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## **BODY CONDITION SCORE IN DAIRY COWS: TARGETS FOR PRODUCTION AND FERTILITY**

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### **Introduction**

Body condition score (BCS) is probably the most useful management tool available to dairy producers for assessing the nutritional status of cows. It provides a rapid indication of levels of body fat reserves at almost zero cost. Body fat reserves play an important biological role in early lactation by buffering the cow against feed shortages whilst she partitions energy mainly towards milk output. However, rapid mobilisation of body fat reserves causes fertility and health problems. It is important, therefore, that appropriate targets for BCS are established, based on the underlying biology of the cow.

The current chapter discusses the use of BCS for assessing body fatness; relationships among BCS, feed intake and negative energy balance; implications of BCS changes for cow health and fertility; and BCS targets at different stages of lactation. Importantly, it will address the question of whether BCS targets should be updated for modern dairy cows.

### **Body condition scoring systems**

Several body condition scoring systems were introduced in the 1970s and 1980s. Lowman, Scott and Somerville (1973) adapted a system used previously for scoring beef cattle, which involves palpation of the lumbar vertebrae and around the tail head. A scale of 1 to 4 was proposed; 1 corresponds to a cow that looks emaciated, has sharp bones and has no palpable fat cover around the tail head; 4 corresponds to a cow that is excessively fat and the spinous processes of the lumbar vertebrae cannot be felt. A similar system with a scale of 0 to 5 was proposed by Mulvany (1977)

in the UK. In Australia, a scale of 1 to 8 was introduced by Earle (1976). In the United States, a scale of 1 to 5 was proposed by Wildman *et al.* (1982). In New Zealand, a scale of 1 to 10 was introduced in 1993 by the Livestock Improvement Corporation (Stockdale, 2001). Different systems place different emphasis on fat cover felt in different anatomical regions; the US system uses visual appraisal with no requirement to palpate cows. Variations among scoring systems can lead to confusion when comparing targets and responses.

The majority of publications in the past decade have used a 1 to 5 scale. A 1 to 5 scale is used also by the UK Department for Environment Food and Rural Affairs in its advisory leaflet on body condition scoring (Defra, 2001), which is cited by the official welfare code for cattle (Defra, 2003). For these reasons, a 1 to 5 scale will be used in the current review, unless specified otherwise. Photographs and descriptions of this scale are available online (see Defra, 2001 and Ferguson, 2006). The following equations were used to convert other scales to a 1 to 5 scale:

$$1-4 \text{ scale: } \text{BCS} \times 4/3 - 1/3$$

$$0-5 \text{ scale: } \text{BCS} \times 4/5 + 1$$

$$1-8 \text{ scale: } \text{BCS} \times 4/7 + 3/7$$

$$1-9 \text{ scale: } \text{BCS}/2 + 1/2$$

$$1-10 \text{ scale: } \text{BCS} \times 4/9 + 5/9$$

The use of simple mathematical conversions between scales might not be accurate if scales are not linear across the whole range of scores. Roche *et al.* (2004) compared a body condition scoring system used in New Zealand (1 to 10 scale) with systems used in USA (1 to 5 scale), Ireland (1 to 5 scale) and Australia (1 to 8 scale). Although there were significant correlations among systems, scores tended to converge at the bottom of the scales. Unfortunately, the study included no cows outside the range 2 to 4.25 (1-5 scale), and different scoring systems were confounded with different operators. Nevertheless, the study highlights the need for some caution in comparisons of studies that have used different scales. At present, however, there is insufficient information available to justify anything other than simple mathematical conversion to a 1-5 scale.

Ferguson *et al.* (2006) compared scores of four observers for 57 cows ranging in BCS from 2.0 to 4.5 and found significant, but small, differences between observers; the greatest difference in mean BCS between observers was 0.29 units. In an earlier study, Ferguson *et al.* (1994) compared scores given to 225 cows by four observers and found that 58 % of the time all observers gave the same score to the same cow. An additional 33% of the time observers gave plus or minus 0.25 to the same cow. Scores among observers were highly correlated ( $r = 0.76$  to  $0.88$ ). In another recent study, Kristensen *et al.* (2006) compared

scores of six highly-trained instructors and 51 practicing veterinarians. Each individual scored approximately 20 cows twice, giving a total of 2,230 scores. Between scoring sessions, the instructors gave training to the veterinarians. Instructors agreed with each other 83% of the time, and repeated scores agreed 72 to 95% of the time. Scores given by veterinarians were more variable and generally lower than instructors' scores at the first session. Following training, however, veterinarians' scores became more homogenous and similar to the instructors' scores. It was concluded that a limited amount of training brought about substantial improvement in the validity and precision of BCS determined by practicing veterinarians, compared with BCS recorded on the same cows by highly trained classifiers.

Although BCS is positively related to live weight, cows in early lactation can often maintain or gain live weight whilst BCS is declining, due to the effect of gut fill on live weight (Garnsworthy, 1988). For this reason, BCS is better than live weight as an indicator of body fat reserves. Several studies have reported strong relationships between BCS and body fat content. For example, Wright and Russel (1984) reported a strong positive relationship between BCS and total fat content in non-pregnant, non-lactating, Friesian cows, as did Garnsworthy *et al.* (1986). Similarly, Otto *et al.* (1991) found that one unit of BCS increase for cull cows corresponded to an increase of 12.65% ether extract (a chemical measure of fat content).

Some reports suggest that the relationship between BCS and fat content might not be linear at the lower end of the scale. Gregory *et al.* (1998) found that over the range of 1.4 to 8.0 (1-8 scale), BCS was positively related to weight of internal fat depots, proportion of fat in a sample joint, and muscle to bone ratio in the sample joint of cull cows; however below BCS 3 (1-8 scale), cows had limited body fat reserves and there was little change in body fat with change in BCS. In a recent study of 146 lactating Holstein-Friesian cows, Yan *et al.* (2005) found that BCS was not related to empty body concentration of crude protein, but BCS was significantly ( $P < 0.001$ ) related to empty body concentrations of lipid (Lipid ( $\text{g/kg}^{0.75}$ ) =  $271.7 \text{ BCS} - 233.7$ ;  $R^2 = 0.57$ ) and energy (Energy ( $\text{MJ/kg}^{0.75}$ ) =  $10.06 \text{ BCS} + 9.9$ ;  $R^2 = 0.58$ ). Although only linear relationships were reported, their figures indicate little change in body fat with change in BCS when BCS was below 2, supporting the observations of Gregory *et al.* (1998).

