Assessment of sugar intake:
validity of the questionnaire method

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1. Assessment of sugar intake by our short questionnaire has been shown to agree well with its assessment by the conventional method in which subjects keep a 7-day diary of their diets. 2. People who have had a myocardial infarct, or are suffering from peripheral vascular disease, tend to reduce their intake of sugar. We believe this is the reason why investigators making dietary studies several months, or years, after infarction have not found the relatively high sugar consumption in patients with infarction that we had reported earlier. 3. The most suitable method for ascertaining dietary intake depends both on the constituent or constituents to be examined, and on the purpose of the investigation.

Several publications from this Department have drawn attention to the possible role of dietary sugar (sucrose) in the aetiology of ischaemic heart disease (IHD) (for summary, see Yudkin, 1963). In 1964, we presented evidence that men with myocardial infarction or peripheral vascular disease had been taking about twice as much sugar in their diet as men of the same age with no apparent disease of the arteries (Yudkin & Roddy, 1964).

Our publications have met with a variety of criticisms. One is on the grounds of the implausibility of the hypothesis that sugar can cause disease, in that it is presumably treated by the body in the same way as any other carbohydrate. However, we have recently summarized evidence from this Department and from others that shows considerable metabolic differences between the two major dietary carbohydrates starch and sugar (Al-Nagdy, Miller, Qureshi & Yudkin, 1966).

A second criticism of our work is that our method of assessing dietary sugar, by a short questionnaire, may not be sufficiently accurate (Marr & Heady, 1964). We have explained the reasoning that led us to develop this method, and why we believe it is adequate for our purpose (Yudkin, 1964). We have now compared our method with a more conventional method, and we present the results here.

A third criticism is that we did not take precautions against subjective bias when we took the dietary history with our questionnaire. We are now completing a further study in which we have avoided this possible source of error. This study confirms the original findings, and the results are to be published shortly.

A fourth suggested reason for scepticism about our results is that other workers have not been able to confirm that patients with IHD have been taking more sugar than persons without the disease (Little, Shanoff, Csima, Redmond & Yano, 1965; Papp, Padilla & Johnson, 1965). We have suggested that the reason for this apparent contradiction is that after an infarct patients are likely to reduce their sugar intake in an attempt to lose weight (Yudkin, 1965). In this paper we adduce evidence in support of this idea.

EXPERIMENTAL AND RESULTS

Validation of method

We tested the validity of our questionnaire by comparing the results with those obtained by a 7-day diary method. The subjects were twenty-three patients with peripheral arterial disease, attending the clinic at St Mary's Hospital, London; five of the patients were included in the group investigated in our first study. The subjects were interviewed with our questionnaire, which asked a few general questions about diet, but many more specific questions related to sugar and items that contain sugar. This interview took from 15 to 20 min. A few days later, they were given a diary in which to enter all the food and drink consumed over a period of 7 days. The diary consisted of forms for each day of the week, and the subjects were instructed how to record every item immediately after each meal or snack or drink. The items were recorded in simple household measures: heaped or flat teaspoonfuls of sugar or lumps of sugar, number of thin or medium or thick slices of bread, number and variety of biscuits or pieces of confectionery. When the forms were returned, we used the values in the food tables of McCance & Widdowson (1960), or information from food manufacturers, to calculate the sugar intake, and compared these findings with those from our short questionnaire.

There is good correlation between the results of the two methods. The average daily intake of sugar assessed by our questionnaire was 71 g, and by the 7-day diary 69 g; the correlation coefficient was 0·75. As we had predicted, people tend to have a fairly regular pattern of taking sugar or sugar-containing foods, so that the intake assessed by questionnaire correlates equally well with the diary record for 3 days of the week (Saturday, Sunday and Thursday) as for the whole 7 days. For the 3 days, the average daily intake of sugar was 68 g, and gave a correlation coefficient with the questionnaire of 0·79. The correlation coefficient of the 3-day record and the 7-day record was 0·92. These correlations are better than those achieved for many individual nutrients assessed by the 7-day diary method during two different periods, even when the foods consumed are weighed (Yudkin, 1951).

