# The Energy Values of Dietary Fibre and Sugar Alcohols for Man

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INTRODUCTION

Much of the uncertainty about availability of energy from foods has centred on the dietary carbohydrates, in particular dietary fibre or non-starch polysaccharides (NSP), and a range of manufactured bulking carbohydrates, some new to our diets including oligosaccharides (OS) and sugar alcohols (SA). Such components have often been thought to have little effect on energy availability but their effects can be quantified and some might influence the development of major diseases and of premature mortality.

LEGISLATION

Energy values of proteins, fats and carbohydrates, applied to foods eaten in different countries or regions, may differ (Périssé, 1983). A detailed understanding of energy availability from diets may enable food energy calculations to be unified across the world. Where European Council Directives (1990a, b) apply, the current energy value for the purpose of food labelling is 10 kJ/g (2.4 kcal/g) for all SA, and for dietary fibre no value has yet been assigned. This review is concerned almost entirely with values for the adult population in Western countries; for other regions information is mostly lacking.

ENERGY AVAILABILITY, BODY WEIGHT AND DISEASE

Obesity is a risk for morbidity and early mortality (Royal College of Physicians, 1983). As little as 0.2 % of food energy intake above requirements over the first four decades of life must result in obesity (Norgan, 1990; Webster, 1992; McCracken, 1992). Achieving such a fine degree of control is helped by changes in body weight, such that higher energy intakes result in higher body weights and higher energy requirements, and vice versa. A change in energy intake of about 80 kJ from one’s energy requirement is sufficient to bring about a new equilibrium, with a body weight change of 1 kg (Jéquier, 1987). Eighty kilojoules is nearly 1 % of the average 68 kg person’s energy requirement (Department of Health, 1991). For obese subjects with a body mass index (w/h²) of 35 it may be calculated, using data from the Royal College of Physicians (1983), that a 1 % change in energy intake would be associated with a nearly 5 % change in the incidence of diabetes, coronary heart disease deaths and overall premature mortality.

Variation in the availability of dietary energy has been related formally to the occurrence, utilization and effects of those dietary carbohydrates which escape small intestinal digestion (Livesey, 1990a). Experimental human diets differ in energy availability by up to 16 % of gross energy (Livesey, 1990a), corresponding to an influence on body weight of 24 kg in a subject with a body mass index of 35 and a potential decrease in overall mortality from about 170 % to approaching 100 % or more of the average for all weights. Such estimates assume no compensatory changes such as higher food intake. In fact, food intake may be lower for diets enriched with specific dietary fibre sources and certain NSP (Blundell & Burley, 1987). Currently, habitual diets differ by no more than 5 % in energy availability, which corresponds to a 25 % difference in mortality for such obese persons in the absence of compensatory changes. The intakes of SA are generally too small to have had a perceptible impact on obesity, averaging about 2 g daily in those surveyed in the United Kingdom (Ministry of Agriculture, Fisheries and Food, 1990), though individual intakes may sometimes be of significance.
TERMINOLOGY

A great deal of confusion arises in the area of energy evaluation because of the use of imprecise carbohydrate terminology. The first substantial study to influence current thinking about energy from carbohydrates that escape small intestinal digestion after eating conventional foods was that of Southgate & Durnin (1970). To describe these carbohydrates, they used the term ‘unavailable carbohydrate’. Since then, the term ‘dietary fibre’ has been introduced, but the suggestion has been made that its use be discontinued in scientific work because of varying and imprecise definition (British Nutrition Foundation, 1990; Department of Health, 1991). The term ‘dietary fibre’ is therefore used sparingly in this review. Certain OS and SA also escape small intestinal digestion, and may therefore be called unavailable carbohydrates. To distinguish these from the polymeric unavailable carbohydrates found in conventional foods, the British Nutrition Foundation (1990) termed the latter ‘unavailable complex carbohydrates’ (UCC). It is important to retain these terms in energy evaluation, at least for the time being, because there are few human studies on the availability of energy from more precisely defined fractions of UCC. Thus, UCC contains both NSP and enzymically (though not physically) resistant starch. There are few published studies on the energy value of NSP alone and few on the starch fraction that escapes digestion in the small intestine. Where studies are undertaken with NSP the carbohydrate can often be defined more precisely, for example as guar gum or crystalline cellulose, or even more precisely in chemical terms (British Nutrition Foundation, 1990). It needs to be recognized that ‘complex carbohydrate’ is sometimes unfortunately (and incorrectly) used as a synonym for starch; here it includes both starch and NSP, as in the Task Force on Complex Carbohydrates (British Nutrition Foundation, 1990).

VALIDITY OF CURRENT FOOD ENERGY CALCULATIONS

Concern over energy availability from dietary fibre led to the Atwater (1910) food energy assessment system being questioned (Life Sciences Research Office, 1983). For diets rich in UCC it is now evident that the general food energy conversion factors of both Atwater (1910) and McCance & Widdowson (1946) do not apply accurately (Livesey, 1990a; British Nutrition Foundation, 1990; Wisker & Feldheim, 1992). This is of considerable importance since the two systems form the basis of food energy assessment used in almost all regions of the world.

Extensive analysis of the literature showed (Livesey, 1990a) that for mixed diets a digestible energy value of 8.4 kJ/g or 2 kcal/g UCC agrees with the McCance & Widdowson (1946) food energy calculation method (but not that of Atwater, 1910) in providing an unbiased prediction of energy availability. The analysis used data on 17 diets. Examination of over 30 diets, by multiple regression analysis with UCC contents ranging from 3 to 93 g daily, also indicated (Livesey, 1991a) that for mixed diets a digestible energy value of 8.4 kJ/g or 2 kcal/g can be assigned. With a heat of combustion of 17 kJ/g UCC, an apparent digestibility of 0.7 for UCC in mixed diets, and a conversion efficiency of 0.3 kJ faecal bacterial energy per kJ carbohydrate fermented, the 8.4 kJ/g or 2 kcal/g value fitted expectations at all intakes of the UCC (Livesey, 1990a).

The finding that the digestible energy value of UCC was related to its apparent digestibility opened the way to assigning specific energy values to individual carbohydrates and to the discounting of energy losses due to fermentation so giving net energy values for maintenance.

Currently, the most precise and least biased methods of calculating digestible, metabolizable and net energy values of diets are based on dietary measurements of gross
energy, UCC and nitrogen content and the application of formulae (Livesey, 1991a, b). A limitation is the lack of information about validity with diets of high UCC intake but low total energy intake, such as may be eaten during slimming. Another limitation is the age at which the formulae begin to apply and whether or not the formulae apply accurately to the aged eating high UCC diets. Further, there remains a theoretical possibility of ethnic differences in the extent to which UCC supply energy, so it is uncertain whether the formulae would apply to regions outside the Western world. Moreover, the formulae do not apply to diets very high in whole grain cereal with intact endospermal tissue (to be discussed) or include terms for OS and SA. In the future, UCC may be replaced by more precise assays of NSP and resistant starch.

**FACTORIAL CALCULATION OF ENERGY VALUES**

**PROPOSALS FOR UNAVAILABLE CARBOHYDRATES AND SUGAR ALCOHOLS**

Proposals of how to use information to calculate net energy values of carbohydrates for maintenance have been described recently by several researchers (Bär, 1990; Bernier & Pascal, 1990; Livesey, 1991b; van Es, 1991) and committees (Dutch Nutrition Council, 1987; British Nutrition Foundation, 1990).

Sugar alcohols

For SA net energy \( (NE_{SA}) \) is given (Dutch Nutrition Council, 1987) by:

\[
NE_{SA} = [(A \times B) + (1-A) \times f] \times H_c,
\]

where \( A \) is the fractional absorption of SA from the small intestine, \( B \) is the fraction of \( A \) unrecovered in the urine and \( H_c \) is the heat of combustion of the SA (Table 1). \( A, B \) and \( H_c \) are variables to be determined experimentally. The term \( f \) is the proportion of carbohydrate energy fermented that becomes available to the host; it is too difficult to determine on a routine basis so the Dutch proposed that their calculated value of 0.5 be used.

