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Sucrose in the diabetic diet

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Introduction

The aims of dietary management of diabetes mellitus are to optimize control of blood glucose levels, to minimize the risk of hypoglycaemia in those treated with insulin, to achieve weight loss in the obese and to reduce the risk of long-term complications.

In the late 1970s and early 1980s, due to accumulating evidence regarding the effects of diet on glucose and lipid metabolism in both diabetics and non-diabetics, the Diabetes Associations of several countries reviewed their dietary recommendations. A recommendation common to all countries was an increased carbohydrate intake with a concomitant decrease in fat intake. In contrast, advice regarding the restriction of glucose and glucose-containing disaccharides (sucrose and lactose) was unchanged. However, the rationale behind the elimination of sucrose and other simple sugars was questioned and there was a general consensus that the harmful effects of sucrose for diabetics had been exaggerated in the past[1-3].

The dietary restriction of glucose, sucrose and lactose is based on the presumption that ingestion of glucose or a glucose-containing disaccharide results in a more rapid blood glucose response than does ingestion of the complex carbohydrate starch[4]. Sucrose has also been implicated in the development or exaggeration of hypertriglyceridemia[5]. However, many diabetics appear unwilling to do without sweet-tasting foods[6,7] and it has been reported that the fat restriction now recommended for both diabetics and non-diabetics, becomes increasingly difficult once sucrose has been removed from the diet[8].

The non-nutritive sweeteners, saccharin and cyclamate, are frequently suggested alternatives to sucrose. However, to 25-33 per cent of the population, saccharin has a bitter metallic after-taste and is therefore an unsatisfactory sweetener. In addition, there is a reported link between cancer and the consumption of saccharin and cyclamate, although this area remains

controversial. Cyclamate is still available for use in Australia, but it has been banned in many countries, notably the UK and the US[9].

The non-glucose nutritive sweeteners, fructose, xylitol and sorbitol are also marketed as being suitable for use by diabetics. However, these sucrose alternatives do not always match the sweetness quality, physical characteristics, price or digestibility of sucrose[10]. The dipeptide, aspartame, is useful as a table-top sweetener, but has limited stability in solution, especially in non-acid conditions, and it loses its sweetness at temperatures used for cooking[11].

Thus, although there is no specific nutritional need for sweetening agents, the availability of an acceptable sweetener may have important psychological and therapeutic implications.

Chemical structure and physical characteristics of sucrose

Sucrose is a disaccharide of glucose and fructose (α-D-Fructofuranosyl-β-D-glucopyranoside)[12]. It has been described as the optimum sweetner and it provides the standard by which the sweetness of other substances is judged. In addition to providing sweetness, sucrose gives substance and texture to baked foods, such as biscuits and cakes, it provides viscosity or 'mouth feel' to soft drinks[10] and it has a preservative function in jams[9]. Its popular use as a table-top sweetener is due both to its palatability and its easy solidification to a dry product. Sucrose provides 16 kJ of energy per gram[10].

Digestion, absorption and metabolism of sucrose

After ingestion, sucrose is very rapidly hydrolysed by the disaccharidase, sucrase, at the outer brush border of the small intestine. The products of sucrose hydrolysis are the monosaccharides, glucose and fructose, in equimolar amounts, although up to 20 per cent of the fructose may be converted to glucose before absorption through the intestinal wall[5,14,15].

While glucose is rapidly absorbed by an active transport mechanism, the absorption of fructose occurs by facilitated diffusion[5,14]. Fructose absorption occurs more slowly than that of glucose, but this difference is considered by some not to be physiologically significant[5]. The limiting factor to absorption is transport of the monosaccharides and not digestion by the disaccharidase[16].