### **Body condition score, dry matter intake and energy balance**

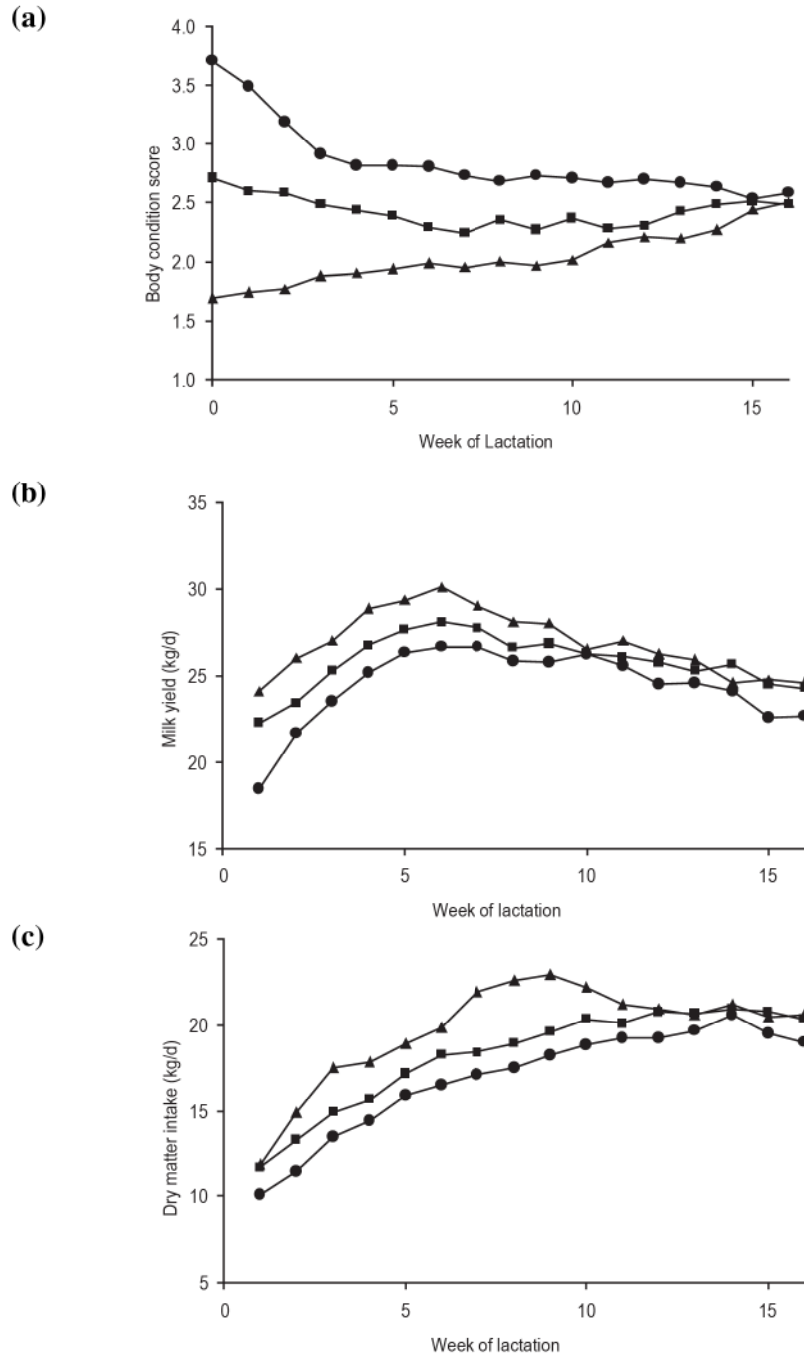
Early recommendations (e.g. Lowman *et al.*, 1973) were for minimum BCS (1-4 scale) of 3.5 at calving, 2.5 at mating and 3.0 at drying off. These recommendations were based on observations that dry matter intake normally

increases at a slower rate than milk yield in early lactation. Under these circumstances, energy output in milk exceeds energy intake from feed, and the cow is in 'negative energy balance'. Cows in negative energy balance mobilise body fat reserves to support milk production. Increasing body fat reserves at calving, by increasing prepartum feed supply, allowed greater fat mobilisation and higher milk yields postpartum (Broster, 1971). This explains the relatively high BCS recommendation of 3.5 at calving; the philosophy was to 'steam up' cows before calving so that they could 'milk off their backs' after calving.

Studies in the 1980s, however, showed that body fat has a negative feedback effect on feed intake. In two experiments, Garnsworthy and Topps (1982) controlled the energy intake of cows before calving so that cows calved with BCS (1-4 scale) of 1.7 (Thin), 2.7 (Medium) or 3.7 (Fat). After calving, all cows were offered a high-energy total-mixed ration (TMR) *ad libitum*. There was no effect of BCS at calving on milk yield. Over the first 12 weeks of lactation, cows that were fat at calving lost 0.9-1.0 BCS units; cows with medium BCS at calving lost 0.5-0.6 BCS units; cows that were thin at calving gained 0.4-0.5 BCS units. BCS tended to converge at 2.5 in week 12-15 of lactation (Figure 1a), suggesting that cows have a target BCS that they try to attain in early lactation. Although milk yield peaked in week 6 of lactation for all groups of cows (Figure 1b), maximum dry matter intake was reached in week 15 for fat cows, week 11 for medium cows and week 9 for thin cows (Figure 1c). This suggested that feed intake was controlled by physiological feedback mechanisms and that level of body fat had a direct effect on feed intake.