Equation 1 correctly implies that all SA entering the large intestine become completely fermented (see below), and that there are no significant interactions with other dietary constituents, which may not be entirely correct. SA cause some small intestinal ‘malabsorption’ of other energy sources in pigs (Livesey, 1990b) and 12 g oral lactose causes losses of protein and fat in lactase deficient subjects investigated by intubation (totalling 0.3 times the gross energy in the unavailable lactose—Debongnie et al. 1979). At present there is too little information to know whether this effect occurs with all SA to an extent that would merit amendment of equation 1.

An interaction occurs between foods and drinks containing SA (Bernier & Pascal, 1990; Livesey, 1991b). Rapid osmotic loading of the small intestine occurs with SA in drinks, compared with foods, leading to intestinal hurry, cramps and diarrhoea. Consequently, SA are not to be permitted in drinks sold to the public within the European Community (European Council Directive, 1990b). This has obvious implications for the design of experimental protocols for energy evaluation.

Unavailable complex carbohydrates

The calculation of net energy for UCC \( (NE_{UCC}) \), advanced in the British Nutrition Foundation (1990), was formalized in Livesey (1991a):

\[
NE_{UCC} = f \times D \times H_c
\]
Table 1. **Heats of combustion** of starch, unavailable complex carbohydrates (UCC), non-starch polysaccharides (NSP), oligosaccharides and sugar alcohols

<table>
<thead>
<tr>
<th></th>
<th>kJ/g</th>
<th>kcal/g</th>
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<tr>
<td><strong>Starch</strong></td>
<td>17.5</td>
<td>4.18</td>
</tr>
<tr>
<td><strong>UCC</strong></td>
<td>17.2</td>
<td>4.1</td>
</tr>
<tr>
<td><strong>Non-starch polysaccharides</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e.g. cereal, mean (range)</td>
<td>17.5</td>
<td>(16.7-18.5)</td>
</tr>
<tr>
<td>vegetable, mean (range)</td>
<td>16.8</td>
<td>(16.6-17.9)</td>
</tr>
<tr>
<td>fruit, mean (range)</td>
<td>16.5</td>
<td>(14.9-17.3)</td>
</tr>
<tr>
<td><strong>Isolated polysaccharide preparations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e.g. cellulose (Solka-floc)</td>
<td>17.5</td>
<td>4.18</td>
</tr>
<tr>
<td>guar gum</td>
<td>17.5</td>
<td>4.13</td>
</tr>
<tr>
<td>gum arabic</td>
<td>17.2</td>
<td>4.11</td>
</tr>
<tr>
<td>locust bean gum</td>
<td>17.9</td>
<td>4.28</td>
</tr>
<tr>
<td>gum karaya</td>
<td>17.2</td>
<td>4.11</td>
</tr>
<tr>
<td>Beta-fibre®</td>
<td>17.6</td>
<td>4.22</td>
</tr>
<tr>
<td>hydroxypropylmethylcellulose</td>
<td>22.0</td>
<td>5.25</td>
</tr>
<tr>
<td><strong>Oligosaccharide bulking agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e.g. Neosugar®</td>
<td>16.9</td>
<td>4.03</td>
</tr>
<tr>
<td>soya bean oligomers, SOE</td>
<td>16.4</td>
<td>3.91</td>
</tr>
<tr>
<td>SOR</td>
<td>16.8</td>
<td>4.02</td>
</tr>
<tr>
<td>Polydextrose®</td>
<td>17.0</td>
<td>4.05</td>
</tr>
<tr>
<td><strong>Sugar alcohols</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e.g. glycerol®</td>
<td>18.0</td>
<td>4.30</td>
</tr>
<tr>
<td>erythritol®</td>
<td>17.2</td>
<td>4.20</td>
</tr>
<tr>
<td>xylitol®</td>
<td>17.0</td>
<td>4.05</td>
</tr>
<tr>
<td>sorbitol®</td>
<td>16.7</td>
<td>3.99</td>
</tr>
<tr>
<td>mannitol®</td>
<td>16.7</td>
<td>3.99</td>
</tr>
<tr>
<td>lactitol®</td>
<td>17.0</td>
<td>4.06</td>
</tr>
<tr>
<td>maltitol®</td>
<td>17.0</td>
<td>4.06</td>
</tr>
<tr>
<td>Isomalt®</td>
<td>17.0</td>
<td>4.06</td>
</tr>
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SOE: soya-bean unpurified extract; SOR: soya-bean refined extract.

a Refers to the standard state (solid, liquid at 25 °C and 1 atmosphere).

b The exact composition of UCC varies, as the ratio of enzymically resistant starch to NSP which make up the UCC is not likely to be constant. The values shown are likely to be about right, and are in keeping with the suggestions of Southgate (1975) and Livesey (1990a).

c Values are calculated based on the heats of combustion of mono and disaccharides and their alcohols (Domalski, 1972), 20 kJ per glycosidic linkage and the regularity in heats of formation of chemical groups. In kJ/g (kcal/g), pentose and hexose 15.6 (3.72); dihexose 16.5 (3.95); trihexose 16.7 (4.00); tetrahexose 16.9 (4.04); pentahexose 17.0 (4.07); polyhexose 17.5 (4.18); polypentose 17.6 (4.20); polydeoxypentose 20.6 (4.93); polyhexuronic acid 13.1 (3.13); polymethyl hexuronic acid mix (35:65) with polyhexuronic acid 15.8 (3.78). NSP compositions are as given by Englyst et al. (1988, 1989), with a ratio of uronic to methyluronic acids in the fruits and vegetables assumed to be 35:65.


e The values for Polydextrose® are for the whole manufactured product (Cooley & Livesey, 1987).

Values to be determined experimentally are D the apparent digestibility of the UCC and, where possible, H, its heat of combustion (Table 1). Again the value of f is 0.5, but it was derived with slightly different quantitative assumptions and a different calculation procedure than was used for the SA by the Dutch Nutrition Council (1987). Equation 2 for UCC is simpler than equation 1 for SA because it is assumed that no energy from UCC is absorbed or lost to urine (see Livesey, 1990a).
For both SA and UCC, $f$ may be obtained as:
\[ f = (1 - a - b - c) \times g. \] (3)

The term $a$ is the proportion of carbohydrate energy lost as microbial mass in faeces; $b$ is the heat of fermentation; $c$ is the efficiency of combustible gas (hydrogen and methane) production; and $g$ is the efficiency of short chain fatty acid metabolism compared with glucose. Values for $a$, $b$, $c$ and $g$ are not known precisely and some may vary. The terms used in the calculation procedures (equations 1–3) are discussed below, beginning with the variation in the heats of combustion of carbohydrates.

**OCCURRENCE, COMPOSITION AND HEATS OF COMBUSTION OF FOOD CARBOHYDRATES**

With some exceptions it is virtually impossible to obtain plant cell wall carbohydrates sufficiently pure for accurate heats of combustion values to be determined by bomb calorimetry. By contrast, SA and some OS can be manufactured in relatively pure form, but bomb calorimetry is not always easily accessible to researchers investigating their metabolism and approximations are sometimes made. Some heats of combustion values are shown in Table 1.

The heat of combustion of starch is well documented but for the unavailable carbohydrates information is scant. The UCC in diets made with conventional foods have been assumed in research publications to have a heat of combustion only slightly less than that for starch (Table 1).

Plant cell wall polysaccharides contain a variety of monosaccharide components: arabinose, xylose, fucose, rhamnose, glucose, galactose, mannose and glucuronic and galacturonic acids (Englyst et al. 1988, 1989). About 35% of the uronic acids in fruits and vegetables may be methylated (and some acetylated), raising their heats of combustion. The NSP in conventional foods are heterogeneous with the proportions of uronic to neutral polysaccharides increasing in the order cereals < vegetables < fruits. Consequently, the heats of combustion of the NSP decrease along this order (Table 1).

