

**Dietary sugars and human disease : report of the Panel on Dietary Sugars,
Committee on Medical Aspects of Food Policy.**

Contributors

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Department of Health

Report on Health and Social Subjects

37



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Report of the Panel on
Dietary Sugars

Committee on Medical Aspects
of Food Policy

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Dietary Sugars and Human Disease

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Dietary Sugars

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London
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Preface

In June 1986 the Committee on Medical Aspects of Food Policy convened a Panel to inquire into and report on the health aspects of the consumption of sugars in United Kingdom diets.

The Panel met on ten occasions and reviewed much published information. It invited and received written submissions from a variety of interested organisations and individuals. This report summarises the findings of the Panel and gives its conclusions and recommendations.

I am very grateful to the members of the Panel who have generously given of their time and knowledge, to those who have prepared and supplied information for the Panel and to the secretariat for their unwavering support.

SIR DONALD ACHESON

Chairman of Committee on Medical Aspects of Food Policy

Committee on Medical Aspects of Food Policy

Panel on Dietary Sugars*

Members

H Keen (Chairman)

A Black
J Durnin
J Garrow
K Heaton
A Jackson

J Mann
D Naismith
E Newsholme
N Read
A Rugg-Gunn
T Silverstone

Observers

J Ablett
D Buss

P Clarke
C Howard

Secretariat

M Wiseman
(Medical)

R Wenlock
(Scientific)

K Follin
(Administrative)

N Bateson (Minutes)
E Lohani (Minutes)
D Roberts (Minutes)

*For title and affiliation see Appendix A, page 86

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1. Introduction

1.1 Background to the Panel

1.1.1 In its Report published in 1984¹ the Panel on Diet in relation to Cardiovascular Disease of the Committee on Medical Aspects of Food Policy (COMA) recommended to the general public that 'intake of simple sugars (sucrose, glucose and fructose) should not be increased further', and that 'these sugars and foods containing them are appreciable sources of food energy and may contribute to obesity. Certain foods containing these sugars may also contain saturated fatty acids (eg cakes and biscuits)'. It also noted that 'restriction of intake of these sugars has been recommended on other health grounds (eg dental caries)'.

1.1.2 COMA has noted the conflicting evidence and changing opinion in recent years about the role of dietary sugars in cardiovascular disease, dental caries and other disorders. It has considered the views expressed in two recent expert reports on sugars in the diet, one in the UK² and the other from the United States.³ In 1986 COMA convened this Panel on Dietary Sugars to review the role of dietary sugars in disease in the United Kingdom.

1.2 Terms of reference of the Panel

'To examine the role of dietary sugars in human disease and to make recommendations'.

1.3 Interpretation of terms of reference

Although the Panel has concerned itself with carbohydrate foods with the chemical characteristics of sugars, it has given particular attention to sucrose as the sugar predominating in the UK diet. Sugars have been considered in relation to a wide spectrum of human diseases and reviewed in the light of published evidence. Obesity as a risk factor for several other conditions (eg diabetes, hypertension) occupied a special position in these considerations. We have mainly considered observations and experiments in humans; animal experiments have been considered only when they appeared to have clear relevance to human disease. Artificial sweeteners have been considered to the extent that they might be used instead of dietary sugars.

1.4 Meetings of the Panel

1.4.1 The Panel was appointed at the end of 1986 at the recommendation of the Committee on Medical Aspects of Food Policy. It met ten times.

1.4.2 We wish to express our appreciation and thanks for expert assistance to the Secretariat. We are grateful to those who have prepared working papers and have made documents available.

1.5 Perspectives

1.5.1 Sugars in berries, fruits and honey have always formed part of the human diet. Sugars became a regular and substantial contributor to food energy intake in broad sections of the UK population only in the middle of the last century when large quantities of sucrose extracted from sugar cane (and later from sugar beet) became available in a cheap and easily transportable form. Sugars are the carbohydrate chemical units which, combined in chains, form the complex carbohydrate molecules which function in plants as a major energy store or in providing a rigid fibrous structure. Starch, the chief storage carbohydrate, is almost entirely broken down in man by the process of digestion to single sugar (glucose) units in which form it is absorbed.

1.5.2 The carbohydrates in the rigid fibrous structure of plants, such as cellulose, largely resist digestion but may be fermented to absorbable fragments by bacteria in the intestine. This poorly absorbed material makes up a large part of so-called 'dietary fibre'.

1.5.3 By contrast with these starchy and structural carbohydrates, sugars are soluble in water and taste sweet. They occur naturally in many foods, including milk, honey, fruit, vegetables and sugar cane and beet. Previously so costly as to be a luxury available only to the wealthy, cane and beet sugar (sucrose) has become relatively inexpensive and has been incorporated into the diets of all sections of the community. Sucrose now represents about one seventh of the average daily food energy supply in the UK (see Table 3).

1.5.4 With the widespread and increasing use of sucrose came claims that its consumption was responsible for the increased prevalence of a number of diseases including dental caries,⁴ diabetes mellitus,⁵ cardiovascular disease,⁵ obesity⁵ and behavioural problems.⁶ These claims have attributed the diseases either directly to effects of sucrose on biological processes or indirectly to the loss of intact cell wall material ('dietary fibre') which occurs during the extraction, concentration and crystallisation process.

1.5.5 Most of these claims have been contested.^{7, 8} For both the general public and informed observers, it has been difficult at times to distinguish between established facts and strongly held opinions. COMA felt in 1986 that the role of sugars in the UK diet required further clarification.

1.6 Form of the Report

1.6.1 We have attempted in this Report to derive practical conclusions from our interpretation of many lines of evidence. We have also tried to identify

those areas where the evidence is inadequate or inconclusive and further research is needed. The data from which our conclusions and recommendations are drawn are summarised in sections 2-13 and the recommendations based upon them in Section 14. References are given in Section 15.

1.6.2 The recommendations were agreed by all members of the Panel.

2. The Nature of Sugars and Terminology of the Report

2.1 Sugars are soluble carbohydrates of fundamental importance in providing energy for the maintenance of life. Combined in chains they form an important food energy store (starch) and provide the structural framework (eg cellulose) of plant life. Plant life builds sugar units from carbon dioxide gas and water by the process of photosynthesis which uses light as the energy source. It is this incorporated energy which gives sugars their major nutrient property. The energy is liberated by regulated metabolic breakdown of sugars, either to meet current needs or to be stored, in animals after conversion to fat or glycogen (para 7.2.3). In this breakdown, sugars are ultimately converted back to carbon dioxide and water.

2.2 Glucose is the most abundant of the sugar units in nature. This single sugar unit is classed as a monosaccharide, as are fructose and galactose and many other less abundant sugars. Disaccharides such as sucrose, lactose and maltose are, as the name suggests, compounds of two monosaccharide moieties. Sucrose, the sugar most widely used as a food in the United Kingdom, is a disaccharide composed of glucose and fructose. Starches, the major carbohydrates in food, comprise a mixture of long straight chains (amylose) and branching chains (amylopectins) of glucose units and are formed by plant life as its energy store. Animals can construct long, branching glucose chains known as glycogen and this provides them with a readily available but relatively small and short-term energy store, the major energy store being fat. Starches in foods such as bread and potatoes as well as disaccharides are broken down by digestion in the intestine to, and absorbed as, their unit monosaccharides.

2.3 In this Report, the plural term 'sugars' has been used to include all the members of the chemical family of mono and disaccharides. When reference has been made to an individual sugar it has been named (eg sucrose, glucose, fructose). The most commonly occurring dietary sugars are listed in table 1 along with some of the foods in which they are found. Glucose is present in many foods, as the monosaccharide and as a component of disaccharides and starches. Lactose is present in milk and milk products, and sucrose, occurring naturally in sugar cane and beet and in fruit is added to many prepared and manufactured foods. Some sources of individual sugars in the diet in the UK are given in table 2.

2.4 Sucrose owes much of its sweetness to its fructose moiety, the sweetest-tasting of all the sugars. Other commercial sweeteners include

glucose syrups and fructose which is increasingly used by itself as a sweetening agent and is prepared commercially by conversion from glucose produced by hydrolysis of starch.²

2.5 There have been many attempts to group dietary sugars; biochemically (monosaccharides, disaccharides, hexoses, pentoses, etc); in manufacturing terms (added, natural); in physical terms (extracellular, intracellular); or in dietetic and physiological terms. The last provides a more useful framework for the consumer, but is necessarily more complex in order to avoid misleading groupings. For instance honey though 'natural' acts as any concentrated solution of the simple sugars glucose and fructose. Milk sugar (lactose) is 'natural' and extracellular but negligibly cariogenic. Sugars in fruit juices are also 'natural' and extracellular, but have different cariogenic potentials and are more rapidly absorbed than those in intact fruit. We have attempted to produce a logical and comprehensive classification without inconsistencies for this Report which future workers may find useful. One disadvantage is that previous dietary studies used a variety of different classifications which are not immediately comparable.

2.6 It is helpful to distinguish sugars naturally integrated into the cellular structure of a food (intrinsic) from those which are free in the food or added to it (extrinsic). This difference in physical location influences their availability for bacterial metabolism in the mouth and the readiness with which they are absorbed after ingestion. The speed with which sugars consumed in the diet become available to the tissues of the body influences some of the metabolic responses to their ingestion, a question considered later in this Report. These availability classes have been defined as follows.

2.6.1 *Intrinsic sugars* Sugars forming an integral part of certain unprocessed foodstuffs, ie enclosed in the cell, the most important being whole fruits and vegetables (containing mainly fructose, glucose and sucrose).

2.6.2 *Extrinsic sugars* Sugars not located within the cellular structure of a food.

Milk sugars Sugars occurring naturally in milk and milk products (almost entirely lactose) are extrinsic sugars but the Panel considered them a special case so that they form a separate sub-group.

Non-milk extrinsic sugars Includes fruit juices and honey and 'added sugars' which comprise recipe and table sugars.

(i) *Recipe sugars* Includes sugars that are added to composite dishes and confections. Recipes and formulations have a characteristic content of sugars, modification of which beyond certain limits results in a different product. Amounts of sugars in different recipes and products range from negligible (eg glazings) to providing 100 per cent of the food energy (eg 'boiled sweets'). Foods richest in recipe sugars include preserves, many

soft drinks, sugar confectionery, biscuits, cakes and puddings. Without recipe sugars some foods such as gooseberries, rhubarb and cooking apples would rarely be eaten and others such as meringues, and various confections and desserts would not exist.

- (ii) *Table sugars* Sugars, pure or reinforced with non-nutritive sweeteners, which the individual may choose to add to foods or drinks.

2.7 Nutrient density The relationship between the energy and the protein, vitamin and mineral contents of different foods, drinks and whole diets is of nutritional importance. The ratio of each nutrient to the energy content is termed the *nutrient density*. It is expressed as the quantity (usually in metric weight) of a specified nutrient per unit of food energy expressed as kilocalories (kcal) or megajoules (MJ) (1 megajoule (MJ)=239 kilocalories (kcal)). Since nutrient density is a ratio, it can provide information on absolute values of nutrient content only when total energy content is also known.

3. Technological Functions of Sugars in Foods

3.1 Sweetness of taste has given sugars their prominent place in human nutrition whether the sugars are intrinsic as, for example, in fruits and berries, or as recipe sugars added in domestic cooking or in commercial processing. In food manufacture the recipe sugars may be integral to the making of the product as in confectionery and meringues, or an essential component of the flavour of sauces or garnishings like tomato ketchup. Sugars, mainly sucrose, are added to jams partly because of their preservative properties. Sucrose is used as a bulking agent in cakes and is added to modify or enhance the flavour of foods and of drinks such as coffee and tea.

3.2 Baked products Sucrose has a major influence on the development of the characteristic structure and texture of cakes. It raises the temperature at which egg proteins denature and delays the gelation of starch, allowing the mixture to rise fully before it is solidified by heat. Glucose and fructose can be used as alternatives to sucrose, although greater quantities are needed to produce cakes of equivalent texture. Sucrose inhibits cake staling by resisting changes in moisture levels. Sugar is also important to the structure of biscuits. During cooking, the sugar dissolves, reducing the availability of water and so restricting the hydration of starch. On cooling it recrystallises and gives biscuits their characteristic crunchy texture.

3.3 Chocolate and sugar confectionery Chocolate is a suspension of sugar crystals (usually sucrose) and ground cocoa solids in cocoa butter. In chocolate the sweetness of sugar acts as a foil to the natural bitterness of the cocoa. Solid sugar ground so fine that the crystals are imperceptible to the tongue is responsible for the characteristic 'snap' of chocolate. In sugar confectionery, sucrose can remain in supersaturated solution with peripheral crystallisation, resulting in a 'sugar glass' characteristic of certain products.

3.4 Diabetic and dietetic confectionery A number of chocolate, cake, dessert and confectionery products have been manufactured using permitted bulk sweeteners (see section 13) instead of sucrose. These products are usually aimed at the diabetic market although some are used more widely, sometimes mistakenly, as slimming aids. Increasing numbers of confectionery products containing artificial sweeteners are likely to become available. Even without sucrose many of these products are relatively high in food energy. Consumed in excess some bulk sweeteners (eg sorbitol) have laxative properties.

3.5 Dairy products and frozen confectionery A principal use for sugars in dairy products is in ice-cream where they contribute to the familiar texture and lower the freezing-point. In soft-scoop ice-creams some of the sucrose is replaced by glucose and fructose syrups and glycerol.

3.6 Desserts Sugars provide sweetness and textural properties to products such as whipped desserts and custards. They have a preservative function in products like pie fillings through their ability to reduce available water and are widely used in compotes with acid-tasting fruits, berries and stems lacking natural sweetness.

3.7 Preserves The water sequestering property of sugars at high concentrations suppresses the growth of some micro-organisms in stored foods. About 70 per cent of the weight of jams usually consists of dissolved sugars, and they do not therefore require artificial preservatives.

3.8 Soft drinks In some soft drinks, carbonated, still, or squash-concentrate, sugars are used to provide sweetness and an acceptable feeling to the mouth. Low calorie 'diet' drink alternatives using intense artificial sweeteners are becoming increasingly widely consumed.

4. Sugars as an Energy Source

4.1 Sugars currently contribute between 10 and 20 per cent of the food energy to the human diet in the UK. The technical meaning of the term 'energy' should not be confused with its popular use indicating physical vigour and vitality. It is often supposed that tiredness and fatigue (ie lack of energy in its popular sense) are due to depletion of food energy in its technical sense. Lack of vitality is sometimes ascribed to abnormally low blood glucose concentration (hypoglycaemia) but this is rarely the case. The argument that consumption of sugars or other energy rich foods will raise the blood glucose concentration and promptly reinvigorate the fatigued individual is false. In healthy individuals the concentration of glucose in the blood is maintained within relatively narrow limits. The availability of energy to the body is also regulated at a fairly constant level and, except in situations of starvation, is virtually independent of food intake. In the normally nourished person, recovery from fatigue will occur during a period of physical rest and is neither accelerated nor delayed by food consumption. Some of the psychological aspects of fatigue may be relieved by the diversion of a refreshing drink or taking food but this effect is not related to its energy content.

4.2 Food energy may be derived from many sources; the human diet is very variable in respect of the relative proportions of its carbohydrate, fat, and to a lesser extent, protein and alcohol content, all of which do provide energy. With a few exceptions people in developing countries obtain the majority, in some cases over 80 per cent, of their food energy from carbohydrate sources. In industrial countries with higher incomes, such as the UK, fat has progressively displaced carbohydrate as an energy source.⁹ One of the principal recommendations of the COMA Panel on Diet and Cardiovascular Disease¹ was that this balance should be redressed by increasing the contribution of carbohydrate at the expense of fats, particularly those rich in saturated fatty acids. That Panel set the goal of reducing the contribution of fat to dietary energy in the UK to 35 per cent or less. Protein in foods, from animal and vegetable sources, contributes 10 to 15 per cent of daily energy consumption.¹⁰ In some people alcohol constitutes an appreciable proportion of energy intake but its usefulness as a food is limited because of its adverse effects on health.¹¹

4.3 Energy is released for biological use by the regulated oxidation of the fats, proteins, carbohydrates and any alcohol in the diet. The amount of energy provided by particular nutrients or by the diet as a whole can be determined directly by measuring it either as the heat released when it is burned in a calorimeter (making allowance for the combustible but non-utilised portion) or as the heat produced by the body following its

consumption. Alternatively the oxygen consumed by the body for its oxidation can provide an indirect measure of energy used (see Appendix D).

4.4 Cellulose and other structural plant materials yield energy when they are burned in a calorimeter, but their contribution to dietary energy is very small. Starches and sugars are almost completely digested and absorbed and the factor used for calculating their energy yields in mixed British diets is 3.75 kcal or 16 kJ per gram of monosaccharide equivalent (see Appendix D).¹² The corresponding factors for protein, fat and alcohol are 4 kcal (17 kJ), 9 kcal (37 kJ) and 7 kcal (29 kJ) per gram respectively.

5. Intakes of Sugars in the UK

5.1 The use of sugars by the public can be estimated at four different points in the food distribution chain. The applications and limitations of each of these estimates are described in the following sections.

- (a) *National food supply statistics* The Ministry of Agriculture, Fisheries and Food (MAFF) publishes estimates of the amount of each basic foodstuff entering the food distribution chain in the UK. This annual series, collected in a consistent manner for nearly 50 years, includes all foods produced in, and imported into, the United Kingdom and is the basis for international comparisons. Estimated deductions for exports and for non-human uses such as animal feed are made and the total further adjusted for changes in year-end stocks. The final totals divided by the mid-year population give an estimate of the supply, usually in kilograms per head per year. These estimates and their trends over the years for sucrose, glucose syrups and honey are shown in Table 3 and Figure 1. They include the sugar sold in packets, the sugars (apart from those naturally present in fruit and vegetables) later incorporated into cakes, biscuits, jam, ice-cream and other commercial foods and those which enter the catering sector. No allowance is made for wastage and the *per caput* estimates may thus be substantially greater than the amounts actually eaten.
- (b) *Food industry statistics* Data on the sales of certain manufactured foods, including sugar and chocolate confectionery, are provided by some segments of the food industry. As with the Government's food supply statistics, they can give no information on consumption by specific groups of individuals such as children, nor by people living in different parts of the country.
- (c) *Household purchases* MAFF's National Food Survey (NFS) has also collected information continuously for almost half a century on the amounts of all foods, apart from sweets and alcoholic drinks, brought into homes throughout Britain. A representative sample of some 7000 households each year records the quantities of all foods they purchase, including packet sugar and sugar-containing foods such as cakes, biscuits and fruits. The results are presented according to geographical region of residence, household composition, income group and other socio-economic descriptors but they do not show the distribution of food intakes among individual members of the household.
- (d) *Intakes by individuals* Some dietary surveys attempt to record all foods eaten inside and outside the home by relatively small groups of selected

individuals. Although not always representative and rarely repeated, such surveys should nevertheless generate the best estimates of the amounts of sugar and sugar-containing foods eaten by those individuals. In practice there are biases of unknown magnitude. Food items may be left unrecorded, snacks and second helpings rejected because of the bother of reporting them; the survey may itself alter consumption, for example, by reinforcing a determination to restrict food intake.^{13, 14}

5.2 Sugars supplies in the UK Figure 1 shows the daily *per caput* national estimates of sugars supplies in the UK from 1850 to 1987.¹⁵⁻¹⁸ Supplies rose steadily up to the beginning of the First World War and increased again until the Second World War. From the low point during the Second World War (85.5g per head per day in 1943), supplies of sucrose plus honey and glucose peaked in 1958, when they averaged 147.5g per head per day contributing an estimated 18.2 per cent of available food energy (Table 3). Between 1958 and 1974 the estimate fluctuated between 137.6 and 145.4g (from 17.2 to 18.8 per cent of estimated total food energy). Since 1974 supplies per head have fallen and in 1987 stood at approximately 120.2g per head (or 15.7 per cent of energy). Within this total, glucose and honey supplies rose steadily until 1978 and subsequently decreased on a *per caput* basis but at a slower rate than sucrose (Table 3).