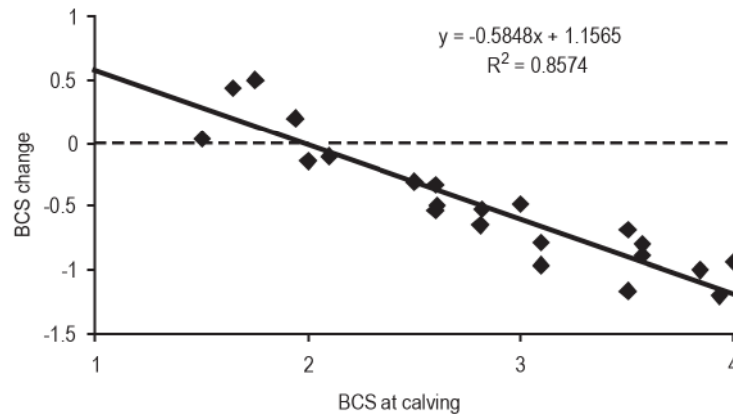
Numerous studies in the 1980s and early 1990s (reviewed by Garnsworthy, 1988; Broster and Broster, 1998) confirmed the strong negative relationship between BCS at calving and change in BCS during early lactation (Figure 2). Each individual dairy cow has a genetically-programmed target BCS that she attempts to reach approximately 10 to 12 weeks after calving. If her BCS is above this target, feed intake is reduced and she loses condition; if her BCS is below this target, feed intake is increased and she gains condition. The biological drive for a cow to attain a target BCS appears to be as strong as the drive to attain a genetically-programmed peak milk yield. The philosophy of getting cows in 'good condition' at calving is, therefore, counter productive. Instead of a high BCS at calving compensating for low feed intake in early lactation, it actually reduces feed intake further and exacerbates negative energy balance.

To quantify effects of BCS on feed intake in early lactation, Garnsworthy (1994) examined data from 143 cows in 8 experiments where weekly values were available for milk yield, BCS and dry matter intake over the first 17



**Figure 1.** Changes in BCS (a), milk yield (b) and dry matter intake (c) for cows calving at BCS (1-4 scale) >3.5 (●), 2.5 – 3.0 (■), or <2.0 (▲). Data are combined means of Experiments 1 and 2 from Garnsworthy and Topps (1982).





**Figure 2.** Relationship between BCS at calving and change in BCS over the first 10 – 12 weeks of lactation. Data (1 – 4 scale) are group means from review by Garnsworthy (1988), and were originally published in Land and Leaver (1980; 1981), Garnsworthy and Topps (1982), Treacher *et al.* (1986), Garnsworthy and Jones (1987) and Bouchier *et al.* (1987)

weeks of lactation. The model assumed that intake would be related primarily to milk energy output, but would be modified by a feedback factor related to the difference between current BCS and target BCS. For high-energy diets ( $>11.5$  MJ ME/kg DM), the best equation for predicting dry matter intake (kg/d) was  $0.458n + 0.538Y - 0.48BCS_d$ , where  $n$  = week of lactation,  $Y$  = milk yield (kg/d) and  $BCS_d$  = BCS in week  $n$  minus BCS target (2.5) ( $R^2 = 0.96$ ; mean square prediction error = 0.38 kg/d). More recently, Ellis *et al.* (2006) used a similar approach to adjust static prediction equations of dry matter intake for use in dynamic modelling of dairy cow performance over a whole lactation. The prediction equations used were those of the Agricultural Research Council (1980), the Cornell Net Carbohydrate and Protein System (Fox *et al.*, 2004), and the National Research Council (2001). These were examined with a dataset comprising 777 data points from 21 sets of lactation performance data. By adjusting dry matter intake with a feedback factor, residual sums of squares were reduced on average by 41% for dry matter intake predictions and by 52% for body weight predictions. The feedback factor was based on the difference between initial body energy reserves and current body energy reserves for each week of lactation. It was assumed that cows would attempt to replenish energy reserves to the level at calving, which would be equivalent to BCS 3.5. Perhaps predictions might have been improved still further had the cows' biological target BCS been used, rather than calving BCS which is determined by management policy.

The ability of cows to reach their target BCS is affected by diet composition. With high-energy diets, thin cows can be in positive energy balance and

increase BCS, but fat cows will be in negative energy balance and decrease BCS. With low-energy diets, feed intake is limited by physical capacity of the rumen and thin cows cannot increase energy intake to match milk energy output; fat cows mobilise body condition at a faster rate to support milk production and are in negative energy balance longer than with high-energy diets (Jones and Garnsworthy, 1989). High-protein diets result in greater loss of body condition by fat cows, but either increase gain or decrease loss of body condition by thin cows which use excess protein for gluconeogenesis (Garnsworthy and Jones, 1987; Jones and Garnsworthy, 1988). Low-fibre, high-starch diets increase BCS gain in thin cows and decrease BCS loss in fat cows, probably by increasing insulin status (Garnsworthy and Jones, 1993). High-fat diets decrease BCS loss in fat cows, but do not affect BCS change in thin cows (Garnsworthy and Huggett, 1992).

The relationship between BCS at calving and change in BCS during early lactation applies across all feeding systems. Studies reviewed by Garnsworthy (1988) and Broster and Broster (1998) included cows fed on TMR, hay plus concentrates, and self-feed silage. Stockdale (2001) extended the review to include six grazing studies, which confirmed that pasture-fed cows exhibit the same strong relationship.

