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Acetate metabolism in lactating sheep

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1. The metabolism of acetate, glucose and D(–)-3-hydroxybutyrate was studied in lactating and non-lactating sheep in vivo. Special consideration was given to the utilization by hind-limb muscle in both groups of sheep and the uptake of nutrients by the lactating mammary gland was also measured.

2. The entry of acetate into the circulation (mmol/h per kg body-weight) was similar in all experimental animals at a given arterial concentration of acetate. However, normal lactation was associated with a reduced extraction of acetate by muscle and the 'spared' acetate was comparable with that removed by the udder. Feeding lactating ewes a 700 g concentrate/kg ration tended to prevent this redistribution of acetate utilization.

3. The muscle of non-lactating ewes utilized sufficient glucose, when corrected for lactate release, to account for 57% of the oxygen utilization by muscle. In lactation this fell to 32%, largely because of an increased lactate production. D(–)-3-Hydroxybutyrate utilization by muscle accounted for 16–17% of the O_2 consumed by the muscle in non-lactating and lactating sheep.

4. Lactating mammary gland metabolism in sheep was similar to published values for dairy cows and goats. Thus the extraction (%) of glucose, O_2, acetate and D(–)-3-hydroxybutyrate was 25, 28, 62 and 53 respectively. Blood flow was 529 ml/min per kg udder and the ratio, blood flow: milk flow was 475. Glucose used by the udder relative to the whole animal utilization rate may be less in sheep than in cows and goats, but the comparable proportion for acetate is as large or larger than in these species.

The importance of acetate as a substrate for both fatty acid synthesis and oxidation in the mammary gland of dairy goats and cows is well documented (Davis & Bauman, 1974; Linzell, 1974). Approximately half the utilized acetate is oxidized and the remainder supplies most of the fatty acids with a chain length of less than that of palmitic acid, and half the palmitate. The supply and utilization of acetate in lactating ewes is not known. Davis & Bickerstaffe (1978) and Davis *et al.* (1978) have measured the metabolism of glucose and amino acids by the sheep mammary gland. They concluded that the sheep had a particularly high mammary blood flow and that metabolism of glucose was relatively less efficient than that of the cow and goat. No other studies of sheep mammary gland metabolism are available.

In lactating cows (Davis & Brown, 1970) and ewes (Oddy, 1978) eating a diet rich in concentrates and low in fibre, the concentration of milk fat may decrease resulting in the low milk-fat syndrome. The factors that can produce this are complex (Annison *et al.* 1974) and have been recently summarized by Engvall (1980). In the latter work, field cases were studied and from the forty-three cows with a low fat content of milk, the milk fatty acids showing significant reduction were those synthesized from acetate and D(–)-3-hydroxybutyrate. It is thus possible that for many field cases acetate may be limiting. Quantitatively acetate is a more important blood-borne precursor of milk than D(–)-3-hydroxybutyrate (Davis & Bauman, 1974). Factors affecting the availability of acetate to the mammary gland include (*a*) the rumen production rate, (*b*) the endogenous secretion rate and (*c*) the utilization of acetate by non-mammary tissue. Davis & Brown (1970) and Annison *et al.* (1974) suggested that despite the large change in the molar proportion of rumen acetate that occurs when cows are given a 900 g concentrate/kg ration, the production rate of acetate in the rumen does not change. The results of Pethick *et al.* (1981)

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suggest that endogenous acetate production may be relatively constant in a variety of circumstances. Thus the last factor, i.e. competition between mammary and non-mammary tissue for acetate, may be the most tenable, although in no studies have there been attempts to contrast the metabolism of acetate by the mammay gland and other tissues. In this study the metabolism of acetate is measured in the whole animal and muscle tissue of non-lactating and lactating sheep. Lactating mammary gland metabolism is reported and contrasted with that of other species. Finally, some indication of the interaction of diet and acetate utilization in lactating ewes is reported. Some of the results have been reported in preliminary form (Pethick & Lindsay, 1981).