Determined heats of combustion of cell wall polymer preparations may differ from expectations based on the NSP composition. Cell walls from wheat bran contain about 12% protein (23.6 kJ gross energy (GE)/g) and 12% phenolic material and lignin (~ 26 kJ GE/g) (Selvendran & Robertson, 1990) which raise the heat of combustion from 17.5 kJ GE/g cereal NSP to 19 kJ GE/g cell wall material. Chemical modification of NSP may also influence the heat of combustion. A value as high as 22.6 kJ/g for hydroxypropylmethyl cellulose arises (Table 1) because of the high heat of formation of the substituent groups.

There is interest in the use of OS, both to increase faecal bulk and to mix with intense sweeteners which otherwise lack the desirable bulk. An example finding early use is Polydextrose*, a citric acid catalysed random condensation product of glucose and sorbitol (9:1). Soya bean OS, held responsible for abdominal discomfort and flatulence, have been prepared as a water soluble extract (SOE) and as a refined product (SOR) (Ohmura et al. 1990). The extract contains more than 95% of the oligosaccharide raffinose (d-galactosyl-α1,6-d-glucosyl-α1,2-d-fructose) and a higher homologue called stachyose (d-galactosyl-α1,6-raffinose). The fructo-OS provide a second homologous series that occur naturally—in onions, asparagus, Jerusalem artichokes, all sources of inulin (Hidaka et al. 1983). The series includes 1-kestose (d-fructosyl-β1,2-sucrose), nystose (d-fructosyl-β1,2,1-kestose) and d-fructo-nystose (linked β1,2). A mixture of these OS is produced commercially as Neosugar® which finds use as a sugar substitute (Hidaka et al. 1982).
Polyols (SA) are classified as bulk sweeteners, though glycerol finds use in parenteral nutrition as an emulsifying agent with lipid feeds used in hospital practice (Livesey & Elia, 1985a) and is used as a plasticizer in confectionery. Erythritol is being developed as a bulk, low calorie sweetener in Japan (Oku & Noda, 1990). Interestingly, erythritol has been defined as a drug that has a mild prolonged action in reducing blood pressure by dilating blood vessels, and was once used to treat hypertension and angina (Concise Medical Dictionary, 1980). Sorbitol (hydrogenated glucose) has long been used in dietetic foods as its metabolism is less dependent on insulin than is glucose. Sorbitol also finds use as an energy source in artificial feeds used in hospital practice (Livesey & Elia, 1985a). Maltitol (D-glucosyl-a:1,4-D-sorbitol) is available in pure form or in mixtures with lesser amounts of sorbitol and maltotritol (D-glucosyl-a:1,4-maltitol) in the products Malbit® and Lycasin 80/55®. Lactitol (D-galactosyl-β1,4-D-sorbitol) is a hydrogenation product of the milk sugar D-lactose (van Velthuijsen, 1979) and Isomalt is a generic name for an approximately equimolar mixed disaccharide alcohol (D-glucosyl-α:1,6-D-sorbitol and D-glucosyl-α:1-D-mannitol) (Sträter, 1988). The latter is also named hydrogenated palatinose, hydrogenated isomaltulose, and Palatinit®.

**LOSSES OF CARBOHYDRATES TO FAECES**

Variable amounts of NSP of plant cell wall origin are recoverable from faeces as reported elsewhere (Cummings, 1981; Livesey, 1990a; see also the section on modelling of NSP degradation in the rat). Factors affecting NSP recovery in faeces have been reviewed (Cummings, 1983).

Unless accompanied by diarrhoea, the losses of dietary SA to faeces in man and model animals are usually negligible as evidenced with xylitol, sorbitol, mannitol, lactitol, maltitol and Isomalt and reviewed elsewhere (Dutch Nutrition Council, 1987; Würsch & Anantharaman, 1989; Bär, 1990; Bernier & Pascal, 1990). Added to this list are glycerol and erythritol (Oku & Noda, 1990). Linear OS bulking agents are also unrecovered in the faeces, for example Neosugar® (Tokunaga et al. 1989) and the oligomers from soyabean (Ohmura et al. 1990). However, about 50% of the carbohydrate of Polydextrose®, a branched and crosslinked OS, is said to be recoverable from rat faeces (Hobbs, 1988), though there is evidence that it may sometimes be less (Cooley & Livesey, 1987).

**LOSSES OF ENERGY DUE TO FERMENTATION**

*Production of microbial mass and faecal energy*

Carbohydrates undergoing fermentation give rise to multiplication of faecal microorganisms, so adding to those faecal energy losses from the undigested unfermented carbohydrates. On the basis of results after ingestion of 50 g lactitol in humans, the Dutch Nutrition Council (1987) suggested a conversion efficiency of about 0.2 kJ faecal bacteria per kJ carbohydrate fermented. In his review on SA, Bär (1990) also adopted a value of 0.2 kJ/kJ. By contrast, for the UCC from mixed diets and for NSP the British Nutrition Foundation (1990) and Livesey (1991a) suggested a value of about 0.3 kJ/kJ. The values were considered to range between 0.15 and 0.25 for SA (Bär, 1990) and between 0.2 and 0.4 kJ/kJ for NSP (Livesey, 1991a).

Microbial cell yields in vitro depend on growth conditions, varying with the same organism and substrate; high cell yield may be accompanied by significant amounts of microbial storage polysaccharides (glucans) (Hungate, 1963). The losses of energy to faeces from SA may also differ between experiments. For example, between 0.10 and 0.35 kJ/kJ Isomalt in both pigs and rats (Sinkeldam, 1983; Kirchgessner & Muller, 1983; Staudacher
There is no information on the interactions between SA and other unavailable carbohydrates or other dietary components affecting the production of microbial mass and faecal energy. Our unpublished work in the rat indicates no interactions between the NSP of uncooked cereal and dietary fat which affects faecal energy losses, but that guar gum interacts with dietary fat (R. F. Faulks, J. C. Brown and G. Livesey, AFRC Institute of Food Research). Possible interactions between UCC and dietary fat affecting faecal energy losses are being studied in man (P. Moe, USDA Human Nutrition Laboratory). Key & Mathers (1992) showed that increasing maize oil from 30 to 170 g/kg diet in rats fed either white or wholemeal bread appeared not to influence NSP digestibility, so effects of fat on the energy values of NSP would not be predicted.

**Hydrogen and methane production**

The efficiency of conversion of fermentable carbohydrate to the combustible gases molecular hydrogen and methane is low in humans compared with ruminants. For conventional foods the value is about 0·02 kJ gas (breath + flatus)/kJ carbohydrate fermented in man (Livesey & Elia, 1988). For 50 g loads of lactitol with food fed to humans, a conversion efficiency of about 0·03 applies (van Es et al. 1986). Based on the data of Fritz et al. (1985) who fed humans with 20, 35 and 50 g doses of Isomalt in food, the present author has calculated a conversion efficiency of almost 0·01 kJ gas per kJ Isomalt estimated to be fermented. This calculation assumed that 80% of Isomalt was fermented and only half of the combustible gases reached the breath. Breath hydrogen production from sorbitol, mannitol, Isomalt and Neosugar® is less than that from lactitol (Würsch et al. 1989). Methane production in man is either absent or small (Bjørneklett & Jenssen, 1982; Segal et al. 1988), so that low conversion efficiencies can be expected more generally. It should be noted that the above estimate of 0·01 kJ gas per kJ Isomalt estimated as fermented is notably less than estimates for other SA; such a low conversion efficiency is also observed in the present author’s unpublished studies on anaerobic fermentation in vitro. With other carbohydrates higher conversion efficiencies, about 0·08, have been observed in the pig (Müller & Kirchgessner, 1983, 1985), which serves only to suggest a possible species difference. The conversion efficiencies of 0·03–0·08 suggested by the Dutch Nutrition Council (1987) may have been influenced by observations in the pig; both theirs and the 0·05 adopted by the British Nutrition Foundation (1990) seem marginally to overemphasize these losses of energy.

