constituent more directly responsible. For instance, there is evidence to suggest that a low level of amino-acids in the digesta of the small intestine may depress the absorption of magnesium from that organ (McCance & Widdowson, 1944), and this level could well be related to the type of fermentation proceeding in the rumen.

This brief account of our investigation of the pattern of rumen fermentation and the metabolism of the cow has indicated the special features and problems connected with the feeding of spring grass, but clearly much further study along these lines is required.

We should like to record our appreciation of the constant advice and criticism of Dr S. J. Rowland throughout the course of our work. Many of the experiments reported have been done in association with Dr C. C. Balch and we are grateful to him, and to Dr A. T. Cowie for establishing the rumen and intestinal fistulas in the cows and sheep.

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The influence of pasture on the vitamin A and carotene in the milk of cows

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Vitamin A and carotene in milk and milk products make a valuable contribution to human nutrition, and this contribution depends on the feeding of the cow.

The daily allowance of vitamin A for an adult man recommended by the (U.S.A.) National Research Council: Food and Nutrition Board (1953) and the British Medical Association: Committee on Nutrition (1950) is 5000 i.u. from sources containing both carotene and vitamin A. A reasonable estimate of the contribution of milk and milk products can be calculated on the assumption, valid for the fat-soluble vitamins at least, that they quantitatively find their way into the diet of the population. The total sale of milk for all purposes in this country is virtually 2×10^9 gal./year, which distributed to a population of 53 million works out at 40 gal./head annually, or 500 g/day. The Milk Marketing Board statistics (Provan, 1955) show that in England and Wales milk contains on the average 3·6% fat, and

the survey in Great Britain of Thompson, Ganguly, Mawson & Kon (1949) that milk fat contains 29 i.u. vitamin A/g (mean of winter and summer values) and hence contributes 520 i.u. vitamin A/head/day. In addition Great Britain imports 280×10^3 tons of butter and 140×10^3 tons of cheese. Assuming that cheese contains 35% fat (Dearden, Henry, Houston, Kon & Thompson, 1945) and butter contains 82% butterfat, these together contribute another 6000 g fat/head in a year, or 480 i.u. vitamin A daily, making with the contribution from home-produced milk a total of 1000 i.u./head/day. Hence the contribution of milk is about 20% of our total need. In the U.S.A. a similar calculation by Cary (1947) showed that milk and milk products contribute one-third of the recommended daily allowance. It is clear that the contribution of milk and milk products is quite considerable, particularly as over half the activity is derived from vitamin A, which is well known to be better utilized than its precursor carotene.

Cattle are independent of an exogenous source of the water-soluble vitamins, in contrast to their dependence on the feed for their supply of fat-soluble vitamins. It is easy to cause large changes in the vitamin A status of cattle and to run the risk of deficiency of vitamin A. F. Blakemore & T. Moore (unpublished results) observed deficiency in store cattle near Cambridge fed mainly on sugar-beet pulp. Blindness due to vitamin A deficiency has been known for some time in cattle, particularly in the Channel Island breeds (de Schweinitz & De Long, 1934). In investigations in this field Moore, Sykes, Jacobson & Wiseman (1948) found that the measurement of the cerebrospinal-fluid pressure was a more sensitive guide to the vitamin A status of calves than the measurement of vitamin A and carotene in the blood.

Evidence for the mechanism and site of conversion of carotene into vitamin A in cattle is not as complete as for the rat, goat, pig and sheep (see review by Kon & Thompson, 1951). Kon, McGillivray & Thompson (1955) have given some information about conversion of carotene in calves and Eden & Sellers (1950) and Kon *et al.* (1955) have shown that the process of absorption of preformed vitamin A is the same as in other species. Briefly, carotene is converted into vitamin A alcohol and esterified in the mucosa of the small intestine, carried via the lymph to the blood and so to the liver, mammary glands or other tissues. Over 97% of the vitamin A in cow's milk is in the ester form (Thompson *et al.* 1949) and is probably normally derived directly from the vitamin A ester circulating in the blood.