Change in sugar intake

As we have said, we believe that people ordinarily tend to take a fairly constant amount of sugar in their diets. But in our experience sugar is one of the first items in the diet that is restricted by people wishing to lose weight. One of us (Yudkin, 1965) has suggested that this is why Little and his collaborators (Little *et al.* 1965) had found that their patients were not taking more sugar than the controls when seen several months or years after an infarct.

In order to test this suggestion, we re-investigated as many of the subjects as we could some 18 months after the investigation (Yudkin & Roddy, 1964) in which we had discovered the high intake of sugar in the patients with arterial disease. At the time of the first investigation, we determined normal home sugar intake within 3 weeks of the first known attack of IHD, or a short time after the diagnosis of peripheral arterial disease.

Several of the patients had died, or had moved, or did not respond to our request for further investigation. As a result, our studies were made on eleven of the twenty original patients with IHD, nine of the twenty-five with peripheral arterial disease, and seventeen of the twenty-five control subjects. In our calculations, we omitted one of the patients of the second group, who had developed overt diabetes since our first investigation and was put on a low-carbohydrate diet by his doctor. In spite of the very small numbers, it is clear that both groups of patients with arterial disease had significantly reduced their sugar intake, whereas the control subjects showed no significant change (Table 1).

Table 1. *Daily sugar intake (g) measured by questionnaire soon after admission to hospital* (S_1) *and 18 months later* (S_2) *of patients with myocardial infarction* (MI) *or peripheral arterial disease* (PA) *and of controls* (C)

Group	No. of subjects	Sugar intake		P (t test)	No. taking		P (sign test)
		S_1	S_2		More sugar	Less sugar	
MI	11	122	86	< 0·05	2	9	< 0·05
PA	8	104	67	< 0·01	2	6	NS
C	17	94	114	NS	10	7	NS

NS, not significant.

DISCUSSION

The method that is most suitable for the assessment of dietary intake depends both on the item(s) being measured, and on the purpose for which the measurement is required.

The total intake of a dietary component is the product of the amounts of foods containing it that are consumed, and the proportion of the component in those foods. For most components of the diet, it is difficult to obtain the former measurement, and impossible to obtain the latter. Let us suppose that one wishes to assess fat intake. Fat is found in almost all of the foods we eat: in bread as well as in butter, in lean meat and in eggs as well as in bacon and in cheese. The only way to be certain of the amounts one eats of these foods is to weigh them, since one man's small piece of bacon is another man's medium piece. One now needs to know the amount of fat in an average piece of bacon, and this one can get by referring to food tables. Even better, one can actually analyse a 'matched' piece of bacon in the laboratory, though this is in most circumstances quite impracticable. But it is clearly impossible to know the proportion of fat in the particular piece of bacon that a man is eating or has just eaten; it cannot be both analysed and eaten.

For sugar, however, the situation is far better in regard to both the quantities of the relevant foods eaten, and the proportion of sugar they contain. Much of the sugar is taken in lumps, or spoonfuls, so that the subject has a reasonable idea of the amount he takes in a cup of tea or on his breakfast cereal. Most of the rest is taken in ice-cream, confectionery, biscuits or drinks that are consumed in discrete and ascertainable amounts. In addition, most people have a quite regular pattern of consumption of sugar and the foods containing it. They usually take the same number of cups of tea

or coffee at meals or between meals, and they usually put in the same amount of sugar. They also have similar items at their meals, ending for example with the same sorts of puddings, or cheese, or fruit. For all these reasons, it is possible to make a reasonable assessment by questionnaire of the total amount of sugar, and of the foods and drinks containing it, that are consumed in a typical day.

Clearly, this method does not give such precise measurements of amounts of food as those given by weighing. But this lack of precision in assessing amounts of food is compensated by the fact that the proportion of sugar in the items containing it is known much more precisely than, say, the amount of fat in meat or cheese or bacon, or the amount of protein, or any of the vitamins. As we have seen, a great deal of sugar is eaten as such, and there is no other item of diet—unless one is drinking something like maize oil—that consists entirely of the constituent one wishes to measure. In addition, very many foods such as biscuits and confectionery contain a fixed proportion of sugar, and this proportion is known or can readily be discovered from the manufacturer.

