THE ROLE OF FAT IN RUMINANT NUTRITION

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Ruminants have been domesticated and exploited by man for two major reasons. Firstly, they are able to digest roughage and thereby derive energy for productive purposes because they possess a functional rumen. Secondly, they produce meat and milk which are of nutritive value to man. Much of this paper will cover aspects pertaining to the above with particular emphasis on fat intake and metabolism within the ruminant animal.

A Function of the Rumen

The young milk-fed calf or lamb is essentially monogastric with regard to its digestive physiology, but by 3-4 months of age, under normal husbandry conditions, i.e. weaned and fed pasture, the preruminant animal develops a digestive system which is essentially ruminant in character.

The adult ruminant possesses a complex stomach comprising four compartments (rumen, reticulum, omasum and abomasum). The rumen contains large numbers of microorganisms which break down the cellulose and hemicellulose in pasture plants and other roughages to volatile fatty acids (VFA). Thus ruminants are able to utilize fibrous material from which non-ruminants would derive negligible quantities of digestible carbohydrate. Unfortunately the rumen microorganisms also break down dietary protein and fat. This is detrimental as far as man is concerned in his role as farmer and consumer. Ruminal degradation of protein results in a relatively inefficient utilization of protein compared to monogastric animals especially if the dietary protein is of high biological value (Buttery and Annison 1972) with the resultant production of VFA and ammonia. Thus rations containing high levels of protein will not provide the same weight gains seen when pigs, for example, are fed these rations.

Complex plant lipids are hydrolysed and hydrogenated by rumen microorganisms such that these glycolipids containing a high proportion of the fatty acids linoleic (18:2) and linolenic (18:3) are converted to fatty acids stearic (18:0) and oleic (18:1) (Garton et *al* 1958; Kemp and Dawson 1968). These unesterified fatty acids are bound largely to particulate matter in the rumen (Lennox *et al* 1968) before being passed into the small intestine where they are absorbed across the gut epithelium and pass into the lymphatic system on route to the blood stream (see Yoffey and Courtice 1970). The hydrogenation process occurring in the rumen has a marked effect on the fatty acid composition of lymph as shown in Table 1.

TABLE 1. Fatty acid composition of dietary lipid, rumen FFA and triglyceride and phospholipid fractions from thoracic duct lymph of sheep. (From results of Felinski *et al* 1964).

Fatty acid	Total lipid of diet	Source of fatty acids			
		Rumen contents		Thoracic-duct lymph	
		FFA	Esterified fatty acids	Triglyceride	Phospholipid
16:0	16.6	9.8	25.9	14.2	18.2
18:0	2.6	73.1	14.9	45.1	23.8
18:1	25.7	11.1	18.9	21.9	16.2
18:2	21.8	0.7	7.7	4.8	20.2
18:3	20.3	trace	3.8	4.5	8.4
other acids	13.0*	5.3*	28.8**	9.5**	13.2**

* mostly fatty acids 12:0, 14:0, and 16:1.

** includes fatty acids 12:0,14:0, fatty acids with odd numbers of carbon atoms, and branched chain fatty acids.

It has been observed in sheep (Felinski et *al* 1964) and cattle (Wadsworth 1968) that the major fatty acid in lymph triglyceride is 18:0 and although desaturase activity has been observed in sheep intestinal epitheliutn (Bickerstaff and Annison 1968), the results presented in Table 1 indicate that the conversion of fatty acid 18:0 to 18:1 in vivo is not quantitatively significant (see also Leat and Harrison 1974). The endogenous contribution of lipid (mainly from bile) also has an effect on the total fatty acid composition of lipid in lymph resulting in an increase in the content of fatty acids 18:2 and 18:3 compared with rumen free fatty acids (FFA). The daily amount of 18:2 and 18:3 absorbed from the intestine of sheep is only 0.3% of the dietary intake which is well below the minimum level recommended for linoleic acid in non-ruminants of 1% of the dietary energy (Leat and Harrison 1972). However, essential fatty acid (EFA) deficiency has never been noted in ruminant animals, suggesting that the ruminant utilizes and conserves its EFA more efficiently than non-ruminants. The overall effect of rumen biohydrogenation of fat is to produce ruminant tissues. which contain saturated fat even though pasture species contain predominantly unsaturated fatty acids. The possible detrimental effects of this for man as consumer of ruminant products will be discussed later.

