THE ENERGY REQUIREMENTS OF THE LACTATING ANIMAL

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SUMMARY

High yielding dairy cows are in negative energy balance for the first 6-8 weeks of lactation and mobilize body energy reserves in order to make good their energy deficit. The paper deals mainly with the metabolism of the dairy cow during this period of early lactation. It discusses the metabolic and endocrinological implications of feeding high levels of concentrates during this period and questions the present interpretation of why the high-yielding cow in negative energy balance will respond to increased absorption of protein.

ENERGY BALANCE DATA

Calorimetric experiments carried out mainly in the last 25 years have provided the basis for making reasonably accurate assessments of the energy requirements of the lactating animal. These have been reviewed on a number of occasions (e.g. Flatt et al. 1972; van Es and van der Honing 1979) and it is not intended to discuss them here except to use the information now available on the efficiency of conversion of metabolizable energy (ME) into milk energy (\(k_c; 0.60-0.65\)) and on the maintenance requirement of lactating animals (490-530 kJ/kg\(^2\)) to derive the estimated energy requirements of a high yielding cow throughout her lactation. Table 1 gives calculated data for one Friesian cow in the Rowett Herd which produced 7500 kg of milk in 1972. From weeks 2-11 of lactation the cow was given ad. lib. access to an experimental diet of 35% hay, 65% concentrate (E. R. Ørskov, L. Issase, G. w. Reid and C. Tait personal communication). Feed intake, milk production and liveweight were recorded daily. Thereafter it was returned to the Institute herd. As yet data on the ME content of the ration are not available and so values of 9 MJ/kg DM for hay (FEU 1978) and 12 MJ/kg DM for concentrate (FEU 1981) have been used in the calculations to illustrate the fact that over the first 6-7 weeks of lactation the cow was in serious negative energy balance and was therefore deriving the extra energy required for milk synthesis from mobilized body tissue, presumably fat. Thereafter voluntary food intake increased and milk production started to decrease and so the cow moved into positive energy balance. The computed energy balance data are reasonably compatible with the changes in liveweight.

Bines (1979) demonstrated a similar relationship between milk production, voluntary feed intake and weight change of Friesian heifers given a 40% roughage 60% concentrate ration (see Figure 1). During the second and third month of lactation the heifers lost approximately 0.5 kg/d body weight, presumably mainly fat (39.5 MJ/kg) and this coincides well with a calculated energy deficiency of 15 to 20 MJ/d. Later in the lactation cycle when milk yield had fallen away from the peak output and voluntary feed intake had increased to a maximum (3 to' 5 months) the animals consumed more than sufficient energy for milk and maintenance requirements and so could then replenish the body reserves used during peak lactation. Bauman and Elliot (1983) give a further example of this same phenomenon in their recent review on the control of nutrient partition.

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TABLE 1 - Energy supply and demand of Friesian cow giving 7500 kg milk over total lactation

<table>
<thead>
<tr>
<th>Wk of lactation</th>
<th>Milk Prod.† (kg/d)</th>
<th>Milk Compos.†</th>
<th>Energy Req.‡ (MJ/d)</th>
<th>Energy Supply†‡ (MJ/d)</th>
<th>Livewt.† (kg)</th>
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<td>3.62</td>
<td>2.22</td>
<td>151</td>
<td>''</td>
</tr>
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</table>

† Data for weeks 2-11 from Expt of E. R. Ørskov, L. Isasse, G. W. Reid and C. Tait (personal communication).

‡ Assuming ME content of 35% hay 65% conc. ration = 11 MJ/kg DM.

‡‡ Data from MMB computerized records.

* Calculated assuming energy content of milk fat = 39.5 kJ/kg; protein = 23.5 kJ/d; milk lactose = 47 g/l; energy content 16.5 kJ/g; $k = 0.65$; maintenance req. = 510 kJ/kg.
Fig. 1. Milk yield, Food Intake and Liveweight of Friesian heifers (Bines 1979).

