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SUGAR INTAKE, OBESITY, AND CORONARY HEART DISEASE

The precise bearing of level of sugar intake on health and disease is a subject upon which there is much difference of opinion. Of course there is no dispute that, as a vehicle of calories, excessive sugar intake promotes overweight and increases the prevalence of many associated diseases. The same opprobrium attaches to excessive consumption of other vehicles of calories, such as carbohydrate staples and foods high in fat concentration. But in addition to its role as a supplier of calories, there is a belief by some that sugar has a specific noxiousness, particularly in respect of obesity, diabetes, and coronary heart disease. On the whole subject there is an enormous volume of literature. It is proposed to illustrate the inadequacy of evidence and confusion of thought prevailing by referring to a few recent contributions, and then to indicate the types of evidence required to establish the specific incrimination of a particular food or food component, such as sugar.

In a recent letter to *The Lancet*, Ziegler[1] has pointed out that in Britain and on the Continent the increase in sugar consumption (the 'sugar climate') since 1860 almost exactly parallels the increases that have occurred in secular height and weight of adult populations. If this correlation is valid generally, then sophisticated populations with the greatest recent gains in weight would be expected to have proportionally the highest sugar intakes. Is this so? In the USA, the average male and female are 19 lb. and 15 lb. heavier, respectively, than their British counterparts;[2,3] yet the annual consumption of sugar *per capita* is less in the USA, namely 90 lb. compared with 115 lb. in Britain.[4] There are obvious limitations to this type of comparison. In the last resort, however, incrimination will be forthcoming only from studying the intake and response in the individual. In a paper just published, Howell and Wilson[5] have reported their investigations on 1,328 men in Britain; no correlation could be detected between total sugar intake and weight gain after the age of 25 years.

Turning to coronary heart disease, there is a body of opinion, of whom the chief protagonist is Yudkin,[6] which regards a high intake of sugar as the chief cause of the disease. In respect of total populations, however, anomalies are immediately encountered. Thus, coronary heart disease is a far more severe menace in the USA compared with Switzerland, although the intake of sugar is the same in each country; this also applies to Germany and France.[6,7] With respect to individuals, Yudkin found a twofold greater intake of sugar in patients who had suffered from a myocardial infarction, compared with control groups.[6] Yet this difference was not observed in similar investigations undertaken by other workers.[5,8-10] In the study by Howell and Wilson[5] just mentioned, no relationship could be traced between the total sugar intake of the subjects (aged 43 - 57 years) and the presence or absence of ischaemic heart disease. In the investigation of Burns-Cox *et al.*, 80 men under 65 years recovering from myocardial infarction were compared with 160 men as controls, matched for age and country of birth; no significant difference in habitual sugar intake could be detected.[10]

Other workers assert that prevalence of coronary heart disease is determined primarily by the level of combined intakes of sugar and of refined cereal products.[11] Cleave *et al.*[11] maintain: 'We ourselves are confident that, if the refined carbohydrates were avoided by reducing the diet to the practical evolutionary level we have set out, the incidence of coronary heart disease would in due course be reduced to almost nil . . .' Yet a major coronary heart disease prevalence study recently published on an urban population in India[12] has yielded results which are at variance with this view. At Chandigarh (100,000 persons), North India, the monthly income of the majority was less than R12; moreover, the high cost of living was emphasized. From other sources, it is gathered that the principal foodstuff is 'unrefined cereals'.[4] The sugar intake has been given as 1 oz. *per diem*,[4] a quarter of that in Britain. Consumption of fat by the Indian masses is low—of the order of 1 oz. *per diem*,[4] providing 10 - 15% of the daily calories (a third of the proportion obtaining in the USA). There is no reason to believe that at Chandigarh the fat intake of the relatively poor people studied was significantly higher than that of the general Indian population. Yet the prevalence of coronary heart disease was revealed to be high, the same as that found in the USA town of Tecumseh.[13] The same electrocardiographic and clinical criteria were used in both studies. The prevalence of coronary heart disease at Chandigarh is certainly far higher than what would be expected on the basis of any of the dietary hypotheses. A further interesting point is that the frequency of cigarette smoking was only slight in the men, and absent in the women. Additionally it is noteworthy that the risk factors (overweight, hypertension, inactivity) that have been elucidated for coronary heart disease in western contexts also operate at Chandigarh. As to activity, for example, no Indian male performing heavy manual work had evidence of coronary heart disease. This fact, by itself, is irreconcilable with a wholly dietary hypothesis. Furthermore, it cannot be excluded that, apart from the role of the environmental factors cited, there may be an ethnic proneness in Indians to coronary heart disease.[14]

To demonstrate unequivocally that a particular nutrient or food—in this case sugar—when consumed to excess is specifically noxious presents many difficulties. The first requirement for incrimination is a good knowledge of the intake of sugar in different groups of the population—male and female, young and old, rich and poor, town and country, etc. Information based simply on the total amount of sugar consumed by humans has little value. The second necessity is a fairly accurate knowledge of the prevalence of the relevant stigmata (e.g. obesity, diabetes, coronary heart disease) in the population groups enumerated. Data gleaned solely from clinic and hospital studies are inadequate. The third need is proof that small population groups within a given context who habitually consume little or much sugar are indeed relatively free, or suffer greatly, from the relevant stigmata. It must be

stressed that proof must be sought from segments of populations within the same context, whether primitive or sophisticated; the direct extrapolation of lessons to be learnt from developing to sophisticated populations has considerable limitations.[15] How may this third requirement be met?

It would be necessary to show, for example, that strict vegetarians in a western context, such as 'vegans' (with their enormous emphasis on unrefined and 'natural' foods), suffer far less than the general population from obesity, diabetes, and coronary heart disease. Conversely, it would have to be shown that a population such as South African Jews, with their very high mortality from coronary heart disease,[16] consumes far more sugar and refined cereal products than does the general population. On neither of these or similar populations is the required definitive information available; indeed, it is disquieting that no serious attempt to obtain such data has been made by the relevant protagonists.

The fourth requirement is that in individuals there are significant differences in habitual consumption of sugar, in persons with and without the relevant stigmata. The contradictory evidence in relation to sugar intake and coronary heart disease in the individual has been mentioned earlier. A corollary of this requirement is the need to demonstrate that when recommended changes in diet are made by affected persons, there is a detectable amelioration of stigmata, provided (*i*) dietary alterations are strictly adhered to, (*ii*) persons are not too old, and (*iii*) the stigmata are not too far advanced.

With respect, therefore, to the bearing of level of sugar intake on health and disease pattern, it is clear that—apart from the obvious role of sugar as a ready and palatable supplier of calories—present knowledge is insufficient to allow firm conclusions on other roles that have been accorded to this food. As indicated, precise information on level of intake and on prevalence of relevant stigmata in different segments of populations is meagre, particularly in the moieties prone and non-prone to the stigmata. Unfortunately, the type of the investigations required, whether retrospective, prospective, or merely cross-sectional, is not popular. It is far easier to speculate from inadequate information than it is to pursue laborious field studies, especially when they are of the type that do not lend themselves to instant answers.

A.R.P.W.

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