Nutritional aspects of fermented milk products *

M.I. Gurr

*Nutrition Consultant, The Milk Marketing Board, Thames Ditton, Surrey, KT7 0EL, UK.
and Visiting Professor in Food Science & Technology, University of Reading, Reading RG6 2AP, UK.*

Key words: Lactic acid bacteria; Yoghurt; Kefir; Cultured buttermilk; Lactose digestion; Protein quality; Vitamin

1. SUMMARY

Fermented milk products are a palatable and economical source of a wide range of nutrients. The nutrient composition is similar to that in milk, but concentrations of vitamins are in general a little lower, with the possible exception of folic acid. Concentrations of lactic acid, galactose, free amino acids and fatty acids are increased as a result of the fermentation. Lactose-intolerant individuals tolerate lactose when it is consumed in yoghurt better than when it is taken in the equivalent quantity of milk. The mechanism of this effect has not been clearly established. By far the greatest proportion of published material freely available in the West concerns yoghurt; reference is made to other cultured products where results are available to indicate interesting differences.

2. INTRODUCTION

Since the time of Metchnikoff [1], fermented milk products have had an image of possessing almost magical health-giving properties but it is only in the last two decades or so that some of these ideas have been subjected to rigorous scientific test. This review will concentrate on two aspects: the nutritive value of fermented milks compared with natural milk and the digestion of lactose in relation to the benefits of cultured products for lactose-intolerant individuals. The major emphasis will be on yoghurt, but other cultured milks will be referred to for comparison.

3. NUTRITIVE VALUE

The products discussed here are based on cow's milk and therefore it can be expected that their nutrient composition will be broadly similar to that of the milk from which they were made. The composition will be modified by (a) changes in milk constituents brought about during the fermentation by the action of the microorganisms upon them, (b) the addition of nutrients and other chemical substances supplied by the organisms during the fermentation, (c) the presence of the microorganisms themselves and their associated enzymes, and (d) materials added in manufacture.

3.1. Energy

The chief sources of energy in milk are fat and lactose.

The energy value of yoghurt is very similar to that of the milk from which it is made. When the solids-not-fat are increased in the basic yoghurt

mix, however, then on a weight for weight basis, yoghurt may provide the consumer with a higher intake of protein, carbohydrate, calcium and certain B group vitamins than milk [2].

It has frequently been claimed that the fat is more digestible in cultured products than in milk because a certain degree of 'predigestion' has taken place [3]. Starter bacteria have limited ability to hydrolyse fat [4]. The presence of free fatty acids in yoghurt [5] should not, theoretically, aid digestion and absorption of fat and could in fact give products of poor organoleptic properties [6]. More recently, Schaafsma (personal communication) could find no significant differences in milk fat digestibility in vivo in rats fed milk, yoghurt or pasteurized yoghurt. It seems unlikely that major claims can be made on the basis of better milk fat digestibility, although it remains a possibility that cultured products could be beneficial to individuals who are unable easily to digest fat and it would be worthwhile testing this experimentally.

3.2. Lactose and its metabolic products

During the fermentation of milk, the microorganisms generally use lactose as a substrate, converting it into lactic acid. As a result, the lactose concentration in yoghurt is lower than in unfermented milk, provided that no supplementation with skim milk powder was made during manufacture.

Whereas fresh milk contains a negligible quantity of lactic acid, the fermentation process results in the conversion of some lactose into lactic acid [5]. Lactic acid may be beneficial by (a) acting as a preservative for the product; (b) contributing a mildly sour and refreshing taste; (c) influencing the physical properties of the casein curd to promote digestibility; (d) improving the utilization of calcium and other minerals and (e) inhibiting the growth of potentially harmful bacteria in the gut. Its energy value is 15 kJ/g compared with 16 kJ/g for lactose. Lactic acid occurs in yoghurt as two isomers; L(+) and D(−) lactic acid. The D(−) isomer is metabolized only very slowly in man compared with L(+) lactic acid [7] and if taken in excess can lead to metabolic disturbances. The World Health Organization recommended not more than 100 mg D(−) lactic acid per kg body

weight should be consumed daily, although Giesecke and Stangassinger [8] claim that only 60 mg/kg/day can be metabolized by man. A large part of the D(−) lactic acid intake is metabolized by the liver or is excreted in the urine by adults. D(−) Lactic acid normally represents about 40–50% of total lactic acid in yoghurt and there is no evidence for metabolic problems associated with the amounts normally consumed. Other fermented milk products contain a much smaller proportion of D(−) lactic acid because of the different metabolism of the starter bacteria used [14]. Thus the proportion of D(−) lactic acid as a percentage of total lactic acid is: Kefir, 2–5; cultured buttermilk, 3–6; soured milk, 4–12; fromage frais, 4–14.

