nardone. 2005. Lymphocyte functions in overconditioned cows around parturition. J. Dairy Sci. 88:2010-2016.

Lucy, M.C. 2000. Regulation of ovarian follicular growth by somatotropin and insulin-like growth factors in cattle. J. Dairy Sci. 83:1635-1647.

McArt, J.A., D.V. Nydam, and G.R. Oetzel. 2012. Epidemiology of subclinical ketosis in early lactation dairy cattle. J. Dairy Sci. 95:5056-5066.

McNamara, J.P. 1991. Regulation of adipose tissue metabolism in support of lactation. J. Dairy Sci. 74:706-719.

Ospina, P.A., D.V. Nydam, T. Stokol, and T.R. Overton. 2010. Associations of elevated nonesterified fatty acids and β-hydroxybutyrate concentrations with early lactation reproductive performance and milk production in transition dairy cattle in the northeastern United States. J. Dairy Sci. 93:1596-1603.

Roche, J.R., N.C. Friggens, J.K. Kay, M.W. Fisher, K.J. Stafford, and D.P. Berry. 2009. Invited review: Body condition score and its association with dairy cow productivity, health, and welfare. J. Dairy Sci. 92:5769-5801.