In non-diabetics, when glucose is absorbed from the intestine, plasma insulin levels increase two to ten fold and there is an influx of glucose into the liver (which removes about 25 per cent of the glucose) and the insulin-sensitive tissues, fat and muscle. Within the liver cells, this glucose is either stored as glycogen or converted to fatty acids and triglycerides. In the peripheral tissues, the remainder is oxidized as fuel or stored as glycogen or fat. Thus, in non-diabetics, these clearance mechanisms for glucose result in the plasma glucose concentration remaining stable within a relatively narrow range, despite intermittent food intake[4,17]. In absolute or relative insulin deficiency, there is a reduced rate of glucose release from liver glycogen stores into the circulation and a reduced rate of glucose removal from the blood by the peripheral tissues. This altered metabolism results in hyperglycaemia[17].

In both diabetics and non-diabetics, nearly all of the fructose absorbed is taken up by the liver, where it is rapidly phosphorylated to fructose-1-phosphate by fructokinase, and then cleared into trioses by liver aldolase. These initial steps of metabolism are insulin-independent, but from this point on, insulin is needed for further metabolism of the trioses to occur. In well controlled diabetics and non-diabetics, the trioses produced from fructose are largely used for the synthesis of glycogen and triglycerides, with the consequence that there is only a modest increase in plasma glucose concentration. In contrast, in poorly controlled diabetics, glycogen synthesis is impaired and the fructose which is converted to glucose in the liver, is rapidly released, resulting in a considerable rise in plasma glucose concentration[15,17].

Dietary sources

The main dietary sources of sucrose in the Australian and other Western diets are those foods to which sucrose has been added during manufacture: sweetened breakfast cereals, cakes and biscuits, confectionery, soft drinks and flavoured dairy products. Naturally-occurring sucrose, which is present in some fruits and vegetables, generally contributes considerably less to total sucrose intake than does the sucrose added to manufactured foods[12,13].

Consumption trends

To date, there is no information on the sucrose intake of groups of individuals in Australia. Hence, estimates of sucrose consumption and of the contribution which sucrose makes to total energy intake have to be obtained directly from the Australian Bureau of Statistics Apparent Food Consumption Data[13].

In Australia, the difference between apparent and actual food intake is unknown. In the UK however, the difference between apparent and actual energy intake has been calculated to be as great as 34 per cent[18].

In Australia, the apparent per caput consumption of sucrose from 1938-39 to 1977-78 remained fairly constant at around 50 kg. From 1977-78 to 1982-83, there was a decrease in consumption of approximately 3 kg, from 49 to 46 kg. Thus 1982-83 consumption is equivalent to a mean daily sucrose consumption per person of 126 g and a contribution to total energy intake of 15 per cent. Between 1938-39 and 1977-78, a considerable shift from the consumption of sucrose as a refined sucrose to the consumption of sucrose-containing manufactured goods occurred. During the period 1977-78 to 1982-83, approximately 70 per cent of total sucrose consumption was from manufactured goods, while the remaining 30 per cent was added as refined sucrose during food preparation or by the individual to food consumed[18].

Other sweeteners in the Australian diet, grouped collectively as glucose, honey and syrups, represented 8.7 per cent of total sugar consumption in 1982-83. Although there are no specific data on corn sweetener (high fructose) syrup consumption in Australia, its contribution to total sugar consumption is minor[18].

Assessment of individual dietary sucrose intake

The sucrose content of many foods is not constant. For example, the sucrose content of a given fruit depends on a variety of factors which include the growth conditions, the time of harvesting and the storage environment up to the time of consumption. In canned fruits, although the initial syrup is predominantly sucrose, considerable inversion to fructose and glucose occurs in the can, so that by the time the fruit is eaten, the sucrose content may be significantly reduced. Similarly, the sucrose content of soft drinks will vary, depending on the acidity and on the time between production and consumption. Hence, values given in food composition tables can only be regarded as typical ones.

In the study of Type-2 diabetics (NIDD) conducted by the authors, individual 24-h sucrose intakes were estimated by using food composition data and simple sugar analyses (HPLC) of 24-h food collections. An approximation was made when the sucrose content of a required food item was unavailable from food composition data. A comparison of the results obtained by the two methods revealed marked differences (Table 1). In most cases, the values obtained by the HPLC method were considerably less than those obtained by food composition data. These results highlight the inadequacy of food composition data with respect to the estimation of dietary sucrose intake.