The low levels of combustible gas production in man compared with ruminants and possibly the pig make it difficult to establish a general stoichiometry for the fermentation process in man. The theoretical amounts of H₂ and CH₄ production needed to balance a stoichiometric equation are more than 10 times the amount observed to be produced (Livesey & Elia, 1988). The occurrence in humans of acetogenic organisms (Lajoie et al. 1988) which use H₂ and CO₂ to form acetic acid seems a possible explanation of the low H₂ + CH₄ production. However, this would require considerably higher rates of acetic acid production, compared with propionic and butyric acids, than is currently thought to be the case. Other means of using the reducing powers of the excess H₂ have been identified (Gibson et al. 1990), yet none appears sufficient to explain the discrepancy. Clearly more information is needed.

**Heat of fermentation**

Stoichiometric considerations indicate the heat of fermentation to be about 0·065 times the energy in carbohydrate fermented in ruminants (Hungate, 1966). For fermentation studies in vitro, Arieli (1986) directly observed a value of 0·063, and a value of 0·07 was obtained in vivo (Webster, 1978).
It is difficult to make estimates for humans based on stoichiometric considerations because of uncertainty in the disposition of hydrogen. The Dutch Nutrition Council (1987) assumed the value would be between 0.02 and 0.05. The approximate value of 0.05 as adopted by the British Nutrition Foundation (1990) would seem about right.

Efficiency of short chain fatty acid metabolism

There is no consensus on the efficiency of converting the chemical energy of short chain fatty acids to ATP. Values of 0.69, 0.8–0.85 and 0.85 were chosen by Japanese workers (Iwakawa, 1989), the Dutch Nutrition Council (1987) and the British Nutrition Foundation (1990) respectively. Both 0.85 and 0.92 are indicated by the data of Blaxter (1989), in his Fig. 12.4 and Table 12.4 respectively. The particularly low value from Japan is based on a figure of uncertain origin for acetic acid with an assumed similarity for the other acids. Values as low as 0.6 have been found for acetic acid under unphysiological circumstances in ruminants (Blaxter, 1989).

Mixtures of acetic, propionic and butyric acids infused into sheep spare body fat with an efficiency close to 0.85, in keeping with an efficiency of oxidation relative to glucose of the same order (see Fig. 12.4 and Table 12.6 of Blaxter, 1989). In the pig (Roth et al. 1988) the efficiency of lean and fat deposition from intracaecal acetic and propionic acids are consistent with these theoretical expectations.

Accurate conversion efficiencies may be supposed difficult to calculate because of uncertainties in mitochondrial stoichiometry (Livesey, 1984). Within a probable range of mitochondrial stoichiometry the impact on the conversion efficiency is only small (Livesey & Elia, 1985a) and the following equation applies in which \( p_{Ac}, p_{Pr} \) and \( p_{Bu} \) are the energy proportions of the fatty acids (kJ acid per kJ total acids):

\[
g_{mix} = 0.848p_{Ac} + 0.865p_{Pr} + 0.920p_{Bu}.
\]

Clearly the conversion efficiency is not much affected by exchanges of propionic with acetic acid. The exchange of butyric with other acids affects \( g_{mix} \). The molar proportions generally found in humans are 0.60:0.24:0.14 (Cummings, 1981), which corresponds to energy proportions of 0.43:0.30:0.27 for \( p_{Ac}, p_{Pr} \) and \( p_{Bu} \) respectively. This mixture of acids gives a value for \( g_{mix} \) of 0.87.

Recommended efficiency values

From equation 3 the fractional loss of energy due to fermentation, \( f \), is given by: \( f = (1-a-b-c) \times g \). Rounding to 5% and based on the above discussion it is evident that for both SA and UCC: \( b = 0.05, c = 0.05 \) and \( g = 0.85 \). For UCC \( a = 0.3 \), consequently the value for \( f \) is 0.5. However, for the SA \( a = 0.2 \), so the corresponding value of \( f \) is 0.60. This contrasts with the 0.5 value recommended by the Dutch Nutrition Council (1987) for SA. The discrepancy arises for two reasons: firstly a lower efficiency of ATP production, 0.8–0.85 (mean 0.825), was selected by the Dutch, and secondly energy losses had been accounted for differently, by subtraction of the proportion 1-g, whereas multiplication of energy gains by \( g \) would have been correct. On the other hand it has been suggested that the Dutch method, like the present calculations, overestimates energy values to the extent that it takes no account of osmotic effects of the SA (Livesey, 1990b; see page 64). In view of this, the results of energy values calculated by the Dutch method would still seem about right.

There is insufficient evidence to recommend a value of \( f \) for use with OS. It seems likely that the value of \( a \) will be no greater than the fermentation energy losses repeatedly observed with Polydextrose® in the rat: 0.24 (Cooley & Livesey, 1987), 0.24 and 0.21 (Livesey, 1987) and 0.18 (Brown et al. 1987), so \( f \) would then be 0.6. However, choosing \( f \)
to be 0.5 for OS, even if not scientifically exact, is least discriminatory, and would make the same value applicable to all carbohydrates undergoing fermentation.

**SMALL INTESTINAL ABSORPTION OF NSP AND OLIGOSACCHARIDES**

The $\beta$-glycosidic linkages of NSP render them indigestible to mammalian enzymes of the small intestine which, except for $\beta$-galactosidase, degrade only $\alpha$-glycosidic linkages. However, bacterial colonization may lead to degradation of NSP, as in the pig where 0.2-0.7 of ingested NSP disappears before the terminal ileum (Millard & Chessen, 1984; Graham et al. 1986; Fadel et al. 1988, 1989) in association with microbial activity (Ratliffe, 1985; Chesson et al. 1985; Liu et al. 1985; Giesecke, 1990). In man 0.0-0.2 of ingested NSP is unrecovered in ileostomal fluid; the loss may be due to colonization of the stoma and fluid with micro-organisms (Sandberg et al. 1981, 1986; Englyst & Cummings, 1985, 1986, 1987). In normal humans there is no reason to believe that NSP undergo significant degradation accompanied by absorption anterior to the colon.

Polydextrose* contains a small fraction (~5%) of glucose which is absorbed; the remainder of the OS is thought to escape small intestinal digestion. It resists O-glucosidases (Rennhard, 1981), and $^{14}$CO$_2$ production from [U-$^{14}$C]polydextrose (freed of monomeric material) is delayed by comparison with glucose when administered orally to man (Fgdor & Bianchine, 1983) and rat (Fgdor & Rennhard, 1981). By contrast, up to 80% of the manufactured Polydextrose* is claimed to disappear rapidly from the alimentary tract of the rat (Lorenz & Grossklaus, 1984), possibly before reaching the large intestine (Krüger et al. 1990). The $\beta$(2-1)-linkage of the storage polysaccharide inulin (GF$_n$) renders this molecule resistant to mammalian digestive enzymes (Lewis 1912; Nilsson & Björck, 1988). Limited hydrolysis (13%) of inulin prior to the ileostoma in seven men who ingested 10 or 30 g each has been observed (Hessov et al. unpublished), but was accompanied by short chain fatty acid production by bacteria which may account for all or some of the loss of inulin. Possible acid hydrolysis of fructosans in the stomach was indicated by McCance & Lawrence (1929) and at pH 1.3 as much as 8% of inulin is converted to fructose in 2 h (Nilsson & Björck, 1988). At the higher pH in the human stomach the hydrolysis of inulin and cereal fructans is not thought to be significant (Flemström & Marsden, 1974; Nilsson & Björck, 1988), though it is difficult to quantify in vivo. Based on the production of breath hydrogen, Rumessen et al. (1990) concluded that almost all of the 10–30 g loads of inulin ingested in man must be fermented. Such evidence is not very reliable because of uncertainty in the stoichiometry of conversion of inulin to hydrogen gas (see below). The fructo-OS called Neosugar* ($\beta$1-2 linked GF$_n$) also appeared to be undigested in the small intestine as judged by the time course of $^{14}$CO$_2$ production in germ-free rats (Oku et al. 1984), in normal rats and in man (Hirayama & Hidaka, 1990) consistent with negligible absorption from the small intestine. Raftilose*, produced by the hydrolysis of inulin to the hetero-oligomer of glucose and fructose (GF$_n$) and the homo-oligomer of fructose, like inulin and Neosugr* is not expected to show significant degradation prior to the colon. The OS of the raffinose family from soya beans also appear to resist mammalian enzymes (Ohmura et al. 1990) and 88% of raffinose escapes the small intestine of man (Saunders & Wiggins, 1981).