Because of the breakdown of lactose during fermentation, the concentration of galactose is higher in cultured products than in unfermented milk. Galactose is normally absorbed very rapidly from the gut and metabolized to glucose in the tissues. A rare inborn error of metabolism, galactosaemia, in which the patient is unable to convert galactose into glucose, resulting in excessively high blood galactose concentrations is associated, among other symptoms, with cataracts [9]. Richter and Duke [10] observed that rats fed an exclusive diet of yoghurt developed cataracts, an effect they attributed to the galactose content of the yoghurt. It should be emphasized, however, that the extrapolation of these animal results to man is invalid because, in the first place, no human beings eat diets consisting entirely of yoghurt and secondly, rats have a limited ability to convert galactose into glucose, whereas, apart from rare cases of galactosaemia, human beings have an abundance of the enzyme needed to metabolize galactose. It can certainly be excluded, therefore, that normal consumption of yoghurt will be associated with cataracts as has sometimes been suggested.

In the fermentation of milk to make kefir, the presence of yeasts as well as bacteria leads to the formation of ethanol from the milk sugar. The final concentration is about 0.17–0.19% [11].

3.3. Proteins and amino acids

The total amino acid content and composition of yoghurt and other fermented milks does not

differ substantially from that of the milk from which they were made, but the free amino acid content is higher due to the proteolytic action of the microorganisms [5,11–13].

The protein quality of milk is already very high and the biological value of yoghurt protein, as measured by a rat growth assay, was not improved significantly above that of milk [14]. Most healthy human beings digest proteins very efficiently and it is unlikely that 'predigestion' of part of the protein in cultured products and the finer coagulation of the curd [15] will result in improved digestibility for them, although there could be benefits for those with impaired digestion.

3.4. Vitamins

Breed, diet, climate, geographical location, stage of lactation and other factors can influence the vitamin content of cow's milk [16,17] which in turn will affect the vitamin content of the cultured product. The amounts of the various vitamins in the milk base from which the cultured products are made will also be influenced to different extents by the heat treatment it receives in the preparative stages of manufacture. More significant will be the influence of the microbial inoculum [18]. While many lactic acid bacteria require B vitamins for growth, several cultures are capable of synthesizing certain vitamins. It is therefore impossible to quote 'typical' values for the vitamin content of cultured products, although an indication of changes that can occur after heat treatment, fermentation and storage is given by IDF [19]. Unfortunately most vitamins are generally present in lower concentrations in yoghurt than in milk with the exception in some cases of folates [14,19], although it is uncertain whether the bacterial folates have biological activity for man. The concentration of folic acid is even greater in cultured sour cream [12] than in yoghurt but not in cultured buttermilk [17].

3.5. Minerals

Fermentation has little effect on the mineral content of milk and like milk, yoghurt is an excellent source of essential minerals, particularly calcium, phosphorus, magnesium and zinc. The nutritive value of a food depends not only on its nutrient composition but on the bioavailability of those nutrients, namely the proportion that can be absorbed and utilized by the body. Lactose improves the absorption of calcium and other minerals [20] and it is important to ask whether the decrease in lactose concentration that occurs during fermentation is associated with lower mineral bioavailability. In recent experiments with rats, Schaafsma (personal communication) showed that reduction of lactose concentration either by fermentation or treatment of milk with lactase resulted in a somewhat lower bioavailability of calcium and other minerals and a small decrease in bone mineral content. This is consistent with other results in rats [21] and lactase-deficient human subjects [22] showing that calcium from yoghurt is not better utilized than that from milk. Nevertheless it should be emphasized that the differences are small and that the bioavailability of calcium from all dairy foods is very much higher than calcium from plant sources.

Many cultured products sold on the supermarket shelf today are not simply the fermented equivalent of milk. They may be fortified by the addition of skim milk powders, caseinates, ultrafiltered concentrates, fruit pulp, stabilizers, flavourings and colourings, many of which will modify the nutritive value by increasing the concentrations of proteins, sugars, polysaccharides and other nutrients [23].

4. LACTOSE DIGESTION AND LACTOSE INTOLERANCE

To be efficiently absorbed from the gut, lactose must be digested into its constituent sugars, glucose and galactose, by the enzyme lactase.