Factors influencing the vitamin A and carotene content of cow's milk

Breed. Palmer & Eckles (1914) were the first to study chromatographically the pigments of cow's-milk fat and to make quantitative measurements of the carotene content. It is only more recently that accurate comparisons have been made on herds of cows of different breeds kept under the same conditions of feeding and management. Typical findings are shown in Table 1. The carotenoids show greater variations between breeds than vitamin A.

Stage of lactation. Colostrum may be ten times richer in carotene and vitamin A than mid-lactation milk (Walker, Thompson, Bartlett & Kon, 1949). By the 4th day

Table 1. *Carotenoid and vitamin A content of milk of different breeds of dairy cattle* (μg/g winter-milk fat)

Country	Breed	Carotenoids	Vitamin A
U.S.A.	Guernsey*	5·8	6·4
	Holstein*	3·4	7·3
	Ayrshire*	4·1	7·8
Britain	Guernsey†	13	3·4
	Shorthorn†	5·4	5·5

* Wise, Atkeson, Caldwell, Parrish & Hughes (1947).
† Thompson *et al.* (1949).

of lactation, when the milk is first used for human consumption, the levels are becoming normal and it is thought that there are no further lactational trends (Hibbs, Krause & Monroe, 1949; Barnicoat, 1947).

Hormonal influences. Plasma carotene and vitamin A decrease around parturition (Sutton, Kaeser & Soldner, 1945; Walker *et al.* 1949; Goodwin & Wilson, 1951). It has been suggested that this decrease might be a reflection of hormonal changes, but Thompson & McGillivray (1957) found that it was due to a general drain of these constituents into colostrum. The thyroid plays some part in carotene metabolism (Chanda & Owen, 1952) but its role and functions are not clearly established.

Food. The greatest variations in the vitamin A activity of milk are due to variations in the carotene content of the cow's food (Booth, Kon, Dann & Moore, 1933; Lord, 1945; Cary, 1947; Thompson *et al.* 1949). Carotene is converted into vitamin A inefficiently: Bauman, Steenbock, Beeson & Rupel (1934) calculated that when on pasture cows convert only 1·3% of the ingested carotene. Hibbs *et al.* (1949) showed that to produce butter of maximum potency the pasture must contain at least 26 mg β-carotene/100 g dry matter. This view is in agreement with Wiseman, Sheppard & Cary's (1949) estimate of the daily intake of carotene for this purpose, 2·5 g.

Table 2. *Daily carotene intake of cows at Shinfield on three typical winter rations*

		Ration no. 1		Ration no. 2		Ration no. 3	
Source of carotene	β-Carotene content (μg/g dry matter)	Dry-matter content (kg)	β-Carotene content (mg)	Dry-matter content (kg)	β-Carotene content (mg)	Dry-matter content (kg)	β-Carotene content (mg)
Grass* and clover†	250	0	0	0	0	0	0
Grass—lucerne silage‡	100	5	500	0	0	0	0
Hay‡	5	5	25	10	50	5	25
Kale and other green crops§	100	0	0	0	0	5	500
Concentrates	0	4	0	4	0	4	0
Total		14	525	14	50	14	525

* Bartlett, Henry, Kon, Osborne, Thompson & Tinsley (1938).
† Moon (1939).
‡ S. Y. Thompson (unpublished).
§ Kivimae (1950).

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An estimate of the levels of carotene intake of cows in the south of England in winter is shown in Table 2. Shorthorn or Friesian cows on pasture during the summer will eat 14 kg dry matter daily, containing about 3·5 g carotene, except during periods of drought. During the winter the intake of carotene will be adequate only while silage and kale are available. Considerable variations occur in this country above and below these levels and are reflected in the variations of carotene and vitamin A in the milk fat of market milk (Thompson *et al.* 1949). These authors found that in Great Britain winter butter contained 18·5 and summer butter 29·0 i.u./g. In actual practice the vitamin A potency of market milk or milk products in various parts of the world is a reflection of feeding and management in these areas, modified by the characteristics of the dominant breed.