Generally it seems that all $\beta$-linked oligo and polysaccharides examined escape absorption in the small intestine, and it is hard to find strong evidence that small percentages are degraded with some absorption occurring prior to the colon in healthy people.
SMALL INTESTINAL ABSORPTION, URINARY LOSSES AND METABOLISM OF SUGAR ALCOHOLS

Experimental approaches to determine the extent of small intestinal digestion and absorption of the SA are several and the reliability of the results, or the means to interpret them, is not always good. Consequently, there has been much discussion on the assessment of small intestinal losses of the SA (Dutch Nutrition Council, 1987; Würsch & Anantharaman, 1989; Bär, 1990; Bernier & Pascal, 1990). Below, some reasonably firm conclusions have been drawn for erythritol, arabitol, mannitol and lactitol when administered as drinks on an empty stomach, however, information is poor with other SA. A European Council Directive (1990b) is to prohibit the use of SA in drinks. Unfortunately, there have been few rigorous or published human studies assessing small intestinal SA absorption from foods. Therefore one can do no better at this time than to examine also the methods for assessing small intestinal absorption of SA.

It is evident in man that molecular size is an important determinant of the extent of SA absorption from the small intestine and subsequent excretion in the urine (Fig. 1). After ingestion with water the urinary excretion of 10–20 g erythritol (C4) is 0·91 (Oku & Noda, 1990), of β-arabitol (C5) is 0·53 (Crick, 1961), of 10–60 g mannitol (C6) is 0·20–0·35 (Nasrallah & Iber, 1969; Laker et al. 1982; Elia et al. 1987; Juby et al. 1989) and of 24–40 g lactitol (C12) is 0·02 (Grimble et al. 1988; Metzger et al. 1988). These data appear reasonably reliable, being corroborated by other evidence. With erythritol the high loss (0·91) is independent of dose (10 or 20 g) and supported by evidence in the rat where 0·94 is excreted in the urine at doses up to 0·05 w/w of the diet (Noda & Oku, 1990). The excretion of β-arabitol in the pig is 0·40 (Näsi & Tanhuopää, 1981) and in the dog is 0·60 (Keller et al. 1978, 1979) – both similar to that in man. The urinary excretion of mannitol when infused intragastrically at high dosage in man, 0·2–1·0 g/min, was also 0·19–0·30 of the dose (Vidon et al. 1983). This is in agreement with the work of Saunders & Wiggins (1981) who administered 10 g mannitol in a mixture with lactulose and raffinose to ileostomists and recovered 0·74 at the terminal ileum. The <0·02 loss of lactitol to urine is similar to the 0·005 loss suggested by earlier apparently unpublished work (see van Velthuijsen, 1979) and compatible with the lack of effect of this SA on the urinary excretion of energy (van Es et al. 1986). Further, jejunal perfusion in vivo with 10–100 mM lactitol in man showed insignificant absorption (Patil et al. 1987).

The use of urinary excretion as a measure of SA absorption is dependent on the lack of their metabolism in the tissues. The loss of 14CO2 from uniformly labelled erythritol administered to rats is delayed in comparison with glycerol and consistent with only a small amount (<0·05) of fermentation (Noda & Oku, 1990). Arabinol is suggested by Bär (1990) not to be metabolized on the basis of reports by Crick (1961) and McCormick & Touster (1961). Intravenous [U-14C]mannitol leads to the excretion of only 0·02 of the dose as 14CO2 in man (Nasrallah & Iber, 1969). Similarly, intravenous [14C]lactitol in man is 0·95 recovered in the urine, as reported in Bernier & Pascal (1990).

Urinary loss is not an index of sorbitol and xylitol absorption because they are metabolized by human tissues. Intravenous infusion of sorbitol and xylitol at very high rates, 0·5 g/h per kg body weight, results in losses to urine of only 0·11 and 0·22 respectively (Heuckenkamp & Zöllner, 1972; Bickel et al. 1973), so demonstrating a substantial metabolic capacity. However, in agreement with Bär (1990) there seem to be no theoretical or practical grounds on which to suppose that sorbitol (C6) should be absorbed differently from mannitol (C6), or that xylitol should be absorbed differently from arabitol.

The disaccharide SA maltitol and Isomalt, like lactitol, are not expected to be absorbed, but may be partly hydrolysed within the small intestine to absorbable glucose and partly
absorbable sorbitol and mannitol (for review see Ziesenitz & Siebert, 1987). About 0.03 of orally administered maltitol (10 g with breakfast) appears in human urine (Rennhard & Bianchine, 1976). The occurrence of Isomalt and its free hexitols in human urine after the ingestion of 50 g Isomalt (in water without food) is < 0.005 of the dose (Grupp & Siebert, 1978), which is consistent with very little small intestinal hydrolysis. Whether urinary mannitol occurs after Isomalt administration with food in man is not known, but there are no such losses in the rat (Siebert et al. 1975).

Various indirect approaches have been used to assess the extent of absorption of SA. Tentative suggestions of the extent of absorption and energy value of SA have been based on breath hydrogen production from sorbitol, lactitol, maltitol and Isomalt (Beaugerie et al. 1989), and from sorbitol, mannitol, maltitol, isomalt, lactitol and Neosugars® (Würsch et al. 1989) when administered on an empty stomach. The suggestions are highly dependent on four assumptions: first, that the rate or extent of fermentation of all substrates reaching the colon is similar; second, that the stoichiometry of hydrogen production from carbohydrate fermentation is constant; third, that the ratio of hydrogen excretion in breath to flatus is constant; fourth, that the SA have no impact on substrate fermentation from the previous day’s food intake. These assumptions have not been tested rigorously. There is only limited information indicating that some sugars (not including SA) give similar amounts of hydrogen in faecal incubations (Burbige et al. 1983; Flourié et al. 1988) whereas a lactitol fermentation (Würsch et al. 1989) and probably a sorbitol fermentation (Würsch & Anantharaman, 1989) yield more hydrogen gas than a lactulose fermentation. Hydrogen production may differ by up to 10-fold between different SA when incubated with human faecal micro-organisms (T. Smith and G. Livesey, unpublished). Consequently, the published between-SA comparisons of breath hydrogen responses seem to be as much misleading as helpful.

A promising technique is ileal intubation, the sampling of terminal ileal digesta and a non-digestible marker through a nasogastric tube. Application of this technique (Beaugerie...
et al. 1990) gave unexpected, so possibly doubtful (van Es, 1991), high values for sorbitol absorption (mean 0.79 after a 10 g load in water with breakfast) and for maltitol digestion and absorption (mean 0.76). From these data Bernier & Pascal (1990) concluded that food increased the amount of SA absorbed from drinks. Sorbitol absorption is passive, so is probably related to water flux through the small intestinal wall. The absorption of available carbohydrates in foods may increase water flux and in turn the rate of sorbitol absorption. Livesey (1990b) had already indicated that food intake lowered the rate of osmotic loading of the small intestine by delaying stomach emptying. Both mechanisms probably operate to increase one’s tolerance to SA and modify energy availability. While the results with the intubation technique are uncertain there are no strong practical or theoretical grounds on which to exclude the data.