Lactose is a normal constituent of human milk as well as cow's milk and in babies of all races the enzyme lactase is present in the gut to digest the milk lactose. In most of the world's races this enzyme is lost during the first or second decade of life and only peoples of Northern European origin, their overseas descendents and some isolated African and Indian communities maintain a high intestinal lactase activity throughout life. It is generally believed that this change is genetically

programmed and that the amount or activity of the enzyme is not influenced by lactose in the diet. People who have a low activity of intestinal lactase may develop gastrointestinal symptoms upon the ingestion of lactose, which may include diarrhoea, flatulence and abdominal pain caused by bacterial fermentation of undigested lactose in the colon and the resultant generation of gases. Different individuals tolerate different amounts of lactose: many in whom lactase activity is low or absent are quite able to tolerate modest amounts of dairy products taken as components of a regular mixed diet; others become ill with quite small amounts of milk products.

Anecdotal evidence suggests that yoghurt is better tolerated than milk by lactase-deficient people and if this were true, then many would find it unnecessary to reject dairy products which are the source of many essential nutrients. The advantage of yoghurt has been attributed either to its low lactose content, or the lactase activity of *Lactobacillus bulgaricus* and *Streptococcus thermophilus* which survive passage through the stomach and might contribute to lactose digestion in the small intestine [24]. As explained earlier, not all yoghurts have a lower lactose concentration than milk, so caution must be exercised in recommending cultured products as 'low lactose products'.

Recently, several reports on the tolerance of yoghurt compared with milk by lactase-deficient human subjects, have provided more rigorous scientific evidence for the benefits of the cultured product [25–27]. For example, Kolars et al. [25] provided 10 healthy lactose-intolerant subjects the following test meals: lactose (20 g in 400 ml water); milk (400 ml containing 18 g lactose); yoghurt (440 g containing 18 g lactose or 270 g containing 11 g lactose). Although the larger amount of yoghurt matched the amount of lactose provided by 400 ml milk, the smaller amount of yoghurt was designed to provide about the same fat and protein load as 400 ml milk. Samples of the subjects' breath were taken every hour for 8 h after consuming the test meal and the concentration of hydrogen in the breath gases was measured. The principle of this technique is that hydrogen is produced by fermentation of unabsorbed carbohydrate substrates (e.g. lactose) re-

aching the colon. A proportion of hydrogen is absorbed and is excreted in the breath, the rate of breath hydrogen excretion being roughly proportional to the amount of carbohydrate reaching the colon. Thus, when lactose is readily digested and the digestion products are absorbed through the small intestine, little goes on to reach the colon and little hydrogen is produced. Therefore, individuals producing large amounts of hydrogen are those who have digested lactose very poorly; low concentrations of hydrogen indicate good digestion.

The amount of hydrogen expelled after ingestion of yoghurt by these lactase-deficient subjects was only one third of that expelled after taking milk despite the fact that the lactose content was the same. Diarrhoea or flatulence was reported by 80% of those drinking milk but only by 20% of those eating yoghurt. The authors measured the lactase activity in samples of duodenal juice aspirated from three of the subjects. Significant activity was measured 20 min after ingestion of yoghurt. In two patients, this activity then returned to its former value over the next 40–60 min, whereas in the third patient the level of activity continued to rise for one hour and then declined. The authors concluded that the microbial lactase activity in the ingested yoghurt was responsible for the improved digestibility of lactose from this food.

Although this study is one of the most convincing published to date, it has two major drawbacks. The number of subjects studied was extremely small. Ten subjects only were studied but in only 3 was lactase activity measured directly and in two of these three the activity was short-lived: within one hour it had returned nearly to baseline levels. The most important criticism was that it was not conducted in a double-blind fashion, in which neither the subjects nor the experimenters knew which diet they were receiving. It cannot, therefore be excluded that there was a 'placebo effect' such that patients were expecting to react better to yoghurt and actually did so. Because of the different tastes and textures of the foods, however, a true double-blind experiment would be difficult to achieve.

Savaiano et al., [26] and Gilliland and Kim [27]

demonstrated that the consumption of heated yoghurt by lactase-deficient subjects resulted in the production of more breath hydrogen than from a meal of unheated yoghurt, indicating a greater digestibility of lactose from unheated than heated yoghurt. The bacterial lactase activity was much lower in the heated product and the results were interpreted as indicating digestion of lactose by the bacterial enzyme. The hydrolytic activity was higher in the presence of bile salts [27] perhaps as a result of disruption of the bacterial cells and a release of enzyme activity.