Table 3*. *Comparison of vitamin A potencies of butter in different countries*

	Vitamin A potency expressed as			
	i.u./g		i.u./lb.	
Country	Winter butter	Summer butter	Winter butter	Summer butter
New Zealand	41·9	33·8	19,010	15,330
Great Britain†	18·5	29·0	8,390	13,150
Sweden‡	15·0	24·2	6,800	10,980
Denmark§	16·5	33·1	7,480	15,010

* From McDowell & McDowall (1953).

† Calculated from values published by Thompson, Ganguly, Mawson & Kon (1949) for carotene and vitamin A contents of milk in Great Britain. The assumption has been made that 80% of the total carotenoids in the summer milks and 75% of the total in the winter milks examined was active β-carotene.

‡ Platon & Swartling (1944).

§ Fridericia (1947).

Table 3, reproduced from a paper by McDowell & McDowall (1953) shows the vitamin A potency of winter and summer butters from various parts of the world. The results of the U.S.A. survey (Cary, 1947) are not included because they are based on a factor of 4·0 instead of the factor of 3·33 now accepted as correct for the conversion of vitamin A expressed in μg into international units. Cary's values, recalculated on the assumption that 20% of the potency is due to carotene, are 9500 i.u./lb. for winter, and 15,600 i.u./lb. for summer, butter.

Variations in the vitamin A potency of milk from cows on pasture

All countries in the northern hemisphere show a similar seasonal fluctuation, with higher values in summer than in winter, depending on the carotene content of the feed. Under New Zealand conditions where cows, predominantly Jerseys, are fed outdoors on pasture throughout the year a uniformly high vitamin A potency might be expected. However, Barnicoat (1947) reported higher values in winter than in summer and, extending this work, McDowell & McDowall (1953) showed the mean annual carotene and vitamin A content of North Island butterfat to be 9·5 and 8·1 μg/g respectively. Mean carotene levels ranged from a maximum of 12·4

μg/g in July to a minimum of 6·6 μg/g in March. Vitamin A maximum and minimum figures were 11·4 and 6·8 μg/g, occurring in July and December respectively. Similar seasonal fluctuations in carotene and vitamin A levels have been reported by Farrer, Balding, Warren & Miller (1949) in districts in Australia where dairying conditions are similar to those in New Zealand. There is also some indication of a similar midsummer decrease in results reported by Thompson *et al.* (1949) for butter from the south-west of England. Barnicoat (1947) suggested that the decreased potency of New Zealand summer butter was due to a lower carotene intake. However, McGillivray (1952) showed that, except under extreme drought conditions, the clover—rye-grass association characteristic of most dairy pasture in New Zealand provides a more than adequate carotene intake even in midsummer and suggested that the explanation was connected with differences in utilization rather than in intake. In this connexion he found that the tocopherol level in summer pasture is slightly lower than at other times of the year, and that this decrease was associated with the emergence of clover as the dominant species. Additional tocopherol raised the vitamin A potency from summer to typical winter levels. Investigating the effect of botanical composition of the pasture on the milk fat, Worker & McGillivray (1957), in experiments with monozygous twins, found marked differences between clover and rye-grass. Although the carotene intake remained virtually the same, the vitamin A potency of the milk fat of animals on a pure stand of white clover averaged only 27 i.u./g whereas that of their twin mates grazing the purest rye-grass available at that time (containing about 18% clover on a dry-matter basis) was 38 i.u./g. McGillivray (1952) has shown that the tocopherol content of the milk fat is also correlated with the vitamin A potency. It would appear that in summer pasture, probably associated with the clover, there are present some factors which decrease the absorption and utilization of not only carotene and tocopherol but probably all fat-soluble materials. The decrease in the level of vitamin A and carotene in milk fat is, however, only a reflection of changes which occur in the level of these substances in the blood plasma of cows on pasture (McGillivray, 1957*a*; Worker, 1956). Carotenoids in the milk fat reflected the changing level of carotenoids in the plasma very closely, but with vitamin A the picture was complicated by the fact that, although the free and esterified forms of vitamin A are present in both milk fat and blood plasma, in the former the esterified form predominates and the alcohol form in the latter. If milk-fat vitamin A arises from the ester form in the blood, the slight decrease in plasma ester over the summer months might be sufficient to explain the decreased levels of vitamin A in milk fat. The lower plasma vitamin A alcohol level might also be due to the animal striving to maintain milk-fat vitamin A levels at the expense of liver reserves but, if this is so, on the basis of the work of Chanda & Owen (1952) some increase in milk-fat vitamin A alcohol would be expected as the ester level decreased. However, McGillivray (1957*a*) found that the winter level of vitamin A alcohol was 0·5 μg/g fat and that this small proportion of vitamin A further decreased slightly during the summer months.