Table 1. Estimates of sucrose content of different 24-h food collections by use of food composition tables and HPLC.

Observation	*Sucrose content of 24-h food collection (g)*	
	Tables	*HPLC*
1	5.1	4.4
2	6.5	8.6
3	29.6	16.5
4	20.1	7.1
5	9.0	5.0
6	6.4	0.5
7	6.6	2.5
8	6.2	Tr
9	55.9	—
10	9.4	11.3
11	23.2	Tr
12	14.6	5.3
13	20.4	7.3
14	0.7	—

(Tr = trace; — none detected)

Recommended use of sweeteners in the diabetic diet

Glucose and glucose-containing disaccharides

The 1979 American Diabetes Association's (ADA) recommendations restated the importance of glucose, lactose and sucrose restrictions, although an actual level of restriction was not given[1]. In 1982, the Finnish Diabetes Association (FinDA) also recommended continued restriction of glucose,

lactose and sucrose, but concluded that a low concentration of these simple sugars (1-3 per cent) was not detrimental in otherwise acceptable foods[3]. In a revised policy statement for the 1980s, the British Diabetic Association (BDA) recommended an exclusion of all rapidly absorbed mono and disaccharides wherever possible, except in cases of illness or hypoglycaemic emergency[2].

More recently, the ADA further revised its dietary recommendations. The consumption of a modest amount of sucrose was considered to be acceptable, contingent upon the maintenance of metabolic control[19].

Non-glucose nutritive sweeteners

In 1979, the ADA reported that the amounts of these sweeteners which would be acceptable in the diabetic diet were uncertain and that there was insufficient evidence to accept or reject the use of these sweeteners by diabetics[1]. Others advised that sorbitol and fructose intakes should be limited to 30-40 g/d, providing their contribution to total energy intake was taken into account. The use of special sorbitol- and fructose-containing foods for diabetics was not recommended[2,3].

Non-nutritive sweeteners

The ADA's recommendations regarding the use of non-glucose nutritive sweeteners hold equally for the use of non-nutritive sweeteners. However, the revised nutrition policy of the BDA endorsed the recommendation taken by the Joint FAO/WHO Expert Committee on Food Additives of an Acceptable Daily Intake of saccharin of 2.5 mg/kg body weight (11-14 tablets/d)[2]. The FinDA considered that saccharin and cyclamate were suitable for sweetening of coffee, tea and soft drinks within the limits recommended by the WHO.

There is limited information on the effects of aspartame on diabetic control. On the basis of short-term studies, it appears to be a safe, low-energy sweetener for diabetics. However, studies investigating the metabolic effects of long-term use by diabetics are needed[12].

Dietary sweeteners, blood glucose and insulin

Acute studies

In 1920, Allen observed that pancreatectomized dogs showed greater glycosuria after glucose ingestion compared to starch digestion. On the basis of this finding, his review of the literature and his own clinical experience, Allen concluded that glucose caused greater glycosuria than did starch. It was assumed that glucose, when compared with the complex carbohydrate, starch, causes a more rapid rise of blood glucose concentration. This poorly based concept was subsequently expanded to include all simple sugars, including the common sugar, sucrose[4].

The belief that meats containing glucose or sucrose result in higher blood glucose concentrations than meats containing isoenergetic amounts of starch has persisted to the present time. Many of the studies which support the belief that simple sugars are more rapidly digested and absorbed than

complex carbohydrates have limitations. For instance, in some studies, the lower glycaemic response after starch ingestion can be explained by the use of uncooked starch[20]. Uncooked starch is not as readily hydrolysed and is therefore more slowly absorbed than cooked starch[21]. Cooked starch, for example, bread, rice and potato, cause glycaemic responses that are similar to, or only slightly less than the glycaemic response to glucose[20,22], and often have been reported to be the same or greater than those observed with sucrose[20,22,23]. Other studies have drawn conclusions from comparisons between test meals of unequal fat content[24] and between liquid test meals containing simple sugars and solid test meals containing starch[25].

