Report of the COMA panel on dietary sugars and human disease: discussion paper

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The recently published Report on dietary sugars has dismissed as unfounded the belief that sugar (sucrose) may be a cause of obesity, diabetes mellitus, or coronary heart disease[1]. A careful perusal of the published work on dietary sucrose shows that the Report's conclusions are unwarranted.

The following comments relate to those items in the Report that I believe are the most flawed.

Intake of sugars in the UK

By far the largest contributor to sugar intake is sucrose. In common with most other western countries, the average daily UK consumption is around 120 g, which includes the mixture of glucose and fructose used mostly in the food industry as a partial replacement of sucrose. This 120 g may be compared with the 7 or 8 g taken in England in the middle of the 18th century.

The Report quotes several studies in which sugar intake was measured. There is only one set of figures that refers to sizeable groups of adult subjects, giving a mean daily intake of 107 g of sucrose for 105 men and 62 g for 112 non-pregnant and non-nursing women[2,3]. The Report quotes these figures as evidence that the quantities used in experiments with volunteers are unrealistically high.

The figures of consumption quoted in the Report give not only the means but also the standard deviations; 107 (SD 58) and 62 (SD 38). If the distribution of sugar consumption were normal, one would have to say that 2½% of men eat less than minus 9 g (107−116) of sugar a day, and 2½% eat more than 223 g (107+116). This is of course a clear demonstration of the fact that the distribution is not normal, but skewed to the right. A similar calculation can be made for the women. It has to be concluded that the daily intake of over 2.5% of men is more than 223 g, and of over 2.5% of women more than 138 g. The quantities of sucrose used in many of the experiments were therefore not outrageously large.

Epidemiology

The Report points out that not all countries with a high sugar intake have a high prevalence of diabetes or cardiovascular disease. Similarly, they say that not all studies have found a relationship in individuals between their sugar consumption and the risk of developing CHD or diabetes.

But it would be expected that comparisons of populations or individuals would not always demonstrate a relationship between sugar and the occurrence of disease unless sugar were the sole or major cause, as occurs between cigarette smoking and lung cancer. This has never been claimed for dietary sugar as a possible cause of CHD or diabetes.

It is universally recognized that, apart from genetic factors, the risk of developing CHD is affected by several environmental factors, including cigarette smoking, lack of exercise and obesity. Even all these risk factors, as well as dietary fat, still do not explain the actual risk of people developing CHD[4].

Sucrose metabolism

Shafrir[5] has reviewed the effects of dietary sucrose in an article with 431 references, nearly 100 of which are in the section entitled 'Deleterious effects of sucrose diets'. The 237 references in the Report do not include a reference to Shafrir's review.

Experimental evidence

'The Panel considered that recommendations regarding sugars should be based on evidence derived from studies in humans'. If this attitude had guided all investigations into human disease, we would have remained ignorant of almost all that we know about nutrition. Eijkman's work with chickens and Hopkins' work with rats led to the discovery of the vitamins, for which they were jointly awarded the Nobel Prize in medicine in 1928.

Obesity

It has not been suggested that it is the metabolism of sucrose that is involved in producing obesity. Its role is the simpler one of its use in producing, for many people, highly attractive foods and drinks that are not only consumed to satisfy hunger and thirst but, because of their high palatability, are consumed also when both hunger and thirst have been satisfied.

The Panel says that '. . . the available evidence is insufficient to establish a link between sugar intake and development of obesity'. Nevertheless, the Report later implies that sugar *can* cause obesity by saying that the risk of developing diabetes, or some cancers, may be increased by obesity caused by dietary sugar.

Diabetes mellitus

Apart from hyperglycaemia and increased glycaemic response, patients with non-insulin-dependent diabetes mellitus (NIDDM) show an increase in blood lipids, uric acid and insulin, a decrease in insulin sensitivity and changes in platelet behaviour. All of these abnormalities are produced by dietary sucrose in about 15% or 20% of apparently healthy people[6].

The Report says that such changes occur 'in short term experiments in which sugars have been given in very large quantities and/or unusual food mixes or formula diets'. In our experiments, quite normal meals were taken but with sugar replacing an equivalent amount of starch. Our original findings were confirmed by Reiser and his colleagues[7,8] from

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the Human Nutrition Laboratory of the US Department of Agriculture.

The quantities of sugar sometimes used by both the American group and ourselves were certainly high, but as we saw these quantities were not beyond those taken by a significant proportion of the population. At a low estimate, the proportion of people who are 'sucrose sensitive' is of the order of 15% of the population. This can hardly be ignored: it amounts to about 8 million people in the UK and about 40 million in the US.

But for some extraordinary reason, the Report omits to say that Reiser's group[8] studied the effect not only of a high sucrose diet but also of a low sucrose diet. One diet provided 18% sucrose calories, about the USA and UK average. A second provided 33% sucrose calories, which produced the increase in insulin and glycaemic response mentioned in the Report. But the third, with 5% sucrose calories, and not mentioned in the Report, produced a decrease in insulin and glycaemic response. As Reiser and his colleagues point out, their results confirmed that an intake of sucrose higher than the average in the US or UK produces in a large number of people abnormalities associated with diabetes. But they also point out that these 'risk factors' are reduced when the intake of sucrose is lower than the average intake. It is difficult to understand why the Panel omitted any reference to these findings.

Coronary heart disease

The Report concluded that 'current consumption of sugars, particularly sucrose, played no direct causal role in the development of cardiovascular disease'. It also quotes the US Food and Drug Administration report on sugars as saying, 'There is no conclusive evidence that dietary sugars are an independent factor for CHD in the general population'. Many people would add that there is no conclusive evidence that dietary fat is an independent risk factor for CHD.

If, however, we extend our horizon beyond a rise in the concentration of cholesterol, we find a similar association between CHD risk and several other biochemical abnormalities. These include a raised blood concentration not only of insulin but also of glucose, uric acid and oestrogen, a decrease in glucose tolerance and insulin sensitivity, and abnormalities in platelet behaviour. These changes can be caused by dietary sucrose: some in all subjects and some in a substantial proportion of the subjects.

This multiplicity of abnormalities, both in diabetes and in CHD, as well as the clinical link between these two conditions and also with gout and peptic ulceration, suggests that the underlying mechanism in the production of these diseases is disturbances in the balance of hormones, perhaps of insulin in particular[9-11].

Duration of experiments

The Panel's criticism of some of the research on the effects of sucrose in human subjects is that they are mostly of short duration; conditions such as NIDDM and CHD on the other hand are likely to be produced only by prolonged exposure to causative factors. There are however three problems that militate against conducting long-term dietary experiments with human subjects. Firstly, it is difficult to persuade people to record precisely every item of food they eat over a long period. Secondly, the abormalities induced by an increase in dietary sucrose occur within 2 or 3 weeks in human subjects. They also occur in short-term and in long-term studies in laboratory animals, often intensifying over time.

Thirdly, there is the ethical problem. Longstanding diabetes often produces nephropathy and retinopathy. Obviously, no one has attempted to produce these abnormalities in human subjects with high sugar diets; they are however produced in laboratory animals with exactly similar histopathology. In short-term experiments in human subjects, increased dietary sucrose produced an increase in the urinary excretion of N-acetylglucosaminidase, an early sign of renal damage[12]. This is a persuasive argument against the continuation of such experiments in human subjects over a long period.

The Report concludes, '... The current consumption of sugars, particularly sucrose, played no direct causal role in the development of cardiovascular disease, or of diabetes'. It is difficult to accept the validity of this conclusion when the evidence presented is derived from the perusal of a small and selected part of the extensive published research.

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