Thus, because one can ascertain the amounts of sugar and sugar-containing foods and drinks fairly well, and the proportion of sugar in them even better, it is not surprising that we get such good accord between the results from our rapid questionnaire, and those from the much more laborious 7-day diary. It is also true that, as has so frequently been demonstrated, there could be no such precision in the assessment of other dietary constituents by a questionnaire of this sort, or by an interview designed to elicit the recall of all the food consumed over the previous 7 days as has been attempted by Papp *et al.* (1965). For some constituents, notably vitamins A, C and D, even weighed records of food intake over 7 days can give very varying results (Yudkin, 1951).

It is not surprising that Little *et al.* (1965) did not find the same high sugar intake in patients as we had found. Their patients were chosen from the hospital files, and had been discharged from hospital at least 3 months previously. Those of Papp and his collaborators (Papp *et al.* 1965) had had their infarct less than 6 months before the dietary study, but we are not told anything more about the interval between the attack and the assessment of diet. In neither investigation is it clear whether the infarct referred to was the first attack or a recurrence. More particularly, it appears that in both studies the diets assessed were those then being consumed by the patients; in our study, we had assessed the diets as soon as possible after diagnosis of the first known attack, and our questions were specifically concerned with the patient's normal diet at home before the illness. The results of the two groups of Canadian workers could have been taken as genuinely contradicting our own only if they had shown that patients do not reduce their sugar consumption after one or more myocardial infarcts. Our own findings now show that the patients do just this.

The intake of sugar calculated from the information collected either by questionnaire or by the diary method is somewhat lower than that expected from the known average sugar consumption in Britain. One possible reason for this discrepancy is that manufacturers put sugar in increasing amounts in some of their preparations such as soups and tomato juice, and this is not always shown in the food tables. In general, too, it is

known that intake measured by dietary history tends to underestimate food consumption (cf. Paul, Lepper, Phelan, Dupertuis, MacMillan, McKean & Park, 1963).

For our purposes, it is not important that the assessment of sugar intake should be accurate in absolute terms, but rather that it should be able to distinguish with reasonable precision between different levels of intake. It is then legitimate to conclude, as we did in our first survey, that one group of individuals is taking significantly less sugar than another group. It would not matter whether the intake as measured were as little as one-half of the real intake, provided that persons eating different amounts of sugar were found to be graded in the correct order. This conclusion can clearly be drawn from our method, which gives a good correlation with the generally accepted method of a 7-day diary record.

On the other hand, it would be important to assess absolute amounts of a dietary constituent if one were attempting to assess dietary adequacy, that is, nutrient intake in relation to nutritional requirement. It would matter if the intake of thiamine as measured were 0·75 mg a day instead of a real intake of 1·5 mg, if we were then to conclude that this is evidence of thiamine deficiency since the requirement is 1·5 mg.

We feel it necessary to reiterate that we consider that a high intake of sugar is only one of the factors causing IHD. Thus, though people who develop IHD have on average been eating appreciably more sugar than people who do not develop the disease, they exhibit a considerable range of sugar intake from quite low to very high, as we have shown. The fact that someone eating little sugar nevertheless has an attack of myocardial infarction must then be due to one or more of the other causative factors. There is no reason why such a person should be any less likely to develop a second infarct than someone in whom the main factor was a high sugar intake. The totality of causative factors that was responsible for his first attack, that is, are just as likely to result in a second attack in someone who eats little sugar as in someone who eats a great amount. It is understandable therefore that Ball *et al.* (1965) have found no difference in the average amount of sugar eaten by those who had a relapse and those who did not. This finding has no bearing on the question of the role of sugar in causing myocardial infarction, or on the question as to whether a reduction in sugar intake would reduce the chances of a first infarct or the chances of a recurrence.

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