5.3 Food industry statistics MAFF estimates that almost half the sucrose moving into human consumption is sold as packet sugar and about half is used by food manufacturers. The major industrial use of sucrose is for the production of confectionery which accounts for about a quarter of the total used in manufacturing, while the soft drinks industry uses about one fifth.¹⁹ Although the weight of confectionery sold has fluctuated, over the last 25 years it has increased by 16 per cent in the UK. The type of product has changed, with an increasing proportion of chocolate, rather than sugar, confectionery. At 34g per person per day, confectionery consumption in the UK in 1984 was the highest in the European Community.²⁰ Sales of soft drinks have increased five-fold in the UK from 52 to 259ml per person per day between 1950 and 1986.^{21, 22}

5.4 National Food Survey The pattern of household purchases of sugar recorded by the NFS¹⁰ is shown in Figure 2. Daily packet sugar purchases per head by households fell slowly from a peak of 75g per person in 1958 to 69g in 1970, then faster to 30g by 1987. This fall has been partly offset by an increase in soft drink consumption, estimated to have risen from 141 to 259ml per head per day between 1979 and 1986, corresponding to a rise from approximately 6 to 11g of sugar per day (MAFF unpublished 1988). People in income group A purchase less than half the sugar bought by those in income group D and by old age pensioners. Young housewives currently purchase less than half as much packet sugar as older housewives.¹⁰

5.5 Dietary survey data Some dietary survey information on consumption of sugars has been commissioned by Government, some collected for research and some for industrial purposes. The surveys differ in the type and

size of population groups selected, and in the methods of dietary assessment. Some of the detailed information, along with additional unpublished data made available to the Panel and covering the period from 1960, is summarised in Tables 4–7. These relate to pre-school, school, adult and elderly age groups respectively. Details of food composition data used for these analyses are given in Appendix D.

5.6 Non-milk extrinsic sugars account for about half the total sugars intake in young children (Table 4) and perhaps a higher proportion in some groups of older children, adults and the elderly (Tables 5, 6, 7 respectively). In children of pre-school age, total sugars intake was 25 to 29 per cent of total daily energy consumption (Table 4) and was higher than that for schoolchildren (19.0 to 25.0 per cent, Table 5). The proportion was, in general, lowest for adults (Table 6) but among these was relatively high in a Dublin population sample of unemployed people (25 and 28.2 per cent in men and women respectively).²³ Among adults there appeared to be a trend for women to take a smaller proportion of non-milk extrinsic sugars in their diets than men, but the differences (in those studies which distinguished between the sexes) were small and inconsistent.

5.7 The main food sources of the intrinsic and extrinsic sugars in the diets of Northumberland schoolchildren and Cambridge adults are listed in Table 8. Table sugar, confectionery and soft drinks together provided 69, 66 and 58 per cent of recorded non-milk extrinsic sugars intake in children, men and women respectively. Biscuits, cakes and puddings accounted for a further 21, 16 and 24 per cent.

5.8 The Panel examined in detail the data of Nelson *et al*^{24–27} derived from 7-day records of the dietary intake of 105 men and 112 women in Cambridge, England along with other available material. In particular they considered the quantities and proportions of sugars in these diets and whether these were associated with consumption of protein, minerals or vitamins so low as to constitute a threat to health.

5.8.1 The relationship between total energy intake and sugars and other nutrient consumption is shown in Table 9. Those people in the highest thirds of energy intakes consumed significantly more of all the nutrients, including sugars, than those in the lowest. Protein provided a somewhat smaller proportion of dietary energy to those in the highest total energy intake group ($p < 0.01$); their proportion from sugars and alcohol was not statistically significantly higher, with the proportion from fat staying relatively constant.

5.8.2 Intakes of nutrients by people grouped into thirds according to the proportion of their dietary energy derived from non-milk extrinsic sugars are shown in Table 10. Those in the highest third consumed an estimated 8 to 11 per cent more food energy overall than those in the lowest, but this difference could have arisen by chance. Since total energy content varied little between these thirds, the proportions of food energy derived from fat and protein necessarily fell, the former from 42.4 to 36.5 per cent for the men and 44.6 to

38.0 per cent for the women, the latter from 13.8 to 12.0 per cent for the men and 15.0 to 12.1 per cent for the women.

5.8.3 *Sugars intake and nutrient density* The Panel considered the possible effects of higher levels of sugars intake on the micronutrients in the diet. The micronutrients shown in Tables 8 and 9 were chosen because Recommended Daily Amounts (RDA) have been set for them in the UK.²⁸ Intakes of zinc and magnesium are also shown, even though no UK RDA is set for them, because there is current interest in these nutrients. Taken together these selected micronutrients give an indication of the nutritional quality of the diet. Intakes of these and all the other nutrients for which data are available are given in full in Appendix E for information. The level of total energy consumption had little influence on the micronutrient density (Table 9). As might be expected micronutrient density was lowest in those diets selected for the highest proportional contribution of non-milk extrinsic sugars to total energy intake (Table 10).

5.8.4 The relation between intake of sugars and the composition of the diet at various levels of total food energy intake is explored in Table 11. This compares nutrient intakes in those Cambridge men and women with consumption of sugars above the median values of intake ('higher sugars') with those at or below the median ('lower sugars') within each third of the distribution of total energy intake. The UK RDA²⁸ are also given along with the US RDA²⁹ for zinc and magnesium. In general the intakes of all nutrients ran parallel with total energy intake. There was a trend for micronutrient intakes to be lower in those with 'higher sugars' than those with 'lower sugars' consumption within each third of total energy intake. This trend was clearest at the lowest energy intakes. Using the RDA as a benchmark, mean micronutrient intake appears to be low with low energy intake in general, but especially in women, much of whose low total came from extrinsic sugars.

5.8.5 Tables 11 and 12 show a reciprocal relationship at any level of energy intake between the proportion of energy in the diet coming from fat and that from sugar.^{26, 30, 31} Restriction of dietary sugars would lead to a reduction in dietary energy unless it were accompanied by increases in consumption of other foods. In this situation the nature of these replacement foods will determine the change in the composition of the diet. People might choose complex carbohydrate or protein to meet this energy deficit, but a compensatory increase in dietary fat is an undesirable change¹ which could result from restricting sugars intakes. Tables 11 and 12 suggest that people who choose a lower sugar diet tend to choose one containing higher proportions of energy from fat and protein but similar intakes of starch.

5.9 In summary:

- (1) On average people with high total energy intake eat more of all nutrients including sugars. (See para 5.8.1).

- (2) At each level of total energy intake, sugars consumption shows a reciprocal relationship to the intakes of fats and protein^{26, 30, 31} (Tables 10 and 11).
- (3) Sugars intake is a weaker predictor of absolute micronutrient intakes than total energy consumption (Table 11 and para 5.8.4).
- (4) At any level of energy intake a higher sugars intake is associated with lower micronutrient intake (paras 5.8.3 and 5.8.4).

5.10 The conclusions from the foregoing study of a Cambridge population sample are in general supported by other data made available to the Panel. There were similar findings in a study of pregnant women in Hackney.^{32, 33} A similar analysis of dietary data collected from a sample of pregnant women in Leeds³⁴ (Table 12) also shows substantially lower micronutrient intakes in the lowest third of total energy intake with a trend to lower micronutrient intakes in those above median values for sugars intake in each of the thirds, most markedly in the lowest. Gibney and Lee (Table 13) report the daily food intake of unemployed families in Dublin.²³ There was relatively little difference in absolute values of micronutrient intake between the lowest and highest fourths of the distribution of table sugar consumption despite large differences in total energy intakes. The Body Mass Index (BMI) of the men with the highest reported table sugar intakes was substantially lower than those with the lowest.

5.11 The Panel concluded that those people who eat a lot of sugars also tend to have high energy and high fat intakes. At all levels of total food energy consumption the intakes of sugars and fat have a reciprocal relationship. Reduction in sugars intake might result in an increase in the amount and the proportion of dietary fats with a consequent possible increase in the risk of cardiovascular disease.¹ Sugars provide an acceptable means of contributing to the very high energy needs of some individuals. In people who eat only small amounts, dietary sugars may compete with other nutrients. For such people appropriate selection of food items is of special importance.

6. Sugars and Dental Caries

6.1 The problem of dental caries in the United Kingdom Dental caries is still very prevalent in the United Kingdom. In spite of increased use of fluorides, the decrease in caries prevalence over the last 15 to 20 years³⁵ appears to have slowed or halted in children.³⁶⁻³⁸ In 1983 by the age of 5 years, 49 per cent of children in the UK had one or more decayed teeth, rising to 93 per cent of 15 year old children, with an average of 5.9 decayed permanent teeth per child.³⁹ The prevalence and severity of dental caries varies with geographic and social factors. Caries prevalence is generally lower in London and South East England than in other regions of the UK^{39, 40} and higher in children from social classes IV and V than in those from social classes I and II.³⁹ Twenty-two per cent of adults of all ages have lost all their natural teeth⁴¹ with at least half of the tooth loss caused by dental caries.^{42, 43} More adults from social classes IV and V have lost teeth than adults from social classes I and II. Adults aged between 35 and 44 years with any remaining natural teeth have, on average, 19 of them decayed, missing or filled.⁴⁰ Adults suffer an average of three days dental pain per year.⁴⁴ In any one year a quarter of the adult population reports dental pain.⁴⁵ In 1987-88 the cost of the general dental services within the NHS in the UK was £934.6 million⁴⁶ the majority incurred in treating caries. This cost excludes hospital, community and armed forces dental services and private practice. Of the 410,000 general anaesthetics given within NHS general dental practice for dental extractions in the UK in 1987-88, 250,000 were given to children under the age of 15 years.⁴⁶

6.2 Aetiology of dental caries As soon as a tooth erupts into the mouth, a dense layer called dental plaque, consisting mainly of bacteria, forms on the tooth surface. In less accessible areas this plaque remains undisturbed. Many plaque organisms metabolise sugars, generating local concentrations of acid in the inner layers of plaque, favouring local dissolution of the mineral structure of the dental enamel. When plaque acidity* lessens (or pH rises) remineralisation of enamel can occur. At first a loss of mineral causes whitening of the enamel, which subsequently gives way to a carious cavity, a stage at which restoration of the tooth usually becomes necessary. Most dietary sugars are easily metabolised to acid by plaque organisms. Some forms of dietary starch can be slowly broken down in the mouth to sugars by salivary amylase. These sugars can then be metabolised by plaque organisms to acids. Sucrose favours the establishment in dental plaque of *Streptococcus mutans*, an organism considered by some to be particularly important in

*Acidity is directly related to the concentration of hydrogen ions $[H^+]$ and is inversely related to pH defined as $\frac{1}{\log [H^+]}$

caries aetiology.⁴⁷ In the majority of cases it takes several years for a carious lesion to become apparent, but a tooth can be destroyed by caries within months of eruption.

6.3 Tooth development Animal experiments have shown that high sucrose diets taken during tooth development result in enamel which is more susceptible to caries than normal enamel,⁴⁸ but this pre-eruptive effect is much less important than its effect on erupted teeth.⁴⁹

6.4 Evidence relating sugars to dental caries The evidence relating dietary sugars to dental caries comes from many types of investigation. Clinical trials provide the best evidence but they are difficult to conduct since long term studies are necessary. Other sources of evidence include: human epidemiological and observational studies, human intervention studies, animal experiments, *in vivo* measurements of plaque acidity, *in vivo* enamel slab experiments, and *in vitro* incubation experiments.

6.4.1 Human epidemiological and observational studies A large number of studies have shown positive correlations between the level of sugar consumption, and the prevalence and severity of dental caries. These associations have been observed in comparisons between countries, communities or individuals. A high correlation was found between sugar availability and caries experience of children in 47 countries,⁵⁰ and many populations have experienced a rise in caries occurrence after an increase in sugar consumption.⁵¹ People eating little sugars have low caries experience⁵² and there were reductions in caries experience coincidental with wartime restrictions which included dietary sugars.⁵³ Positive correlations have been observed both in cross-sectional and longitudinal studies between caries experience of individuals and their consumption of sucrose or confectionery.⁴⁹ In contrast, diets containing high levels of starch but low levels of sugars are associated with low caries experience. This has been observed both in populations^{51, 54-56} and in smaller groups of people.^{52, 57-59}

6.4.2 Human intervention studies The Vipeholm study⁶⁰ showed that eating sugary foods between meals was associated with markedly higher risk of dental caries than when sugar consumption was restricted to meal times. In the Turku study, substitution of dietary sugars by xylitol in free-living adults was followed by a greatly reduced caries occurrence.⁶¹⁻⁶² The intakes of dietary starch by the sucrose and the xylitol groups in the Turku study were similar and it can be concluded that sucrose but not starch was associated with high caries occurrence.

6.4.3 Animal experiments Important local (intra-oral) effects of sucrose have been demonstrated.⁶³ There is a positive correlation between caries severity in rats and the frequency with which they are fed a high sucrose diet, independently of the amount eaten.⁶⁴ Concentration of sucrose in food and weight of sucrose eaten are also positively correlated with caries development independently of eating frequency.⁶⁵⁻⁶⁷ When the frequency of feeding was standardised, cooked starch or starchy foods (eg bread) have been shown to

be less cariogenic than sucrose.⁶⁸ Caries development increased as the proportion of sucrose in sucrose/starch diets increased.⁶⁷ Baked starch/sucrose mixtures are very cariogenic.⁶⁹

6.4.4 Plaque acidity studies These experiments, which measure the acidity of dental plaque and not caries development, show that each time sugars are consumed there is a marked increase in plaque acidity.^{70, 71} Cooked starch or starchy foods were less acidogenic than sugars or sugary foods when using the sampling method of assessing plaque acidity⁷²⁻⁷⁴ but similarly acidogenic when the indwelling glass electrode method was used.^{70, 75}

6.4.5 Enamel slab experiments In these experiments, slabs of dental enamel are mounted in intra-oral appliances which remain in the mouth while dental plaque accumulates. The plaque-covered slabs demineralise when exposed to solutions of sugars or sugary foods.⁷⁶ Two studies have shown that cooked starch or starchy foods cause about a quarter of the demineralisation of enamel slabs that occurs with sugars or sugary foods.^{77, 78}

6.4.6 Incubation experiments Plaque organisms rapidly metabolise sugars to acids *in vitro*. Tooth enamel dissolves in solutions where the acidity rises above the critical level.⁷⁹ Unlike sugars, starch is not transported across the cell membrane of plaque micro-organisms⁸⁰ and must be split into sugars before it can be used by the cell. The rate at which this happens will depend on the salivary amylase levels and the nature of the starchy food.

6.5 Different dietary sugars Of the different dietary sugars, sucrose appears to be the most cariogenic. Glucose, fructose, and maltose may be only marginally less so.^{49, 62, 70, 76} Lactose and galactose are substantially less cariogenic than other sugars.^{49, 76}

6.6 Weight of sugars consumed, concentration of sugars and frequency of consumption Frequency of eating sugars and caries development are positively related.^{60, 64} In animals there is evidence that the mass of sugars consumed and their concentration in foods are also positively related to caries development, independently of eating frequency.⁶⁵⁻⁶⁷ In free-living people these three variables, mass, concentration and frequency, are all closely and positively related to each other. Caries experience is low where sugars supplies are on average less than 15-20 kg/person/year.⁵⁰ In 1987, sugars supplies in the UK were 44kg per person per year (Fig 1; Table 3) of which sucrose contributed 36kg.

6.7 Sugars naturally present in foods Although lactose alone is moderately cariogenic, milk also contains factors which protect against dental caries, so that milk without added sugars may be considered to be virtually non-cariogenic.⁸¹⁻⁸³ Fresh fruits, as eaten by humans, also appear to be of low cariogenicity.⁸⁴ There is no reason to suppose that natural syrups (eg honey, maple syrup, concentrated fruit juices) are not cariogenic.

6.8 Dietary starch From several different studies of the relative cariogenicity of dietary starch and sugars,⁸⁴ it appears that:

- (1) the cariogenicity of uncooked starch is very low;
- (2) finely ground and heat-treated starch can cause caries, but less than sugars;
- (3) cooked staple starchy foods such as rice, potatoes and bread are of low cariogenicity in humans;
- (4) the addition of sugars increases the cariogenicity of cooked starchy foods;
- (5) in their natural state, fibre and phosphates in starch-containing foods may help to protect against dental caries.

6.9 Non-sugar sweeteners The permitted intense sweeteners (saccharin, aspartame, acesulfame K and thaumatin; see Section 13) are non-cariogenic.⁸⁵ Information about cariogenicity of permitted bulk sweeteners is variable; xylitol and sorbitol have been subjected to human clinical trials and there are substantial data on hydrogenated glucose syrup and isomalt, but less on mannitol. Current evidence suggests that bulk sweeteners have negligible cariogenicity compared with sugars⁸⁵ and that substitution of sugars by alternative sweeteners could substantially reduce caries development.⁶¹ The greatest gain would be expected to occur if they were used to replace sugars in foods ingested frequently, such as sweet snacks, drinks, and liquid medicines.

6.10 Groups of the population at greatest risk of caries Dental caries can occur at any age. It is especially prevalent in pre-school children and has been associated with frequent and prolonged use of sweetened comforters⁸⁶ and sugared medicines.⁸⁷ The addition of sugars to feeding or comforter bottles containing fruit-flavoured drinks or milk is especially common in lower socio-economic groups and some ethnic minorities.^{36, 88} Caries increases in adolescence,³⁹ and the elderly are at risk because of diminishing salivary flow and exposure of roots of teeth due to gingival recession.⁸⁹

6.11 Sucrose is sometimes added to infant foods to prevent constipation in babies. Usually, all that is required to avoid constipation is to give more water. Rosehip syrups, blackcurrant syrups and various fruit juices have been widely promoted as vitamin supplements for babies but they may contain high levels of sugars. Sucrose-free vitamin drops are available at low cost at child health centres.

6.12 Methods for caries prevention Apart from reducing consumption of sugars the three other practical methods for caries prevention are plaque control, fluoride, and fissure sealants. Fluoride has been shown to be the most effective agent for caries prevention and is at its most efficient when incorporated in water supplies and toothpaste. Its use is considered the most

important reason for the decline in the prevalence of dental caries in Northern Europe, North America and Australia.^{90, 91} Plaque control (without the use of fluoride) is the least effective method.⁹² Fissure sealants are effective but demand skilled application, costly in manpower.⁹³ The Panel recognised that caries had declined in the UK from 'high' prior to the mid 1970s to 'moderate' in the 1980s,³⁵ but considerable dental morbidity remains (see 6.1); the decline appears to have slowed or halted.³⁶⁻³⁸ Even in areas of the UK where the drinking water has been fluoridated or has a natural fluoride content at the optimum level and with nearly all toothpastes containing fluoride, children with diets high in sugars still develop dental caries. In fluoridated north-east England in 1987, 50 per cent of 5 year olds had tooth decay, with 16 per cent of the children having five or more decayed teeth.³⁸ The protective effects of reduction of dietary sugars and the preventive influence of fluoride appear to be independent.⁹⁴⁻⁹⁶

6.13 The Panel concluded that the prevalence of dental caries in the UK is of social, medical and economic importance. The evidence relating dietary sugars to dental caries is very extensive and has been obtained from several types of investigation. Without sugars around plaque-covered tooth surfaces caries development is very limited. Caries risk can be reduced by non-dietary means, particularly the use of fluoride, but these methods can be expensive and are not completely effective. If the prevalence of dental caries in the UK is to be reduced further it will be necessary to reduce the amount and frequency of consumption of non-milk extrinsic sugars.

7. Metabolism of Sugars

7.1 *Metabolism* describes the many linked chemical changes which support the processes of life; it includes the transformation and breakdown of foods after their digestion and absorption from the intestine. Disturbance of any of the processes of metabolism may result in clinical disorders. In some cases this arises from the impact of a specific component of the diet upon the constitution of particular individuals.