METABOLIC CONSIDERATIONS

Precursors of milk constituents. The data presented in Table 1 and Figure 1 illustrate the critical nature of the balance between energy supply and demand in the first two to three months of lactation. It is generally accepted that secretory ability of the mammary gland is determined by events which occur both before and after puberty and during pregnancy (Baldwin 1966; Cowie et al. 1980) and that the ability of animals to realise their potential depends on the availability of nutrients presented to the mammary gland. We therefore need to consider what governs this supply, not just in terms of energy per se but in terms of specific metabolites needed for the biosynthesis of milk constituents.

Again there are many research publications and reviews which discuss milk composition (e.g. Balch 1972; Oldham and Sutton 1979) and the biochemical and cytological mechanisms associated with the biosynthesis of milk constituents by the mammary gland (e.g. see 17th Nottingham Easter School; Falconer 1971; or Lactation, A Comprehensive Treatise; Larson & Smith 1974; or Biochemistry of Lactation; Mepham 1983) and it is not intended to discuss these mechanisms in detail here. It is necessary however to consider the various roles of the major precursors of milk constituents and how these may interrelate when alterations in milk volume and composition are brought about by manipulation of the
diet. Figure 2 presents a simplified diagram of the main metabolic pathways leading to the formulation of milk constituents.

Most of the lipid in milk (98%) is in the form of triglycerides. Approximately 50-60% of these are obtained from preformed plasma glycerides, mainly the chylomicrona and low-density lipoproteins (see Figure 1, Reaction 1). The remaining 50-50% are formed in the mammary gland from acetate and \( \beta \)-hydroxybutyrate (Reactions 2 & 3). Glucose is not a precursor of fatty acids in ruminant mammary tissue, but it is needed as a precursor of some if not most of the NADPH required for the synthesis of fatty acids (there is still some debate as to the relative contributions of the pentose cycle and the isocitrate dehydrogenase mechanism in the provision of NADPH for fatty acid synthesis in ruminant mammary tissue; see Smith et al. 1983) as well as for the provision of glycerol-P for the subsequent esterification of the fatty acids. Most of the milk proteins (90 to 95%) are synthesised in the mammary gland from a free amino acid pool which is in equilibrium with that of the blood stream (Reaction 5). The remainder are taken up as preformed proteins from blood (e.g. serum albumin and immuno-globulins (Reaction 6). Milk lactose is synthesised from glucose and UDP-galactose, itself derived from glucose (Reaction 7 & 8) and here there is evidence to suggest that the rate of biosynthesis of lactose is controlled by a complex mechanism at the level of the Golgi apparatus involving \( \alpha \)-lactalbumin which exerts an influence on the enzyme galactosyltransferase.

The biosynthesis of milk therefore requires provision at the mammary gland of the correct balance of

(i) Acetate, \( \beta \)-hydroxybutyrate and blood lipids to produce milk lipid

(ii) Glucose or glucogenic precursors to provide milk lactose and at least some of the reducing equivalents as well as the glycerol phosphate required for synthesis de novo of milk lipids.

(iii) Amino acids for the synthesis of milk protein

(iv) Precursors of TCA cycle intermediates to provide the energy needed for the synthetic process.

The supply of these precursors is influenced both by the digestive physiology of the animal and its endocrinological status. The following section attempts to indicate why problems can arise when the normal pattern of metabolite availability is distorted.

Low-fat Syndrome. Problems with voluntary feed intake in early lactation cause the availability of energy from dietary sources to be limiting (see Table 1) and so it is usual to maximize energy intake by providing considerable quantities of energydense feedingstuffs, i.e. concentrates. Problems can arise with such rations in that milk fat levels may fall. At first sight this might appear to be caused by a simple alteration in the balance of nutrients being presented to the mammary gland. Such rations tend to cause a reduction in the molar ratio of acetate and an increase in the molar ratio of propionate present in the rumen and this might be expected to increase the glucose and decrease the acetate which is presented to the mammary gland. However Bauman et al. (1971) showed that such changes in the molar ratio of the VFAs do not necessarily mean that the production rate of acetate is reduced on a high grain ration. Rather the production rate of propionate is substantially increased and this is the main reason for the low milk fat syndrome. The propionate
Fig. 2. SIMPLIFIED DIAGRAM OF METABOLIC PATHWAYS INVOLVED IN BIOSYNTHESIS OF MILK CONSTITUENTS