Most of the early studies that compared the blood glucose response following the ingestion of simple sugars to that following the complex carbohydrate, starch, were performed in non-diabetics. More recently, comparisons have been made between starch and the different dietary sweeteners in both diabetics and non-diabetics.

Non-diabetics. For both simple sugar solutions and mixed meals, the ingestion of the non-glucose sweetners, fructose and sorbitol, results in significantly lower blood glucose and insulin responses than does the ingestion of sucrose or dextrose (glucose)[26,27]. In contrast, when sucrose and dextrose are compared with starch, the findings are less consistent, and often difficult to explain in terms of the known digestive and absorptive processes of each of the carbohydrates. However, it does appear that the isoenergetic substitution of starch by sucrose, at moderate intake levels and in meals composed of commonly eaten foods, has no significant effect on either blood glucose or plasma insulin responses in non-diabetics[28].

Non-insulin-dependent diabetics (NIDD). The differences in blood glucose and insulin responses between sucrose- and fructose-sweetened foods and meals, are smaller in NIDD than in non-diabetics[26-28]. As reported in studies of non-diabetics, the isoenergetic exchange of sucrose and starch, at moderate intake levels and in mixed meals composed of commonly eaten foods, has no significant effect on either blood glucose or plasma insulin levels in NIDD[7,28]. This is clearly shown in a recent study by the authors, where the isoenergetic exchange of sucrose and starch plus saccharin, at a level of 5 to 15 per cent of total energy intake, had no significant effect on either the blood glucose or plasma insulin responses to a test meal (Figs 1 and 2)[30].

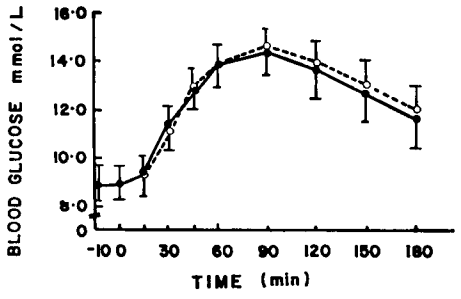

Fig. 1. Mean blood glucose responses to a test meal supplemented with sucrose (8% of total energy) (●——●) and saccharin (1 × 22.8 mg tablet) + starch (8% of total energy) (○- - - -○). Results are plotted as mean ± s.e.m. (n = 17).

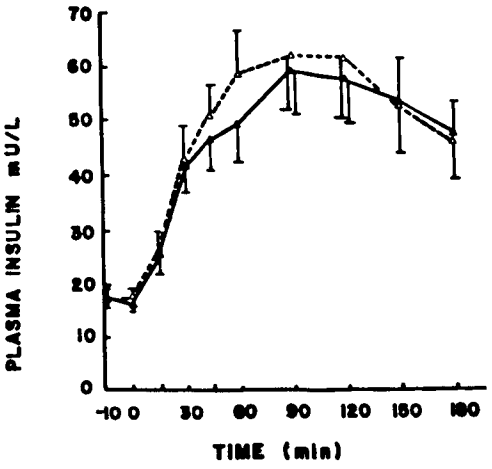

Fig. 2. Mean blood insulin responses to a test meal supplemented with sucrose (8% of total energy) (▲——▲) and saccharin (1 × 22.8 mg) + starch (8% of total energy) (△- - - -△). Results are plotted as mean ± s.e.m. (n = 16).

There is little evidence to support the use of sorbitol as a preferred sweetener to sucrose in the diet of NIDD[6].

Insulin-dependent diabetics (IDD). Evidence suggests that in IDD there are no significant differences in blood glucose response between sucrose-, fructose- and sorbitol-sweetened foods or meals[32]. Similarly, the isoenergetic exchange of sucrose and starch, at moderate intake levels, has no significant effect on blood glucose response in IDD[7,28,32]. Although one study has reported a smaller insulin requirement with xylitol ingestion than with sucrose ingestion, the use of xylitol as an alternative sweetener to sucrose has generally been curtailed[33].