METHODS AND MATERIALS

Animals and diet

Clun Forest sheep, aged between 3 and 5 years, were used throughout. Non-lactating sheep (mean body-weight 53 kg) were fed a daily ration of 1000 g chopped hay (g/kg; 500 lucerne, 500 cocksfoot) and 100 g rolled oats fed as two equal sized portions daily (09.00 and 16.00 hours) with an estimated metabolizable energy intake of 0·15 MJ/kg body-weight (Ministry of Agriculture Fisheries and Food, 1975). Lactating ewes (mean body-weight 63 kg) were offered 1000 g chopped hay and 200 g concentrates at 09.00 hours and 1000 g long hay and 200 g concentrates at 16.00 hours. The hay was not always completely eaten, but the estimated metabolizable energy intake of the ewes was 0·26 MJ/kg body-weight. One lactating ewe was given the ration in a relatively constant manner, i.e. one-quarter of the ration at each of the following times: 08.00, 13.00, 18.00 and 23.00 hours. Three lactating ewes (mean body-weight 57 kg) were given a 700 g concentrate/kg diet consisting of 200 g chopped hay and 500 g concentrates fed at 09.00 hours and 16.00 hours. Estimated metabolizable energy intake was 0·25 MJ/kg body-weight per d and was fed for 3–4 weeks before experimentation. The concentrate was a pelleted mixture by relative weight: ground barley 0·59, flaked maize 0·1, rolled oats 0·1, soya-bean meal 0·1, molassine meal 0·1, plus a supplement of vitamins plus minerals. Sheep were housed at least 6 weeks before experimentation.

All sheep were prepared with indwelling cannulas in arterial and recurrent tarsal veins as described by Domanski *et al.* (1974). Mammary venous blood was sampled by placing a cannula in a superficial caudal epigastric ('mammary' or 'milk' vein) vessel. In Clun Forest sheep venous drainage from the udder occurs generally through several epigastric veins as well as the external pudics. It is probably not practicable to channel drainage through a single superficial vessel as has been done in the goat and cow. The cannula was, therefore, passed caudally to the centre of the udder, so that the tip lay near the confluence of the epigastric and pudic vessels. Cannulation was carried out under general anaesthesia, induced by Pentothal (Abbott Laboratories Ltd, Queenborough, Kent) and maintained with Halothane (Imperial Chemical Industries Ltd, Macclesfield, Cheshire) 2–3 d before experiments. The cannulas were tunnelled subcutaneously to emerge on the flank, in order to avoid interference by the lambs.

Treatments

Non-lactating sheep were housed in Babraham-type metabolism cages (Harrison, 1974) for 1 week before experiments. Lactating ewes were housed, 24 h before experiments, in a system employing two metabolism cages joined together to allow confinement of the mother and ready contact of mother and offspring. Experiments were done 4–6 weeks after parturition. Time was required to train ewe and lamb to adjust to the metabolism cages. While the experiments may not have been made at the peak of lactation, the lambs were eating no appreciable amount of solids.

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Experiments generally involved the infusion of approximately 100 μCi [U-^{14}C] acetate (40–60 mCi/mmol, The Radiochemical Centre, Amersham, Bucks) into the jugular vein for 40–90 min. During the last 20 min of infusion five sets of blood samples were collected simultaneously from arterial, recurrent tarsal and (when available) mammary veins for the measurement of blood or plasma acetate specific radioactivity and concentration, blood ketone body, lactate and glucose concentrations. Infusions were performed either before or 4–7 h after the morning feed; at both times the concentration of circulating metabolites was fairly stable. At least five simultaneous samples were taken from all sampling sites during the day for estimation of blood oxygen and sometimes acetate concentration. Mammary gland and muscle blood flow were measured simultaneously after the infusion of [U-^{14}C] acetate. Milk yield in three ewes was measured in the 2–3 d period encompassing the experiment.

Methods of analysis

Metabolites. Specific radioactivity of circulating acetate and concentrations of volatile fatty acids (VFA), glucose, D(−)-3-hydroxybutyrate and acetoacetate were measured as described by Pethick *et al.* (1981). Rumen VFA concentrations were measured by direct analysis of samples of acidified centrifuged rumen samples (obtained by stomach tube or at necropsy) using the same gas–liquid chromatographic method described for blood VFA. Blood L-lactate concentrations were measured by the method of Gutmann & Wahlefeld (1974).