Radiorespirometry with 10 g [U-14C]maltitol after breakfast provides a breath 14CO2 response consistent with a high absorption of maltitol in man (Rennhard & Bianchine, 1976). Two peaks of 14CO2 are produced, one corresponding to small intestinal absorption and one at a later time corresponding to large intestinal fermentation. Unfortunately, fusion of the two peaks makes it difficult to quantify accurately how much of the maltitol reaches the large intestine. Moreover, when maltitol is given with water alone, the initial peak seems to be accompanied by increased amounts of breath hydrogen (Tsuji et al. 1990) so the second peak may underemphasize the proportion undergoing fermentation. Despite these difficulties the early exhalation of labelled CO2 from uniformly labelled maltitol by man is much higher when given with a breakfast (Rennhard & Bianchine, 1976) than when given with water (Tsuji et al. 1990), again suggesting that food increases the fractional absorption of maltitol.

The conclusion that food enhances the fractional small intestinal absorption of sorbitol and maltitol may not apply to Isomalt. In cannulated pigs fed 0.05 to 0.20 of the weight of the diet as Isomalt, the ileal recovery was > 0.60 independent of dosage (van Weerden et al. 1984a, b; see also Livesey, 1990b). This value compares with 0.3–0.8 for NSP in pigs as already noted and in part may be due to the colonization of the pig stomach and small intestine with microflora (Dutch Nutrition Council, 1987). Recovery of 0.58 of Isomalt from human ileostomists was observed when administered with water alone (Kroneberg et al. 1979). Incomplete passage of digesta through the stomach and small intestine and colonization with fermentative bacteria probably contribute to the incomplete recovery.

MODELLING OF NSP DEGRADATION IN THE RAT

Obtaining precise information on the apparent digestibility of NSP in man is not more technically difficult than in the rat. There are numerous examples of such studies (see Cummings, 1981; Livesey, 1990a). Reasons for choosing the rat are several. Food grade material may not be available or too little may be available for study in man. More frequent is the consideration of study costs being less in the rat, and that results can be obtained more rapidly – important when information is needed urgently. Further, there may be a need for the simultaneous investigation of aspects of metabolism requiring invasive or destructive techniques (Davies et al. 1991).

The apparent digestibilities of NSP appear similar over a wide range of NSP fermentation in man and rat (Fig. 2). There are, however, limitations to the interpretation of the data in Fig. 2.

Clear species differences exist for some of the individual anhydrosugars of NSP (Nyman et al. 1986). Comparative data on NSP rich in uronic acids are limited. Uronic acids from apple, cabbage, and carrots are > 87% utilized in the rat (Nyman et al. 1986) and pure pectins are almost completely fermented in man (Cummings et al. 1979). In the diet studies
of Hanson, Feldheim and Wisker (see Mathers, 1991) the apparent digestibility of uronic acids was similar in rat and man at low UCC intakes, whereas at high intakes a slightly lower (~10%) value in man compared with the rat was found; the difference if real was no greater than a similar small difference found for the neutral NSP.

When considering the variation about the line of identity in Fig. 2 there are several possible causes. For example, there may be differences in the analytical methods for NSP applied in one compared with another species (Nyman et al. 1986), differences in the basal diets and differences in the duration of adaptation to the particular NSP diet. Further, precise interpretation of the data is difficult because of the occurrence of faecal polysaccharides from mucus and bacteria. For example, 0.98 of guar gum when fed to man appears to be fermented, but this increases faecal losses of xylose, glucose, rhamnose and fucose whereas these are either absent (xylose, rhamnose and fucose) or are only a minor constituent (<1%) of the gum, which is predominantly (>95%) a galactomannan.

At first, studies comparing fermentation in man and other animals led to the suggestion that small animals, such as the rat, are of no use when studying the problems of human nutrition (Van Soest et al. 1983). Small animals were seen to need more food energy per unit body weight, so require faster transit through the gastrointestinal tract which would lower the extent of cellulose fermentation. Mathers (1991) also suggested possible, though not great, man–rat differences with higher intakes of NSP and shorter transit times in the rat. These suggestions imply either a lack of adaptive mechanisms or a low capacity for adaptation to higher rates of fermentation.

Both man and rat can adapt to high rates of fermentation such as occur with intakes of UCC or NSP as high as 13% of dietary gross energy (Livesey, 1990a, and unpublished; Key, 1990), much higher than with habitual diets of Western subjects. In the rat, an expansion of the caecum occurs. There is little information on the mechanism of adaptation...
in humans; it may be that with Western diets the colon operates well below capacity. At least two other adaptive mechanisms have been suggested for animals. One is the adoption of an exceptionally low metabolic rate as in monotremes, marsupials, hyraxes and sloths (Björnhag, 1987). Another is the development of a mechanism which retains easily fermented particles and allows passage of coarse particles difficult to degrade—the so-called colonic separation mechanism (Sperber, 1985).

The observation in Fig. 2 is fortunate—it differences do exist amongst other species. For example the pig obtains substantial amounts of energy from the extensive degradation of NSP (Keys et al. 1970; Longland, 1990) by bacterial activity in the stomach and ileum (Chesson et al. 1985; Liu et al. 1985; Ratcliffe, 1985; Giesecke, 1990) and the substantial fermentation capacity of the large coiled colon (Argenzio & Stevens, 1984). Poultry represent an opposite extreme, being poorly able to ferment NSP (Carré & Leclercq, 1885; Longstaff & McNab, 1987). This is common with most avian species (Morton, 1978), exceptions being Canada geese (Buchsbaum et al. 1986) and the Australian wood duck (Dawson et al. 1989). The findings with poultry are important, showing that they are not suitable for the assay of carbohydrate fermentability and energy value; thus the studies of White et al. (1988) will have seriously underestimated the energy values of randomly bonded glucose and Polydextrose® for application in humans.

The man–rat similarity (Figure 2) may find use. Currently, the rat is being examined as a possible model for prediction of the fermentability and energy values of isolated NSP in humans, at the same time as a possible in vitro technique with the same goal; each is part of a paneuropean interlaboratory study (International Life Sciences Institute (Europe), Brussels).

**SPECIFIC DIGESTIBLE AND NET ENERGY VALUES**

*Unavailable complex carbohydrates in mixed diets*

At this time it is not possible to estimate the energy values for the UCC in all or even many foods. General values can be assigned: as discussed already these are 8.4 kJ digestible energy (DE)/g (or 2 kcal DE/g) and 6 kJ NE/g (1.5 kcal NE/g) for mixed diets. As shown in Fig. 2 these values appear independent of daily intake. Similar values can be calculated from the observations of Hansen, Wisker and Feldheim (reported in Mathers, 1991) for NSP fed to humans at two dose levels (Fig. 2).

*Specific non-starch polysaccharides*

Often NSP are chosen for their faecal bulking characteristics, so have a low apparent digestibility, or for their high solubility so tend to have high apparent digestibilities. A whole range of energy values is then possible as shown in Fig. 2. These and other specific energy values for NSP are given in Table 2.