Other recent studies throw some doubt on whether the lactase activity of yoghurt bacteria contributes to lactose digestion in the gut. Because of the difficulty in making direct measurements of lactose digesting activity in human beings, many research workers have turned to the adult laboratory rat as a model for human lactose intolerance, since the young rat quickly loses its intestinal lactase after weaning on to solid food. Schaafsma et al. (personal communication) fed five groups of rats, for six weeks, diets based on milk, lactase-treated milk, yoghurt, pasteurized yoghurt or a commercial rat diet. Since galactose released from lactose by digestion in the small intestine is rapidly absorbed, and is present to a limited extent in foods other than those based on milk, its appearance in the blood following a meal that contains lactose can provide a measure of lactose digestion. Schaafsma and colleagues found that, using blood galactose appearance as a measure of lactose digestion, the lactase-treated milk demonstrated the highest digestibility, while the lactose in yoghurt and *pasteurized yoghurt* also showed significant digestibility. Milk lactose was poorly digested while no galactose was detected in the blood following a meal of commercial diet that did not contain galactose. It should be noted, however, that these blood galactose profiles reflect not only the galactose released by digestion of lactose in the gut but also the galactose present in the yoghurt and pasteurized yoghurt as a result of the hydrolysis of lactose during the fermentation. It is likely that a large part of the rise in blood galactose concentration can be accounted for by absorption of this free galactose in the diet. The release of some galactose from unfermented milk,

which does not contain free galactose, found by these authors provides evidence that part of the blood galactose appearance is due to hydrolytic activity in the gut.

The same authors demonstrated that pasteurization had inactivated the bacterial lactase which was present in abundance in the unpasteurized yoghurt. Since apparent lactose digestibility was no less from pasteurized yoghurt than from non-pasteurized yoghurt, we can rule out the possibility that bacterial lactase in the cultured product contributes significantly to lactose digestion in the gut.

Garvie et al. [28] fed rats a yoghurt diet and compared the activities of enzymes which hydrolyse lactose in the gut contents and the mucosal cells with the activities in control groups fed either on the base milk from which the yoghurt was made or on a standard rat chow diet. There were no changes in lactase activity in the gut mucosa, suggesting that the yoghurt in the diet does not stimulate the animal's inate ability to hydrolyse lactose, although there were large increases in lactose-hydrolysing activity in the gut contents due to the presence of the bacterial enzyme. This is consistent with the general view that dietary lactose does not induce lactase activity in the gut mucosa [29]. The literature, however, is not clear on this matter since Besnier et al. [30] found that mucosal lactase of mice could be stimulated by yoghurt feeding. There is also some evidence that intestinal lactase can be stimulated in man [31].

In summary, it is not yet possible to offer a satisfactory explanation of why lactose digestion (as measured by appearance of galactose in the blood or decreased concentrations of breath hydrogen) appears to be more efficient when the sugar is ingested as yoghurt than in the form of unfermented milk. Despite some discrepancies in the literature, the results presented here do not substantiate the hypothesis that the digestion is brought about by the microbial enzyme in the product, or by stimulation of the gut enzyme. Nor can the benefits of yoghurt be attributed necessarily to an intrinsically lower content of lactose in the food. The following hypothesis may be worth pursuing: that cultured products, because of their acidity and the finer dispersion of protein in

the stomach, retard the emptying of the gastric contents into the duodenum. Any lactose hydrolysing activity present, whether from indigenous or bacterial origin would have longer contact with the substrate and digestion would be more efficient even when the specific activity of the enzyme was low.

REFERENCES

[1] Metchnikoff, E. (1908) The Prolongation of Life, G.P. Putnam's & Sons, New York.

[2] Robinson, R.K. (1977) Yoghurt and health. Brit. Nutr. Found. Nutr. Bull. 21, 191–197.

[3] Wasserfall, F. (1972) Sauermilchprodukte und ihre Bedeutung als Nahrungsmittel, Ernährungsumschau, 19, 155–158.

[4] Stadhouders, J. and Veringa, H.A. (1973) Fat hydrolysis by lactic acid bacteria in cheese. Neth. Milk Dairy J. 27, 77.

[5] Rasic, J.L. and Kurmann, J.A. (1978) Yoghurt: Scientific Grounds, Technology, Manufacture and Preparation. Tech. Dairy Publ. House, Copenhagen.

[6] Tuckey, S.L. and Stadhouders, J. (1968) Increase in the sensitivity of the organoleptic detection of lipolysis in cow's milk by culturing or direct acidification. Neth. Milk Dairy J. 21, 158–165.

[7] Kielwein, G. and Daun, U. (1979) Vorkommen und Bedeutung von D(−) Milchsäure in fermentierten Milcherzeugnissen unter besonderer Berücksichtigung von Milcherzeugnissen auf Joghurtbasis. Dtsch. Molkerei Ztg. 101, 290–293.