7.2 Utilisation

7.2.1 *Digestion* The breakdown of dietary starches, higher sugars and disaccharides into monosaccharides in the intestine before absorption has already been described (paras 1.5.1 and 2.1). This breakdown is accelerated by enzymes secreted by the pancreas and cells lining the intestine. The sugar units so produced are then absorbed across the lining of the small intestine into the blood stream.

7.2.2 *Absorption* Sugars are carried across the intestinal wall by passive diffusion, by active transport or after breakdown to lactate within the cells. Sugars other than glucose and fructose are absorbed when their concentration in the intestine rises and they progressively diffuse into the fine blood vessels lying in the intestinal wall and so enter the circulation. Glucose and fructose are actively transported along with sodium ions into cells lining the intestinal wall. This energy-requiring process concentrates glucose and fructose in the intestinal cells to a level higher than that in the circulation, accelerating diffusion into the blood. The glucose and fructose absorbed may be metabolised to lactate in the intestinal wall and enter the blood stream in this form. Lactate is extracted and converted to glucose by the liver where it is stored as glycogen or released into the circulation for metabolism by the rest of the body.

7.2.3 *Metabolism of glucose* Glucose is distributed to the tissues where it fuels many chemical processes. Its energy is transferred in a finely regulated sequence of breakdown steps and it is ultimately converted back to carbon dioxide and water. Although fatty acids released from the depot fat can meet most of the body's energy needs especially under conditions of partial or complete starvation, the brain and nervous system have an obligatory requirement for glucose as an energy source. Maintenance of a constant blood glucose concentration in the face of variable or low dietary carbohydrate intake is achieved by continuous release of glucose from liver glycogen or by gluconeogenesis from amino acids, glycerol and lactate.

7.2.4 *Metabolism of fructose* Fructose is less rapidly absorbed from the intestine than glucose, but is readily converted to glucose by the liver which

may release it or store it as glycogen. Some fructose directly enters the pathway of sugar breakdown and like glucose its fragments may be converted to fatty acids and deposited in fat stores or broken down to carbon dioxide and water. Fructose may also have a role in the metabolism of trace nutrients; in particular copper status may be compromised by high fructose diets.⁹⁷

7.2.5 Regulation of glucose metabolism The concentration of glucose in the blood is normally held within remarkably stable limits, which provides an assured supply for brain function. This stability of concentration, maintained in the face of variable input from food and after prolonged fasting, is effected by the reciprocal interplay of hormonal and nervous stimuli. When circulating glucose falls too low, these stimuli trigger the release of glucose from liver glycogen, or, when it rises, promote its uptake by tissues and restrain its release from the liver by prolonging insulin secretion. If insufficient insulin is secreted or its action is impeded, unregulated release of glucose into the blood from the liver and reduced uptake of glucose from the blood by the body tissues leads to a rise in blood glucose concentration and the state of diabetes mellitus considered in section 9 of this Report. The state of hypoglycaemia, when glucose concentration falls too low, is considered in section 11. Reduced uptake of glucose by body tissues may lead to chronically raised compensatory levels of circulating insulin, a state of 'insulin resistance' which may be involved in disease pathogenesis (see para 7.3.1).

7.2.6 Lactose The principal sugar in human milk is lactose or milk sugar and is present at a concentration of 7g per 100ml in human milk.⁹⁸ It is also present in cow milk and most infant formulas. This sugar provides about 40 per cent of the infant's dietary energy and enhances the beneficial growth of lactobacilli in the gut. In adults lactose is a less important dietary sugar. Individuals who lack the intestinal enzyme lactase fail to digest it, leading to gastrointestinal disturbance (see para 12.5.1).

7.3 Metabolic responses to sugars intake

7.3.1 The ingestion of carbohydrates is followed by a sequence of hormonal responses and metabolic changes. Glucose and insulin levels in the blood are normally closely interrelated and regulated. A breakdown in this relationship is associated with clinical disorders such as diabetes mellitus and hypoglycaemia. Insulin has many other biological roles, including the metabolism of fatty acids, depot fats and proteins. Unduly high or low insulin levels may also be associated with the development of some pathological conditions.⁹⁹⁻¹⁰¹ There has been special interest in the effects of dietary sugars either directly, or indirectly through insulin responses, on the patterns of the fats (lipids) and on the concentrations of glucose and insulin in the blood because of their possible implications for arterial disease. Lipids (cholesterol and triglycerides) are carried in the circulation attached to specific proteins (the lipoproteins) and disordered patterns of the lipoproteins in blood have been linked to the development of arterial disease.¹

7.3.2 The effect of experimentally altering intakes of sugars on blood lipids has been much studied in animals. While of considerable scientific interest

these responses vary considerably among mammalian species and so cannot be extrapolated directly to man. The Panel considered that recommendations regarding sugars should be based on evidence derived from studies in humans.

7.3.3 Most of the studies in man have been performed on small numbers of participants and have been of short duration. The effects of altering dietary sugars depend not only upon the size of such alterations and the nature of rest of the diet, but also upon the subjects studied, their previous diet and their constitutional liability to exaggerated metabolic changes.

7.3.4 The relevance of the metabolic responses observed lies in their possible association with a number of common disease states. For example the higher the total blood cholesterol concentration, the greater is the risk of cardiovascular disease.¹ Dietary changes that raise the blood cholesterol level might thus be expected to increase the risk, while those that lower cholesterol levels to diminish it. Dietary changes can affect other constituents of the blood such as triglycerides and insulin and these may be implicated in cardiovascular and other diseases.

7.3.5 In a study in which sucrose was replaced isocalorically by complex carbohydrates for one week, blood cholesterol did not change when the replacement was based on bread and potatoes but fell when it consisted of leguminous carbohydrate.¹⁰² Similarly, in a two week study in which dietary sucrose was replaced by potato and rice there was no change in blood cholesterol.¹⁰³

7.3.6 A long-term study of the effect of replacing moderate levels of dietary sucrose with glucose for one year was carried out by Lock *et al.*¹⁰⁴ Inspection of their results suggests relatively little overall change in blood cholesterol although they claimed that it fell in the sub-group which at the end of the study was not found not to have gained weight. The two year Turku study in Finland¹⁰⁵ compared blood lipids, glucose and insulin in population groups consuming sucrose and fructose and found no differences.

7.3.7 When sucrose is eliminated from the diet, blood triglyceride concentration falls. Total cholesterol does not change appreciably¹⁰⁶⁻¹⁰⁸ but HDL cholesterol may rise.¹⁰⁸ It is difficult to dissociate these effects from that of the accompanying weight loss. In fact when sucrose replaces starch isocalorically fasting triglyceride concentrations usually do not change.^{103, 109}

7.3.8 When Crapo substituted fructose for sucrose in 11 normal people on a low fat test diet for 2 weeks, she found no systematic alteration in blood triglycerides, glucose or insulin although total blood cholesterol fell by 8 per cent.¹¹⁰ In another study in people with initially high blood triglyceride concentrations a further rise was recorded in subjects adding 80g of fructose per day to their normally sucrose-free diet, but this was not sustained at 28 days despite continuation of the diet.¹¹¹

7.3.9 The rise in blood glucose and insulin concentrations following intake of carbohydrate foods is related to the rapidity with which they are digested and absorbed. Thus, blood glucose and insulin responses to a food differ with the physical forms in which it is consumed (eg whole fruit versus fruit pulp versus fruit juice).^{112, 113} This blood glucose response to a given food, termed its glycaemic index, compares the excursion of blood glucose following different carbohydrate containing foods with a standard oral glucose load. This index is better established for individual foods than for mixed meals or diets. Rapid glucose absorption is associated with higher insulin levels and this may be relevant to the claims that hyperinsulinaemia is associated with increased risk of various diseases.^{101, 114-116}

7.3.10 Reiser has described a group of what he terms carbohydrate sensitive individuals who he estimates may constitute 10 to 15 per cent of the United States population. He defines them by their response of blood insulin to a sucrose load and they characteristically show exaggerated responses of blood cholesterol, triglyceride, glucose and insulin to sucrose intake than to equivalent amounts of starch.¹¹⁷⁻¹²⁰ These responses may be due, in part at least, to overweight.

7.3.11 In experiments on normal people whose daily sucrose intake exceeded more than 200g or constituted more than 30 per cent of dietary energy increases in blood insulin and lipids have been demonstrated.^{121, 122} Since intakes of this magnitude are rarely achieved, these findings are of doubtful significance in relation to those with normal dietary habits.

7.4 Thiamin and carbohydrate metabolism The metabolic pathways by which glucose is metabolised by the body tissues depend on a large number of enzymes, many of which require co-factors and elements derived from vitamins and minerals consumed in the diet. The metabolism of glucose specifically requires the vitamin thiamin. It has been suggested that the thiamin requirement is 0.3 mg per 1000 kcal (4.2 MJ) of carbohydrate energy consumed.¹²³ With a normal mixed diet, the intake of minerals and vitamins such as thiamin increases with increasing total food energy intake. However the UK dietary pattern at present provides sufficient thiamin to meet the widely varying quantities of carbohydrate contributing to energy intakes.¹⁰ Deficiencies are likely to arise only if dietary patterns are grossly disturbed as in excessive and continued alcohol consumption with restriction of normal food intake.

8. Sugars and Obesity

8.1 Obesity describes a state of excessive fatness resulting from periods when food energy consumption has exceeded energy expenditure. In this Report we have accepted the general, if arbitrary, definition of obesity as a Body Mass Index (BMI: weight (kg)/height(m)²) of 30 or more. This does not take the distribution of body fat into account. Obesity so defined is associated with an increased risk for a number of diseases (eg diabetes, hypertension, cardiovascular disease, gallstones). Obesity and its related diseases all become more prevalent with increasing age^{124, 125} (Table 14). This section explores the role of dietary sugars in the development and in the maintenance of obesity.

8.2 For the development of obesity the availability of food is a necessary precondition. Alone it cannot be sufficient as not all people living in apparently similar environments appear to be equally susceptible. Obesity tends to run in families.¹²⁵ Both genetic and environmental factors play a part in determining who will become obese.

8.3 It is very difficult to ascertain the long term dietary behaviour of individuals and we have only scanty information about the diets of people who later become obese compared with the diets of those who remain at normal weight. When people who are already obese are questioned they characteristically report lower total food energy intakes than lean people. However, this is unlikely to be representative of their habitual dietary behaviour because when estimates of energy expenditure have been made on obese people, it is greater than that of lean people.^{13, 126-129} Reports of lower energy intakes in obese than in lean people may be due to unrepresentative behaviour patterns during the period of measurement.¹³⁰⁻¹³³ They may be more likely to underrecord their intake than the lean, or they may be currently restricting food intake to reduce weight, their determination to do so perhaps being reinforced by the process of enquiry itself. In any case the food intakes and energy expenditures of people who have become obese may differ from those during the period of active weight gain.¹³⁴ It is even more difficult to relate the past intake of a specific food item such as sugar or fat to the development and maintenance of obesity.

8.4 Theoretically there are three ways in which sugars could contribute to obesity. Energy expenditure might be lowered by a high intake of sugars; the energy derived from intake of sugars might be specially directed to fat storage; or sugars could provoke overconsumption of energy containing foods. There is no evidence that sugars intake reduces energy expenditure. An effect by displacement of protein from the diet would be very small, of the order of one per cent or less of the total energy expenditure.¹³⁵ Nor is there

evidence that food energy derived from sugars is more or less likely to be stored as fat than energy from any other food source.¹¹⁷ There are however *a priori* reasons for supposing that dietary sugars might provoke an increased food energy intake. Some people find the sweetness of sugars attractive¹³⁶ and this could promote in them the consumption of certain foods. Sugars are added to foods and dissolved in drinks which might otherwise be consumed without them (eg breakfast cereals, tea and coffee).

8.5 People with a 'sweet tooth' who derive particular pleasure or satisfaction from sweet foods might be thought particularly likely to overconsume them and so become fat. However, there is no evidence from psychological studies that obese people have a greater preference for sugars or sweetness in their food than those of normal weight.^{136, 137}

8.6 As with total food energy intakes (see para 8.4) when samples of the population are asked about their consumption of sugars and their reported intake is related to their degree of adiposity, most studies report that the fatter the person, the less sugar they consume, an inverse relationship which is found even across the normal range of BMI.^{130, 132} Many factors may operate to distort this relationship. Fatter people may have altered their diets, and cut out sugars; there may be systematic reporting bias and there may be imperfect recall of past eating habits. Although such studies fail to support the hypothesis that obesity is due to high sugars intake for the above reasons they cannot be taken to refute it.

8.7 The most direct experimental investigations of the effect of sucrose on energy intake were those of Porikos.^{138, 139} In the first study sucrose was covertly replaced by aspartame in the diets of 8 obese subjects who normally obtained 25 per cent of their energy from added sucrose. In spite of access to unlimited amounts of palatable food, there was a 23 per cent fall in dietary energy consumption during the first 3 days of replacement, which reduced to 14 per cent in the next 3 days. The second study with 6 normal-weight male subjects, showed a similar pattern during 12 days of replacement of sucrose by aspartame. Of the scanty experimental data bearing on the effect of sucrose on energy intake these short-term studies are the best. Their relevance to people under normal conditions over longer periods remains to be demonstrated.

8.8 In studies in which a variety of dietary modifications included sugars restriction, several authors have noted incidental weight loss.^{106-108, 140} Weight loss achieved by restriction of sucrose intake is probably not different from that which would follow similar restriction of any energy providing food.¹⁴¹ Fat restriction may have a special role in the reduction of cardiovascular risk.¹

8.9 The only controlled trial of the effectiveness of aspartame in assisting obese patients to lose weight is that of Kanders *et al.*¹⁴² Half of a group of obese subjects who were recommended identical reducing diets were given aspartame sweetened foods and half were not. There was no significant

difference in weight loss after 12 weeks. This small trial suggests that aspartame is not particularly effective as an adjunct to weight loss in obese people who are already dieting.

8.10 The Panel acknowledged the absence of sound evidence for a specific role for any single dietary constituent as a general cause of obesity. When sugars contribute energy to the diet they would necessarily be part of any total excessive intake. Although the arguments reviewed above indicate that sugars may favour food energy intake, the available evidence is insufficient to establish a link between sugars intake and the development of obesity. More intensive research into the constitutional, behavioural, environmental and nutritional aspects of human obesity is necessary.

8.11 Restriction of sugars intake alone is unlikely to be adequate to correct obesity and people seeking to lose weight should adopt this as part of a more general dietary strategy.

8.12 The Panel concluded that in obese people and those becoming obese, restriction of sugars intake is a sensible contribution to calorie restriction.

9. Sugars and Diabetes Mellitus

9.1 The diabetic state is characterised by an abnormally high concentration of glucose in the blood (see para 7.2.5) and its loss in the urine, and is accompanied by many other disorders of blood constituents and bodily functions. It often leads to a characteristic pattern of damage to the kidney, the retina and lens of the eye, the peripheral nerves and the arteries—the so-called complications of diabetes. Diabetes is due to inadequate production or hindered action of the hormone insulin, which *inter alia* is centrally involved in regulating the storage, utilisation and production of glucose by the body. If insulin action is inadequate more glucose is produced by the liver and less taken up by the tissues for storage and metabolism.

9.2 There are two major clinical types of diabetes.¹⁴³ In the insulin-dependent form, usually starting in childhood or youth, insulin production by the body is grossly defective and insulin must be injected regularly to maintain life and health. The non-insulin dependent form, usually diagnosed later in life, is often associated with obesity. This form may respond to dietary treatment alone but may also require oral medication or sometimes insulin injections.

9.3 Although there are many other disturbances of the body's chemistry¹⁴⁴ the raised glucose is the most obvious and the most easily measured, in blood and in urine. Since excess circulating glucose is a cardinal feature of diabetes, excessive sugar in the diet might seem a plausible cause. The relationship of dietary sugars to the development of glucose intolerance and diabetes mellitus has been extensively reviewed. It has not been proposed that dietary sugars play a part in the pathogenesis of the insulin-dependent form of diabetes. The Panel therefore concentrated its attention on the non-insulin dependent form of the disorder.

9.4 The extensive literature on the evidence relating consumption of sugars to the development of diabetes was reviewed in detail by West¹⁴⁵ who considered both epidemiological and experimental data in man and the results of laboratory experiments in animals. The epidemiological evidence from studies relating sugar intake and diabetes prevalence in different populations and from studies over time in individual populations led him to conclude that 'in the aggregate, the evidence from these two approaches would appear to exclude sugar consumption as a dominant and universal factor'. This view was endorsed in the Report of the Food and Drug Administration's Sugars Task Force³—'epidemiological studies were unable to show a connection between high sugars consumption and diabetes'.

9.5 Experimental studies of the effect of dietary sugars on blood glucose concentration and insulin responses in man have, in the main, been

short-term and have administered sugars in very large quantities and/or in unusual food mixes or 'formula diets'. Results have been inconsistent and difficult to interpret but do not suggest that a diabetic state is provoked by such manoeuvres. In the longest term study reviewed, blood glucose concentrations were closely similar when 30 per cent of the food energy intake was supplied as sucrose or as starch, each for a six week period,¹¹⁸ though some elevation of the glycaemic response in selected individuals and higher insulin levels were found with sucrose in a more recent study.¹⁴⁶

9.6 Animals fed diets very high in sugars may show rises in blood glucose levels. However, the quantities of sugar fed are so high (70 per cent of dietary energy in the highly inbred selected sub-strain reported by Cohen,¹⁴⁷) or the interpretation complicated by accompanying obesity, that the application of these findings to disease in man is questionable.

9.7 The approach to diet in the patient with established diabetes has changed over the last decade. Carbohydrate is now proposed as the major source of food energy (50 to 55 per cent), which compensates for a decrease in fat intake to 35 per cent or less.¹ It is recommended that the additional carbohydrate should be largely unrefined, but compliance with such a diet may be improved by taking part of the carbohydrate as simple sugars. Intakes of sucrose up to about 50g a day taken with mixed meals as part of a diet high in complex carbohydrate and low in fat have no detrimental effect on diabetic control, at least in the short term.¹⁴⁸

9.8 There are a number of foods sweetened with fructose and sorbitol which are marketed for consumption by diabetic individuals. Such products are not now permitted to contain more food energy than their conventional equivalents and tend to contain marginally less. Consuming more than 30 or 40g of sorbitol per day may provoke troublesome osmotic diarrhoea.¹⁴⁹ Fructose fed in large amounts has also been shown to have similar effects to sucrose on the regulation of blood glucose in the diabetic. Allowing limited amounts of sucrose in the diabetic diet further reduces the already limited place of these expensive 'diabetic foods' in the diabetic regimen. The British Diabetic Association recommendations allow up to 25g of sucrose as part of a prudent diet.

9.9 Non-insulin dependent diabetes mellitus is associated with obesity, and the relationship between dietary sugars and obesity is discussed in Chapter 8. The Panel concluded that there was no evidence supporting a direct causal role, independent of this, for dietary sugars in the development of diabetes.

10. Sugars and Cardiovascular Disease

10.1 There have been suggestions that the intake of sucrose is causally related to development of atherosclerotic cardiovascular disease.⁵ Both a direct effect and an indirect link via obesity have been proposed. The relationship between sucrose intake and obesity has already been considered in chapter 8. Further consideration has been given to the possibility of a direct causal connection between sucrose intake and cardiovascular disease.

10.2 The strongest assertions that sucrose consumption was an important factor in the aetiology of coronary heart disease (CHD) were made by Yudkin.¹⁵⁰⁻¹⁵² His conclusions were based upon the arguments that

- (1) reported national mortality rates for CHD in different countries correlated with their estimated sucrose consumption;
- (2) the increasing mortality from CHD over the past 80 years or so has been associated with increasing consumption of sugar;
- (3) men who develop CHD report a higher consumption of sucrose than healthy controls; and
- (4) high sucrose consumption causes hyperlipidaemia and hyperinsulinaemia.