When the feedback effect of BCS on feed intake was established in the 1980s, the regulatory mechanism was unknown, although lipostatic control of feed intake had been proposed many years earlier (Kennedy, 1953). Subsequently, the main signalling factor was suggested to be leptin, a peptide hormone secreted by adipose tissue and discovered in 1994. Plasma leptin concentrations vary with mass of adipose tissue and leptin has been shown to decrease appetite by direct action on the hypothalamic satiety centre (Vernon *et al.*, 2001). However, the overall regulatory mechanism is complex. In addition to its effects on feed intake, leptin has been found to modulate nutrient transfer and partitioning by interaction with other hormones including insulin, glucagon, glucocorticoids, growth hormone, insulin-like growth factor-I, cytokines and thyroid hormones (Hill, 2004). Other factors secreted by adipose tissue (tumour necrosis factor  $\alpha$  and resistin) have also been shown to interact with leptin in regulation of adiposity (Vernon *et al.*, 2001). Meikle *et al.* (2004) found that BCS at parturition affected leptin concentrations. Leptin concentrations during late pregnancy and the first two weeks of lactation were higher in cows with higher BCS. Leptin concentrations decreased at a faster rate postpartum in fat cows (BCS  $\geq 3$ ) than in lean cows (BCS  $< 3$ ), and the leptin nadir was reached 10 days earlier in lean cows than in fat cows. However, although leptin concentrations were positively related to BCS both before and after parturition in fat cows, the relationship was only significant before parturition in lean cows. This is in agreement with the

data of Yan *et al.* (2005), mentioned in the previous section, where there was little change in body fat with change in BCS when BCS was below 2.0.

### **Body condition score, health and reproduction**

Cows that are excessively fat at calving are more likely to develop fatty liver and ketosis because high BCS depresses appetite severely and body fat is mobilised too rapidly (Reid *et al.*, 1986). Such cows exhibit severe negative energy balance, poor reproductive performance and increased incidence of diseases (Treacher *et al.*, 1986). Fatty liver is characterised by accumulation of triglycerides (TAG) in hepatocytes due to overloading of fatty acid oxidation and lipoprotein synthesis systems within the liver. Fatty liver is defined as >20% fat in liver cells observed by microscopy, or >50 g TAG/kg liver tissue by chemical analysis (Newbold, 2005). In a survey of 9 herds in the Netherlands, Jorritsma *et al.* (2001) found that 54% of cows had >50 g TAG/kg liver tissue when sampled between 6 and 17 days postpartum. Causes, prevention and alleviation of fatty liver were reviewed by Newbold (2005), who stated “central to any approach to the prevention of fatty liver is control of body condition: more specifically, the avoidance of fat cows at calving”. In terms of BCS, it appears from the data of Treacher *et al.* (1986) and Jorritsma *et al.* (2001) that risk of fatty liver increases considerably when BCS is above 3.5 at calving.

Fatty liver is often associated with ketosis, although fatty liver usually precedes ketosis and ketosis is associated more with carbohydrate metabolism than fat metabolism (Grummer, 1992). In a study of 732 moderate-yielding Norwegian cows, Gillund *et al.* (2001) found that cows calving with a BCS of 3.5 or greater were 2.3 to 2.8 times more likely to experience ketosis compared with a BCS of 3.25 or lower. In an analysis of 3,586 lactations covering a 17-year period at the Langhill Dairy Cattle Research Centre, Rasmussen *et al.* (1999) found that cows with a BCS at calving of 3.5 had double the risk of developing ketosis compared with cows with a BCS at calving of 2.0, all other things being equal.

In addition to fatty liver and ketosis, other disease problems have been linked with BCS. Relationships are variable and inconsistent, but excessive loss of BCS in the dry period or early lactation, low BCS at drying off, and high BCS at calving, have been associated with increased risks of dystocia, retained placenta, metritis, milk fever, mastitis and lameness (see for example: Treacher *et al.*, 1986; Gearhart *et al.*, 1990; Markusfeld, 1997). Blood biochemistry can be used as an indicator of nutritional status and disease risk; Ward *et al.* (1995) found that  $\beta$ -hydroxybutyrate (BHBA) and bile acids

were higher, and glucose was lower, for the first 40 days of lactation in cows with BCS above 3.0 at calving, compared with cows below 3.0. Although no data on mineral and vitamin status could be found in the literature for fat versus thin cows, it is possible that reduced intake by fat cows could compromise their micro-nutrient reserves.

Even moderate levels of fat mobilisation are associated with negative energy balance and reduced fertility. Several studies have shown that high genetic merit, negative energy balance, body fat mobilisation, high plasma non-esterified fatty acids (NEFA), and low plasma insulin are associated with delayed first ovulation postpartum and reduced pregnancy rates (see reviews by Garnsworthy and Webb, 1999; Butler, 2003; Pryce *et al.*, 2004; Butler, 2005). Butler (2005) reported that cows losing less than 0.5 BCS over the first 30 days postpartum took an average of 30 days from calving to first ovulation; cows losing 0.5 to 1.0 BCS took 36 days; cows losing more than 1.0 BCS took 50 days. Bouchier *et al.* (1987) surveyed 2000 cows in high-yielding herds and found a significant effect of BCS change on conception rate to first service: cows gaining condition during the first 12 weeks of lactation had a 67 % conception rate; cows losing 0.5 to 1.0 BCS had a 55 % conception rate; cows losing more than 1.0 BCS had a 47 % conception rate. A similar relationship was found by Butler (2005), who concluded from several studies that conception rate decreases by 10 % per 0.5 unit BCS loss. In reviewing physiological mechanisms, Butler (2003) reported that negative energy balance is strongly associated with attenuation of LH pulse frequency and low levels of blood glucose, insulin and IGF-I that collectively limit oestrogen production by dominant follicles; with diminished quality of oocytes and capability for embryo development; and with reduced serum progesterone concentrations. This is supported by studies at the University of Nottingham, which showed that dietary manipulation of insulin status affects postpartum anoestrus (Gong *et al.*, 2002) and oocyte quality (Fouladi-Nashta *et al.*, 2005).

