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Review Articles

Sweetness in the Diabetic Diet

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"Let the patient eat food easy of digestion such as veal, mutton and the like and abstain from all sorts of fruits and garden stuff"
Thomas Sydenham (1624–1689)

Alterations of the dietary content, particularly that of carbohydrate, in an attempt to treat diabetes have a long history and the advice contained in the quotation above was applied by Sydenham to patients suffering from that disease. Dietary therapy continues to play an important part in the management of diabetes mellitus, either alone or as an adjunct to insulin injection or treatment with other hypoglycaemic drugs. The therapeutic aims of the diabetic diet can be stated as follows: (a) to aid control by preventing rapid changes in blood glucose concentrations after carbohydrate ingestion, (b) to avoid hypoglycaemia in the Type 1 (insulin-dependent) diabetic patient; (c) to induce weight loss in the obese patient. To achieve these aims emphasis has been laid on restriction of the intake of certain monosaccharides and disaccharides, particularly glucose and sucrose, and on the timing of dietary carbohydrate intake in relation to insulin administration [1–3].

In recent years, the nature of the diabetic diet and its place in the therapy of diabetes mellitus have been the subject of scrutiny. For example, in 1979 the American Diabetes Association published a report prepared by their Food and Nutrition Committee on the dietary management of diabetic patients [4]. In the United Kingdom the Nutrition Sub-Committee of the British Diabetic Association's Medical Advisory Committee has reconsidered dietary treatment in relation to the British diabetic patient [5]. The purpose of this Editorial is to discuss the restriction of carbohydrate intake within the diabetic diet, with particular reference to the desire of the patient for 'sweetness' in the diet and the therapeutic alternatives to overall carbohydrate restriction.

In developed countries dietary carbohydrate restriction for the diabetic patient takes place within societies in which the average daily energy intake is at least 20% greater than that required [6]. The consumption of simple sugars, as distinct from complex polysaccharides, is also high. For example, in the United Kingdom about half of the daily energy intake is derived from carbohydrates, the average per capita intake of sucrose alone being at least 0.9 kg/week [7]. In the United Status sucrose and lactose consumption account for 20% of the total energy intake [8].

Thus 'sweetness' is an established property of the Western diet and there is evidence that if the intake of sucrose is restricted as part of diabetic treatment the majority of patients feel that an alternative sweetener is a necessity. In one study [9] a survey of 500 diabetics revealed that if no other sweetener were available in the diet, 83% of those questioned would use sucrose for this purpose. Another justification for sweetness has been the belief that the diet is more palatable if it contains carbohydrates which are sweet to the taste [10]. If these facts are representative of diabetic patients in general, and if we believe that good control of the blood sugar concentration cannot occur without dietary carbohydrate restriction, a number of therapeutic alternatives are available.

The first alternative is the use of nutritive sweeteners in the diabetic diet to satisfy the need for 'sweetness' without disturbing diabetic control. To be useful such a compound would have to be sweet to the human taste, easily assimilated and metabolized by man and have no deleterious effect upon diabetic control. In addition, it would have to be cheap to produce and have physicochemical properties such that it could be incorporated into various foods without degradation during cooking or storage.

The first of these, fructose, is a normal constituent of the Western diet, being found in the disaccharide sucrose and widely distributed as the free monosaccharide among fruits and vegetables and in honey. In the United Kingdom the diet provides 50–100 g of fructose/day [11]. It is sweeter than sucrose, easy to produce and can be used in cooking and canning without deterioration. After absorption from the intestine, the major metabolic fate of fructose is its metabolism to glucose and glycogen [12, 13]. The diabetic state does not alter the

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ability of man to metabolize fructose rapidly [14]; in the adult Type 1 diabetic patient the isocaloric replacement of dietary glucose by fructose in doses up to 90 g/day does not worsen diabetic control and in some cases may improve it [15, 16]. Others have shown that fructose can be utilized by diabetic children at a rate of 0.5 $g \cdot kg^{-1} \cdot day$ as an isocaloric carbohydrate substitute [17]. On this evidence fructose seems ideal but, as has been carefully argued in an Editorial in this journal [18], if it is administered in large quantities the pathway of fructose metabolism is such as to create metabolic disturbances which may not be advantageous to the diabetic patient. In particular little is known of the long term effects of oral administration upon lipid metabolism.