Blood flow. Blood flow in both muscle tissue and the mammary gland was measured by infusing ^3H_2O using the tissue saturation technique (Pappenheimer & Setchell, 1972). Bickerstaffe *et al.* (1974) have used a similar technique to determine mammary gland blood flow in the cow udder using urea as a marker.

Milk yield. The daily turnover of water in the offspring of lactating ewes was used to estimate milk yield as described by MacFarlane *et al.* (1969).

RESULTS

Mammary gland metabolism in sheep

Assessment of mammary gland substrate utilization by arteriovenous difference measurements in ruminants is complicated by the nature of the mammary venous drainage (Linzell, 1974). In the present study the sampling cannula was passed to the middle of the udder and vasoconstriction plus some thrombus formation (noted *post mortem*) in the cannulated superficial epigastric vessel probably limited flow considerably in this vein. This was desirable because sampling near the confluence of the superficial epigastric and external pudic vessels was thereby almost entirely that draining the mammary tissue. With the technique used for flow measurement, provided a representative venous sample was obtained, it was not necessary for total flow to drain through one vessel.

Table 1 shows the results for four lactating ewes for metabolite extractions, udder weights (obtained *post mortem*), blood flow and milk yield. The fractional extraction of glucose, 3-hydroxybutyrate and acetate; milk yield/g udder; and ratio, blood flow: milk flow are all comparable with values previously published for the goat and the cow. This supports the validity of the technique used for studying mammary metabolism.

Acetate metabolism in the whole animal

The entry rate of acetate in all experiments is summarized in Fig. 1. At a given arterial concentration of acetate the entry rate expressed per kg body-weight is similar in many different physiological states.

Feeding a 700 g concentrate/kg diet altered the molar proportions of the rumen VFA only slightly. Thus the molar proportions of acetate, propionate and butyrate were

Table 1. *Indices of mammary gland metabolism in lactating ewes and comparison with values in cows and goats*

(Mean values with their standard errors for four animals, except for milk yield and blood flow where mean values are for three animals)

	Mean	SE	Goats*†	Cows†
Udder wt (kg)	0·98	0·04	—	—
Milk yield (l/d)	1·57	0·07	—	—
Blood flow (ml/min per kg tissue)	529	13	400–700	420
Glucose				
Arterial concentration (mM)	2·88	0·18	—	—
Fractional extraction	0·26	0·03	0·33	0·28
Glucose uptake/lactose output‡	1·27	—	1·46	1·28
Acetate				
Arterial concentration (mM)	1·34	0·21	—	—
Fractional extraction	0·62	0·03	0·63	0·61
D(−)-3-Hydroxybutyrate				
Arterial concentration (mM)	0·41	0·06	—	—
Fractional extraction	0·53	0·06	0·57	0·39
Oxygen				
Uptake (mmol/h per kg tissue)	51·6	2·4	62	41
Fractional extraction	0·28	—	0·47	0·29

* From Linzell (1974).
† From Bickerstaffe *et al.* (1974).
‡ Assuming milk lactose was 4·8% (Jenness & Sloan, 1970).