Lopez-Gatius *et al.* (2003) performed a meta-analysis of 15 papers corresponding to nearly 8,000 cows to examine relationships between BCS and reproductive performance. Effects of BCS at calving, and at first insemination, on pregnancy rate were highly heterogeneous, which was attributed to variation among farms in voluntary wait period and oestrous detection rate, combined with the dynamic nature of BCS change. Cows not served until their second or third ovulation postpartum, due to either farm policy or undetected oestrus, are more likely to conceive and to have higher BCS than cows served at their first ovulation. On the other hand, effects of BCS on number of days open were consistent among studies. Compared with cows losing 0 to 0.5 BCS, cows losing 0.5 to 1.0 BCS took 3.5 days longer to conceive, and cows losing >1.0 BCS took 10.6 days longer to

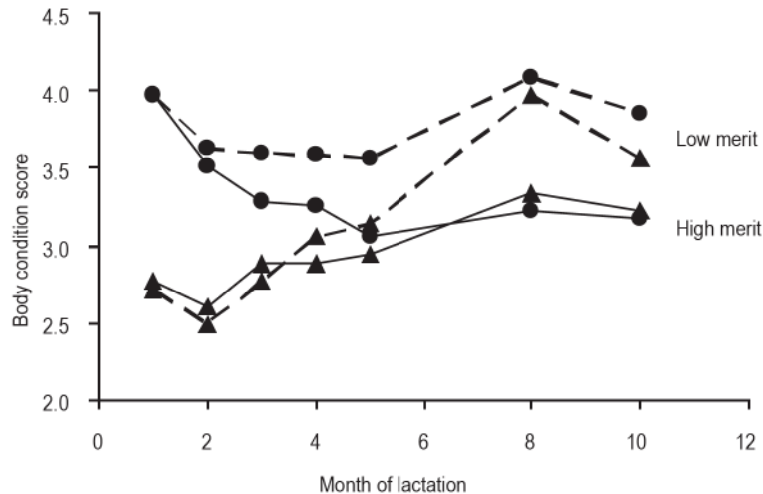
conceive; cows gaining BCS took 3.7 days less to conceive.

Increased incidence of diseases and reduced fertility in cows that are mobilising body fat could be linked to lymphocyte function and oxidative stress. Lacetera *et al.* (2005) found that BCS at calving affected the ability of cows to mount an immune response; when challenged with mitogens, lymphocytes from thin (BCS 2) cows produced more immunoglobulin M and interferon- $\gamma$  than fat (BCS 4) cows. Bernabucci *et al.* (2005) measured oxidative stress indicators in cows calving with BCS <2.5 (thin), 2.6-3.0 (medium), or >3.0 (fat). Over the first 30 days of lactation, cows in the fat group lost 0.6 BCS units; cows in the other groups lost 0.3 units. Plasma concentrations of BHBA and NEFA were twice as high in cows from the fat group as in cows from the other groups, and significant correlations were found among BCS loss, BHBA and NEFA. No difference was found in oxidative status between the thin and medium BCS groups. Cows in the fat BCS group, however, had significantly elevated plasma concentrations of reactive oxygen metabolites (ROM) and thiobarbituric acid-reactive substances (TBARS), and significantly lower erythrocyte thiol groups (ESH) and plasma superoxide dismutase (SOD). BHBA and NEFA were positively related to ROM, and NEFA were negatively related to ESH and SOD. None of the cows in this study showed clinical signs of disease or disorders. The ROM and TBARS data suggest that cows with high BCS at calving and high lipid mobilisation are more likely to suffer oxidative stress; the ESH and SOD data suggest that these cows are less able to mount an antioxidant defence. It should be noted that cows in the fat BCS group were not excessively fat; the average BCS at calving for this group was  $3.2 \pm 0.2$ , which is within the normally-recommended range and would not be expected to induce clinical signs of fatty liver.

### **Body condition score and genetic merit**

As stated previously, the biological drive for a cow to attain a target BCS appears to be as strong as the drive to attain a genetically-programmed peak milk yield. It also appears that a cow's biological target BCS is determined by genetics. In a classic study reported by Holmes (1988), cows of high genetic merit showed a lower target BCS than cows of low genetic merit; both groups of cows achieved their targets, whether they had a high or low BCS at calving (Figure 3). In a recent study, Roche *et al.* (2006) compared Holstein-Friesian cows of New Zealand origin (NZ) with higher genetic merit cows of North American origin (NA). Cows were managed to calve with similar BCS; 270-day milk yields were 5818 L for NZ cows and 6748 L for

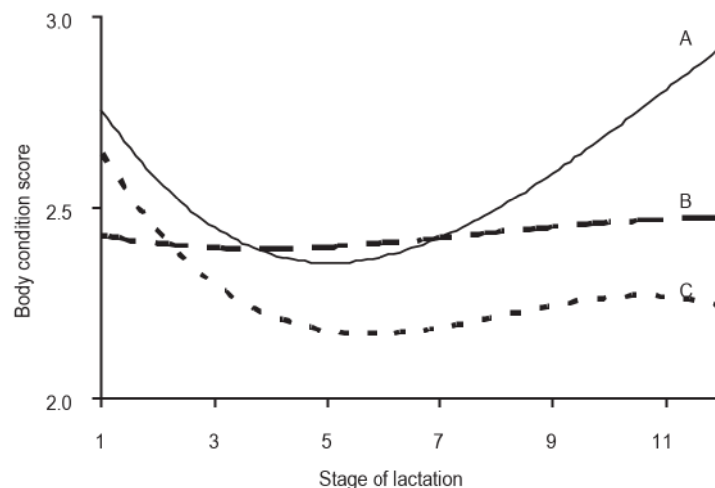
NA cows. There was no effect of genetic merit on rate of BCS loss in early lactation, but NA cows lost BCS for 14 days longer ( $P<0.01$ ) and reached a lower BCS nadir ( $P<0.001$ ) than NZ cows. Also, NA cows gained less BCS than NZ cows in later lactation.



**Figure 3.** Changes in BCS for cows of high (solid lines) or low (dashed lines) genetic merit, with high (●) or low (▲) BCS at calving. Data are from Holmes (1988) and have been converted to a 1 – 5 BCS scale.