Medium-term studies

There is conflicting but limited information on the reported medium-term effects of dietary sweeteners on parameters of glucose tolerance. These differences may be attributed to factors such as the percentage of sweetener in the diet, the duration of the diet, the composition of the background diet and individual genetic differences. Sucrose has been the most frequently studied sweetener.

Non-diabetics. In medium-term studies of non-diabetics, the isoenergetic replacement of dietary starch with sucrose, at a level of between 23 and 32 percent of total energy intake, has no effect of the blood glucose or insulin response to either a test meal or an oral glucose tolerance test[23,34,35]. Although one study has reported that the insulin response to a sucrose load (2 g/kg body weight) was always greater after a sucrose diet (30 per cent sucrose) than after a starch diet (30 per cent starch), these differences only reached significance at one hour post-prandially ($P < 0.005$)[24].

Diabetics. In a recent study of NIDD conducted by the authors, the isoenergetic exchange of sucrose and starch, over a six-week dietary period

and at a level of 5 to 15 percent of total energy intake had no effect on the blood glucose or plasma insulin responses to a sucrose or a saccharin-plus-starch-supplemented test meal[30]. Fasting blood glucose and plasma insulin levels were similarly unaffected by either dietary period (Table 2).

Table 2. Mean fasting blood glucose (BG) and plasma insulin (INS) at the commencement of the study (pre-study), after the sucrose diet (post-sucrose) and after the saccharin diet (post-saccharin). Data (mean values ± s.e.m.: n = 17) are from the study be Cooper[30].

Visit	*Fasting BG (mmol/l)*	*Fasting Insulin (mu/L)*
Pre-study	8.9 ± 0.7	14.4 ± 1.7
Post-sucrose	9.2 ± 0.7	16.5 ± 1.9
Post-saccharine	8.9 ± 0.8	17.3 ± 1.7

A literature search failed to reveal any other studies that have examined the medium-term metabolic effects of the isoenergetic exchange of dietary starch and sucrose in diabetics. However, the effects of dietary intake of added sucrose versus the use of sodium cyclamate were studied in a group of ten well-controlled IDD on continuous subcutaneous insulin infusion therapy. Neither mean daily blood glucose concentrations nor mean daily insulin requirements were altered by the addition of either sweetener. Similarly glycosylated haemoglobin, serum lipids and body weight remained unchanged and within normal ranges throughout the study.[56].

The effects of prolonged use of fructose were studied in a group of young IDD. There was no impairment of diabetic control when fructose was substituted for 20 per cent of total energy intake[36].

Dietary sweeteners and serum lipids

Most of the studies which have examined the effect of different dietary carbohydrates on serum lipids have been in non-diabetics. The most common comparison has been between sucrose and starch. The effect of fructose on serum lipids in both diabetics and non-diabetics has also been examined.

Serum cholesterol

After extensive reviews of the literature Grande and McGandy both concluded that the effect of different carbohydrates on fasting serum cholesterol was modest and of no practical significance[37,38]. Several recent studies also report that moderate sucrose and/or fructose intakes, relative to starch, do not elevate serum cholesterol levels[35,39]. Although these studies were all in non-diabetics, we observed a similar result in NIDD[30].

Serum triglycerides

Sucrose. The role of sucrose in the development of elevated fasting triglyceride levels has been extensively investigated. There is no evidence that sucrose causes greater fasting serum triglyceride levels, relative to starch, in normolipidemic men who are in energy balance. This holds true

particularly when the percentage of sucrose in the diet is comparable to the average sucrose intake of most Western populations (15 to 18 per cent of total energy intake)[23,34,39,40].