*Oligosaccharides*

The calculated (eqn 1) energy values for each OS (Table 2) tend to be similar because they are essentially non-absorbed and usually undergo complete fermentation. A possible exception is Polydextrose®, some of which may escape fermentation to enter faeces. A net energy value of about 6 kJ/g (or 1.5 kcal/g) has been suggested for Polydextrose® (Bernier & Pascal, 1990) which is based on observations with a small scale radiochemical preparation in man (Figdor & Rennhard, 1981) and rat (Figdor & Bianchine, 1983). The latter authors claim a value of 4 kJ/g or 1 kcal/g for Polydextrose®, but there are no published data on the energy value of the manufactured product in humans! Data obtained
Table 2. Specific digestible and net energy values of starch, unavailable complex carbohydrates (UCC), non-starch polysaccharides (NSP) and oligosaccharides

<table>
<thead>
<tr>
<th>Carbohydrate</th>
<th>Digestible energy (DE)</th>
<th>Net energy (NE)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>kJ/g</td>
<td>kcal/g</td>
</tr>
<tr>
<td>Polysaccharides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Available starch – as starch</td>
<td>17.5</td>
<td>4.2</td>
</tr>
<tr>
<td>– as monosaccharide</td>
<td>15.7</td>
<td>3.75</td>
</tr>
<tr>
<td>UCC in mixed diet&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.4</td>
<td>2.0</td>
</tr>
<tr>
<td>Solka-floc cellulose&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>HPMC&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.8</td>
<td>0.2</td>
</tr>
<tr>
<td>Gum karaya&lt;sup&gt;d&lt;/sup&gt;</td>
<td>2.4</td>
<td>0.6</td>
</tr>
<tr>
<td>Psyllium gum&lt;sup&gt;e&lt;/sup&gt;</td>
<td>40</td>
<td>1.0</td>
</tr>
<tr>
<td>Wheat bran NSP&lt;sup&gt;f&lt;/sup&gt;</td>
<td>42</td>
<td>1.0</td>
</tr>
<tr>
<td>Apple NSP&lt;sup&gt;g&lt;/sup&gt;</td>
<td>8.2</td>
<td>2.0</td>
</tr>
<tr>
<td>Carrot NSP&lt;sup&gt;h&lt;/sup&gt;</td>
<td>8.8</td>
<td>2.1</td>
</tr>
<tr>
<td>Beta-fibre&lt;sup&gt;i&lt;/sup&gt; NSP</td>
<td>9.1</td>
<td>2.2</td>
</tr>
<tr>
<td>Cabbage NSP&lt;sup&gt;j&lt;/sup&gt;</td>
<td>11</td>
<td>2.6</td>
</tr>
<tr>
<td>Guar gum&lt;sup&gt;k&lt;/sup&gt;</td>
<td>12</td>
<td>2.9</td>
</tr>
<tr>
<td>Gum arabic&lt;sup&gt;l&lt;/sup&gt;</td>
<td>12</td>
<td>2.9</td>
</tr>
<tr>
<td>Oligosaccharides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polydextrose&lt;sup&gt;me&lt;/sup&gt;</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>Neosugar&lt;sup&gt;me&lt;/sup&gt;</td>
<td>13</td>
<td>3.2</td>
</tr>
<tr>
<td>Raftilose&lt;sup&gt;me&lt;/sup&gt;</td>
<td>13</td>
<td>3.2</td>
</tr>
<tr>
<td>Soyabeen SOE&lt;sup&gt;m&lt;/sup&gt;</td>
<td>13</td>
<td>3.1</td>
</tr>
<tr>
<td>SOR&lt;sup&gt;m&lt;/sup&gt;</td>
<td>13</td>
<td>3.2</td>
</tr>
</tbody>
</table>

HPMC, hydroxypropylmethylcellulose; SOE, soya-bean unpurified extract; SOR, soya-bean refined extract.

<sup>a</sup> Refers to the UCC in diets for which data was analysed by Livesey (1990a, 1991a).

<sup>b</sup> Harley et al. (1989).

<sup>c</sup> From the UCC study of Davies (1990). Assumes NE = 0.7 x DE.

<sup>d</sup> Based on the calculations of Livesey (1990a). Assumes NE = 0.7 x DE.

<sup>e</sup> Based on the human feeding studies of Nyman et al. (1986), assumes DE = 0.7 x D x H and NE = 0.5 x D x H, where D = digestibility and H = heat of combustion. The digestible energy value for wheat bran NSP compares with a determined value of 4 kJ/g in man (Livesey, 1990a). Directly determined estimates of NE for guar gum in the rat are less than indicated in the table due to a putative thermogenic effect of this gum (Davies et al. 1991).

<sup>f</sup> From the rat study of Davies (1990). Assumes NE = 0.7 x DE.

<sup>g</sup> Based on the calculations of Livesey (1990a). Assumes NE = 0.7 x DE.

<sup>h</sup> Based on the human feeding studies of livesey (1990a). Assumes NE = 0.7 x DE.

<sup>i</sup> Small difference for SOE compared with other OS is due to slightly smaller heat of combustion (Table 1).

<sup>j</sup> From the energy balance studies of Johnson et al. (1990) in the rat. Assumes NE = 0.7 x DE.

<sup>k</sup> From the energy balance studies of Harley et al. (1989) in the rat. Assumes NE = 0.7 x DE.

<sup>l</sup> No information on the manufactured material is available for man in vivo! The upper limit assumes Polydextrose, as manufactured, to be undigested in the small intestine. A limit of > 6.5 kJ/g or > 1.5 kcal/g is suggested on the basis of observations with the manufactured Polydextrose<sup>me</sup>: fermentation with human faecal micro-organisms and studies in the rat (see the text).

with the large scale manufactured product in the rat suggest much higher net energy values than are claimed for the radiochemical product. Assuming NE = 0.7 x DE, the 13.5 kJ DE/g or 3.2 kcal DE/g determined in the rat (Cooley & Livesey, 1987) becomes 9.5 kJ NE/g or 2.3 kcal NE/g. Values > 8.4 kJ NE/g or > 2 kcal NE/g are also claimed for the manufactured product both by Lorenz & Grossklaus (1984) and by Krüger et al. (1990). There is no expectation that man and rat should be very different (Fig. 2). Our unpublished work on the resistance of the manufactured product to both human faecal and rat caecal micro-organisms in vitro suggests a net energy value for manufactured Polydextrose<sup>me</sup> of about 8 kJ/g or 2 kcal/g. In the absence of rigorous studies in humans with the manufactured product, it is possible to be confident about an upper limit only (Table 2).
Table 3. The net energy values of sugar alcohols (SA)

<table>
<thead>
<tr>
<th>Sugar alcohol</th>
<th>A&lt;sup&gt;a&lt;/sup&gt;</th>
<th>B&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Net energy values from equation 1</th>
<th>Net energy values from other approaches&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycerol</td>
<td>1.0</td>
<td>1.0</td>
<td>18 kJ/g 4.3 kcal/g</td>
<td>-- --</td>
</tr>
<tr>
<td>Erythritol</td>
<td>0.9</td>
<td>0.0</td>
<td>0.9 kJ/g 0.2 kcal/g</td>
<td>-- --</td>
</tr>
<tr>
<td>Arabinol</td>
<td>&gt; 0.5</td>
<td>0.0</td>
<td>&lt; 5 kJ/g 1.5 kcal/g</td>
<td>-- --</td>
</tr>
<tr>
<td>Xylitol</td>
<td>&gt; 0.5</td>
<td>1.0</td>
<td>&gt; 12 kJ/g 3 kcal/g</td>
<td>-- --</td>
</tr>
<tr>
<td>Mannitol</td>
<td>&gt; 0.2</td>
<td>0.0</td>
<td>&lt; 7 kJ/g 2 kcal/g</td>
<td>-- --</td>
</tr>
<tr>
<td>Sorbitol</td>
<td>&gt; 0.2 &lt; 0.8</td>
<td>1.0</td>
<td>&gt; 10 &lt; 15 kJ/g 2 &lt; 3.7 kcal/g</td>
<td>-- --</td>
</tr>
<tr>
<td>Lactitol</td>
<td>0.0</td>
<td>0.0</td>
<td>8.5 kJ/g 2.0 kcal/g</td>
<td>7 1.7</td>
</tr>
<tr>
<td>Malitol</td>
<td>0.8</td>
<td>1.0</td>
<td>15.3 kJ/g 3.7 kcal/g</td>
<td>-- --</td>
</tr>
<tr>
<td>Isomalt</td>
<td>&lt; 0.4</td>
<td>&gt; 0.75</td>
<td>&lt; 12&lt;sup&gt;d&lt;/sup&gt; kJ/g 2&lt;sup&gt;d&lt;/sup&gt; kcal/g</td>
<td>8.4&lt;sup&gt;d&lt;/sup&gt; 2.0&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup> A is the fractional small intestinal absorption of energy from the SA.