Pasture diets rarely exceed 3-4% (w/w) of fat and any supplementation of rations to give a total fat content of the diet above 7-8% results in a reduction in food intake with cellulose digestion in the rumen being reduced (Johnson and McClure 1973; Scott and Cook 1975). The reason for this is not known although Annison (1972) suggested that adsorption of fat or fatty acids onto partly digested food particles may interfere with microbial metabolism or physically protect cellulose,

surfaces from microbial attack. There are several practical reasons for adding small quantities of fat to ruminant rations. It helps alleviate dust problems in finely ground feeds. Pelleted feeds containing fat look more attractive to the buyer. The pellets have better binding qualities and because of the lubricating properties of fat, less wear on machinery is observed.

B. Mammary Gland Metabolism

The ruminant mammary gland serves the unique role of providing milk both for survival of its young and for the nutrition of humans. It is small wonder that this gland has attracted the attention of a large number of research workers over the past decades.

During lactation the mammary gland consists of a large mass, of glandular tissue richly supplied with blood. This organ is capable of secreting up to 3 kg of protein, fat and lactose per day (Linzell and Peaker 1971). The high metabolic demands imposed by lactation are well illustrated by the work of Annison and Linzell (1964) who showed that the lactating mammary gland of the goat utilizes 60–85% of glucose (from gluconeogenesis), 14–41% of acetate and a significant proportion of the amino acids available to the animal. The respiratory quotient of non-lactating mammary glands is less than 1.0 indicating that fat, glucose or acetate are being oxidised (see Lascelles 1970). In non-lactating cows there is a significant arterio-venous difference across the mammary gland for acetate but not for glucose or triglyceride (Hartmann and Lascelles 1964) which suggests that acetate is the principle substrate metabolised at this time. In the fed, lactating animal the respiratory quotient is greater than one indicating a change in metabolism associated with active synthetic activity (Annison *et al* 1968).

It is well established that approximately *60%* of milk-fat fatty acids of ruminants are derived preformed from the blood and that the remainder are derived by *de novo* synthesis within the glandular epithelium from acetate or β-hydroxybutyrate (see Linzell and Peaker 1971).

1. Preformed fatty acids

Although Hartmann and Lascelles (1966) demonstrated that large quantities of chylomicron triglyceride are transported to the blood stream *via* the thoracic duct lymph in the pasture-fed, lactating cow, most of the triglyceride taken up by the mammary gland is derived from VLDL (Gooden and Lascelles 1973). It was suggested that chylomicrons are rapidly removed from the circulation of the cow and contribute fatty acids to the triglyceride of VLDL presumably formed in the liver. Triglyceride is the predominant form of blood lipid removed by the mammary gland since there is no significant arterio-venous difference for FFA, cholesterol ester, cholesterol or phospholipid (Linzell *et al* 1967; Gooden and Lascelles 1973). However, Bickerstaff *et al* (1974) showed, using labelled palmitic acid (16:0), that there was a fall in specific activity of FFA across the mammary gland indicating that there was an exchange of FFA between plasma and mammary tissue. Approximately half the fatty acid 16:0 and all the longer chain fatty acids in ruminant milk fat are derived from the blood triglyceride (Linzell and Peaker 1971).