Genetic correlations between BCS and milk yield are always negative (e.g. Veerkamp *et al.*, 2001; Berry *et al.*, 2003; Pryce and Harris, 2006) and the genetic propensity for a modern Holstein cow to lose condition is well known amongst dairy producers. Unfortunately, geneticists do not seem to be able to extract target BCS values from their databases for individual cows because BCS is usually evaluated only once, and at different stages of lactation for different cows (Jones *et al.*, 1999). It is possible, however, to evaluate bulls for changes in average BCS of their daughters at different stages of lactation. Using daughter BCS measurements for six bulls with >1,500 daughters, Jones *et al.* (1999) found significant phenotypic and genetic differences among bulls in the shape of the BCS curves followed by their daughters. Differences were found among bulls in BCS at the beginning and end of lactation, the rate of BCS loss in early lactation, the amount of BCS loss, the stage at which BCS was lowest and the subsequent rate at which BCS was regained during the latter part of the lactation. Three contrasting examples are shown in Figure 4. Daughters of bull A appeared to have a higher target BCS than daughters of bull C. Daughters of bull B experienced relatively little change in BCS

throughout lactation. These data suggest that it should be possible to select bulls on the basis of BCS curves of their daughters, but whether genetic BCS curves are related to fertility remains to be seen.



**Figure 4.** Changes in BCS throughout lactation for daughters of three bulls. Curves are derived from cubic regression coefficients of daughter BCS values. Data are from Jones *et al.* (1999), converted to a 1 – 5 scale.

Dechow *et al.* (2002) examined correlations between BCS and BCS loss in a data set of 310,000 lactation records where multiple BCS evaluations were available for individual cows. Genetics and environment appeared to produce conflicting relationships. Phenotypically, an increase in BCS at calving was associated with more BCS loss in early lactation (correlation 0.54), as expected from the biological principles outlined in this review. Genetically, however, a higher BCS at calving was correlated with less BCS loss during early lactation (correlation -0.15 to -0.48). Management and environmental conditions that increased BCS at calving resulted in more BCS loss in early lactation. However, cows that were genetically inclined to have higher BCS at calving appeared to maintain more BCS in early lactation than genetically thin cows. Therefore, selection for increased BCS at calving would be expected to reduce BCS loss in early lactation and to improve fertility, but to reduce milk yield.

Differences between phenotypic and genetic relationships are seen also for fertility traits and BCS. Pryce *et al.* (2001) studied the effects of BCS change from week 1 to week 10 of lactation on fertility in cows of high and average genetic merit. Phenotypically, an increase of one unit in BCS loss resulted in increases of 21 days to first heat in high-merit cows and 14 days



to first heat in average-merit cows; days to first service increased by 14 days per unit BCS loss in high-merit cows, but there was no effect on days to first service in average-merit cows. Genetically, however, BCS at week 10 of lactation was more strongly related to fertility traits than was BCS change because BCS change is influenced greatly by management. In addition, BCS at week 10 had a higher heritability (0.27) than BCS change (0.09). It is likely that BCS at week 10 represents the cow's target BCS, which would have a higher genetic component than BCS change.

These conflicting genetic and phenotypic results emphasise the importance of distinguishing between genetic fatness and phenotypic fatness when comparing studies or making recommendations regarding BCS. Intervention studies, in which cows are matched for breeding value or previous milk yield and in which BCS at calving is manipulated by feeding, will generally reveal phenotypic effects. Large-scale surveys will generally reveal genetic effects, whilst surveys of a small number of farms might include both genetic and phenotypic effects. Genetically thin cows will suffer greater negative energy balance than genetically fat cows if both types have the same BCS at calving. There is a strong case, therefore, for recommending a lower BCS at calving for genetically thin animals.

### **Body condition score in later lactation and the dry period**

The emphasis in the current review, as in most studies, has been on BCS at calving and changes during early lactation. This is because the first 10 to 20 weeks of lactation are the most important for lactation, health and reproduction, and metabolic stress is most likely during this period. There is an abundance of evidence to indicate that a cow has a target BCS that she attempts to reach between 10 and 20 weeks into lactation; her ability to reach that target is governed by feed constraints. In mid- to late-lactation, however, BCS usually increases (e.g. Mao *et al.*, 2004), but is sometimes maintained at target levels (e.g. Yan *et al.*, 2006), and might decrease if feed supply is restricted (e.g. Pryce and Harris, 2006).

Genetic variation is found in the shape of BCS curves, particularly the rate and extent of decline in early lactation (Jones *et al.*, 1999; Mao *et al.*, 2004; Pryce and Harris, 2006), which are clearly related to genetic BCS targets. Genetic and nutritional effects in later lactation are less obvious. In the study of Mao *et al.* (2004), three breeds of cow, fed on two planes of nutrition, reached different BCS nadirs, at different times postpartum. However, the rate of increase in BCS post-nadir was similar for all breeds and feeding systems. On the other hand, rate of increase in BCS post-nadir was influenced



by parity; from the first to later parities, BCS nadirs became progressively deeper, but BCS increased post-nadir at faster rates for cows in later parities, especially cows on a low feed level. Chilliard *et al.* (1991) also found no difference in BCS gain between two groups of cows consuming concentrates at 0.27 kg/d or 1.26 kg/d whilst grazing between weeks 20 and 39 of lactation. These results suggest that absolute BCS in later lactation is related to a cow's genetic BCS target in early lactation, but rate of increase in BCS is fixed.

Some studies have observed little or no change in BCS in later lactation, even with different levels of feeding. For example, Yan *et al.* (2006) examined energy partitioning throughout lactation in high-merit Holstein (H) and Norwegian (N) breeds of cow fed on high (H) or low (L) planes of nutrition. From week 15 to week 44 of lactation, HH cows maintained BCS at 2.5; NH cows maintained BCS at 3.1; NL cows maintained BCS at 2.8; in HL cows, BCS decreased from 2.3 to 2.2. These results suggest that BCS targets do not change as lactation progresses in high-merit cows. In later lactation, energy intake was regulated by energy output and the desire to maintain a constant BCS, except in HL cows, where low dietary energy concentration limited energy intake. Similar effects were observed by Chilliard *et al.* (1991) in cows treated with bovine somatotropin (BST). Compared with controls, cows treated with BST lost more BCS in early lactation and maintained this lower BCS until the end of lactation when fed on a high concentrate allowance.