<sup>b</sup> B is the fractional availability of A.

<sup>c</sup> For discussion on the other approaches see the text.

<sup>d</sup> In consideration of the energy value of Isomalt, a value of 8.4 kJ or 2 kcal per g has been judged as prudent for humans (Livesey, 1990b, c); also see text.

Sugar alcohols

Net energy values, calculated according to eqn 1, are given in Table 3 together with a summary of information on their fractional absorption from the small intestine (A) and the fractional availability of this absorbed energy (B). The values refer to SA present in foodstuffs.

Values for glycerol, erythritol and lactitol are based entirely on human studies and the calculated net energy values are considered reasonably reliable. In the case of lactitol, the value has been validated approximately in a complete energy balance trial using 8 male volunteers in which a 7 (SEM 0.7) kJ NE/g value was obtained (van Es et al. 1986).

With arabinol, xylitol, mannitol and sorbitol information is limited. It is assumed that the fractional absorption of xylitol is like that of arabinol, and sorbitol like mannitol (as discussed). Minimum values are given for fractional absorption based on data in Fig. 1 given that food would increase their absorption (as discussed) to an extent unknown except for sorbitol where fractional absorption after ingestion with food has been determined and may be as high as 0.8 (Beaugerie et al. 1990). It is noteworthy that an increased fractional absorption of arabinol and mannitol would lower their energy values as these SA are diverted away from fermentation towards urine. By contrast an increased absorption of xylitol and of sorbitol would increase their energy values since these SA are metabolized in preference to being excreted. These assumptions need to be tested with experiments.

The calculated net energy value of maltitol is based on two human studies with differing approaches to estimating the fractional absorption after ingestion with food, about 0.8. One is the intubation study of Beaugerie et al. (1990), the other is the 14CO2 method of Rennhard & Bianchine (1976). As already discussed, neither method is thought highly reliable, hence the value needs confirmation.

At present there is no published information on the fractional absorption of Isomalt from foods fed to humans. Because of a higher contribution of micro-organisms to small intestinal activities in pig than in man, the fractional absorption of this SA in man is probably not greater than in the pig, 0.4 (see Livesey, 1990b), so an upper limit to fractional absorption would be prudent.
absorption is suggested for Isomalt (Table 3). Glucose and sorbitol absorbed from Isomalt are fully available, but the mannitol absorbed is expected to be excreted in the urine. Consequently, the availability of energy from that fraction of Isomalt which is absorbed from the small intestine cannot be less than 0.75 of the absorbed fraction, and is likely to be greater (> 0.75, Table 3) since the monosaccharide alcohols are absorbed more slowly than glucose. On this basis an upper limit to the energy value of Isomalt would be 12 kJ or 2.9 kcal/g. However, in practice it may be much less as indicated by estimates made in other ways. Studies in which the terminal ileum of the pig was cannulated also show ileal energy losses to be 0.99 times the additional gross energy intake from 0, 5, 10 and 20% Isomalt present in the feed (Livesey, 1990b). Moreover, the loss of energy to the faeces average 0.22 kJ/kJ Isomalt ingested in both rat and pig (see page 67); this also is consistent with an average zero absorption of Isomalt. All these data suggest that Isomalt has a net energy value of about 8.4 kJ/g (or 2 kcal/g). Further, based on the changes in body composition on feeding 0, 10 and 20% Isomalt in a basal diet, with each dose fed at several dietary intakes to achieve body weight changes in the rat of −10, 0, +10 and +20% of initial body weight, a value of 8.1 kJ NE/g Isomalt was determined (Livesey, 1990c). These observations form the basis of the net energy value given to Isomalt in the right-hand columns of Table 3, which needs to be confirmed in humans.

**PREDICTABILITY OF ENERGY FROM CEREALS**

A statistical analysis of much of the world pool of data on the digestibility of energy in human diets indicates (Livesey, 1991b) that diets high in whole grain cereals result in energy losses to faeces greater than with diets of mixed sources of fibre and diets high in fruits and vegetables of comparable UCC content as analysed. Diets very high in kibbled barley (Judd, 1982), wheat grain (Judd, 1982; Wisker et al. 1988), and brown rice (Miyoshi et al. 1986) appear to be digested less well, by 11, 4 and 5% of gross dietary energy respectively. More than one mechanism seems to be involved. In one, cereal cell walls protect starch from the usual process of digestion, as observed with barley in ileostomists where the total amount of UCC is at least twice that indicated by the barley NSP, the remainder being physically resistant starch (Livesey, 1991c). Milling of the flaked barley to disrupt the cell walls prevents the ileostomal losses of the starch. The rat is different from man: cell walls of flaked barley are not responsible for the loss of starch at the terminal ileum (Livesey, 1991c), possibly because the rat finely chews the flaked cereals and possibly because smaller particles of digesta are formed in the stomach and intestine of the rat compared with man. For this reason the rat may prove to be a poor model for the determination of starch availability from whole grain cereal products, and possibly other foods where physical form is a determinant of nutrient availability.

Starch reaching the large intestine from the barley flake in man, in addition to the rat (Livesey, 1991c), is too small to explain the low energy value of the barley diets, which implies an additional mechanism operating to increase faecal energy losses in both species when eating this cereal.

By contrast with the barley studies in which the starch is present mostly in an ungelatinized form (Livesey, 1991c) the ileostomal losses of starch with cooked oats (Englyst & Cummings, 1985) is small, at about 0.02 of the ingested starch. More consideration needs to be given to the extent of starch malabsorption with different cereals and the influence of food processing on physical form and starch availability.

It is not yet possible to predict net energy availability from high cereal diets. Energy losses to faeces may be predictable with knowledge of the intake and apparent digestibility
of the UCC by use of a formula derived by regression analysis (eqn 8 of Livesey, 1990a), but the formula is not yet validated with an adequate number of observations on cereal diets and provides no means to calculate net energy values. Clearly more information is needed on the balance of energy across the colon and events in the colon when high cereal diets are eaten.

**COMPATIBILITY OF DIETARY ENERGY VALUES**

An important use of food energy values is for the calculation of the energy values of whole diets. Previously, energy values for UCC were determined by a method (Southgate & Durnin, 1970) which gave a value termed the partial digestible energy value (Livesey, 1991a). Such values varied depending on the amount of UCC eaten and took into account the influence of UCC on energy availability from the whole diet. However, as noted before (Livesey, 1990a), caution is needed: partial digestible energy values are not compatible with the British (or the Atwater, 1910) system of food energy assessment. This is because the UCC at moderate intakes cause losses of energy to faeces as protein and fat in amounts in excess of that expected from the microbial matter generated due to UCC fermentation, amounts that are already accounted for in the protein and fat energy values (see Livesey, 1990a). Both the digestible and net energy values reported here (Tables 2 and 3) are compatible with the British system of food energy assessment, that is with the energy conversion factors of 16-7, 37-5 and 15-7 kJ (4, 9 and 3-75 kcal) per g of protein, fat and carbohydrate expressed as monosaccharide respectively. While both the British (McCance & Widdowson, 1946) and the Atwater (1910) energy conversion factor systems currently receive widespread usage, it should be recognized that other, empirical systems exist and offer greater accuracy and precision when calculating whole diet energy values and so should receive preferential use when possible (Livesey, 1990a, 1991a). The digestible and net energy values given in Tables 2 and 3 are compatible with the empirical calculation methods of Livesey (1991a), though not with all empirical calculation methods (see Livesey, 1990a).

**REFERENCES**


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*Printed in Great Britain*