2. *De* novo synthesis

Arterio-venous difference studies in ruminants, using intravenously injected ^{14}C-labelled acetate, have shown that the fatty acids 4:0 to 16:0 are synthesized by the mammary gland from acetate derived from rumen fermentation (McClymont 1949; Popjak *et al* 1951). In non-ruminants, glucose is the major precursor of long-chain fatty acids in mammary tissue (Jones 1969). This striking difference between ruminant and non-ruminant metabolism stems from the roles of glucose and acetate as precursors of acetyl CoA, the key intermediate in fat synthesis (Ballard *et al* 1969). Ruminant mammary tissues possess the enzyme acetyl-CoA synthetase, and therefore are able to form acetyl CoA directly from acetate removed from the blood. The sequence of steps and the formation of the longer chain fatty acids from 4:0 to 16:0 (Lynen 1974) can be summarised as follows:

$$\text{Acetate} + \text{CoA} \xrightarrow[\text{Acetyl-CoA synthetase}]{\text{ATP}} \text{Acetyl CoA}$$

$$\text{Acetyl CoA} + CO_2 \xrightarrow[\text{Acetyl-CoA Carboxylase}]{\text{ATP}} \text{Malonyl CoA}$$

$$\text{Acetyl CoA} + \text{n-malonyl-CoA} \xrightarrow[\text{Fatty Acid Synthetase}]{\text{NADPH}} \text{Fatty acid chain}$$

β-hydroxybutyrate makes a significant contribution to the formation of milk-fat fatty acids although there is some disagreement as to whether it is utilized as a four (Kumar *et al* 1965) or as a two carbon unit (Linzell *et al* 1967).

C. Protected Fat Supplements

The discovery that ruminants fed 10-12% fat in a protected form resultedinincreased production of milk fat,has initiated increased interest in fat metabolism in these animals. The protection of fat droplets, by coating with formalin-treated protein (Scott *et al* 1970; Scott and Cook 1973), is an extension of earlier work on protected protein supplements (Ferguson *et al* 1967). These protected fats appear to be inert in the rumen and therefore relatively large amounts can be ingested without risk of the reduction in food intake commonly observed when large quantities of unprotected fat are fed to cows (Hartmann *et al* 1966). It is important to remember, however, when considering the protection of dietary components, that the rumen microorganisms must not be deprived of energy-yielding nutrients or cellulose digestion and microbial protein synthesis will be curtailed. The feeding of protected fat to a ruminant effectively converts its fat digesting mechanisms in the small intestine towards those of a monogastric animal; triglyceride rather than FFA being the form of lipid presented to the small intestine (Gooden, unpublished 1974).

1. Polyunsaturated Fat and Atherosclerosis

In ruminants carcass fat and milk fatty acids are more

saturated than occurs in monogastric animals (Linzell and Peaker 1971; Hegsted *et al* 1965). As already discussed this is due to ruminal bio-hydrogenation which prevents dietary unsaturated fat from reaching adipose or mammary tissue.

There are two ways in which ruminant tissues can be presented with polyunsaturated fatty acids. They both rely on bypassing the rumen microorganisms. The first is an intra-duodenal infusion of oil containing high levels of either fatty acid 18:2 or 18:3. This approach was used by Bickerstaffe and Annison (1971) who infused increasing amounts of sunflower oil into the duodenum of a lactating cow, fed a conventional ration containing 380 g of fat/day. The amounts of sunflower oil were increased at each successive infusion period (over 4-5 days) to a maximum of 651 g fat/day. Milk yield remained unchanged at 22 litres/day, but fat output increased from 750 g to a maximum of 1050 g/day and the content of fatty acid 18:2 in milk fat rose from 3 to 27 moles %.

It is clear from this data that the feeding of protected polyunsaturated fat (the second way of overcoming rumen hydrogenation) to lactating cows has great potential. There are, however, conflicting reports in the literature regarding the production of milk and milk-fat during the feeding of protected polyunsaturated fat to lactating cows. On the one hand Gooden and Lascelles (1973) showed no significant increase in either parameter although the level of fatty acid 18:2 in milk fat rose from 2.6% to 24.6% over a 7 day period. The composition of the major fatty acids in milk fat during control (protected casein) and protected lipid feeding from the above study is shown in Table 2.

TABLE 2. Fatty acid composition of milk during the feeding of protected casein and protected lipid to three lactating cows. Values (expressed as percentage by weight of total fatty acids ≤ 18:3) are means ± standard errors for three cows.