10.3 Many countries do not fit the pattern of high sugars consumption and high CHD mortality, particularly those in which high consumption of sugars is not associated with high fat intake. For instance countries like Cuba, Venezuela, Colombia, Costa Rica and Honduras have very high consumption of sucrose and low rates of CHD.^{153, 154} Sweden with a higher sucrose consumption *per caput* than Finland, has a lower age-specific mortality from CHD.^{155, 156} Further, a statistically significant relationship between CHD and sucrose consumption is not found when it is sought within countries.

10.4 There are anomalies in the comparison of national trends in sucrose consumption and CHD over the past half century. In the US, data from the Department of Agriculture show little change in average annual sucrose consumption. It was estimated as 51.7kg per person in the 1920's and 50.2kg in the 1960's, a period during which there was a marked rise in mortality from CHD.

10.5 Yudkin's claim that sucrose consumption was higher than normal in patients with cardiovascular disease was based on a study of 20 survivors of myocardial infarction and patients with peripheral vascular disease compared with a healthy control group.^{157, 158} Dietary data gathered by questionnaire reported mean sucrose intake in the patients closely resembling the average *per caput* consumption in the UK. Their apparent excess intake was attributable to the unusually low sucrose intake reported by the healthy controls.

10.6 The possibility that sugars may have effects on serum cholesterol and triglycerides, risk factors for cardiovascular disease, has been considered in section 8.

10.7 The general conclusion of most workers is that Yudkin's hypothesis is unsupported by subsequent clinical, epidemiological and experimental enquiries,^{157, 159} and that present evidence does not support any direct link between sugar consumption and the risk of CHD.

10.8 Similar conclusions have been reached by other expert committees who have also considered the evidence bearing upon this relationship. The joint FAO/WHO Report on carbohydrates in human nutrition¹⁶⁰ quotes the conclusions of Grande 'that a relationship between sucrose intake and the development of CHD is not supported by the available clinical and epidemiological data'.¹⁰² A report of the Finnish Nutrition Committee¹⁶¹ established to study the interrelationships of nutrition and public health concluded that 'no reliable evidence exists of the correlation between the carbohydrate content in the diet and coronary artery disease'. The Report of the COMA Panel on Diet and Cardiovascular Disease¹ could find no evidence incriminating sugar in CHD. The report of the US FDA's Sugars Task Force³ concludes that 'no evidence was found to support the contention that current dietary intake of sugars contributes to the development of hypertension'; and that 'there is no conclusive evidence that dietary sugars are an independent risk factor for coronary artery disease in the general population'. The report of the British Nutrition Foundation's task force on Sugars and Syrups² concluded that 'there is no evidence of a direct association between sugars consumption and these other diseases (ie heart disease and non-insulin dependent diabetes)'.

10.9 The COMA Report on Diet and Cardiovascular Disease¹ recommends that the consumption of saturated fatty acids and of fat in the United Kingdom should be decreased to 15 and 35 per cent of food energy respectively and the ratio of polyunsaturated to saturated fatty acids in the diet may be increased to 0.45. Reduced fat intakes should be compensated for by increasing consumption of fibre rich carbohydrates (bread, cereals, fruit, vegetables). Even allowing for the difficulties in interpreting epidemiological studies in which sugars intakes varied along with many other cultural, economic and physical characteristics of the respective populations and the shortcomings of the experimental studies, the Panel did not find sufficient

cause to make a specific dietary recommendation with respect to cardiovascular disease and simple sugars going beyond that in the 1984 COMA Report¹ that the consumption of simple sugars (sucrose, glucose and fructose) should not be increased further.

11. Behavioural Aspects of Consumption of Sugars

11.1 Sugars are popularly felt to have a calming or tranquillising effect; hot sweet drinks are often recommended to treat emotional or traumatic shock, but there is little evidence for any benefit. On the other hand it has also been claimed, mainly on the basis of anecdotal reports noted in reviews and commentaries on the subject,¹⁶² that the consumption of sucrose by certain people may be responsible for disordered and delinquent behaviour.^{6, 163}

11.2 One explanation offered for sugar-induced behavioural disturbance is that high sucrose meals overstimulate insulin production with a consequent depression of blood glucose to abnormally low levels (hypoglycaemia). It has been proposed that this may lead to brain glucose deprivation and to the release of hormones such as adrenalin to restore the blood glucose level, both of which might disturb normal thought processes or lead to altered behaviour.

11.3 In rare conditions in which blood glucose release from the liver is hindered, production of insulin may drive the blood glucose down to low levels (eg below 2.0 mmol per litre), but this process does not occur spontaneously in otherwise healthy individuals. Sugars may affect behaviour less directly by an insulin-mediated modification of the transport and metabolism of the amino acids tryptophan and tyrosine. These are the precursors of the monoamines (eg catecholamines, serotonin and dopamine) responsible for transmission of signals in the nervous system and which are thought to play an important role in the regulation of mood. In experimental animals administration of sucrose is reported to cause changes in brain neurotransmitter levels.¹⁶⁴ However no consistent effects on behaviour have been demonstrated. The data on which a link between sugars consumption and abnormal behaviour is proposed are open to criticism. For instance abnormal psychological processes of some individuals could well lead to peculiar eating patterns rather than *vice versa*. The Panel concluded that there are no adequately designed and controlled studies in humans which convincingly establish the existence of a causal link between consumption of sugars and abnormal behaviour.

11.4 It has been suggested that sucrose consumption may be important in producing a 'hyperkinetic syndrome', characterised by restlessness and impulsive, demonstrative behaviour patterns. Others have asserted that sucrose aggravates symptoms only in children who are already hyperkinetic.¹⁶⁵ The available evidence, mostly based upon uncontrolled unblinded experiments, did not convince the Panel of the validity of either of these claims.

11.5 Many of the reports of adverse reactions following consumption of meals high in carbohydrate and sugars describe symptoms characteristic of hypoglycaemia (ie sweating, shakiness and forceful beating of the heart). In some reports these symptoms are accompanied by, and in others consist entirely of, vague but disturbing psychological symptoms such as mood change, anxiety, depression, muddled thinking, and even aggressive or delinquent behaviour. The attribution of these symptoms to hypoglycaemia is often the result of diagnosis by unqualified individuals or by the affected persons themselves. It is rarely supported by the demonstration of low blood glucose concentrations, which, if measured at all, are usually within the normal range. There are, however, a number of extremely rare specific biochemical disorders which can result in hypoglycaemia, with symptoms which may include behavioural disturbance. In some cases, the symptoms are comparable to those induced by hunger and may represent normal arousal responses in hypersensitive people. Symptoms may be lessened by dietary change aimed to reduce carbohydrate induced oscillations in blood glucose concentration, though it is far from certain that such measures have specific effects.

11.6 The term Seasonal Affective Disorder (SAD) has been used to describe a condition in which recurring bouts of depression occur at a particular season of the year, typically late autumn and early winter.¹⁶⁶ During these depressive episodes sufferers are said to experience an increased desire for sweet, carbohydrate containing foods in many cases associated with substantial weight gain. It is claimed that 'carbohydrate craving' is due to a relative lack of brain 5-hydroxytryptamine, and that subjects seek carbohydrates to replenish this lack, thereby elevating their depressed mood.¹⁶⁷ The evidence for this is unconvincing, nor does it follow that sucrose has any consistent mood elevating activity.

11.7 **The 'Sweet Tooth'** Infants like sweet things, an attribute demonstrable even in new-born infants of 30–36 weeks gestation.¹⁶⁸ There is a wide variety of items of diet that most children with free access will seek out and demand. In explaining the development of food preferences and in the genesis of the 'sweet tooth', experimental studies with human infants and children suggest that children tend to prefer and accept familiar foods and flavours more readily than new ones¹⁶⁹ and there is some¹⁷⁰ but uncertain¹⁷¹ concordance between the likes and dislikes of particular foods within families.¹⁷² Traditionally confectionery and sweet food have been used for rewards and celebrations which may add psychological and social reinforcements to later choices of food for consumption.

11.8 The Panel therefore concluded that the evidence that the intake of sugars as a cause of emotional or behaviour disturbance, either by provoking hypoglycaemia or by any other mechanism, is inadequate to justify any general dietary recommendations on this account.

12. Sugars and Other Diseases

12.1 The role of sugars intakes in a number of other diseases has been considered but there is only limited published material available upon which to base judgements and conclusions.

12.2 Gallstones

12.2.1 The majority of gallstones are composed of cholesterol monohydrate which crystallises with calcium salts from bile when their concentrations are excessive and when proteins are present which promote nucleation. Sometimes impaired gallbladder contraction plays a part.

12.2.2 Accurate figures for prevalence of gallstones by ultrasonographic surveys are available only for a few communities in Italy and Denmark.¹⁷³⁻¹⁷⁶ Incidence data are not available. Autopsy and clinical data strongly suggest that gallstones predominantly affect people with western cultural patterns.

12.2.3 The relationship of obesity and raised plasma triglycerides, both risk factors for gallstones, to sugars intake has been considered earlier in this Report (paras 7.3.7, 8.10). A number of studies has examined the relationship between dietary sugars and gallstones. Some found a higher intake of sugars in gallstone patients^{177, 178} while others did not.¹⁷⁹⁻¹⁸¹

12.2.4 Sugar rich diets may increase cholesterol saturation of bile,^{140, 182} but reduction in the intake of 'added' sugars (with the addition of bran to the diet) does not prevent gallstones from recurring in overweight patients after the stones have been dissolved by medical treatment.¹⁸³

12.2.5 The Panel agreed that more data were required to explain the nature and extent of any relationship between sugars consumption and gallstones. Sugars may be associated with gallstones to the extent that they may contribute to high energy intakes, obesity and hypertriglyceridaemia.

12.3 Crohn's Disease

12.3.1 Crohn's disease is a chronic inflammatory condition of the intestine which chiefly affects the terminal ileum and colon and which often starts in young adults. Its cause is unknown but is probably related to the contents of the intestine; their diversion by surgical bypass usually improves the condition.

12.3.2 A number of studies have shown an association, perhaps restricted to non-smokers, between the occurrence of Crohn's disease and diets, one of the

features of which was higher non-milk extrinsic sugars content.¹⁸⁴ A high intake of sugars has been shown experimentally, in the short term, to increase intestinal permeability¹⁸⁵ a non-specific finding which is an early feature of Crohn's disease.¹⁸⁶ However, there is currently no evidence for a specific relationship between the disease and intake of sugars.

12.3.3 The Panel concluded that the association between dietary sugars and Crohn's disease deserved fuller study.

12.4 Sugars and cancer

12.4.1 Nutritional factors are recognised as contributing to cancer risk.¹⁸⁷ Obesity has been related to cancers of bowel, breast, uterus, kidney and prostate. Only in the first of these has a specific role for dietary sugars been proposed.

12.4.2 *Colorectal cancer* Populations with higher energy intakes often have higher prevalence of colorectal cancer.¹⁸⁸ In the UK,¹⁸⁹ USA,^{190, 191} and Australia¹⁹² but not in France^{193, 194} patients with large bowel cancer reported higher energy intakes than local control populations. Cancer of the colon is more common in overweight people.¹⁹⁵

12.4.3 The intakes of protein, carbohydrate and fat are so closely correlated with each other and with total food energy intake that their independent effects are difficult to assess.^{189, 190} In one study¹⁸⁹ 'sugars depleted in fibre' and combined with fat, contributed disproportionately to the higher energy intake of patients with colorectal cancer when compared with controls.

12.4.4 *Breast cancer* Breast cancer rates are higher in people consuming a 'western diet'¹⁸⁸ and in second generation immigrants reared on such a diet.¹⁹⁶ Breast cancer in older women in different countries correlates with the consumption of many nutrients, including sugary foods.¹⁹⁷ Obese women have an increased risk of breast cancer¹⁹⁶ associated with increased circulating levels of oestrogens.¹⁹⁸ There is no evidence of a specific role for sugars in the development of breast cancer.

12.4.5 The Panel concluded that there was little evidence of a direct contribution of dietary sugars to cancer risk. However, the link between some cancers and obesity and high energy intakes warrants further investigation of the role of individual dietary components, including sugars.

12.5 Sugars and Kidney Stones

12.5.1 Oral ingestion of glucose or sucrose loads may acutely increase calcium and oxalate excretion in the urine leading to the suggestion that the relatively high incidence of renal lithiasis in developed countries may be related to high intake of refined sugars.^{199, 200} The increase in the consumption of sucrose (as well as of fat, meat and milk) that occurred in Japan between 1950 and 1975 coincided with a sharp rise in incidence of urinary stones.²⁰¹ These effects may be secondary to obesity, itself associated with increased risk for urinary tract stones.^{202, 203}

12.5.2 The urinary calcium excretion rate in a group of men with renal lithiasis and their close relatives was significantly higher, on average, than that in 11 'normal' controls both before and after an oral glucose or sucrose load. There was, however, considerable overlap between the two groups.²⁰⁴ Fasting insulin levels may be higher²⁰⁵ and, after a standard oral carbohydrate challenge, the glucose falls lower²⁰⁶ in stone formers than in control subjects. These responses may be indirect because stone formers tend to be obese.^{202, 203}

12.5.3 Sucrose supplementation of the diet increases the urinary excretion of n-acetyl glucosaminidase (NAG), an enzyme produced by the renal tubules.¹²¹ Patients with renal stones excrete more NAG in the urine than normal controls before and after challenge with cola and confectionery.²⁰⁷ The significance of these urinary changes is not clear.

12.5.4 The Panel concluded that there was some epidemiological and experimental evidence linking urinary stone formation with nutritional factors including sugars intake. More investigation is required to confirm this relationship and the extent to which it is independent of accompanying obesity.

12.6 Inborn errors of metabolism

12.6.1 Deficient production or activity of one or more of the key enzymes catalysing metabolism may occur as the result of a genetic abnormality, inherited from one or both parents. Dietary sugars do not cause these conditions but the metabolism of specific sugars may be blocked leading to an accumulation of the sugar and its products preceding the block and a deficiency of those beyond it.

12.6.2 *Lactase deficiency* Lactose, the main sugar in milk, is broken down in the intestine by the enzyme lactase into glucose and galactose. As much as 75 per cent of the adult non-white population in Britain develop a degree of lactase deficiency.²⁰⁸ If large quantities of lactose escape breakdown and absorption in the small intestine, this may cause bowel disturbances.

12.6.3 *Sucrase-isomaltase deficiency* This very rare defect results in deficiency of the enzyme sucrase which catalyses the breakdown of sucrose to glucose and fructose. Sucrase deficiency may first become evident in babies who develop chronic diarrhoea and fail to thrive after weaning to a diet containing sucrose. Exclusion of sucrose from the diet relieves the symptoms.

12.6.4 *Fructosaemia* This rare condition occurs when the liver fails to produce the enzyme fructokinase. If the next enzyme in the metabolic chain, fructose-biphosphate aldolase, is deficient then the major consequence is abnormally low blood glucose levels (hypoglycaemia) after ingestion of fructose. This is due to accumulation of fructose-1-phosphate which inhibits the production of glucose both from smaller molecules (gluconeogenesis) and from the enzyme catalysing the breakdown of storage glycogen (glycogenolysis).

12.6.5 Galactosaemia This is also a rare condition in which the liver fails to produce the enzyme glucose-1-phosphate uridyl transferase which is essential for the metabolism of the galactose moiety of lactose. As a result galactose accumulates in the blood causing toxic symptoms which appear soon after birth. The infant's urine contains galactose. Without immediate dietetic treatment death occurs rapidly. The infant should be fed special infant formula in which lactose is replaced by dextrans, dextrose or maltose.

13. Artificial and Alternative Sweeteners

13.1 Restriction of sucrose consumption is recommended for the prevention of dental caries and in the clinical management of conditions such as obesity and diabetes mellitus (see chapters 6, 8 and 9). Much dietary sucrose is added to food and drink by the consumer, so that its omission is relatively simple. Adjustment to the loss of sweetness is much less easy and for this reason, alternative sweeteners have been developed. Chemically synthesized or extracted from natural sources, artificial sweeteners are substituted for sucrose in many manufactured foods.

13.2 Non-nutritive sugar substitutes are intensely sweet, having from 30 to 3000 times the sweetness of sucrose. Most are excreted unchanged in the urine. Even if they are metabolised wholly or in part they make an insignificant contribution to total food energy. Sucrose, however, may contribute substantially to the energy content of some foods and drinks. For instance one can of carbonated soft drink has an energy value of about 140 kcal (577 kJ), derived solely from its sugars content. Replacement of sucrose in such products with a low calorie alternative is often part of weight reduction regimens.

13.3 Most artificial sweeteners in common use have some disadvantage, in taste (or aftertaste) or in stability during food processing. Different sweeteners are sometimes blended so that the limitations of each are compensated for by others. Sweeteners of different classes used in combination may have a synergistic sweetening effect.

13.4 A major question for any new artificial sweetener is its long-term safety. The Acceptable Daily Intakes, used by national and international regulatory authorities, are levels of intakes of food additives considered to be safe for a lifetime of exposure. Advice on toxicity, based on a scientific review of the available toxicological data, is provided by the Joint FAO/WHO Expert Committee on Food Additives. Progress towards a single market within the European Community in 1992 will require the harmonization of the laws of its member states in relation to additives (including sweeteners) in foodstuffs. At present four intensely sweet substitutes for sucrose are permitted for use in the manufacture of foods and drinks in the UK. These are saccharin, acesulfame K, aspartame and thaumatin. Cyclamate, although used widely throughout Europe, is not permitted in the UK.

13.4.1 *Saccharin* has been used commercially to sweeten food and beverages since the beginning of the century. It is 200–500 times sweeter than

sucrose but, for some, has an unacceptable aftertaste. It is not metabolized and, being stable to heat, has a wide range of applications in food preparation and in beverages.

13.4.2 *Acesulfame K* is also about 200 times as sweet as sucrose, is not metabolized, and is very stable in many food preparations. When used alone a slight aftertaste is perceptible.

13.4.3 *Aspartame* has an acceptable taste, and acts as a flavour enhancer, particularly with citrus and other fruits. It is a compound of two amino acids, aspartic acid and phenylalanine. It contributes negligible energy to the diet and is 200 times sweeter than sucrose. It should not be used by people with the rare hereditary disorder phenylketonuria who need to restrict their intake of phenylalanine. Aspartame loses its sweet taste on heating, and so is used primarily in beverages and in foods that require little or no cooking.

13.4.4 *Thaumatococcus*, the most potent intense sweetener (2000–3000 times sweeter than sucrose), is a protein extracted from the West African fruit *Kateme* (*Thaumatococcus daniellii*). The perception of sweetness is not immediate and is long lasting, leaving a liquorice-like aftertaste. It is unstable to heat, and is used in combination with other sweeteners in a variety of foods and drinks.

13.4.5 *Cyclamate* is the least potent of the synthetic sweeteners (30 times the sweetness of sucrose). Metabolized to a limited extent in the intestine (to cyclohexylamine), after absorption it is excreted unchanged in the urine. It is heat stable and so has many applications in food and drink production, often in combination with saccharin. Although banned in the USA in 1969 and later in the UK on the basis of limited evidence, it is in use in many European countries following the publication of more extensive experimental data. The use of cyclamate in the UK is currently under review by the Ministry of Agriculture, Fisheries and Food.

13.5 In addition to these non-nutritive sweeteners, a number of 'bulk' sweeteners are approved for use in the UK in foods often formulated for people with diabetes. These are modified sugars or alternative sugars eg sorbitol, isomalt, mannitol, xylitol, hydrogenated glucose syrups. All have about the same energy value as sucrose weight for weight, but insulin is not directly required for their metabolism. Sixteen per cent of the chewing gum sold is now 'sugar-free' containing sorbitol, mannitol and xylitol and approximately 30 per cent of syrup medicines sold in the UK are also 'sugar-free' containing sorbitol, saccharin and hydrogenated glucose syrup.