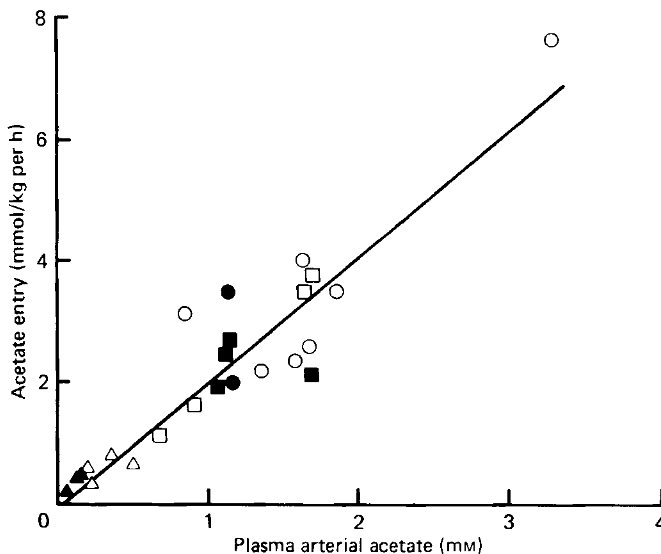

Fig. 1. The entry rate of acetate in sheep as a function of arterial plasma acetate concentration. If x is the arterial plasma acetate concentration (mM) and y is the entry rate of acetate (mmol/h per kg body-weight), then $y = 2{\cdot}06x - 0{\cdot}10$ (r^2 0·94; $P < 0{\cdot}001$). For details of animals and diets, see p. 320. The results for insulin-stabilized alloxan-diabetic ewes and diabetic fasted ewes (from which food and insulin had been withdrawn for 36 h) have been taken from Pethick *et al.* (1981). Fed non-lactating sheep: (■), insulin-stabilized alloxan-diabetic ewes (four animals); (□), wethers (four animals). Fasted non-lactating sheep (▲), wethers (3 d fasted) (three animals); (△), diabetic ewes (four animals). Fed lactating sheep: (○), normal diet (seven animals); (●), high-concentrate diet (two animals).

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Table 2. *Utilization of glucose, lactate and* D(−)*-3-hydroxybutyrate by the muscle of sheep*

(Mean values with their standard errors; no. of animals given in parentheses)

Ewe... Diet...	Non-lactating 'Normal'‖		Lactating 'Normal'		Lactating 700 g concentrates/kg	
	Mean	SE	Mean	SE	Mean	SE
Blood glucose						
Arterial concentration (mM)	3·81 (5)	0·36	2·97 (9)*	0·12	ND	
Net utilization (mmol/h per kg muscle)	2.56 (5)	0·8	2·06 (9)	0·25	ND	
Blood L-lactate						
Net utilization† (mmol/h per kg muscle)	0·02 (5)	0·002	−0·96 (5)*	0·23	ND	
Blood D(−)-3-hydroxybutyrate						
Arterial concentration (mM)	0·35 (4)	0·04	0·41 (5)	0·04	0·55 (3)	0·02
Net utilization (mmol/h per kg muscle)	1·0 (4)	0·2	0·9 (5)	0·2	1·19 (3)	0·19
Maximum contribution to oxygen uptake by muscle						
Glucose‡	57		32		ND	
D(−)-3-Hydroxybutyrate	16 (4)		17 (5)		17 (3)	

ND, values not determined.

* Significantly different from those for non-lactating ewes: $P < 0{\cdot}05$.

† Glucose utilization has been corrected for lactate output; for complete oxidation of 1 mol glucose and D(−)-3-hydroxybutyrate, 6 and 4·5 mol O_2 are needed respectively.

‡ Negative net utilization represents net output.

‖ The ration for these animals (1000 g chopped hay, 100 g oats) was divided into twelve equal portions, one of which was fed every 2 h.

(mean ± SE, three observations): 0·67 ± 0·01, 0·18 ± 0·02 and 0·1 ± 0·01 respectively in lactating ewes eating a normal diet; and 0·65, 0·18 and 0·14 respectively (two ewes which gave identical proportions) in lactating ewes eating the concentrate diet. The largest change was a 40% increase in the molar proportion of butyrate. Circulating D(−)-3-hydroxybutyrate, which is largely derived from rumen butyrate in fed sheep (Leng & West, 1969) also increased by 34% ($P = 0{\cdot}06$), see Table 2.

Acetate metabolism in muscle

The net utilization of acetate by muscle in all experiments is shown in Fig. 2 plotted as a function of circulating arterial plasma acetate concentration. In three lactating ewes whole blood and plasma acetate concentration and specific activity were compared. Both the concentration and arteriovenous difference for acetate in blood were 82 ± 2% of the plasma values (packed cell volume 0·3). Acetate specific radioactivity was similar whether measured using whole blood or plasma. Thus plasma acetate concentration was corrected to blood values for the calculation of acetate utilization by muscle. In Fig. 2 the results have been pooled into three groups: fed non-lactating, fed lactating and fasted non-lactating sheep. Fed non-lactating sheep include wethers, non-pregnant, non-lactating ewes and alloxan-diabetic insulin-stabilized ewes (results for the latter group have been taken from Pethick *et al.* 1981). Fed lactating sheep were eating the normal diet as described previously (see