Diet	Protected casein	Protected lipid
No. of samples	4	3
Fatty acid		
< 16:0	27.1 ± 0.5	20.6 ± 0.4
16:0	30.4 ± 1.9	15.6 ± 2.6
16:1	1.9 ± 0.5	1.1 ± 0.3
18:0	12.1 ± 0.8	11.5 ± 0.5
18:1	24.2 ± 0.5	24.2 ± 1.1
18:2	2.6 ± 0.5	24.6 ± 1.6
18:3	1.7 ± 0.1	2.4 ± 0.2

Cows were fed 1 kg of protected safflower oil/day or 330 g of protected casein.

On the other hand Pan et *al* (1972) and Bitman et *al* (1973) showed marked increases in both milk-fat production and in the degree of unsaturation of milk fat following feeding of protected fat. Feeding of protected fat over prolonged periods (6 weeks) substantially changes the fatty acid compodition of ruminant adipose tissue (Scott et *al* 1971; Hood and Thornton 1976; Hogan and Hogan 1976).

Although the feeding of protected polyunsaturated fat to ruminants is known to decrease the oxidative stability of milk (Scott and Cook 1975), little work has been done to investigate the natural antioxidant status of these animals especially during prolonged periods of feeding.

Much of the interest in protected polyunsaturated fat has been generated by increased awareness and publicity of the possible role of polyunsaturated fatty acids in lowering human blood cholesterol levels and thereby reducing the incidence of atherosclerosis. It seems clear that consumption of polyunsaturated fat rather than saturated fat will lower blood cholesterol levels (Reiser 1973; Miettinen et *al* 1972; Kaunitz 1975), but it is not known if this reduces the incidence of the disease.

In 1973, medical and animal production scientists from New Zealand held a joint symposium (N.Z. Society of Animal Production and Nutrition Society of N.Z. 1973) which concluded that the medical profession in New Zealand, although in a position to identify a sizable proportion of people at special risk from coronary heart disease, had made relatively little progress in doing so. Doctors were in agreement that at least some degree of dietary intervention was justified in people at special risk. Scott (1974) who summarized the symposium stated that the majority of medical practitioners at that meeting doubted whether current evidence warranted major dietary intervention for the New Zealand population as a whole with one *proviso*. The total calorie consumption of many New Zealanders was higher than it should be and measures to reduce overall dietary intake was not unreasonable advice to give at the present time, particularly in relation to children.

D. Overfatness in Export Lamb

One of the ways of lowering human fat intake from ruminant meat is to reduce the total content of meat fat. Excess fat in ruminants is wasteful and expensive both to the producer and to the consumer. As world markets become more selective in their requirements for lamb (and indeed beef), Australia and New Zealand will need to reduce carcass fat while maintaining maximal quantities of red meat. The Australian Meat Board is currently upgrading their meat classification scheme. In New Zealand the proportion of export lamb carcasses down-graded because of excess fat is approximately 1% at the present time but according to Kirton (1974) the problem is greater than this figure suggests. The New Zealand Meat Board has anticipated an increase in demand for leaner lamb in the near future by proposing to reduce the allowable fat depth across the 12th rib.

Breeding programmes designed to reduce the fatness of ruminants have not appeared in the literature. However, it would seem feasible that ruminant fatness could be reduced by a breeding programme (Bowman 1967; Willham 1976) as has proved possible with pigs (Hetzer and Harvey 1.967). The fat content could be controlled genetically by selecting for the rate of attaining maturity or for the rate of fat deposition. As early maturing animals tend to produce fat at a lower body weight (Berg 1967), larger, later maturing animals should obviously be selected. Killing at lower body weight is another means of lowering total body fat.

D.S.I.R. and Massey University, New Zealand are currently conducting a breeding trial to evaluate the heritability of fat cover in Southdown and Romney sheep. An important aid to any selection programme is the ability to measure fat depth in the live animal. An ultrasonic probe, originally designed to measure distances across the human brain, has been modified for use with sheep and lambs and can measure fat depths to an accuracy of 0.5 mm (Beach and Gooden, unpublished 1977). Of particular interest are the underlying mechanisms whereby two sheep grazing the same pasture can have very different rates of fat deposition. According to Bauman and Davis (1974) the regulation of lipogenesis and lipolysis in ruminants is not well understood.

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