Cows selected under conditions of regular feed restriction appear to respond differently to those selected where feed is not restricted. Pryce and Harris (2006) studied BCS changes in New Zealand cows, which often experience periods of reduced pasture availability in late lactation. Under these conditions, cows that maintained relatively high BCS in early lactation had higher total yields of milk solids because they had body reserves available for mobilisation in late lactation. Roche *et al.* (2006) found that New Zealand Holsteins reached their BCS nadir 14 days earlier than North American Holsteins, and then gained BCS at a faster rate for the rest of lactation, particularly with high concentrate supplementation. These results suggest that New Zealand cows are more sensitive to feed supply and give greater priority to energy reserves when partitioning nutrients.

It is not clear whether BCS usually increases in later lactation because BCS targets vary with stage of lactation, the feedback signal is down-regulated, or different constraints assume priority. Friggens (2003) interpreted increases in BCS during mid- to late-lactation as the cow's investment in future offspring. However, this does not explain increases in BCS observed in non-pregnant cows, nor does it account for the lack of change in BCS observed in some studies. A more likely explanation is that accretion of adipose tissue is passive during mid- to late-lactation. Declining milk yield, coupled with relatively

high intake of dry matter, would lead to increases in insulin status. Increasing insulin would encourage deposition of body fat, but feedback from leptin and its associated factors would have a delayed effect on dry matter intake. Chilliard (2001) pointed out that there are several time-scales in ruminant tissue leptin yield and plasma leptin regulation: months for the effect of body fatness; weeks for physiological status or environmental conditions; days for the daily level of intake; and hours or minutes for meal intake or acute metabolic and hormonal regulations.

During the dry period, BCS is more likely to increase than during lactation because plasma insulin is considerably higher in dry cows (Grum *et al.*, 1996). For this reason, producers who wish to increase BCS of cows that are thin at drying off often feed dry cows above their requirements for maintenance and pregnancy. On the other hand, cows with a high BCS at drying off are often fed on a low plane of nutrition so that body fat is mobilised in late gestation. Such practices should be avoided. As shown in previous sections, there is no benefit from higher BCS at calving, which will increase negative energy balance and disease susceptibility. Cows that lose BCS during the dry period are more prone to dystocia (Gearhart *et al.*, 1990; Keady *et al.*, 2005) and are more likely to be culled in their subsequent lactation (Gearhart *et al.*, 1990). The only sensible strategy is to monitor BCS in late lactation and ensure that the cow is dried off with a BCS that is desirable at calving (see next section). Recently there has been interest in reducing the length of the dry period from the traditional 60 days to 30 days or less. Preliminary studies suggest that shortening the dry period improves cow health and reproduction; part of this effect is probably due to reduced BCS gain in the dry period and reduced BCS loss during lactation, as days dry decreased (Rastani and Grummer, 2005).

### **Body condition score targets for production and fertility**

In most countries, increases in milk yield over the past 30 years have been accompanied by reductions in fertility because of unfavourable genetic correlations between milk yield and reproductive traits (Pryce *et al.*, 2004). Poor reproductive performance in high-yielding dairy cows is usually attributed to negative energy balance (Butler, 2003; 2005). For this reason, dairy producers are usually advised to avoid over-fat cows and to feed diets with a high energy concentration. Many papers state that negative energy balance is unavoidable in dairy cows of high genetic merit. As shown in previous sections of the current review, however, this is not true. Cows that calve with a BCS below their target BCS can be in positive energy balance.

Because BCS has a major influence on feed intake and negative energy balance, it is important to evaluate BCS recommendations in relation to genetic targets of cows. If genetic targets decrease with selection for higher milk yield, negative energy balance will become greater if BCS at calving remains high.

According to a brief survey of websites, current UK recommendations for BCS at calving (1-5 scale) are 3.0 (MDC, 2006; DARDNI, 2006) and 2.5 to 3.0 (Defra, 2001). Recommendations in USA and Canada are somewhat higher, but recommendations in New Zealand and Australia are similar to UK values (Table 1). Most recommend that BCS does not fall below 2 to 2.5 at service. Importantly, they also recommend that BCS is the same at drying off and at calving. UK recommendations are 1.3 to 1.8 units (1-5 scale) lower than those made in the 1970s, reflecting either increased awareness of effects of BCS at calving on negative energy balance and fertility, or the difficulty of getting Holstein cows to reach the previous targets.

**Table 1.** Recommended BCS for dairy cows at drying off, calving and service, published on official web sites accessed in September 2006.

<i>Country, organisation</i>	<i>BCS at</i>		<i>Reference</i>
	<i>Drying off/ calving</i>	<i>Service</i>	
UK, Defra	2.5 – 3.0	2.0 – 3.0	Defra (2001)
UK, MDC	≤ 3.0	2.0 – 2.5	MDC (2006)
UK, DARDNI	≤ 3.0		DARDNI (2006)
USA, Nebraska	3.5 (3.0 – 4.0)	2.0 (1.5–2.0)	Nebraska (2005)
USA, Penn State	3.25 – 3.5	2.5 – 3.5	Ferguson (2006)
USA, Wisconsin	3.0 – 3.5	2.0	Wattiaux (1999)
Canada, Ontario MAFRA	3.5 (3.0 – 4.0)	2.5 – 3.5	Parker (1996)
New Zealand, Dexcel	2.8 – 3.0 <sup>1</sup>		MacDonald and Roche (2004)
Australia, Victoria DPI	3.3 – 3.9 <sup>2</sup>		Moran (2006)