[8] Giesecke, D. and Stangassinger, M. (1977) ^{14}C-Versuchen über den Stoffwechsel von D(−)-Milchsäure, Ernährungsumschau 24, 363–364.

[9] Segal, S. (1978) In: The Metabolic Basis of Inherited Disease (Stanbury, J.B., Wyngaarden, J.B. and Fredrickson, D.S., Eds.). McGraw-Hill, New York.

[10] Richter, C.P. and Duke, J.R. (1970) Cataract produced in rats by yoghurt. Science 168, 1372–1374.

[11] Mann, E.J. (1985) Kefir and koumiss. Dairy Ind. Int. 50, 11–12.

[12] Shahani, K.M. and Shandan, R.C. (1979) Nutritional and healthful aspects of cultured and culture containing dairy foods. J. Dairy Sc. 62, 1685–1694.

[13] Miller, I., Martin, H. and Kandler, O. (1964) Das Aminosäurespektrum. Milchwissenschaft 19, 18–25.

[14] Hewitt, D. and Bancroft, H.J. (1985) The nutritional value of yoghurt. J. Dairy Res. 53, 197–207.

[15] Renner, E. (1983) Milk and Dairy Products in Human Nutrition. Volkswirtschaftlicher Verlag, Munich.

[16] Gregory, M.E. (1967) Nutritive value of milk and milk products: water soluble vitamins in milk and milk product. J. Dairy Res., 34, 169–181.

[17] Scott, K.J. and Bishop, D.R. (1986) Nutrient content of milk and milk products: vitamins of the B complex and vitamin C in retail market milk and milk products. J. Soc. Dairy Technol. 39, 32–35.

[18] Hartman, A.M. and Dryden, L.P. (1974) Vitamins in milk and milk products. In: Fundamentals of Dairy Chemistry, 2nd edition (Webb, B.H., Johnson, A.H. and Arnold, J.A., Eds.), pp. 325–401.

[19] International Dairy Federation (1983) Cultured Dairy Foods in Human Nutrition. IDF Document 159. IDF, Brussels.

[20] Schaafsma, G. (1983) The significance of milk as a source of calcium. IDF Document 166, pp. 19–32. IDF, Brussels.

[21] Dupuis, Y., Gambier, J. and Fournier, P. (1985) Études comparées de la bioresponsibilité de calcium de lait, de yaourt et du fromage fondu. Sci. Aliments, 5, 559–585.

[22] Smith, T.M., Kolars, J.C., Savaiano, D.A. and Levitt, M.B. (1985) Absorption of calcium from milk and yoghurt. Am. J. Clin. Nutr. 42, 1197–1200.

[23] Deeth, H.C. and Tamime, A.Y. (1981) Yoghurt: nutritive and therapeutic aspects. J. Food Protect. 44, 78–86.

[24] Kilara, A. and Shahani, K.M. (1976) Lactase activity of cultured and acidified dairy products. J. Dairy Sci., 59, 2031–2035.

[25] Kolars, J.C., Levitt, M.D., Aouji, M. and Savaiano, D.A. (1984) Yoghurt: an autodigesting source of lactose. N. Engl. J. Med., 310, 1–3.

[26] Savaiano, D.A., Abou El Anouar, A., Smith, D.E. and Levitt, M.D. (1983) Lactose malabsorption from yoghurt, pasteurized yoghurt, sweet acidophilus milk, and cultured milk in lactase deficient individuals. Am. J. Clin. Nutr. 40, 1219–1223.

[27] Gilliland, S.E. and Kim, H.S. (1984) Effect of viable starter culture bacteria in yoghurt on lactose utilization in humans. J. Dairy Sci., 67, 1–6.

[28] Garvie, E.I., Cole, C.B., Fuller, R. and Hewitt, D. (1984) The effect of yoghurt on the gut microflora and metabolism of lactose in the rat. J. Appl. Bacteriol. 56, 237–245.

[29] De Groot, A.P. and Hoogendoorn, P. (1957) Detrimental effect of lactose, 2. Quantitative lactase determinations in various mammals. Neth. Milk Dairy J. 11, 290–303.

[30] Besnier, M.O., Bourlioux, P., Fourniat, J., Ducluzeau, R. and Aumaitre, A. (1983) Influence de l'ingestion de yaourt sur l'acidité lactasique intestinale chez le souris axénique ou holoxénique. Ann. Microbiol., 134A, 219–230.

[31] Buts, J.P., Bernasconi, P., Van Craynest, M.P., Meldaque, P. and Demeyer, R. (1986) Responses of human and rat small intestinal mucosa to oral administration of Saccharomyces boulardi. Pediatr. Res. 20, 495–498.