The evidence that at parturition (Thompson & McGillivray, 1957) and during periods of low carotene intake (McGillivray, 1957*b*) milk-fat vitamin A ester may be

derived from the liver stores via the vitamin A alcohol in the plasma agrees with the recent findings of Chanda, Clapham & Owen (1955) that the mammary gland can esterify plasma vitamin A alcohol. The plasma vitamin A is taken up as the alcohol form but is secreted in the colostral fat mainly as the ester (Thompson & McGillivray, 1957). In experiments in which cows were transferred from pasture to a carotene-free diet, McGillivray (1957*b*) showed that there was no immediate change in levels of carotene in the plasma, yet the milk-fat carotene decreased from 7·2 to 3·0 μg/g in 7 days. It seems reasonable to suggest therefore, on analogy with plasma vitamin A alcohol and ester, that there may be two distinct forms of carotene in the blood plasma. One, the physiologically normal form corresponding to vitamin A alcohol and associated with plasma proteins, contributes little carotene to milk fat. The other form of plasma carotene is present only when the animals are on a diet containing carotene and represents a transport form, probably associated with the vitamin A ester and dietary fat in the chylomicrons which can be taken up directly by the mammary gland and used for synthesis of milk fat.

Metabolic disturbances associated with pasture feeding

There is no doubt that quickly grown improved pasture has produced a variety of new metabolic disorders, some of which appear to be related to the problem of the decreased vitamin A potency of New Zealand summer butter. The more important from an economic point of view are under active investigation in that country. White-muscle disease is found in young sheep (Dodd, 1954) and, although there is adequate tocopherol in the pasture, responds to high doses of tocopherol. Rickets is troublesome in young lambs and it has been shown that there is probably an antivitamin D factor present in pasture (Grant, 1954). The high rate of wear in the teeth of sheep limits their useful life. This wear is thought to be due to the high tricarboxylic-acid content of rapidly growing grass and clover (Barnicoat, 1957). Bloat in cows is a serious problem; the cause is not known, but spraying the pasture or drenching the cows with oil of animal or vegetable origin is the most effective treatment (Johns, 1954). The major interest is, however, centred on facial eczema which takes, in some years, a large toll of sheep and has been reported in cattle. The cause of death is liver damage. This damage, which probably develops as soon as the toxic pasture is eaten, only becomes apparent externally 3 weeks later as a photosensitization of the exposed areas of the skin by porphyrins liberated from the damaged liver causing facial eczema, and at this stage it is too late either to save the animal or to collect the toxic pasture (Clare, 1952). Hence the experimental problem is to obtain toxic pasture for study.

Fortunately for other primary-producing countries, some of these metabolic problems are associated with highly improved pastures grown under climatic conditions favouring rapid growth and the production of an abundance of lush feed throughout the year and may well be peculiar to New Zealand.

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