- **LACTOSE**
  - UDP-Galactose
  - Glucose (g-6-p) → NADP

- **Blood lipids**
  - TRIGLYCERIDES

- **Tissue Catabolism**
  - Aspartate → Oxaloacetate → Citrate

- **Absorbed Protein**
  - Amino acids
  - Essential
  - Non-essential
  - Protein

- **Blood protein**
  - Glutamate

- **Acetate**
  - 8-Hydroxybutyrate

- **ATP**
  - Isocitrate
  - α-Ketoglutarate
  - CoA

- **Blood lipids**
  - Fatty acids

- **UDP**
  - 6-Phosphogluconate
  - Glycerol-P

- **Blood lipids**
  - G-3-p
once it is absorbed is converted to glucose in the liver. When this glucose is released into the blood stream it causes an increased insulin secretion which then partitions not only the glucose but also glycerides and acetate towards lipogenesis in adipose tissue; Obstvedt et al. (1967) reported that the lipogenic enzymes in adipose tissue are elevated in lactating cows given high grain diets which stimulate high circulating levels of blood insulin. 

This concept of insulin partitioning nutrients away from the mammary gland is by no means new. Indeed it is fitting to recall that work in the Department of Nutrition and Chemical Pathology in the University of New England in the late 1950s demonstrated a reduction in milk fat when intravenous glucose (2 kg/d for 48 h) was given to lactating cows (Vallance & McClymont 1959). Three years later these workers were perhaps the first to suggest that the low-fat syndrome might be mediated via an insulin response (McClymont & Vallance 1962) which would result in a reduction in the mobilization of tissue glycerides. Indeed they reported a reduction in the plasma glyceride level in one cow whose milk fat had been reduced during an intravenous glucose infusion.

Twenty years on from that early work recent studies at the NIRD are perhaps starting to provide an indication of how this insulin effect can be alleviated to some extent. Sutton, et al. (1982) fed lactating Friesian cows four rations containing hay plus 60, 70, 80 and 90% concentrates under two different feeding regimens. In one the animals were given hay plus concentrates twice daily at 0600 and 1630 h and in the other the animals received their allocations of hay at 0600 and 1645 h but their allocation of concentrates were automatically dispensed in six equal amounts at 4 h intervals. With all but the highest concentrate rations the more frequent feeding gave more milk and with a higher fat content. Cows on the 90% concentrate ration gave less milk on the more frequent feeding (21.4 versus 23 kg/d) but their milk fat content was markedly higher (29.7 versus 17.9 g/kg). Rumen samples showed a progressive decrease in the molar proportion of acetate and an increase in the molar proportion of propionate as level of concentrate was increased. There was no attempt to study the diurnal patterns of VFA production on the two feeding regimens but frequently taken blood samples showed that the patterns and levels of plasma insulin were markedly different in animals given the two feeding regimens. Four hourly feeding of concentrates halved plasma insulin concentrations on the 90% concentrate diet and on the other diets it greatly reduced the post-feeding "spikes" of insulin presumably because propionate absorption was more evenly distributed throughout the 24 h.