14. Conclusions and Recommendations

14.1 The Panel's remit was to review the evidence relating sugars in the diet to health.

14.2 The Panel found no evidence that the consumption of most sugars naturally incorporated in the cellular structure of foods (intrinsic sugars) represented a threat to health. Consideration was therefore mainly directed towards the dietary use of sugars not so incorporated (extrinsic sugars). Non-milk extrinsic sugars, principally sucrose, at present constitute about 15 to 20 per cent of the average daily food energy supply in the UK.

14.3 Dental caries

14.3.1 Dental caries remains prevalent in the UK. It is of social, medical and economic importance. Extensive evidence suggests that sugars are the most important dietary factor in the cause of dental caries. Their presence at plaque-covered tooth surfaces is essential for more than very limited caries development. Caries experience is positively related to the amount of non-milk extrinsic sugars in the diet and the frequency of their consumption. Staple starchy foods, intrinsic sugars in whole fruit and milk sugars are negligible causes of dental caries. Non-sugar bulk and intense sweeteners are non-cariogenic or virtually so. A reduction in the consumption of non-milk extrinsic sugars would be expected further to reduce the prevalence of dental caries in the UK.

14.3.2 Dental caries can occur at any age but those at greatest risk are children, adolescents and the elderly. Caries risk can be reduced by non-dietary means, particularly the use of fluoride, but these methods offer incomplete protection and some are expensive.

14.3.3 In order to reduce the risk of dental caries, the Panel *recommends* that consumption of non-milk extrinsic sugars by the population should be decreased. These sugars should be replaced by fresh fruit, vegetables and starchy foods.

14.3.4 Those providing food for families and communities should seek to reduce the frequency with which sugary snacks are consumed.

14.3.5 For infants and young children simple sugars (eg sucrose, glucose, fructose) should not be added to bottle feeds; sugared drinks should not be given in feeders where they may be in contact with the teeth for prolonged

periods; dummies or comforters should not be dipped in sugars or sugary drinks.

14.3.6 Older children need to be aware of the importance of diet and nutrition in relation to dental as well as general health. The Panel *recommends* that schools should promote healthy eating patterns both by nutrition education and by providing and encouraging nutritionally sound food choices.

14.3.7 Elderly people with teeth should restrict the amount and frequency of consumption of non-milk extrinsic sugars because their teeth are more likely to decay due to exposure of tooth roots and declining salivary flow.

14.3.8 An increasing number of liquid medicines are available in 'sugar free' formulations. When medicines are needed, particularly long-term, such alternatives should be selected by parents and medical practitioners. The Panel *recommends* that Government should seek the means to reduce the use of sugared liquid medicines.

14.3.9 Dental practitioners should give dietary advice, including reduction of non-milk extrinsic sugars consumption, as an important part of their health education to patients, particularly to those who are especially prone to dental caries. To facilitate this, the Panel *recommends* that teaching of nutrition during dental training should be increased, and professional relations between dietitians and dental practitioners be encouraged.

14.4 Obesity

14.4.1 Dietary sugars may contribute to the general excess food energy consumption responsible for the development of obesity. This condition plays an important part in the aetiology of a number of diseases, eg diabetes, raised blood pressure, hyperlipidaemia and arterial disease and gallstones (see Para 8.9).

14.4.2 Omission of sugars from the diet, though safe, is not usually sufficient as a weight reducing regimen. Restriction of fats in the diet is also important in reducing obesity and has other health advantages.¹ The Panel endorses the need for the obese to reduce energy consumption and *recommends* that the reduction of non-milk extrinsic sugars intake should be part of a general reduction in dietary energy intake. Overweight people who wish to lose weight and those becoming obese should follow the same approach to dietary energy restriction as that recommended for obese people. The Panel *recommends* intensification of research into the determinants of food choices and the mechanisms of the variety of individual responses to food intakes.

14.5 Metabolism

14.5.1 There is no evidence for a direct adverse effect in most people on blood levels of cholesterol, triglycerides, glucose or insulin when sucrose is

substituted isocalorically for starch up to about 150g per day or 25 per cent of total food energy. A subgroup, possibly genetically determined and often overweight, comprising 10 to 15 per cent of the population, may respond with hyperlipidaemia to high normal intakes of sugars. At high levels of sucrose intake (about 200g per day or 30 per cent of food energy) substituted isocalorically for starch, undesirable elevations may occur in fasting plasma lipids, insulin and glucose.

14.5.2 A relatively small group of people with metabolic disorders may need to restrict or regulate their intake of particular sugars. This includes people with diabetes and those with certain rare inherited disorders.

14.5.3 For the majority of the population, who have normal plasma lipids and normal glucose tolerance, the consumption of sugars within the present range in the UK carries no special metabolic risks. Those members of the population consuming more than about 200g per day should replace the excess with starch. The Panel *recommends* that those with special medical problems such as diabetes and hypertriglyceridaemia should restrict non-milk extrinsic sugar intake to less than about 25 to 50g per day unless otherwise instructed by their own physician or dietitian.

14.6 Links between sucrose intake and certain other diseases (eg colo-rectal cancer, renal and biliary calculi, Crohn's disease) have been proposed. Although the Panel did not feel the evidence was adequate to justify any general dietary recommendations, it *recommends* the intensification of research in these areas. The Panel *recommends* that human nutrition should form an integral part of the training of medical students and of other health professionals.

14.7 The Panel concluded that current consumption of sugars, particularly sucrose, played no direct causal role in the development of cardiovascular (atherosclerotic coronary, peripheral or cerebral vascular) disease, of essential hypertension, or of diabetes mellitus (either insulin-dependent or non-insulin dependent). It further concluded that sucrose had no significant specific effects on behaviour or psychological function.

14.8 Artificial and alternative sweeteners can be considered non-cariogenic or virtually so and are useful sugar substitutes within limits prescribed by other Expert Advisory Committees. The Panel *recommends* that food manufacturers produce 'low sugars' or 'sugars-free' alternatives to existing sugar-rich products, particularly those for children.

14.9 Those wishing to regulate their sugars consumption need information of the sugars content of foods. The Panel *recommends* that manufacturers adopt current proposals for labelling foods including their total sugars content. The Panel further *recommends* that the Government seek the means for analysis and labelling of non-milk extrinsic sugars if practicable.

14.10 The Panel *recommends* that the Government should monitor average and extreme intakes of non-milk extrinsic sugars by members of the population in relation to the general and dental health of the public.

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Table 1 *Mono and disaccharides in foods*

Sucrose

- The following foods normally contain more than 10g sucrose per 100g: drinking chocolate powder, chocolate, condensed milk, sweetened fruit drinks, cakes, biscuits, sugar coated breakfast cereals, sugar confectionery, sweetened alcoholic beverages, preserves, many puddings, ice-cream and ripe bananas.
- Small amounts are added to sauces, many yogurts and other milk desserts, fruit products, processed vegetables and soups.
- Small amounts also occur naturally in cereal grains and flours, fruit and vegetables.

Glucose

- Present in honey, sugar confectionery, preserves, some alcoholic beverages, breakfast cereals, some cakes, sweet biscuits, soft drinks (except low calorie soft drinks), fruit and fruit juices, sauces, soups, sweetened yogurts, ice-cream and vegetables.
- Trace amounts are present in cereal grains and flours.

Fructose

- Mainly present in honey, and in fruit and products containing fruit eg fruit cakes, mueslis, fruit juices, yogurts.
- Small amounts are also present in some alcoholic beverages, soft drinks (not low calorie soft drinks) most vegetables and soups.
- Fructose is added as a sweetener to some diabetic products.

Lactose

- Mainly present in milk (including human milk) and milk products, and also occurs in processed items in which milk, cream, skimmed milk powder or whey is an ingredient eg various mueslis, milk chocolate, instant potato, cream cakes and buns, some puddings, pastries, biscuits and creamed soups.

Maltose

- Present in foods made with glucose syrups eg sugar confectionery items and some infant formulas.
- Small amounts are also present in some biscuits, breakfast cereals.

Foods may contain a number of other sugars including arabinose and xylose (eg in white wine, beer), galactose (fermented milk products), mannose

Table 1 (continued)

(fruit), fucose (human milk and bran), rhamnose (wheat bran), raffinose (some vegetables), stachyose (beans other than green beans) and verbascose (oat bran, and legumes). Rarer sugars may also be found in specific foods eg mannoheptulose in avocado pear, mycose in mushrooms and melezitose and erlose in honey. Maltotriose and other higher polymers of glucose are also found in foods made with glucose syrups (maltodextrins) including sugar confectionery and some infant formulas.

Table 2 Contribution of food groups to intakes of individual sugars* from the National Food Survey 1987¹⁰ (g/person/day)

	Glucose	Fructose	Sucrose	Maltose	Lactose	Other
Liquid whole milk	0	0	0	0	11.0	0
Other milk & cream	0.2	0.1	0.8	tr	4.5	0.2
Cheese	0	0	0	0	tr	0
Carcass meat	0	0	0	0	tr	0
All other meat & meat products	0.1	tr	0.1	0.2	tr	tr
Fish & fish products	0	tr	tr	tr	0.1	0
Eggs	0	0	0	0	0	0
Fats	tr	0	0.1	tr	0.2	0.2
Sugar & preserves	2.1	1.4	33.1	0.5	0	0
Potatoes	0.4	0.3	0.6	0	0	0
Fresh green vegetables	0.3	0.3	0.2	0	0	0
Other fresh vegetables	0.8	0.7	0.9	0	0	0.2
Processed vegetables	0.3	0.2	1.7	0	tr	tr
Fresh fruit	1.6	2.8	2.4	0	0	0
Other fruit & fruit products	2.7	2.7	2.8	0.2	0	0
Bread	2.4	0.1	0.6	0.1	tr	0
Flour	0.1	0.1	0.1	tr	0	0
Cakes and biscuits	0.7	0.5	8.2	0.2	0.3	0
Other cereals	0.5	0.4	2.6	0.4	0.3	0
Beverages	tr	0	0.5	0.2	tr	0.1
Miscellaneous	1.0	0.8	2.5	0.1	0.4	tr
TOTAL, ALL FOODS	13.2	10.4	57.0	1.9	16.9	0.8

* Excludes confectionery, alcoholic and soft drinks.

Table 3 Sucrose, honey and glucose syrups and lactose available for human consumption in the UK¹⁵⁻¹⁸

Year	Sucrose g/day A	Honey plus glucose** g/day B	Lactose g/day A+B	Energy kcal/day	Energy from sucrose as per cent of total energy from food*	Energy from sucrose, honey & glucose as per cent of total
Pre-War	120	6.6	126.6	3050	15.5	16.3
1940	89.4	6.6	96.0	2890	12.2	13.0
1941	81.9	3.7	85.6	2900	11.1	11.6
1942	82.8	3.1	85.9	2930	11.2	11.6
1943	82.6	2.9	85.5	2920	11.1	11.5
1944	87.7	3.2	90.9	3060	11.3	11.7
1945	83.1	3.4	86.5	3010	10.9	11.3
1946	95.1	3.4	98.5	2940	12.7	13.2
1947	100.3	3.9	104.2	2940	13.4	13.9
1948	102.2	3.8	106.0	3000	13.4	13.9
1949	112.6	5.1	117.7	3120	14.2	14.8
1950	103.5	4.5	108.0	3120	13.1	13.6
1951	114.7	5.0	119.7	3080	14.7	15.3
1952	108.7	4.8	113.5	3030	14.1	14.7
1953	119.3	7.1	126.4	3100	15.1	16.0
1954	128.1	7.1	135.2	3190	15.8	16.6
1955	131.4	7.6	139.0	3170	16.3	17.2
1956	132.5	7.4	139.9	3170	16.5	17.3
1957	135.2	7.8	143.0	3180	16.7	17.7
1958	139.7	7.8	147.5	3180	17.3	18.2
1959	134.3	7.6	141.9	3130	16.9	17.8
1960	134.6	8.1	142.7	3140	16.9	17.8
1961	137.1	8.3	145.4	3150	17.1	18.1
1962	134.0	8.3	142.3	3170	16.6	17.6
1963	134.6	8.9	143.5	3180	16.7	17.7
1964	129.3	9.2	138.5	3160	16.1	17.2

Table 3 (Continued)

Year	Sucrose g/day A	Honey plus glucose** g/day B	Lactose g/day A+B	Lactose g/day	Energy kcal/day	Energy from sucrose as per cent of total energy from food*	Energy from sucrose, honey & glucose as per cent of total
1965	130.1	10.3	140.4		3140	16.3	17.5
1966	131.5	10.6	142.1		3160	16.4	17.6
1967	128.7	11.1	139.8		3080	16.5	17.8
1968	126.7	11.8	138.5		3090	16.1	17.5
1969	128.1	12.8	140.9		3110	16.2	17.8
1970	126.8	13.2	140.0		3110	16.1	17.6
1971	124.1	13.5	137.6		3080	15.9	17.5
1972	125.4	14.2	139.6		3060	16.1	17.9
1973	123.3	15.6	138.9	22	3040	16.0	17.9
1974	125.5	16.7	142.2	22	2960	16.7	18.8
1975	98.6	19.2	117.8	21	2917	13.3	15.8
1976	112.3	20.8	133.1	23	3003	14.7	17.3
1977	112.1	19.5	131.6	23	3061	14.4	16.8
1978	111.5	21.4	132.9	23	2988	14.7	17.4
1979	108.5	20.0	128.5	23	2980	14.3	16.9
1980	101.1	17.5	118.6	22.1	2851	14.0	16.3
1981	100.5	16.2	116.7	22.9	2863	13.8	15.9
1982	106.8	16.2	123.0	22.5	2883	14.6	16.7
1983	102.5	16.2	118.7	23.2	2872	14.1	16.2
1984	103.6	17.2	120.8	24.5	2891	14.1	16.3
1985	101.6	16.7	118.3	24.4	2919	13.7	15.9
1986	101.9	17.3	119.2	21.3	2970	13.5	15.7
1987	103.8	16.4	120.2	22.7	2992	13.7	15.7

* Excludes energy from alcoholic drinks.

** Excluding glucose and other sugars in fruit and vegetables.

Table 4 Sugars intake by pre-school children Mean (s.d.)

Author, Location, date	Ages	Sample number gender	Intake per day				Percent energy		
			Energy MJ	Energy kcal	Total sugars g	Non-milk extrinsic sugars g	Total sugars %	Non-milk extrinsic sugars %	Non-milk extrinsic sugars %
Ministry of Health, ²⁰⁹ England, 1963	1-2y	88 M&F	4.69 (1.18)	1117 (281)	—	43 (18.8)	—	—	14.4
	2-3y	92 M&F	5.67 (1.53)	1349 (364)	—	57 (28.8)	—	—	15.8
	3-4y	84 M&F	5.63 (1.08)	1341 (256)	—	60 (18.3)	—	—	16.8
	4-5y	79 M&F	6.49 (1.45)	1545 (346)	—	68 (24.7)	—	—	16.5
	6-18m	201 M&F	4.41 (1.06)	1050 (252)	—	48.0 (22.0)	—	—	18.3
DHSS, ²¹⁰ England, Wales & Scotland, 1967/68	1.5-2.5y	394 M&F	5.30 (1.34)	1262 (320)	—	56.8 (23.0)	—	—	18.0
	2.5-3.5y	407 M&F	5.88 (1.51)	1401 (360)	—	64.7 (24.6)	—	—	18.5
	3.5-4.5y	319 M&F	6.16 (1.59)	1468 (378)	—	69.4 (23.6)	—	—	18.9
	15m	36 M&F	3.93 (0.92)	936 (219)	64 (25)	31 (20)	25.7 (8.8)	—	12.4 (8.1)
Paul <i>et al</i> ^{211,212} and unpublished Cambridge, 1978-82 (Longitudinal study)	18m	34 M&F	3.99 (1.07)	948 (255)	72 (25)	37 (23)	28.5 (7.6)	—	14.4 (8.3)
	24m	30 M&F	4.35 (0.75)	1034 (180)	79 (19)	44 (19)	29.0 (7.5)	—	16.4 (7.9)
	36m	39 M&F	4.87 (0.65)	1159 (154)	86 (24)	50 (24)	27.9 (7.3)	—	16.1 (7.8)
	1-4y	35 M	6.19 (1.47)	1474 (350)	102 (34)	—	26.0	—	—
Nelson, ^{25,213} Cambridge, 1977-79		34 F	5.70 (1.35)	1357 (321)	101 (31)	—	27.8	—	—

Table 5 Sugars intake by schoolchildren Mean (s.d.)

Author, Location, date	Ages	Sample number gender	Intake per day			Percent energy		
			MJ	Energy kcal	Total sugars g	Non-milk extrinsic sugars g	Total sugars %	Non-milk extrinsic sugars %
Cook <i>et al</i> ²¹⁴ Kent, 1968/70	8-15y	355 M	10.68	2543	—	113.2	—	16.7
		341 F	8.50	2024	—	90.4	—	16.7
	8-11y 12-15y	352 M+F	8.99	2141	—	97.4	—	17.1
		344 M+F	10.25	2441	—	107.8	—	16.4
Darke and Disselduff, ²¹⁵ Bristol, Croydon & Sheffield, 1971	10-11y	163 M	9.08 (1.63)	2169 (390)	—	95	—	16.4
		158 F	8.02 (1.56)	1916 (372)	—	85	—	16.7
Nelson ^{25,213} Cambridge 1977/79	5-10y	56 M	7.86 (1.77)	1871 (420)	118 (38)	—	23.7	—
	11-17y	45 M	10.33 (2.19)	2460 (521)	139 (43)	—	21.2	—
	5-10y	53 F	7.14 (1.52)	1700 (362)	112 (35)	—	24.7	—
	11-17y	35 F	8.71 (1.89)	2074 (450)	118 (39)	—	21.3	—
Hackett <i>et al</i> ²¹⁶ Rugg Gunn <i>et al</i> ²¹⁷ Northumberland 1979/81	11.5- 13.5y	184 M 191 F	9.4 (1.4) 8.5 (1.4)	2238 (333) 2023 (333)	123 (27) 114 (30)	84.3 (21.3) 77.6 (22.6)	20.9 21.5	14.3 14.6
Nelson <i>et al</i> ^{218,219} Southampton 1982	13-15y	63 M	10.29 (2.33)	2450 (555)	133 (43)	—	20.4	—
	13-15y	34 F	8.03 (1.94)	1912 (462)	97 (38)	—	19.0	—

Table 6 Sugars intake by adults Mean (s.d.)