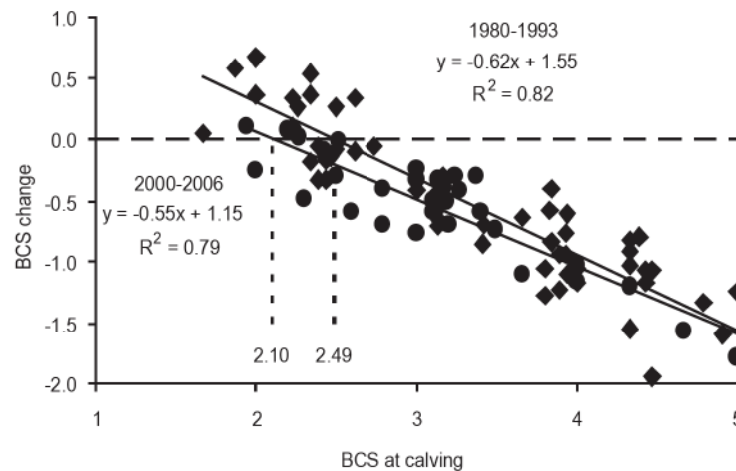
<sup>1</sup> Published recommendation: 5–5.5 on 1–10 scale.

<sup>2</sup> Published recommendation: 5–6 on 1–8 scale.

There is evidence from carcass studies that Holstein dairy cows have less external fat and more internal fat than beef breeds (Baber *et al.*, 1984) and, possibly, than Friesians (Kempster *et al.*, 1988). It is likely, therefore, that a modern Holstein cow will have a greater total body fat content than a Friesian cow at the same BCS. In view of the numerical dominance of the Holstein breed, the relevance of BCS could be questioned because BCS assesses subcutaneous fat rather than internal fat. However, strong relationships

between BCS and total body fat suggest that BCS is still the best method for monitoring changes in fat reserves.

Recent studies suggest that the lower UK targets are appropriate for optimising production, health and fertility in high-yielding Holstein cows. The relationship between BCS at calving and change in BCS during the first 10-12 weeks of lactation is similar to that seen 20 years ago (Figure 5). BCS at calving predicted to give no change in early lactation has decreased from 2.49 in older studies to 2.10 in recent studies, suggesting that current recommendations are slightly above average biological targets. However, a loss of 0.5 BCS units is considered acceptable and provides a safety margin to allow for variation among cows within a herd. As with any biological parameter, there will be a spread of BCS values and responses among cows. Therefore, it is desirable to assess every cow in a herd at monthly intervals. Unfortunately, few dairy farmers are prepared to make such a commitment. Developments in automatic monitoring of BCS (e.g. Coffey *et al.*, 2003) or a remote BCS service based on digital images (Ferguson *et al.*, 2006) might alleviate the situation and provide early warnings of inappropriate nutrition. In the meantime, attention should be focussed on cows in late lactation and the dry period to ensure that they calve with a BCS of 2.5 to 3.0.



**Figure 5.** Relationship between BCS at calving and change in BCS over the first 10 – 12 weeks of lactation for studies published 1980 – 1993 (◆) compared with studies published 2000 – 2006 (●). Vertical dotted lines show BCS at calving that result in zero change in BCS, which has decreased from 2.49 in earlier studies to 2.10 in recent studies. Data are group means (converted to a 1 – 5 scale) from Bernabucci *et al.* (2005), Bouchier *et al.* (1987), Garnsworthy and Huggett (1992), Garnsworthy and Jones (1987; 1993), Garnsworthy and Topps (1982), Garnsworthy, Webb, *et al.* (2006: unpublished), Grainger *et al.* (1982), Horan (2005), Jones and Garnsworthy (1988; 1989), Lactera *et al.* (2005), Land and Leaver (1980; 1981), MacMillan *et al.* (1982), Meikle *et al.* (2004), Reist *et al.* (2003), Roche *et al.* (2006), Stockdale (2000; 2004; 2005), Treacher *et al.* (1986), Yan *et al.* (2006).

## **Conclusions**

As stated in the introduction, BCS is a useful management tool for assessing the nutritional status of dairy cows. The numerical scale used is not important within farms or studies, although further comparisons of scales might be useful for comparing studies and for comparing animals across national boundaries. Even though BCS is subjective, consistency can be achieved within and between operators, especially with training. BCS correlates well with body fat content and provides a better indication of energy reserves than live weight.

Changes in BCS during early lactation highlight the role of body fat in controlling feed intake. The strong relationship between BCS at calving and change in BCS provides compelling evidence that cows have a target BCS in early lactation. Cows that are fatter than their target BCS mobilise body fat; cows that are thinner than their target BCS gain body fat. The rate at which a cow changes BCS towards its target is affected by diet composition as well as current BCS; low energy and high protein diets increase BCS loss in cows that are above target BCS; low energy diets reduce BCS gain in cows that are below target BCS.

Recent reports indicate that average target BCS is lower now than it was in the 1980s. This is because cows selected for higher milk yield over the past thirty years are genetically thinner, so they have a stronger drive to mobilise body fat. Consequently, cows of high genetic merit are likely to experience deeper and more prolonged negative energy balance in early lactation. Negative energy balance is undesirable because it reduces reproductive performance and increases susceptibility to diseases. Therefore, short-term financial gains in extra milk production from cows that are fat at calving will be offset by longer-term financial losses through premature culling. Changes in BCS during the dry period are undesirable, so BCS should be monitored and adjusted in late lactation to ensure that cows are dried off with a BCS appropriate for calving.

To reduce the impact of negative energy balance on cow health and performance, BCS at calving should be no more than 0.5 BCS units above a cow's target BCS. Cows of low genetic merit for milk yield (target BCS 2.5-3.0) should calve with BCS of 3.0 or less; cows of high genetic merit for milk yield (target BCS 2.0-2.25) should calve with BCS of 2.75 or less. The most important message for producers is that increasing BCS at calving exacerbates negative energy balance problems instead of overcoming them.

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