Supplementation at Pasture. The NIRD experiments which illustrate the benefits of feeding concentrates little and often could have considerable significance to the grazing cow. Recent studies on the digestive physiology of grazing ruminants might suggest that frequent feeding of concentrates could have the added advantage in the grazing situation of trapping more ammonia as bacterial protein in the rumen. Grazing experiments with steers at the Grassland Research Institute (Lazardo et al. 1982) tend to confirm earlier findings with sheep in New Zealand (MacRae & Ulyatt 1974) that ruminants given fresh pasture can absorb (waste) a considerable proportion of their dietary nitrogen anterior to the duodenum (20-35% in sheep, MacRae & Ulyatt 1974; 20-50% in steers, Lazardo et al. 1982). If it were practicable, feeding concentrates little and often whilst the animals graze instead of say twice daily at milking would help to even out the energy supply to the rumen microbes
and improve the efficiency with which they convert the available NPN into microbial protein (see discussion by MacRae & Reeds 1979) and thus the amounts of protein which pass out of the rumen. This in itself could have a beneficial effect on milk production, particularly in early lactation when the animal is in negative energy balance.

Protein Supplementation in early lactation. There are now many reports that additional protein supplied to the intestines of cows, sheep and goats either as abomasal infusions or given as dietary supplements of relatively undegrada ble fish meal or protected protein can increase milk yield (see Table 2) yet it is doubtful whether the mechanisms by which the protein is stimulating the increased yield are fully understood. The effect is generally attributed to the provision of extra essential amino acids for milk protein synthesis (see Clark 1975; Ørskov et al. 1977) and experiments such as those reported by Ørskov et al. (1977) in cattle and Ranaway & Kellaway (1977) in goats which indicated that the increase in milk yield resulting from abomasal infusions of casein did not occur when glucose was infused have been cited to substantiate this theory. Certainly in some of the studies given in Table 2 the diets fed were low in protein (e.g. Ranaway & Kellaway 1977a, 1977b 11% crude protein; Ørskov et al. 1977 13% crude protein) and might have caused a protein limiting state, but in many others the crude protein content of the basal ration was above 15% (e.g. Broderick et al. 1970; Tyrrell et al. 1972; Derrig et al. 1974).

It is also interesting to note that the so called "limiting amino acid effect" only seems to occur in animals drawing heavily on body reserves, when the extra protein appears to stimulate the mobilization of depot fat to enhance the energy supply to the animal; this was demonstrated in the comparative slaughter experiments of Cowan et al. (1981). Furthermore the stimulation of additional milk protein has seldom been very high relative to the amount of protein administered (usually 10-25%) whereas because the extra milk produced also contains extra fat and lactose the stimulation of extra energy secreted in milk per unit of protein energy administered is generally much higher (30-90%). In one experiment reported as part of a PhD thesis (Spechter, 1972) lower than experimentally usual levels of protein were infused into the abomasum of two cows giving 20 kg of milk/d (170 g casein/d as compared to the usual 400 g/d). In that experiment the efficiency of conversion of protein into milk protein was much higher (82%), with the corresponding stimulation of milk energy output per unit of energy supplied as casein being 315%.

It is tempting therefore to speculate as to whether the current concepts on protein supplementation are exclusively correct or whether the additional protein given to the energy-deficient lactating animal may in fact be stimulating milk output by other mechanisms associated perhaps with the provision of additional nutrients other than amino acids and/or with the endocrinological responses elicited in the animals.

One possibility is that the glucogenic amino acids of casein are providing extra glycolytic intermediates. This theory requires the consideration of the central role of the four carbon intermediate oxaloacetate. It can be seen from Figure 2 that oxaloacetate has the dual function of both supplying energy to the whole animal (i.e. it is the catabolic intermediate which condenses with acetyl-CoA to form citrate in the initial steps of the energy yielding reactions of the TCA cycle
<table>
<thead>
<tr>
<th>Species</th>
<th>Method of administration</th>
<th>Milk Production kg/d</th>
<th>% Increase</th>
<th>References</th>
</tr>
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<tr>
<td>cows</td>
<td>casein per abomasum</td>
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(Reaction 9)) and acting as an obligatory intermediate in the synthesis of glucose, glycerol phosphate and the $\text{NADPH}_2$ produced in the pentose phosphate pathway from all the important precursors other than glycerol itself (Reaction 10). In lactation the animal's requirements for both glucose, as a precursor of milk lactose and energy for general synthetic processes associated with milk production increase very considerably and the availability of oxaloacetate may become a critical factor in the overall performance of the animal. Extra oxaloacetate could provide both extra milk lactose (which along with $\text{K}^+$ and to a lesser extent $\text{Na}^+$ and $\text{Cl}^-$ provide the main osmoregulation of milk volume (Wheelock et al. 1965)) and additional energy, particularly where a proportion of that energy is derived from B-oxidized fatty acids.