Author, Location, date	Ages	Sample number gender	Intake per day			Percent energy		
			Energy MJ	Energy kcal	Total sugars g	Non-milk extrinsic sugars g	Total sugars %	Non-milk extrinsic sugars %
COMA, ²²⁰ Throughout UK 1967-68	Pregnant 2nd trimester	435 F	9.04	2152		70		12.2
Bingham <i>et al</i> ²²¹ and unpublished	20-39y	13 M	10.23 (1.80)	2435 (428)	122 (46)	83.8 (38.8)	18.9 (5.7)	12.8 (5.3)
Cambridgeshire 1977	40-59y	12 M	9.86 (3.03)	2348 (721)	129 (63)	87.2 (46.5)	21.2 (7.5)	14.1 (7.0)
	20-39y	10 F	9.17 (1.90)	2183 (452)	118 (37)	72.1 (29.4)	21.5 (6.0)	12.8 (6.0)
	40-59y	14 F	8.34 (1.84)	1986 (438)	108 (40)	60.4 (33.9)	20.4 (5.0)	10.7 (5.3)
Smithells <i>et al</i> ³⁴ and unpublished	Pregnant 1st trimester	195 F	7.83 (1.82)	1864 (434)	101 (41)	61 (34)	20.1 (5.9)	12.1 (5.5)
Leeds, 1969-76								
Black <i>et al</i> ³¹ and unpublished	20-59y	42 F	7.67 (1.63)	1827 (389)	98 (29)	37 (19)	20.4 (5.4)	7.3 (2.7)
Throughout UK 1977	Dieticians							
Nelson, ^{24,25} and unpublished	18-57y	105 M	11.68 (2.67)	2786 (637)	151 (63)	107 (58)	20.1 (6.5)	14.2 (6.5)
Cambridge 1977-79	18-53y	112 F	8.16 (1.87)	1946 (445)	103 (43)	62 (38)	19.5 (6.5)	11.7 (6.5)
Barber <i>et al</i> ^{2,222}	18-82y	40 M	12.97 (2.50)	3098 (593)	131*	—	16*	—
Orkney, 1980	19-75y	78 F	8.6 (1.70)	2053 (413)	83*	—	17*	—
Fehily <i>et al</i> ^{223,224} and unpublished	45-59y	665 M	10.00 (2.5)	2395 (585)	109 (56)	—	17.1	—
Caerphilly, S Wales 1980-83	40-59y	49 F	6.68 (1.5)	1600 (366)	67 (35)	—	15.7	—

Table 6 (Continued)

Author, Location, date	Ages	Sample number gender	Intake per day			Percent energy		
			Energy MJ	Total sugars g	Non-milk extrinsic sugars g	Total sugars %	Non-milk extrinsic sugars %	
Thomson <i>et al</i> ^{2,225} Fifeshire, 1980-82	45-54y	164 M	11.3 (2.1)	2700 (510)	141	19.5	—	—
Black <i>et al</i> ²²⁶ and unpublished, Cambridge 1978-1980 (Longitudinal Study)	Pregnant	63 F	8.69 (1.39)	2072 (331)	106 (28)	19.2 (3.5)	8.9 (3.0)	
	Lactating	47 F	10.04 (1.77)	2393 (422)	125 (33)	19.4 (3.2)	8.7 (2.8)	
Schofield <i>et al</i> ^{2,227} and unpublished Edinburgh and London, 1983-85	Non pregnant							
	Non lactating	56 F	7.93 (1.64)	1892 (390)	90 (35)	17.6 (4.9)	8.8 (4.9)	
	18-35y	260 F	8.7	2080	90*	16.0*	—	
Cole-Hamilton <i>et al</i> ²¹⁹ Throughout UK 1984	Pregnant							
		142 M	11.25 (1.76)	2679 (419)	125 (36)	17.5	8.3 (3.3)	
		209 F	7.99 (1.54)	1902 (367)	98 (28)	19.3	8.9 (3.9)	
Gibney <i>et al</i> ²²⁹ Kilkenny, 1985	35-44y	30 M	12.5 (2.6)	2976 (619)	164	20.7	—	
		30 F	8.4 (2.2)	2000 (523)	102	19.1	—	
Gibney and Lee ²³ Dublin, 1987	Adults	42 M	13.1 (3.0)	3119 (714)	209	25.1	—	
		55 F	8.5 (2.8)	2023 (666)	153	28.4	—	
Doyle <i>et al</i> ³² and unpublished London, 1988	Pregnant 1st Trimester	419 F	8.4 (1.8)	2011 (426)	102 (35)	18.9	10.2	

Table 6 (Continued)

Author, Location, date	Ages	Sample number gender	Intake per day				Percent energy	
			Energy		Total sugars		Total sugars %	Non-milk extrinsic sugars %
			MJ	kcal	g	g		
Cade <i>et al</i> 229 unpublished Ipswich, Stoke and Wakefield, 1987	35-54y	1115 M	10.95 (3.58)	2607 (852)	119.7 (60)	—	18.6 (6.7)	—
		1225 F	7.11 (2.57)	1693 (612)	78.2 (46)	—	18.6 (7.0)	—

* excluding lactose

Table 7 Sugars consumption by some elderly groups Mean (s.d.)

Author, Location, date	Ages	Sample number gender	MJ	Energy kcal	Intake per day			Percent energy		
					Total sugars g	Non-milk extrinsic sugars g	Total sugars %	Total sugars %	Non-milk extrinsic sugars %	Non-milk extrinsic sugars %
DHSS, ²³⁰ 6 areas of England and Scotland, 1967-68	over 65y	827 M&F	8.2	1962	—	63	—	—	—	12-14
Davies, ²³¹ Portsmouth, 1970	65-74y 75+y	10 M 16 M	9.0 (6.1-12.5) 9.0 (6.5-11.1)	2160 (1450-3000) 2150 (1550-2660)	—	105 (52-198) 87 (13-147)	—	—	—	18.2 15.2
Recipients of meals on-wheels	65-74y 75+y	24 F 50 F	6.7 (4.2-9.8) 7.3 (5.1-11.0)	1600 (1000-2340) 1750 (1210-2640)	—	53 (6-112) 66 (5-160)	—	—	—	12.4 14.1
DHSS, ²³² 6 areas of England and Scotland, 1972-73	70-80y Over 80y 70-80y Over 80y	111 M 58 M 125 F 71 F	9.3 (2.2) 8.5 (2.1) 7.0 (1.7) 6.5 (1.5)	2217 (522) 2024 (498) 1679 (416) 1559 (358)	—	71.6 (35.7) 66.0 (33.0) 48.8 (27.0) 48.5 (28.3)	—	—	—	12.7 (5.3) 13.2 (6.8) 11.5 (5.5) 12.2 (6.0)
Bingham <i>et al</i> ²²¹ Cambridgeshire, 1977	60-79y	7 M 7 F	9.90 (2.57) 6.42 (1.92)	2357 (612) 1529 (457)	156 (66) 61 (16)	111.0 (62.2) 27.2 (13.6)	25.2 (5.1) 16.5 (4.9)	—	—	16.8 (5.3) 6.6 (3.0)

Table 8 The contribution of various food groups to the sugars content of the diet

Groups of foods	Northumberland schoolchildren 1986 ²¹⁷ (n = 405, both sexes)		Cambridge adults*			
			Women (n = 112)		Men (n = 105)	
	Non-milk extrinsic g/d	Sugars Intrinsic plus milk sugars g/d	Non-milk extrinsic g/d	Sugars Intrinsic plus milk sugars g/d	Non-milk extrinsic g/d	Sugars Intrinsic plus milk sugars g/d
Confectionery	23.0	1.3	4.2	0.2	3.3	0.2
Table sugar	19.6	—	27.3	—	63.4	—
Soft drinks and fruit juices	13.6	2.4	4.7	1.8	3.8	1.3
Biscuits and cakes	10.1	2.9	9.7	3.5	11.2	5.1
Sweet puddings, including fruit pies	7.2	5.5	5.0	1.9	6.3	2.7
Syrups and preserves	2.8	1.5	3.8	1.6	4.4	2.0
Breakfast cereals	2.3	0.5	0.7	0.7	0.8	0.8
Milk, butter, cheese and yogurt	0	11.8	2.4	17.4	2.4	17.7
Fruit	0	5.5	1.3	6.7	1.0	5.3
Rice, pasta, bread			0	1.6	0.1	2.4
Potatoes and vegetables	2.7	5.7	0.9	3.2	1.2	4.0
Others			2.1	1.7	9.3	1.7
Totals	81.3	37.1	62.1	40.3	107.1	43.2

* data of Nelson and Paul (Unpublished)

Table 9 *Nutrient intake of 105 men and 112 women in Cambridge by thirds of the distribution of energy intake*

	Men			Women		
Total intake (per day)	Lowest 35	Middle 35	Highest 35	Lowest 37	Middle 38	Highest 37
Number in third						
Energy, kcal range	2168 1443-2418	2673 2427-2933	3448 3003-5042	1475 794-1777	1944 1791-2134	2416 2150-3318
P				+++		+++
Protein g	74	85	105	54	66	75
Fat g	94	115	151	69	88	110
Carbohydrate g	254	323	397	163	226	284
Alcohol g	9	12	25	4	5	8
Total sugars g	107	153	188	72	103	134
Non-milk extrinsic sugars g	74	107	138	42	61	83
P				+++		+++
Magnesium mg	266	321	392	188	241	292
Zinc mg	9.2	10.8	13.2	6.9	8.0	9.5
Iron mg	11.1	13.1	16.4	8.5	9.9	12.2
Thiamin mg	1.11	1.30	1.53	0.78	1.04	1.14
Calcium mg	832	1147	1350	667	934	1080
Vitamin C mg	48	59	63	42	51	69
P				+++		++
Intakes/1000 kcal						
Protein g	34.0	31.6	30.5	36.7	33.7	31.0
Fat g	43.5	43.0	43.9	46.5	45.5	45.6
Carbohydrate g	117.3	120.6	115.0	110.2	116.2	117.7
Alcohol g	4.2	4.5	7.1	2.6	2.4	3.2
Total sugars g	49.2	57.3	54.6	48.9	52.7	55.5
Non-milk extrinsic sugars g	34.1	40.0	40.1	28.5	31.4	34.3
P				ns		ns

Table 9 (Continued)

	Men			Women		
	Lowest 35	Middle 35	Highest 35	Lowest 37	Middle 38	Highest 37
Total intake (per day)						
Number in third						
Energy, kcal range	2168 1443-2418	2673 2427-2933	3448 3003-5042	1475 794-1777	1944 1791-2134	2416 2150-3318
						p
						+++
Magnesium mg	123	120	114	127	124	121
Zinc mg	4.25	4.04	3.83	4.67	4.12	3.92
Iron mg	5.12	4.89	4.77	5.75	5.09	5.05
Thiamin mg	0.51	0.49	0.44	0.53	0.53	0.47
Calcium mg	384	429	392	452	480	447
Vitamin C mg	22	22	18	28	26	29
						ns
						ns

+++ $p < 0.001$; ++ $p < 0.01$; + $p < 0.05$; ns—not significant (lowest vs highest)
data of Nelson and Paul (unpublished) (see Appendix E)

Table 10 (Continued)

	Men			Women		
	Lowest 35	Middle 35	Highest 35	Lowest 37	Middle 38	Highest 37
Number in third						
Non-milk extrinsic sugars g per 1000 kcal range	20.4 9.0-30.6	38.6 31.2-43.1	55.8 43.9-109.6	16.2 5.2-22.3	28.8 22.4-36.5	49.6 36.9-111.2
			p +++			p +++
Magnesium mg	125	122	107	132	124	115
Zinc mg	4.40	3.91	3.80	4.66	4.17	3.74
Iron mg	5.40	4.68	4.68	5.82	5.33	4.61
Thiamin mg	0.52	0.46	0.46	0.54	0.51	0.46
Calcium mg	426	393	382	496	463	422
Vitamin C mg	22	20	20	30	26	27
			ns			ns

+++ p<0.001; ++ p<0.01; + p<0.05; ns—not significant (lowest vs highest)
data of Nelson and Paul (unpublished) (see Appendix E)

Table 11 Nutrient intake according to intake of non-milk extrinsic sugars within three levels of energy intake in Cambridge adults (kcal per day)

CAMBRIDGE MEN (n=105)									
Energy intake (kcal)		Highest (3003-5042)			Middle (2427-2953)			Lowest (1443-2418)	
Non-milk extrinsic sugars intake (g per 1000 kcal)		Higher	p	Lower	Higher	p	Lower	Higher	Lower
number		41.1-99.4		19.0-41.0	41.5-109.6		9.0-41.3	32.8-84.1	9.6-32.0
		18		17	18		17	18	17
Nutrient intake									
Energy kcal		3625	ns	3260	2690	ns	2656	2180	2155
Protein g		104	ns	106	83	ns	87	70	78
Fat g		150	ns	152	110	+	120	89	100
Total CHO g		448	+++	343	350	++	294	279	228
Total sugars g		231	+++	144	190	+++	115	137	75
Non-milk extrinsic sugars g		180	+++	94	141	+++	71	105	41
Alcohol g		25	ns	25	7	ns	18	7	12
<i>RDA</i> ^{27, 28}									
Magnesium mg	350	401	ns	383	307	ns	335	261	270
Zinc mg	15	12.9	ns	13.6	10.3	ns	11.3	8.7	9.8
Iron mg	10	16.5	ns	16.4	12.1	+	14.1	10.8	11.5
Calcium mg	500	1388	ns	1309	1154	ns	1139	820	845
Thiamin mg	1.0-1.3	1.50	ns	1.56	1.30	ns	1.30	1.02	1.20
Vitamin C mg	30	66	ns	59	61	ns	58	42	53
<i>Per cent of energy</i>									
Protein		11.5	+++	13.0	12.3	ns	13.0	12.8	14.5
Fat		37.3	+	42.0	36.9	+	40.7	36.8	41.8
Total carbohydrate		46.3	+++	39.4	48.8	+++	41.5	48.0	39.7
Total sugars		23.9	+++	16.6	26.5	+++	16.3	23.6	13.2
Non-milk extrinsic sugars		18.6	+++	10.8	19.7	+++	10.0	18.0	7.2

Table 11 (Continued)

CAMBRIDGE WOMEN (n=112)									
Energy intake (kcal)		Highest (2150-3318)		Middle (1791-2134)		Lower		Lowest (794-1777)	
Non-milk extrinsic sugars intake (g per 1000 kcal)		Higher	p	Higher	p	Higher	p	Higher	Lower
Number		18		19		19		18	19
<i>Nutrient intake</i>									
Energy kcal		2507	+	2337	ns	1934	ns	1433	ns
Protein g		72	ns	77	+	63	+	49	+
Fat g		110	ns	110	+	83	+	61	+
Total CHO g		318	+++	253	+++	243	+++	177	+
Total sugars g		160	+++	106	+++	119	+++	89	+
Non-milk extrinsic sugars g		109	+++	52	+++	80	+++	65	+++
Alcohol g		6	+	11	+	3	+	2	ns
<i>RDA^{27, 28}</i>									
Magnesium mg	300	289	ns	247	ns	241	ns	166	+
Zinc mg	15	9.0	ns	10.0	ns	7.7	ns	6.2	+
Iron mg	12	11.6	ns	12.9	+	9.2	+	7.4	+
Calcium mg	500	1080	ns	1079	ns	881	ns	591	ns
Thiamin mg	0.9	1.10	ns	1.18	ns	1.05	ns	0.74	ns
Vitamin C mg	30	74	ns	64	+	45	+	37	ns
<i>Per cent of energy</i>									
Protein		11.6	++	13.3	++	13.0	ns	13.6	+
Fat		39.6	+	42.5	+	38.7	+++	38.5	+++
Total carbohydrate		47.5	+++	40.7	+++	47.1	+++	46.3	+++
Total sugars		23.9	+++	17.1	+++	23.1	+++	23.5	+++
Non-milk extrinsic sugars		16.3	+++	8.5	+++	15.5	+++	17.0	+++

+++ p<0.001; ++ p<0.01; + p<0.05; ns—not significant

Data of Nelson and Paul, unpublished (see Appendix E).

Table 12 *Nutrient intake of pregnant women in Leeds according to intake of non-milk extrinsic sugars per 1000 kcal within three levels of energy intake*

Energy intake		Highest		Middle		Lowest	
Non-milk extrinsic sugars intake (g per 1000 kcal)		Higher 43.4	Lower 23.2	Higher 43.1	Lower 24.0	Higher 42.4	Lower 18.0
Range		30.8-105.9	10.7-30.5	32.8-74.9	12.3-32.8	28.4-77.4	3.1-27.0
Number		32	33	33	33	32	33
		p		p		p	
<i>Nutrient intake</i>							
Energy kcal		2314	2362	1880	1913	1421	1390
Protein g		78	87	63	70	49	58
Fat g		100	111	80	89	60	66
Total carbohydrate g		290	265	237	219	178	151
Total sugars g		154	109	122	88	88	52
Non-milk extrinsic sugars g		102	55	81	46	60	26
Alcohol g		3	3	2	2	2	0
		ns		ns		ns	
Magnesium mg		258	265	212	219	153	159
Zinc mg		9.4	10.4	7.9	8.3	5.6	7.0
Iron mg		10.7	11.5	9.2	9.3	6.5	7.7
Calcium mg		1115	1150	902	930	619	653
Thiamin mg		1.22	1.32	0.99	1.08	0.75	0.85
Vitamin C mg		78	76	58	60	41	41
		ns		ns		ns	

Table 12 (Continued)

Energy intake	Highest		Middle		Lowest	
	Higher 43.4	p	Lower 23.2	Higher 43.1	Lower 24.0	Higher 42.4
Non-milk extrinsic sugars intake (g per 1000 kcal)						
						p
<i>Per cent of energy</i>						
Protein	13.5	+	14.7	13.4	14.7	13.7
Fat	38.9	+++	42.3	38.4	41.7	38.0
Total carbohydrate	47.1	+++	42.1	47.3	42.9	47.4
Total sugars	24.9	+++	17.3	24.4	17.2	23.2
Non-milk extrinsic sugars	16.5	+++	8.7	16.1	9.0	15.9
						16.6
						42.5
						40.8
						14.0
						6.9

+++ $p < 0.001$; ++ $p < 0.01$; + $p < 0.05$; ns—not significantData of Smithells *et al*³⁴ and unpublished (see Appendix E).

Table 13 Daily intake of nutrients of unemployed families in Dublin

Nutrient	Men		Women		Men		Women	
	Mean	(sd)	Mean	(sd)	Lowest	Highest	Lowest	Highest
	42		55		10	10	15	12
Intake:								
Energy (MJ)	13.1	(3.0)	8.5	(2.8)	11.7	15.0	7.2	11.6
Protein (g)	100	(25)	64	(18)	91	100	64	69
Fat (g)	128	(33)	81	(24)	127	123	81	83
Carbohydrate (g)	409	(128)	275	(131)	312	552	191	460
Starch (g)	200	(62)	122	(48)	198	190	120	105
Fibre (g)	21.5	(6.4)	13.9	(5.0)	19.6	21.1	13.8	14.1
Iron (mg)	13.5	(3.2)	8.1	(2.3)	13.1	12.8	7.5	8.7
Zinc (mg)	12.9	(3.6)	8.3	(2.5)	12.2	12.6	8.4	8.6
Vitamin C (mg)	60	(21)	45	(18)	57	60	43	49
Retinol (μ g)	1507	(1299)	1070	(1032)	1484	1339	1050	1317
Table sugar (g)	95	(99)	73	(100)	0	242	0	231
Body mass index (kg/m^2)	24.5	(3.6)	23.2	(4.3)	25.8	23.9	23.1	23.5

Data of Gibney and Lee²³

Table 14 *Percentage distribution of Quetelet index by age and sex among British adults*

Body Mass Index	All ages										
	16-64	16-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64
Men											
20 or under	10	33	14	9	7	8	3	3	5	7	6
Over 20-25	51	56	64	61	53	51	47	43	44	43	39
Over 25-30	33	9	19	26	34	35	42	44	45	42	44
Over 30-35	5	2	3	3	5	5	7	9	5	7	10
Over 35-40	1	—	—	0	1	1	1	1	0	1	0
Over 40	0	—	—	—	—	0	0	—	—	—	0
Base	4,499	480	516	537	520	453	433	394	417	427	316
Average value	24.3	21.4	23.0	23.8	24.5	24.7	25.3	25.7	25.2	25.2	25.4
Median value	24.5	21.5	22.5	23.5	24.5	24.5	25.3	25.5	25.5	25.5	25.5
Standard error of average	0.05	0.15	0.15	0.13	0.13	0.19	0.15	0.19	0.17	0.17	0.25

Table 14 (Continued)

Body Mass Index	All ages										
	16-64	16-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64
Women											
20 or under	14	33	23	18	16	11	7	7	9	6	8
Over 20-25	54	53	56	62	57	61	57	54	48	46	41
Over 25-30	24	11	16	14	22	20	28	28	30	34	36
Over 30-35	6	2	4	4	4	6	6	7	10	12	10
Over 35-40	2	0	1	1	0	2	2	2	3	1	4
Over 40	0	0	0	0	0	0	1	1	1	1	1
Base	4,935	497	547	556	559	492	481	413	491	484	398
Average value	23.9	21.8	22.7	22.9	23.4	23.9	24.6	24.9	25.2	25.4	25.7
Median value	23.5	21.5	22.5	21.5	22.5	23.5	23.5	24.5	24.5	24.5	25.5
Standard error of average	0.08	0.14	0.18	0.17	0.15	0.16	0.19	0.19	0.24	0.18	0.25

Data of Knight¹²⁴

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Figure 1. Supplies of selected sugars since 1850 expressed as grams per head of the population per day