A number of studies have reported that sucrose causes higher fasting triglyceridemia[35,42-44]. However, the triglyceride-elevating effect of sucrose is influenced by a number of factors. These factors include the amount and nature of the accompanying fat (polyunsaturated or saturated) the age and sex of the subject, the duration of the study, the total energy intake and the initial triglyceride levels[44-47].

In the study conducted by the authors in NIDD, fasting serum triglyceride levels were not elevated by the isoenergetic exchanges of dietary starch with sucrose at a level of 5 to 15 per cent of total energy intake[30].

Fructose. Several studies have reported that moderate increases in fructose intake (15 to 20 per cent of total energy) do not significantly affect fasting serum triglyceride levels in either normal subjects[39,48], subjects with hypertriglyceridemia[49] or in diabetics[50].

Serum lipoproteins

Several studies have examined the effect of dietary sucrose on fasting serum lipoproteins in non-diabetics. Lui and colleagues reported that an increase in sucrose intake, from 9 to 15 per cent of total energy, had no significant effect on either fasting LDL or HDL cholesterol concentration[35]. Similarly, sucrose intakes of 11 to 16 per cent[39] and 30 per cent[51] of total energy intake have not been associated with any significant changes in fasting LDL or HDL cholesterol concentration. In NIDD, neither the LDL or HDL cholesterol concentration was affected by the isoenergetic exchange of starch and sucrose, at a level of 5 to 15 per cent of total energy intake[30].

In contrast, one study has reported a significant lowering of fasting HDL cholesterol concentration when dietary carbohydrate was increased from 40 to 60 per cent of total energy intake, while sucrose intake remained constant at 13 per cent of total energy[35].

Overall, however, these findings suggest that, in both diabetics and non-diabetics, moderate sucrose ingestion has no significant effect on fasting LDL or HDL cholesterol concentrations.

Dietary sweeteners and blood pressure

The effects of various dietary factors on sympathetic nervous system activity and blood pressure have been studied in both animals and humans. While numerous studies have investigated the effect of dietary sodium on blood pressure, the effects of sucrose and other dietary sweeteners are less extensively researched.

Substitution of starch by sucrose at moderate levels (10 to 20 per cent of total energy intake) has been reported to significantly elevate blood pressure in rats[52]. In addition, sucrose has been shown to augment the hypertensive effects of salt in experimental animals[53].

In humans, acute carbohydrate administration (glucose or sucrose) has been shown to increase sympathetic nervous system activity and to lead to evidence of cardiovascular stimulation[54]. It has also been reported that sucrose and glucose, when given in a sugar solution (1 g/kg body weight, 22 per cent w/v), elevated blood pressure transiently in humans who had been fasting. In contrast, neither fructose, galactose nor lactose solutions produced any significant changes in blood pressure. In the same study, solutions of glucose, fructose, sucrose and lactose all caused significantly greater salt retention than water alone[55].

It has been suggested that the sympathetic stimulation associated with acute carbohydrate administration may be mediated by plasma glucose and/or insulin[54].

In the medium-term study of NIDD conducted by the authors, neither blood pressure nor urinary Na^+/K^+ excretion were affected by the isoenergetic exchange of dietary starch with sucrose at a level of 5 to 15 per cent of total energy intake[30]. A literature search failed to reveal any other medium-term studies which examined the effecy of the isoenergetic exchange of starch and sucrose on blood pressure in humans.

Conclusion

Sucrose is the least studied component of the diabetic diet and contrary to popular belief, there is no evidence that modest use of added sucrose is detrimental to diabetic control. Although there is a wide range of both nutritive and non-nutritive sweeteners which are marketed as being suitable for diabetics, these sucrose alternatives do not always match the sweetness quality, physical characteristics, price or digestibility of sucrose. The results of the study of NIDD conducted by the authors indicate that there are no medium-term metabolic contraindications to using a moderate amount of sucrose (up to 28 g ≡ 7 teaspoons) as a sweetener in the diabetic diet. Further studies are required to determine if this conslusion can be extrapolated to the longer term.

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RECENT ADVANCES IN CLINICAL NUTRITION: 2

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