This postulate might be more compatible than the "limiting amino acid" theory with the observation that when protein is infused into the abomasum the ratio of essential to non-essential amino acids in peripheral blood rises (Broderick et al. 1970; Derrig et al. 1974; Ranaway & Kellaway 1977a). Most physiologists would nowadays advise extreme caution when interpreting blood profile data however such an observation would be more consistent with a possible preferential utilization of non-essential amino acids in the "glycolytic intermediate theory" rather than with a preferential utilization of essential amino acids which might be expected with the "limiting amino acid theory?"

On the other hand, it could be argued that if the protein is merely a way of providing extra glycolytic intermediates the provision of extra glucose or propionate would be as effective if not more so than the extra protein (see Fig. 2), yet the reasons put forward to support the "limiting amino acid" theory is that glucose given per abomasum will not stimulate milk production as does protein (Ørskov et al. 1977; Ranaway & Kellaway 1977b).

Such reasoning does not of course take into account the endocrinological responses which different infusions will cause in the animals. We have discussed earlier the increased levels of circulating insulin which result from additional glucose entering the blood stream and the fact that this tends to partition nutrients away from the mammary gland and towards peripheral tissues. Absorption of additional amino acids from the small intestine will also tend to increase circulating insulin levels (Fajans et al. 1967; Barry et al. 1982). However, at the same time it will tend to increase the circulating levels of glucagon (Unger et al. 1969; Barry et al. 1982), growth hormone (Swan 1976) and also possibly the glucocorticoids. These other hormones are known to counteract the nutrient partition effects of insulin and so the advantage of protein over glucose or propionate might be associated with its ability to provide some extra amino acid for protein synthesis and some extra oxaloacetate without disrupting the hormonal balance to one which is unfavourable to the provision of all nutrients to the mammary gland.

Yet a further alternative effect of additional protein—might simply be its stimulation of circulating levels of growth hormone. Swan (1976) commented that when his cows were given casein infusions which stimulated an extra 3 kg milk per day the response of circulating growth hormone was "startling" (levels rose from 5 to 30 ng/ml within 1 h of starting the casein infusion. Tyrrell et al. (1983) recently demonstrated that 51 iu of growth hormone given subcutaneously to cows which were giving 27.5 kg milk/d increased their energy output in milk by 25% and caused tissue energy balance to fall from -1.6% of energy intake to -13.7% of energy
It has been argued that the nutrient partition effect of extra growth hormone is more prevalent in mid lactation than in early lactation because of a saturation of the system with growth hormone in early lactation (Bines & Hart 1982). However the experiment reported by Swan (1976) was conducted in early lactation and Peel et al. (1983) recently obtained a 15% increase in milk yield when growth hormone was administered in early lactation.

In conclusion, it can be stated that the calorimetric data referred to in the first part of this paper provides a reasonably accurate means of determining the energy requirements of the dairy cow and so can be used as the basis for a ration formulation system. However such studies are unlikely to increase our understanding of the control mechanisms which regulate the efficiency and productivity of the modern dairy cow. If we are to continue to improve individual animal and whole herd performance in the future we will need to gain a better understanding of these mechanisms. The latter part of the paper illustrates the way in which research studies carried out on the digestive physiology, endocrinology and metabolism of the lactating cow over the last 20 years have started to provide the basis for determining the rationale behind at least some of the practical observations of dairy farmers and applied research workers. We are obviously some way from a complete understanding of all the control mechanisms and future research on the mechanisms which mobilize body reserves in early lactation and why certain supplements stimulate this mobilization might be particularly fruitful.

REFERENCES