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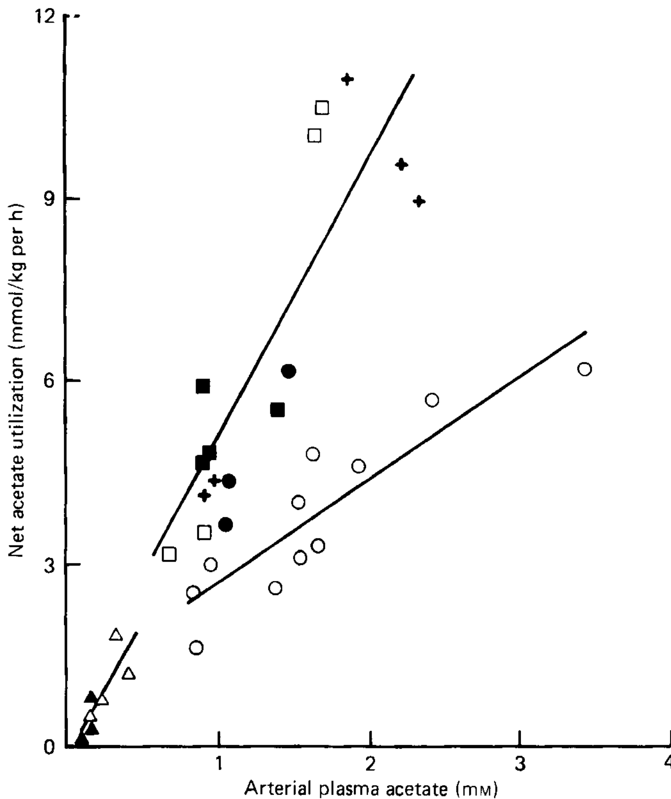

Fig. 2. The net utilization of acetate by sheep muscle. If x is the arterial concentration of plasma (mM) and y is the net utilization of acetate by muscle (mmol/kg muscle per h), then for fed non-lactating sheep $y = 4{\cdot}49\ (\pm 0{\cdot}76) + 0{\cdot}6$ (r^2 0·87; $P < 0{\cdot}001$); for fasted non-lactating sheep $y = 4{\cdot}31x\ (\pm 1{\cdot}39) - 0{\cdot}14$ (r^2 0·81; $P < 0{\cdot}01$); for lactating sheep given the 'normal' diet, $y = 1{\cdot}69x\ (\pm 0{\cdot}28) + 0{\cdot}96$ (r^2 0·9, $P < 0{\cdot}001$). Because of the evidence of heterogenous variances, regression coefficients for fed non-lactating, and lactating sheep on the 'normal' diet, were compared using the Behrens–Fisher test. The difference was found to be significant at $P < 0{\cdot}01$. For details of animals and diets, see p. 320. The results for insulin-stabilized alloxan-diabetic ewes and diabetic fasted ewes have been taken from Pethick *et al.* (1981). Fed non-lactating sheep; (■), insulin-stabilized alloxan-diabetic ewes (four animals); (□), wethers (four animals). Fasted non-lactating sheep: (▲), wethers (3 d fasted) (three animals); (△), diabetic ewes (four animals). Fed lactating sheep: (○), normal diet (eleven animals); (●), high concentrate diet (three animals). (+) Dry ewes (five animals).

Animals and diet) and the fasted sheep included wethers fasted for 3 d and alloxan-diabetic sheep from which food and insulin had been withdrawn for 36 h (results for the latter group have been taken from Pethick *et al.* 1981). No obvious differences were observed within individual groups (see Fig. 2). For each group a significant linear regression was found and the slope for the fed non-lactating sheep was nearly threefold greater than that for the fed lactating sheep.

The maximum contribution of acetate to the energy needs (O_2 consumption) of muscle varied in proportion to the arterial concentration of acetate. If y is the maximal fractional contribution of acetate to the O_2 consumption by muscle and x is the arterial acetate concentration, then in fed non-lactating sheep $y = 0{\cdot}22x + 0{\cdot}08$ (r^2 0·71; $P \simeq 0{\cdot}01$) and for fed lactating sheep $y = 0{\cdot}13x + 0{\cdot}06$ (r^2 0·82; $P \simeq 0{\cdot}002$). Feeding three lactating ewes a 700 g concentrate/kg diet tended to increase the utilization of acetate by muscle at a given arterial concentration to a value found in non-lactating ewes (Fig. 2).