The second compound, sorbitol, is about half as sweet as sucrose and is absorbed from the gut and then metabolized to fructose. The total daily intake is limited to between 30 and 50 g as higher doses cause osmotic diarrhoea. As with fructose, there is some evidence that isocaloric sorbitol substitution in the diabetic diet does not alter diabetic control [19, 20]. However, it is metabolized to fructose and the arguments marshalled against that compound are thus applicable to sorbitol.

The third alternative is xylitol. This pentahydric alcohol is as sweet as sucrose and is widely distributed among vegetables and fruits (greengages contain about 1% of their dry weight as xylitol). It can be produced in quantity by the hydrolysis of xylans occurring in hardwoods and can be used in food manufacture without difficulty [21]. The oral intake of xylitol, and hence its usefulness as an isocaloric dietary carbohydrate substitute, is limited by the occurrence of osmotic diarrhoea. The amount tolerated depends on how it is administered. In a single dose of 30–40 g it causes diarrhoea [22], but when given in divided doses over 50 g can be ingested without adverse effect [23] and there is evidence that tolerance increases with its continued use [24]. The metabolic toxicity of intravenous xylitol in man is well documented but short term oral administration appears to be safe [24]. However, there is little information of its effect on diabetic control during long-term oral administration, and in animals chronic feeding of xylitol has been associated with the induction of tumours [25].

None of these nutritive sweeteners can be recommended without reservation and the most serious gap in our knowledge is the lack of information as to the long-term effects of their ingestion in large quantities by diabetic subjects.

If nutritive sweeteners are unsuitable, what are the other alternatives open to the doctor managing diabetic patients? One would be to re-educate diabetic patients to avoid sweetness in their diet. If the evidence cited above is correct and representative of diabetics in general, this may meet considerable resistance because of the pressures brought to bear on the patients by social factors. A second alternative is the use of non-nutritive sweeteners. In the United Kingdom saccharin is the only compound of this type which is available. The safety of this compound has been questioned because of experimental evidence showing that its administration in large amounts is associated with an increase in the incidence of bladder tumours in rats [26]. Diabetic subjects have a high saccharin intake, and recently a lowering of the acceptable daily intake of saccharin has been recommended. This now stands at 2.5 $mg \cdot kg^{-1} \cdot day^{-1}$. As the British Diabetic Association report points out [5], this is equivalent to between 11–14 saccharin tablets/day, but because of the difficulty in defining any particular level of intake as toxic, their recommendation is that no limitation of saccharin intake should be proposed. Other dietary manipulations that retain 'sweetness' and palatability by altering the relative proportions of different carbohydrates in the diet [27] or by adding complex carbohydrates and leguminous fibre to alter the pattern and extent of carbohydrate absorption from the gut lumen [28] have both been shown to succeed in terms of retaining good control of blood glucose concentrations.

However, is the shibboleth of carbohydrate restriction soundly based? In 1883 Bouchardat [29] noted a decreased prevalence of diabetes during the starvation which accompanied the seige of Paris during the Franco-Prussian War of 1870 and similar findings have been recorded during other conflicts in this century together with a lowered mortality from diabetes [30]. While a low total food intake cannot be the sole factor involved, this and other evidence suggests that the most important dietary factor increasing the risk for diabetes is the total energy intake, irrespective of its source, over and above that required. In addition to their high total daily energy intake, the citizens of the developed world have a total dietary protein intake 40% greater than that of people in under-developed countries where protein is scarce and expensive [6]. In such countries the diet may contain up to 80% of its energy content as carbohydrate, but in spite of this, good diabetic control can be achieved in Type 1 diabetic patients [31]. These observations and the possible mechanism involved in their production are discussed by the British Diabetic Association's Nutritional Sub-Committee in their document, 'Dietary Recommendations for Diabetics for the 1980's'. Their recommendations are comprehensive and in the context of this paper they stress the importance of total daily energy intake rather than the proportion of that intake derived from carbohydrate. The report emphasizes the importance of including complex carbohydrates (polysaccharides) in the diet and discouraging the use of the sweet mono- and disaccharides. The nutritive sweeteners discussed above are criticised, particularly their inclusion in diabetic speciality foods. The conclusions are clear: while it is possible to maintain good diabetic control on a high carbohydrate diet, that carbohydrate is best eaten in the form of complex polysaccharides and the 'sweetness' needed by some patients satisfied

through the use of non-nutritive sweeteners, such as saccharin.

A logical development from the emphasis on total energy intake in the diabetic diet would be to design a diet sheet based upon energy exchanges rather than carbohydrate exchanges. However, we would surmise that the use of such a diet may present difficulties, particularly in relation to the provision of adequate carbohydrate to prevent insulin-induced hypoglycaemia.

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