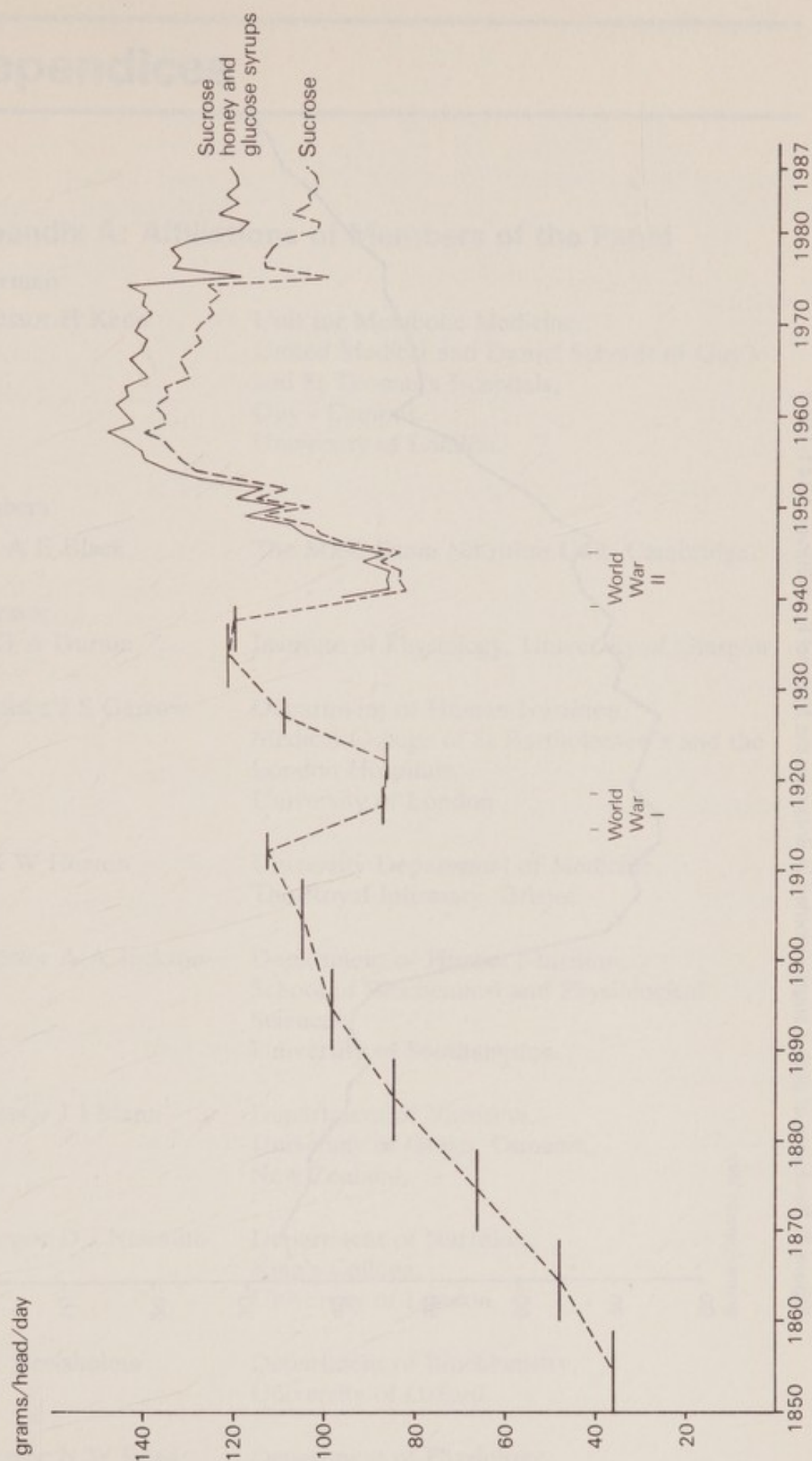
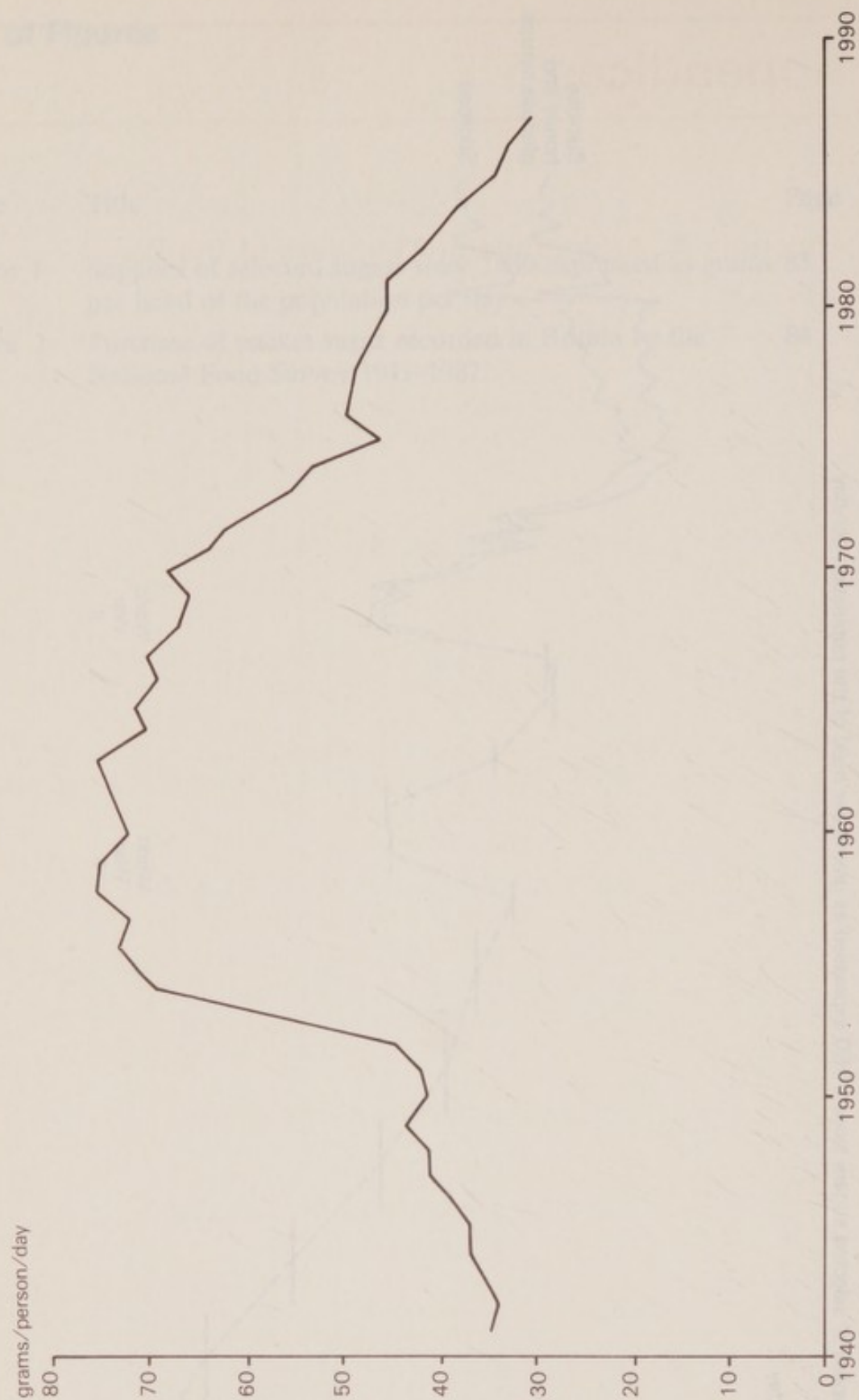


Figure 2. Purchases of packet sugar recorded in Britain by the National Food Survey 1941-1987



Appendices

Appendix A: Affiliations of Members of the Panel

Chairman

Professor H Keen	Unit for Metabolic Medicine, United Medical and Dental Schools of Guy's and St Thomas's Hospitals, Guy's Campus, University of London.
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Members

Miss A E Black	The MRC Dunn Nutrition Unit, Cambridge.
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Professor J V G A Durnin	Institute of Physiology, University of Glasgow.
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Professor J S Garrow	Department of Human Nutrition, Medical College of St Bartholomew's and the London Hospitals, University of London.
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Dr K W Heaton	University Department of Medicine, The Royal Infirmary, Bristol.
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Professor A A Jackson	Department of Human Nutrition, School of Biochemical and Physiological Sciences, University of Southampton.
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Professor J I Mann	Department of Nutrition, University of Otago, Dunedin, New Zealand.
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Professor D J Naismith	Department of Nutrition, King's College, University of London.
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Dr E Newsholme	Department of Biochemistry, University of Oxford.
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Professor N W Read	Department of Physiology, University of Sheffield.
--------------------	---

Professor A J Rugg-Gunn Departments of Child Dental Health and Oral
Biology,
The Dental School,
University of Newcastle Upon Tyne.

Professor T Silverstone Department of Psychological Medicine,
Medical College of St Bartholomew's Hospital,
University of London.

Observers

Dr J G Ablett Department of Health, London.

Dr D H Buss Ministry of Agriculture, Fisheries and Food,
London.

Dr P C Clarke Department of Health, London.

Mr C Howard Department of Health, London.

Secretariat

Dr M J Wiseman
(Medical) Department of Health, London.

Mr R W Wenlock
(Scientific) Department of Health, London.

Mr K L G Follin
(Administrative) Department of Health, London.

Mrs N Bateson
(Administrative) Department of Health, London.

Mrs E Lohani
(Administrative) Department of Health, London.

Mr D Roberts
(Administrative) Department of Health, London.

Appendix B: Individuals and Organisations who submitted evidence to the Panel

We wish to record our indebtedness to the following individuals and organisations who have assisted us by contributing evidence and to express our thanks for their help.

Dr D Benton Department of Psychology,
University College,
Swansea

Dr B Bibby Eastman Dental Centre,
New York

Billingtons
Edward Billington (Sugar) Ltd

Biscuit, Cake, Chocolate
and Confectionary
Alliance

Janet Cade and others MRC Environmental Epidemiology Unit,
Southampton

M J Campbell Department of Medical Statistics and Computing,
University of Southampton

Coronary Prevention Group

Dr A M Cushing For Action and Information on Sugars

Finn Sugar Xyrofin

Food and Drink Federation

Food Policy Research University of Bradford
Unit

A D French British Association for the Study of Community
Dentistry

J R Goodman The British Paedodontic Society, London

Irish Sugar PLC

Professor W P T James The Rowett Research Institute, Aberdeen

Dr S Kreitzman	University of Cambridge Clinical School
M McClusky	Kenilworth, Warwickshire
M Midda	University of Bristol Dental Hospital, Bristol
Miss J Scott	Community Dietician Representing The Community Nutrition Group of the British Dietetic Association
Professor A Sheiham	Department of Community Dental Health and Dental Practice, School of Medicine, University College Hospital London
Society for the Understanding of Nutrition	
Sugar Bureau	
Tate and Lyle PLC	
Dr A R P Walker	Human Biochemistry Research Unit, South African Institute for Medical Research, Johannesburgh, South Africa,
Dr J T Winkler	King's Fund Institute, London
Professor J Yudkin	Emeritus Professor of Nutrition, King's College (KQC), Campden Hill Road, London

Appendix C: Control of Claims Relating to the Presence or Absence of Sugars in Foods

1. Various claims about the nutritional value of foods are controlled by the Food Labelling Regulations 1984,²³³ which were made under the Food and Drugs Act 1955 (now the Food Act 1984). The general provisions of the Act itself prohibit the use of any label or advertisement which falsely describes food or misleads as to its nature, substance or quality, including its nutritional or dietary value.

2. Food Labelling Regulations 1984²³³ then lay down more specific controls over, for example, claims that a food is an aid to slimming, has a reduced energy value, or is suitable for diabetics. Some of these controls implement the European Community Council Directive relating to the labelling, presentation and advertising of foodstuffs.

3. More recently, claims have been made that certain foods are low, high, reduced, higher in or free from certain nutrients eg "low sugar", "free from added sugar", and there is evidence that these claims are not always soundly based. The Food Advisory Committee considered this issue and has recommended that before certain claims for sugars, fats, fibre and salt are made, either expressly or by implication, certain conditions should be met. For sugars these are as follows:

Claim	Conditions
A reduced sugar(s) content	Total sugars per 100g (or 100ml) must not be more than three quarters of that of a similar food for which no such claim is made.
Low in sugar(s)	Total sugars must not exceed 5g per 100g (or 100ml) <i>and</i> total sugars in a normal serving must not exceed 5g.
Sugar(s) – free	Total sugars must not exceed 0.1g per 100g (or per 100ml).
No added sugar/ unsweetened	No mono- or di-saccharides or foods composed mainly of these sugars should have been added to the food or any of its ingredients.

4. The Committee also recommended that when any of these claims were made on a label or in an advertisement, the food should carry full nutritional labelling, showing per 100g (or 100ml) its energy, protein, carbohydrate, total sugars, fat, saturated fatty acids, sodium and fibre contents.

5. These recommendations have been issued by the Ministry of Agriculture, Fisheries and Food for consultation with interested parties.

Appendix D: Evaluation of the Main Dietary Carbohydrates

The following energy yields have been found experimentally for the main carbohydrates in food when burned in a calorimeter.²³⁴

	Per gram		Per gram of monosaccharide equivalent	
	kcal	kJ	kcal	kJ
Glucose	3.72	15.6	3.72	15.6
Sucrose	3.94	16.5	3.74	15.7
Starch	4.18	17.5	3.76	15.7

The yields per gram differ because of the different proportions of carbon, hydrogen and oxygen in monosaccharides, disaccharides and polysaccharides but when each is converted to its monosaccharide equivalent (the form in which it is absorbed) the energy yields are essentially identical. This is the basis for the UK practice for expressing the energy value of carbohydrate-containing foods in food composition tables and for food labelling.^{12, 233.}

Before 1978, intakes in the UK shown in tables 3 to 6 were calculated from unpublished tables of nutrient composition compiled in 1968 by the Ministry of Health. They were based on the third edition (1960) of McCance and Widdowson's *The Composition of Foods*²³⁵ with additional recipes and manufacturer's information. These provided values for total carbohydrate and for extrinsic sugars (defined in para 2.15.2) which were expressed as monosaccharide units. After 1978, nutrient intakes were calculated from the fourth edition of *McCance and Widdowson's The Composition of Foods*¹² with some additional recipe data²³⁶ and manufacturer's information. These provided values for total carbohydrate and total sugars including the concentrations of sucrose, lactose, glucose, fructose and maltose. The estimates of total energy intake in the fourth edition are on average 3.5 per cent lower, and of carbohydrate intakes 6.0 per cent lower, than those in the third edition.²³⁷ Some of the studies in this Report have included values for non-milk extrinsic ('added') sugars (recipe sugars plus table sugars as defined in para 2.17) in the database derived from the fourth edition¹² and have calculated intakes of both total sugars and non-milk extrinsic ('added') sugars.^{24, 25, 32, 33, 34, 211, 212, 216, 217, 221, 227, 230.}

Appendix E: Nutrient Intakes and Sugars Consumption

The full data calculated for tables 9 to 12 are presented in the appendices to each table on pages 92–101.

Appendix to Table 9: Nutrient intake of 105 men and 112 women in Cambridge according to thirds of the distribution of energy intake

	Men				Women			
	Lower	Middle	Upper	t	Lower	Middle	Upper	t
<i>Total intake (per day)</i>								
Number	35	35	35	(df=68)	37	38	37	(df=72)
Energy, kcal	2168	2673	3448	12.6+++	1475	1944	2416	15.5+++
Range	1443-2418	2427-2953	3003-5024		794-1775	1791-2134	2150-3318	
<i>Nutrient intake</i>								
Potassium mg	3167	3652	4216	4.6+++	2224	2867	3254	9.0+++
Calcium mg	832	1147	1350	7.1+++	667	934	1080	6.8+++
Magnesium mg	266	321	392	5.2+++	188	241	292	8.7+++
Phosphorus mg	1192	1473	1786	9.2+++	911	1166	1346	7.2+++
Iron mg	11.1	13.1	16.4	8.4+++	8.5	9.9	12.2	5.5+++
Copper mg	1.65	1.73	2.41	3.5+++	1.23	1.41	1.53	2.6+
Zinc mg	9.2	10.8	13.2	7.3+++	6.9	8.0	9.5	5.5+++
Chloride mg	4017	5303	6914	10.0+++	3039	3577	4431	6.4+++
Thiamin mg	1.11	1.30	1.53	5.6+++	0.78	1.04	1.14	6.2+++
Riboflavin mg	1.74	2.17	2.61	5.2+++	1.52	1.81	1.92	2.7++
Nicotinic acid mg	16.6	18.6	22.8	4.7+++	11.8	13.5	15.4	4.0+++
Tryptophan/60 mg	15.6	18.1	22.8	8.8+++	11.4	13.9	15.9	7.3+++
Vitamin B6 mg	1.20	1.33	1.64	4.9+++	0.82	1.05	1.14	7.1+++
Folic acid µg	155	194	233	5.0+++	120	139	171	3.9+++
Vitamin B12 µg	7.28	7.18	12.36	2.4+	6.86	6.01	5.77	0.8ns
Vitamin C mg	48	59	63	2.9++	42	51	69	3.0++
Vitamin E mg	4.31	5.44	6.73	7.0+++	3.41	4.21	5.37	6.1+++
Vitamin D µg	2.33	2.83	4.36	3.7+++	2.22	2.12	2.82	1.4ns
Retinol µg	1453	1204	2220	1.5ns	1406	1212	1145	0.8ns
Carotene µg	2667	3286	2481	0.3ns	2125	2225	2774	1.4ns

Appendix to Table 9: (Continued)

	Men				Women			
	Lower	Middle	Upper	t	Lower	Middle	Upper	t
<i>Total intake (per day)</i>								
Number	35	35	35	(df=68)	37	38	37	(df=72)
Energy, kcal	2168	2673	3448	12.6+++	1475	1944	2416	15.5+++
Range	1443-2418	2427-2953	3003-5024		794-1775	1791-2134	2150-3318	
<i>Intake/1000 kcal</i>								
Potassium mg	1461	1366	1223	4.5+++	1508	1474	1347	2.9++
Calcium mg	284	429	392	0.4ns	452	480	447	0.1ns
Magnesium mg	123	120	114	1.6ns	127	124	121	0.9ns
Phosphorus mg	550	551	518	1.5ns	618	600	557	2.1+
Iron mg	5.12	4.90	4.77	1.2ns	5.75	5.09	5.05	1.9ns
Copper mg	0.76	0.65	0.70	1.1ns	0.83	0.73	0.63	2.7++
Zinc mg	4.25	4.04	3.83	2.3+	4.67	4.12	3.92	3.0++
Chloride mg	1853	1984	2005	1.9ns	2060	1891	1834	1.8ns
Thiamin mg	0.51	0.49	0.44	2.0+	0.53	0.53	0.47	1.8ns
Riboflavin mg	0.80	0.81	0.76	0.7ns	1.03	0.93	0.79	2.7++
Nicotinic acid mg	7.7	6.9	6.6	2.4+	8.0	6.9	6.4	3.3++
Tryptophan/60 mg	7.2	6.8	6.6	2.8++	7.8	7.1	6.6	3.9+++
Vitamin B6 mg	0.55	0.50	0.47	2.9++	0.56	0.54	0.47	3.5+++
Folic acid µg	71	72	67	0.9ns	81	72	71	1.3ns
Vitamin B12 µg	3.35	2.69	3.58	0.3ns	4.65	3.09	2.39	2.8++
Vitamin C mg	22	22	18	1.7ns	28	26	29	0.1ns
Vitamin E mg	1.99	2.04	1.95	0.0ns	2.31	2.17	2.22	0.3ns
Vitamin D µg	1.08	1.06	1.26	1.1ns	1.50	1.09	1.17	1.2ns
Retinol µg	670	450	644	0.0ns	993	623	474	2.5+
Carotene µg	1230	1229	720	2.5+	1441	1144	1148	0.9ns