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In most experiments a simultaneous utilization and production of acetate was detected. The production of acetate by the muscle tissue was 0·65 ± 0·25 and 0·57 ± 0·14 (mmol/h per kg muscle) for fed non-lactating and lactating sheep respectively. Consequently, differences of acetate net utilization were not associated with differences in endogenous production in muscle.

Utilization of acetate by the mammary gland was considerable. The mean (± SE) extraction (%) of arterial acetate was 62 ± 3 compared with 23 ± 2 and 43 ± 2 for muscle tissue in lactating and non-lactating sheep respectively. Total mammary gland utilization of acetate represented 17% of the entry rate of acetate. Mammary gland utilization was linearly related to arterial supply so that if y is the mammary utilization of acetate (mmol/h per kg tissue) and x is the arterial plasma acetate concentration then $y = 22{\cdot}2x - 3{\cdot}6$ (r^2 0·91; $P \simeq 0{\cdot}03$).

Glucose and ketone body metabolism

The concentration and utilization by muscle of D(–)-3-hydroxybutyrate is little affected by lactation in sheep (see Table 2). When a high-concentrate ration was fed, there was a significant ($P < 0{\cdot}05$) increase in arterial D(–)-3-hydroxybutyrate concentration and a proportional increase in the mean utilization rate, although this change was not significant ($P > 0{\cdot}1$). Arterial concentration of acetoacetate was less than 0·03 mM in all fed animals and so it cannot represent a significant metabolite with regard to energy supply.

Net glucose utilization declined only slightly in lactating ewes; however, when the glucose utilized was corrected by the amount of lactate produced then the maximum contribution of glucose to the energy metabolism of muscle declined from 57 to 32% in fed non-lactating ewes (see Table 2).

DISCUSSION

This study emphasized the significance of contrasting whole animal and regional tissue metabolism. Thus, while the entry rate of acetate at a given arterial concentration is unaffected by lactation, there is considerable redistribution of acetate metabolism amongst the tissues. Acetate utilization rates are summarized in hypothetical non-lactating and lactating ewes at an arterial plasma concentration of 1·25 mM (Table 3). Most striking is the halving of acetate utilization by muscle in lactating ewes with the spared acetate being substantially utilized by the mammary gland. This adaptation would direct significant acetate to the mammary gland for the oxidative and synthetic requirements of milk production.

The significance of these results needs careful consideration since the lactating ewes were eating approximately 70% more metabolizable energy per unit body-weight than the non-lactating sheep. In the lactating ewe given food in a roughly constant manner the mean daily arterial plasma concentration of acetate was 2·4 mM. Using this value a mean entry rate of 4·8 mmol/h per kg body-weight is predicted from Fig. 1. The same estimate is also predicted assuming 25% of the digestible energy is absorbed as acetate and 25% of the entry rate is derived from endogenous sources (Pethick *et al.* 1981). Oddy (1978) found a similar rate of entry of acetate in lactating ewes on a similar diet (4·9 mmol/h per kg body-weight). In Table 3 indices of acetate metabolism have been estimated in a lactating ewe eating the 'normal' ration using an arterial plasma acetate of 2·4 mM (lactating ewe B). In essence the energy requirement of the muscle is met by acetate to a similar extent in lactating ewes and in maintenance-fed non-lactating sheep providing the former are eating sufficiently to result in an arterial plasma acetate concentration of 2·4 mM. These changes suggest some homeostasis resulting from the interaction of dietary intake of feedstuffs and tissue metabolism of acetate.

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Table 3. *Estimated whole animal and tissue metabolism of acetate in fed non-lactating and lactating ewes*

(Values for non-lactating and lactating ewes eating 'normal' diets are derived from the relationships shown on pp. 321 and 323–325 or in Figs. 1 and 2 using the plasma acetate concentration shown)

Ewe... Diet... Index of acetate metabolism	Non-lactating*	Lactating A†‡ 'Normal'	Lactating B‡ 'Normal'	Lactating‖ 700 g concentrate/kg
Concentration plasma acetate (mM)	1·25	1·25	2·4	1·25
Net muscle uptake§ (mmol/h)	73	38	63	58
Percentage contribution to muscle oxygen uptake	35	22	37	33
Net mammary uptake (mmol/h)	ND	24	49	ND
Entry rate (mmol/h per 50 kg)	124	124	241	133

ND, values not determined.

* Non-lactating ewes given a diet of 1000 g chopped hay and 100 g oats as twelve equal portions daily had a mean arterial plasma acetate concentration of 1·25 mM (see Pethick *et al.* 1981).

† Utilization rates for lactating ewe A eating the normal diet have been calculated at an arterial plasma acetate concentration of 1·25 mM for ease of comparison.

‡ Lactating ewes given a diet of 1000 g chopped hay, 1000 g long hay and 400 g concentrate mixture in four equal portions daily have a mean daily arterial plasma acetate concentration of 2·4 mM (lactating ewe B eating a normal diet).

‖ The rates for lactating ewes eating a 700 g concentrate/kg are means of three animals.

§ Muscle is assumed to represent 25% body-weight (Pethick *et al.* 1981).

Although only limited studies were made with lactating sheep receiving a high-concentrate ration, the findings may have implications for the low-fat syndrome. Oddy (1978) has shown that in sheep, as in cows, a high-concentrate diet can result in a fall in milk-fat content. Although we could not determine milk fat in these experiments, the increased utilization of acetate by muscle and resultant limit to the availability of acetate for milk-fat synthesis would, in the absence of other changes, be expected to affect the fat content of milk. It is clear that this cannot be the only cause of this condition, since this would not account for the decrease in C_{18} acids (of dietary or adipose tissue origin) which can occur. Nevertheless, the observations of Engvall (1980) do suggest that a decrease in the fat derived from short-chain fatty acids may be a significant factor, particularly in field conditions.

In Table 4 the significance of the udder in relation to a variety of whole animal factors is shown. The relative glucose demand in the lactating ewe would appear to be less than in the dairy goat or cow. This is not surprising since the total mammary gland weight and O_2 consumption is two to three times lower in sheep and indicates a relatively small drain on total metabolism. In accordance with this, the redistribution of glucose metabolism between muscle and mammary gland is not large, with the major effect being a stimulation of the Cori Cycle in muscle.

In contrast, acetate uptake by the lactating sheep udder represents a greater drain on acetate supply than the udder of dairy cows and is at least comparable to dairy goats, as reported by Bickerstaffe *et al.* (1974). This might be related to the relatively large output of fat by the lactating mammary gland in ewes. Assuming milk fat was 75 g/l in the ewes in the present study (Jenness & Sloan, 1970), then the ewes secreted 6·5 g fat/d per kg

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Table 4. *Quantitative significance of the udder in relation to metabolism of the whole animal, in goats, cattle and sheep*
(Value for udder as percentage for whole animal)

	Goat*	Cow†	Sheep‡
Glucose utilization rate	66	66	41‖
Acetate utilization rate	20	10	17
Oxygen utilization rate	14§	10¶	4§
Wt	4·1	3·1	1·5

* From Linzell (1974).
† Results of Bickerstaffe *et al.* (1974).
‡ Results of the present study.
‖ Whole animal entry rate of glucose calculated from empirical relation with metabolizable energy intake for values greater than maintenance.
§ It is assumed that oxygen consumption of the whole animal is 1·5-fold greater than non-lactating sheep (Pethick *et al.* 1981) as suggested by the respiratory carbon dioxide production figures of Bergman & Hogue (1967).
¶ Whole animal utilization taken from Flatt *et al.* (1969).

body-weight$^{0.75}$, a value not much below the 7·5 g/d per kg body-weight$^{0.75}$ for the dairy cows of Bickerstaffe *et al.* (1974).