+++ p<0.001; ++ p<0.01; + p<0.05; ns = not significant (lower vs upper)

Data of Nelson and Paul (unpublished)

Appendix to Table 10: *Nutrient intake of 105 men and 112 women in Cambridge according to the proportion of sugar in the diet (expressed as grams of extrinsic sugar per 1000 kcal)*

	Men				Women			
	Lower	Middle	Upper	t	Lower	Middle	Upper	t
Number	35	35	35	(df=68)	37	38	37	(df=72)
Extrinsic sugars g per 1000 kcal	20.4	38.6	55.4	13.0+++	15.6	28.8	49.6	14.5+++
Range	9.0-30.6	31.2-43.1	43.9-109.6		5.2-22.3	22.4-36.5	36.9-111.2	
<i>Nutrient intake</i>								
Potassium mg	3571	3951	3514	0.3ns	2685	2915	2744	0.3ns
Calcium mg	1122	1185	1023	1.1ns	906	937	837	0.8ns
Magnesium mg	324	367	287	2.2+	241	251	228	0.8ns
Phosphorus mg	1499	1592	1359	1.7ns	1171	1197	1054	1.5ns
Iron mg	14.0	14.1	12.5	1.7ns	10.6	10.8	9.2	2.0+
Copper mg	1.86	2.10	1.83	0.1ns	1.39	1.50	1.28	0.9ns
Zinc mg	11.3	11.8	10.2	1.8ns	8.5	8.4	7.4	2.0+
Chloride mg	5298	5974	4961	0.9ns	3874	3768	3504	1.3ns
Thiamin mg	1.32	1.40	1.22	1.1ns	0.98	1.05	0.92	0.8ns
Riboflavin mg	2.11	2.25	2.15	0.2ns	1.80	1.82	1.63	1.0ns
Nicotinic acid mg	19.5	20.6	17.8	1.3ns	13.8	14.3	12.6	1.3ns
Tryptophan/60 mg	18.9	20.3	17.2	1.8ns	14.1	14.4	12.7	1.8ns
Vitamin B6 mg	1.34	1.51	1.31	0.4ns	0.99	1.05	0.98	0.1ns
Folic acid µg	193	220	169	1.6ns	158	150	122	2.6++
Vitamin B12 µg	8.52	8.83	9.46	0.4ns	7.46	6.36	4.81	2.1++
Vitamin C mg	55	62	53	0.4ns	54	53	54	0.0ns
Vitamin E mg	5.78	5.83	4.86	2.2+	4.50	4.41	4.07	1.1ns
Vitamin D µg	3.58	0.22	2.72	1.5ns	2.52	2.27	2.36	0.3ns
Retinol µg	1578	1421	1878	0.6ns	1568	1249	944	2.0+
Carotene µg	3339	2944	2151	2.6++	2641	2743	1725	2.4+

Appendix to Table 10: (Continued)

	Men				Women			
	Lower	Middle	Upper	t	Lower	Middle	Upper	t
Number	35	35	35	(df=68)	37	38	37	(df=72)
Extrinsic sugars g per 1000 kcal	20.4	38.6	55.4	13.0	15.6	28.8	49.6	14.5++
Range	9.0-30.6	31.2-43.1	43.9-109.6		5.2-22.3	22.4-36.5	36.9-111.2	
<i>Intake/1000 kcal</i>								
Potassium mg	1376	1310	1312	1.0ns	1471	1441	1382	1.3ns
Calcium mg	433	393	382	1.5ns	496	463	422	2.3+
Magnesium mg	125	122	107	4.0+++	132	124	115	2.3++
Phosphorus mg	578	528	507	3.8+++	642	591	530	3.7+++
Iron mg	5.38	4.68	4.68	3.4++	5.82	5.33	4.61	4.1+++
Copper mg	0.72	0.70	0.68	0.8ns	0.76	0.74	0.64	1.1ns
Zinc mg	4.36	3.91	3.80	3.8+++	4.66	4.17	3.74	3.8+++
Chloride mg	2042	1981	1852	2.6+	2122	1862	1765	3.6+++
Thiamin mg	0.51	0.46	0.46	2.0+	0.54	0.51	0.46	2.4+
Riboflavin mg	0.81	0.75	0.80	0.0ns	0.99	0.89	0.82	1.7ns
Nicotinic acid mg	7.5	6.8	6.7	2.2+	7.5	7.1	6.3	2.5+
Tryptophan/60 mg	7.3	6.7	6.4	4.8+++	7.8	7.1	6.4	4.7+++
Vitamin B6 mg	0.52	0.50	0.49	1.3ns	0.54	0.52	0.49	1.7ns
Folic acid µg	74	73	63	2.7++	87	74	62	3.0++
Vitamin B12 µg	3.28	2.92	3.53	0.2ns	4.08	3.14	2.42	1.5ns
Vitamin C mg	21	20	20	1.0ns	30	26	27	0.6ns
Vitamin E mg	2.22	1.93	1.81	3.1++	2.46	2.18	2.05	2.9++
Vitamin D µg	1.38	1.07	1.01	1.7ns	1.38	1.12	1.19	0.9ns
Retinol µg	608	471	701	1.3ns	859	617	476	1.7ns
Carotene µg	1287	976	803	2.7++	1447	1356	869	2.6+

+++p<0.001; ++<0.01; +p<0.05; ns = not significant (lower vs upper)

Data of Nelson and Paul (unpublished)

Appendix to Table 11: Nutrient intake according to intake of non-milk extrinsic sugars within three levels of energy intake in Cambridge adults

CAMBRIDGE MEN (n=105)										
Energy intake (kcal)	Highest (3003-5042)			Middle (2427-2953)			Lowest (1443-2418)			
	Higher	p	Lower	Higher	p	Lower	Higher	p	Lower	
Non-milk extrinsic sugars intake (g per 1000 kcal)	41.1-99.4		19.0-41.0	41.5-109.6		9.0-41.3	32.8-84.1		9.6-32.0	
number	18		17	18		17	18		17	
<i>Nutrient intake</i>										
Potassium mg	4381	0.8ns	4042	3687	0.3ns	3614	3027	1.3ns	3316	
Calcium mg	1388	0.6ns	1309	1154	0.1ns	1139	820	0.2ns	845	
Magnesium mg	401	0.3ns	383	307	1.6ns	335	261	0.4ns	270	
Phosphorus mg	1795	0.1ns	1776	1452	0.5ns	1495	1159	0.8ns	1226	
Iron mg	16.5	0.1ns	16.4	12.1	2.1+	14.1	10.77	0.7ns	11.5	
Copper mg	2.56	0.8ns	2.24	1.72	0.1ns	1.75	1.58	0.6ns	1.73	
Zinc mg	12.9	0.7ns	13.6	10.3	1.4ns	11.3	8.66	1.8ns	9.8	
Chloride mg	7017	0.4ns	6804	5106	1.3ns	5512	3947	0.5ns	4091	
Thiamin mg	1.50	0.5ns	1.56	1.30	0.0ns	1.30	1.02	1.9ns	1.20	
Riboflavin mg	2.67	0.5ns	2.55	2.23	0.5ns	2.11	1.74	0.0ns	1.73	
Nicotinic acid mg	22.6	0.1ns	23.0	17.7	1.4ns	19.5	15.8	1.1ns	17.5	
Tryptophan/60 mg	22.7	0.1ns	22.9	17.6	1.3ns	18.6	14.8	1.9ns	16.4	
Vitamin B6 mg	1.64	0.0ns	1.64	1.33	0.0ns	1.33	1.15	0.9ns	1.25	
Folic acid µg	238	0.3ns	229	183	1.2ns	205	147	1.3ns	164	
Vitamin B12 µg	13.18	0.5ns	11.48	8.24	0.9ns	6.06	6.34	0.7ns	8.27	
Vitamin C mg	66	0.8ns	59	61	0.3ns	58	42	1.6ns	53	
Vitamin E mg	6.46	1.0ns	7.82	4.90	2.5+	6.00	4.22	0.4ns	4.40	
Vitamin D µg	4.29	0.1ns	4.43	2.33	2.1+	3.45	2.35	0.1ns	2.31	
Retinol µg	2468	0.7ns	1957	1364	0.7ns	1035	1478	0.0ns	1428	
Carotene µg	2718	0.8ns	2230	2469	2.3+	4151	1889	2.1+	3490	

Appendix to Table 11: (Continued)

Energy intake (kcal)	CAMBRIDGE MEN (n=105)							
	Highest (3003-5042)		Middle (2427-2953)		Lowest (1443-2418)		p	Lower
	Higher 41.1-99.4 18	Lower 19.0-41.0 17	Higher 41.5-109.6 18	Lower 9.0-41.3 17	Higher 32.8-84.1 18	Lower 9.6-32.0 17		
Non-milk extrinsic sugars intake (g per 1000 kcal) number								
<i>Intake/1000 kcal</i>								
Potassium mg	1191	1240	1376	1361	1382	1539	1.9ns	
Calcium mg	384	402	429	428	383	386	0.0ns	
Magnesium mg	109	118	115	126	119	126	0.9ns	
Phosphorus mg	496	546	540	562	532	566	1.1ns	
Iron mg	4.65	5.02	4.50	5.31	4.87	5.33	1.4ns	
Copper mg	0.68	0.69	0.64	0.66	0.71	0.80	0.9ns	
Zinc mg	3.58	4.16	3.83	4.27	3.95	4.56	2.6+	
Chloride mg	1936	2086	1893	2080	1819	1902	0.7ns	
Thiamin mg	0.42	0.48	0.48	0.49	0.47	0.56	2.0+	
Riboflavin mg	0.73	0.79	0.83	0.80	0.80	0.80	0.0ns	
Nicotinic acid mg	6.2	7.1	6.6	7.4	7.21	8.14	1.1ns	
Tryptophan/60 mg	6.2	7.0	6.6	7.0	6.8	7.6	2.9++	
Vitamin B6 mg	0.45	0.50	0.50	0.50	0.52	0.58	1.4ns	
Folic acid µg	65	71	68	77	67	76	1.6ns	
Vitamin B12 µg	3.56	3.53	3.05	2.30	2.86	3.79	0.8ns	
Vitamin C mg	19	18	23	22	19	25	1.7ns	
Vitamin E mg	1.85	2.16	1.82	2.26	1.95	2.04	0.4ns	
Vitamin D µg	1.24	1.34	0.86	1.26	1.10	1.08	0.1ns	
Retinol µg	683	603	502	390	659	645	0.0ns	
Carotene µg	762	686	916	1554	853	1592	2.1+	

++ +p<0.001; +p<0.01; +p<0.05; ns = not significant

Data of Nelson and Paul (unpublished)

Appendix to Table 11: (continued)

CAMBRIDGE WOMEN (n=112)									
Energy intake (kcal) Non-milk extrinsic sugars intake (g per 1000 kcal)	Highest (2150-3318)		Middle (1791-2134)		Lowest (794-1777)				
	Higher	Lower	Higher	Lower	Higher	Lower	p	p	Lower
Number	18	19	19	19	18	19			19
	32.8-84.4	10.9-31.8	26.2-71.0	12.0-25.9	23.8-111.1	5.2-19.9			
	p		p						
<i>Nutrient intake</i>									
Potassium mg	3246	3261	2962	2773	2085	2355	1.4ns	1.7ns	2355
Calcium mg	1080	1079	881	986	591	740	1.2ns	1.8ns	740
Magnesium mg	289	297	241	241	166	208	0.0ns	2.4+	208
Phosphorus mg	1310	1381	1122	1210	814	1003	1.2ns	2.3+	1003
Iron mg	11.6	12.9	9.2	10.6	7.4	9.5	2.4ns	2.1+	9.5
Copper mg	1.51	1.56	1.28	1.54	1.22	1.23	1.5ns	0.0ns	1.23
Zinc mg	9.0	10.0	7.7	8.3	6.2	7.6	1.5ns	2.2+	7.6
Chloride mg	4414	4451	3564	3791	2676	3383	1.6ns	2.1+	3383
Thiamin mg	1.10	1.18	1.05	1.02	0.74	0.83	0.4ns	0.9ns	0.83
Riboflavin mg	1.93	1.91	1.73	1.89	1.44	1.60	0.9ns	0.7ns	1.60
Nicotinic acid mg	14.8	16.0	13.4	13.6	10.90	12.6	0.2ns	1.2ns	12.6
Tryptophan/60 mg	15.5	16.4	13.3	14.4	10.3	12.5	1.7ns	2.4+	12.5
Vitamin B6 mg	1.13	1.15	1.04	1.06	0.78	0.86	0.2ns	1.1ns	0.86
Folic acid µg	152	193	133	145	104	136	1.0ns	1.5ns	136
Vitamin B12 µg	4.85	6.65	4.36	7.65	6.59	7.11	1.9ns	0.2ns	7.11
Vitamin C mg	74	64	45	56	37	46	1.6ns	1.0ns	46
Vitamin E mg	5.28	5.45	3.87	4.55	3.02	3.78	2.1+	1.9ns	3.78
Vitamin D µg	2.82	2.82	1.91	2.33	1.93	2.89	1.5ns	0.7ns	2.89
Retinol µg	1118	1173	679	1745	1297	1509	2.7+	0.3ns	1509
Carotene µg	1842	3657	2118	2333	2023	2221	0.0ns	0.3ns	2221

Appendix to Table 11: (Continued)

CAMBRIDGE WOMEN (n=112)						
Energy intake (kcal) Non-milk extrinsic sugars intake (g per 1000 kcal)	Highest (2150-3318)		Middle (1791-2134)		Lowest (794-1777)	
	Higher	Lower	Higher	Lower	Higher	Lower
Number	18	19	19	19	18	19
	p		p		p	
<i>Intake/1000 kcal</i>						
Potassium mg	1304	1407	1534	1423	1470	1567
Calcium mg	403	466	455	504	412	488
Magnesium mg	116	129	125	124	116	137
Phosphorus mg	525	593	597	620	571	663
Iron mg	4.63	5.56	4.78	5.41	5.20	6.21
Copper mg	0.60	0.67	0.66	0.79	0.92	0.80
Zinc mg	3.62	4.34	3.96	4.27	4.34	5.03
Chloride mg	1769	1924	1841	1944	1859	2199
Thiamin mg	0.44	0.51	0.55	0.52	0.52	0.55
Riboflavin mg	0.77	0.82	0.89	0.97	1.04	1.06
Nicotinic acid mg	5.9	6.9	6.9	7.0	7.78	8.37
Tryptophan/60 mg	6.2	7.0	6.9	7.4	7.2	8.3
Vitamin B6 mg	0.45	0.50	0.54	0.54	0.55	0.57
Folic acid µg	61	83	69	75	73	89
Vitamin B12 µg	4.05	2.78	2.28	3.97	5.21	4.47
Vitamin C mg	30	28	23	29	26	30
Vitamin E mg	2.12	2.34	2.00	2.33	2.08	2.46
Vitamin D µg	1.13	1.19	0.99	1.19	1.26	1.61
Retinol µg	429	486	354	903	1037	948
Carotene µg	750	1598	1081	1205	1373	1462

+++p<0.001; ++p<0.01; +p<0.05; ns = not significant
Data of Nelson and Paul (unpublished)

Appendix to Table 12: *Nutrient intake of 195 women in the first trimester of pregnancy in Leeds according to intake of extrinsic sugars within three levels of energy intake*

Energy intake	Highest			Middle			Lowest		
	Higher 43.4	p	Lower 23.2	Higher 43.1	p	Lower 24.0	Higher 42.4	p	Lower 18.0
<i>Nutrient intake</i>									
Potassium mg	3088	1.3ns	3267	2485	2.8++	2766	1834	1.5ns	2025
Calcium mg	1115	0.5ns	1150	902	0.5ns	930	619	0.6ns	653
Magnesium mg	258	0.6ns	265	212	0.6ns	219	153	0.7ns	159
Phosphorus mg	1353	1.2ns	1429	1116	1.0ns	1170	797	2.1+	900
Iron mg	10.7	1.6ns	11.5	9.2	0.1ns	9.3	6.5	3.1++	7.7
Copper mg	1.64	1.1ns	1.81	1.34	0.4ns	1.30	1.00	1.2ns	1.12
Zinc mg	9.4	2.0+	10.4	7.9	0.9ns	8.3	5.6	3.4++	7.0
Chloride mg	4443	1.8ns	4828	3711	2.0+	4044	2788	1.7ns	3052
Thiamin mg	1.22	1.6ns	1.32	0.99	1.6ns	1.08	0.75	2.0+	0.85
Riboflavin mg	2.07	0.3ns	2.12	1.73	0.3ns	1.68	1.14	2.3+	1.39
Nicotinic acid mg	14.6	1.1ns	15.7	12.7	0.0ns	12.8	9.1	3.3++	11.4
Tryptophan/60 mg	16.5	2.6+	18.2	13.5	2.7++	15.0	10.2	3.2++	12.3
Vitamin B6 mg	1.26	1.9ns	1.38	1.05	1.6ns	1.13	0.79	2.3+	0.92
Folic acid µg	173	1.6ns	191	144	0.5ns	148	111	1.2ns	123
Vitamin B12 µg	7.20	0.7ns	8.15	6.34	1.3ns	5.09	4.73	1.5ns	6.43
Vitamin C mg	78	0.1ns	76	58	0.2ns	60	41	0.0ns	41
Vitamin E mg	4.70	1.9ns	5.23	3.88	1.5ns	4.20	2.82	1.3ns	3.14
Vitamin D µg	3.10	0.5ns	2.90	2.18	0.0ns	2.18	1.77	0.8ns	2.02
Retinol µg	1178	1.1ns	1502	1020	1.5ns	680	715	1.2ns	1020
Carotene µg	1903	1.4ns	2517	1785	0.8ns	1545	935	0.5ns	1054

Appendix to Table 12: Nutrient intake of 195 women in the first trimester of pregnancy in Leeds according to intake of extrinsic sugars within three levels of energy intake

Energy intake	Highest		Middle		Lowest	
	Higher 43.4	p	Higher 43.1	p	Higher 42.4	Lower 18.0
<i>Intake/1000 kcal</i>						
Potassium mg	1334	1.1ns	1321	2.4+	1280	2.8++
Calcium mg	485	0.0ns	480	0.2ns	436	1.0ns
Magnesium mg	111	0.0ns	113	0.3ns	107	2.2+
Phosphorus mg	587	0.7ns	593	0.7ns	562	3.3++
Iron mg	4.64	1.3ns	4.87	0.1ns	4.57	4.1+++
Copper mg	0.71	0.9ns	0.71	0.6ns	0.71	1.6ns
Zinc mg	4.07	1.7ns	4.20	0.6ns	3.95	3.9+++
Chloride mg	1933	1.3ns	1969	1.9ns	1970	2.4+
Thiamin mg	0.53	1.2ns	0.52	1.7ns	0.53	2.8++
Riboflavin mg	0.90	0.0ns	0.92	0.5ns	0.81	2.9++
Nicotinic acid mg	6.3	0.9ns	6.7	0.0ns	6.4	4.1+++
Tryptophan/60 mg	7.2	2.1+	7.2	2.4+	7.2	4.1+++
Vitamin B6 mg	0.55	1.7ns	0.56	1.3ns	0.55	3.6+++
Folic acid µg	75	1.3ns	76	0.3ns	78	1.0ns
Vitamin B12 µg	3.09	0.7ns	3.38	1.4ns	3.34	1.7ns
Vitamin C mg	34	0.4ns	31	0.1ns	29	0.1ns
Vitamin E mg	2.03	1.7ns	2.06	1.2ns	2.01	1.5ns
Vitamin D µg	1.32	0.5ns	1.15	0.1ns	1.25	1.1ns
Retinol µg	504	1.0ns	544	1.6ns	508	1.3ns
Carotene µg	829	1.3ns	939	1.0ns	638	0.9ns

+++p<0.00; ++p<0.01; +p<0.05; ns = not significant

Data of Smithells *et al*³⁰ and unpublished









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