The comparisons shown in Table 4 depend on the estimate of mammary blood flow being accurate. It has been suggested (Linzell, 1974) that the ^3H_2O diffusion technique may over-estimate mammary flow because of evaporative losses. This seems unlikely within the 25 min period required for flow measurement. Moreover, the agreement we have found for the blood flow : milk output ratio among sheep, goats and cattle suggests our estimates are unlikely to be substantially in error. Davis & Bickerstaffe (1978) have also studied mammary gland blood flow in ewes. They estimated blood flow using the Fick principle by comparing the mammary arteriovenous difference for plasma methionine and its output in milk. Their estimate of flow is twice that of the present study, and much higher than that obtained for goats and cows. This discrepancy could be related to their use of plasma methionine as indicator. If whole blood methionine concentration is 1·25 times greater than plasma methionine, as our (P. J. Buttery, D. B. Lindsay & D. W. Pethick, unpublished results) preliminary observations suggest, assuming all methionine is exchangeable this would reduce the value for blood flow, using the Davis & Bickerstaffe (1978) equation, to 633 ml/min per kg tissue and the value for blood flow : milk flow to 495, a value similar to those shown in Table 1.

REFERENCES

Annison, E. F., Bickerstaffe, R. & Linzell, J. L. (1974). *J. agric. Sci., Camb.* **82**, 87.
Bergman, E. N. & Hogue, D. E. (1967). *Am. J. Physiol.* **213**, 1378.
Bickerstaffe, R., Annison, E. F. & Linzell, J. L. (1974). *J. agric. Sci., Camb.* **82**, 71.
Davis, C. L. & Bauman, D. E. (1974). In *Lactation, a Comprehensive Treatise*, vol. 2, p. 3 [B. L. Larson and V. R. Smith, editors]. New York and London: Academic Press.
Davis, C. L. & Brown, R. E. (1970). In *Physiology of Digestion and Metabolism in the Ruminant*, p. 545 [A. T. Phillipson, editor]. Newcastle upon Tyne: Oriel Press.
Davis, S. R. & Bickerstaffe, R. (1978). *Aust. J. biol. Sci.* **31**, 133.
Davis, S. R., Bickerstaffe, R. & Hart, D. S. (1978). *Aust. J. biol. Sci.* **31**, 123.
Domanski, A., Lindsay, D. B. & Setchell, B. P. (1974). *J. Physiol., Lond.* **242**, 28P.
Engvall, A. (1980). *Acta. Vet. scand.* **72**, Suppl. 1.
Flatt, W. P., Moe, P. W., Munson, A. W. & Cooper, T. (1969). In *Energy Metabolism of Farm Animals*, p. 235 [K. L. Blaxter, J. Kielanowski and G. Thorbek, editors]. Newcastle upon Tyne: Oriel Press.

https://doi.org/10.1079/BJN19820116 Published online by Cambridge University Press

Gutmann, I. & Wahlefeld, A. W. (1974). In *Methods of Enzymatic Analysis*, vol. 3, p. 1464, 2nd ed. [H. V. Bergmeyer, editor]. New York and London: Academic Press.
Harrison, F. A. (1974). *J. Physiol., Lond.* **242**, 20P.
Jenness, R. & Sloan, R. E. (1970). *Dairy Sci. Abstr.* **32**, 599.
Leng, R. A. & West, C. E. (1969). *Res. vet. Sci.* **10**, 57.
Linzell, J. L. (1974). In *Lactation, a Comprehensive Treatise*, vol. 1, p. 143 [B. L. Larson and V. R. Smith, editors]. New York and London: Academic Press.
MacFarlane, W. V., Howard, B. & Siebert, S. B. (1969). *Nature, Lond.* **221**, 578.
Ministry of Agriculture, Fisheries and Food (1975). *Energy Allowance and Feeding Systems for Ruminants*, Technical Bulletin 33. London: HMSO.
Oddy, V. H. (1978). *Proc. Aust. Soc. Anim. Prod.* **12**, 145.
Pappenheimer, J. R. & Setchell, B. P. (1972). *J. Physiol., Lond.* **226**, 48P.
Pethick, D. W. & Lindsay, D. B. (1981). In *Metabolic Disorders in Farm Animals, Proceedings of 4th International Conference on Production Disease in Farm Animals*, p. 123 [D. Giesecke, G. Dirksen and M. Stangassinger, editors]. Munich: Institut für Physiologie, Physiologische Chemie u Ernährungsphysiologie der Universität München.
Pethick, D. W., Lindsay, D. B., Barker, P. J. & Northrop, A. J. (1981). *Br. J. Nutr.* **46**, 97.

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