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SUCROSE IN THE DIET AND CORONARY HEART DISEASE

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SUMMARY

The widely publicized theory that sucrose in the diet is a major factor in the development of coronary heart disease has been examined. The theory is not supported by acceptable clinical, epidemiological, theoretical or experimental evidence.

It has been claimed that the theory is supported by international statistics, by the time trend of the incidence rate, by comparison of dietary habits of coronary patients and "controls," and by experiments. Not one of these claims is justified by the actual evidence.

Key words: *Coronary epidemiology – Dietary sugar – Patient habits*

INTRODUCTION

The claim that sucrose in the diet is a major cause of coronary heart disease (CHD) has been so much publicized that it must be difficult for the nonspecialist to escape belief that "there must be something to it". When the Professor of Nutrition of the University of London writes, "There is now quite strong evidence that dietary sugar—sucrose—is an important factor in the etiology of coronary heart disease"[1], it is natural to think there really is substantial reason to incriminate sugar in the coronary "epidemic". Unchallenged repetitions of claims of this nature naturally must impress the uncritical reader.

Most students of the etiology of CHD have recognized that the arguments for a role of sucrose in CHD are tendentious and the evidence flimsy indeed: they tend to ignore the occasional reiterations in the popular press. But such neglect leaves the way open for exploitation. A brief review seems overdue.

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YUDKIN based his indictment of sucrose in the diet on four arguments. First, he said that there are big differences among countries in mortality attributed to CHD and these differences are closely correlated with differences in the per capita consumption of sugar in those countries. Second, he said that a great increase in the consumption of sucrose has occurred over time and that this change has been reflected in an enormous increase in the incidence of CHD. Third, he said that men afflicted with CHD are characterized by an unusually high use of sugar in the diet. Finally, he offered possible explanations in terms of mechanism of the way in which dietary sucrose could wreak its havoc. Let us examine each of these claims.

YUDKIN first proposed a causal role of dietary sucrose in the etiology of CHD in an article in which he stated that comparisons of official vital statistics from fifteen countries showed a close correlation between the death rate ascribed to CHD and the amount of sugar in the national diet as estimated from the agricultural statistics[2]. No actual dietary data were offered — the figures on sugar simply concerned the disappearance of sugar, taking account of local production, import, and export, divided by the number of people in the population. And no correlations were calculated; the reader was asked to accept the author's conclusions as supported by the visual evidence of scatter diagrams. The countries selected for the comparison did not include those with very high sugar consumption, such as Cuba, Colombia and Venezuela, which also happen to have a very low incidence of CHD.

YUDKIN assumed that his fifteen selected countries were comparable in the quality of medical diagnoses and in criteria in the early 1930s to which his data pertained. While that assumption is patently unwarranted for some of those countries, a few of them would seem to be strictly comparable, notably the Scandinavian countries with their common tradition of very high medical standards, similar systems of health care and death reporting, and frequent communication and exchange among their physicians. Yet it is in Scandinavia that a fatal flaw to the sugar story appears. The per capita consumption of sugar was, and still is, much greater in Sweden than in next-door Finland, but the age-specific CHD death rate in Sweden is not much more than half that in Finland.

The association between sucrose disappearance and CHD mortality in YUDKIN's data are readily explained by association between sucrose and animal fats or saturated fatty acids in the national diets. YUDKIN had frequently made statements such as, "the prevalence of this disease is more closely associated with the level of sugar consumption than with the level of *any other* dietary component"[1]. Since he was at pains to put "any other" in italics, the trusting reader may accept the statement at face value. In fact, the "any other" claim is not nor was it ever justifiable, even with YUDKIN's own data. The most that could be said about that selected material is that rate of death attributed to CHD seemed to be similarly associated with the national disappearance rates of sugar and of fat.

YUDKIN has never paid attention to the critical differences in effects of different fats and fatty acids. Yet the necessity of distinguishing between fatty acids in the diet has been clear since 1957[3,4]. Other analyses of official statistics on CHD mortality

and national diets, far more extensive and sophisticated than YUDKIN's, have produced a very different picture[5–8].

But comparisons based only on national vital statistics and estimates by agriculturalists of food disappearance can never be more than suggestive, a possible source of clues to be followed up in more critical studies. The obvious need is for data on actual food consumption by persons in defined samples and on the incidence of CHD as judged by international teams following and periodically examining those samples in the countries of interest. So far, only one large-scale study of this kind has been attempted; it concerns middle-aged men in seven countries whose entry characteristics and 5-year CHD experience have been reported[9,10]. Detailed dietary studies have been made on random sub-samples of the cohorts; they included repeated 7-day food weighings in different seasons, with chemical analysis of replicate meals eaten by the men under study[11,12].

Figs. 1, 2, and 3 summarize this study in regard to the incidence of CHD and the amount of sugar and saturated fatty acids in the diet. The age-specific incidence of CHD among men judged to be CHD-free at entry proved to be significantly related to the average percentage of calories supplied by sucrose in the diet; the correlation coefficient was $r = 0.78$. But CHD incidence proved to be even more highly correlated with the percentage of dietary calories supplied by saturated fatty acids, with $r = 0.86$. The correlation between sucrose and saturated fatty acids in the diets, $r = 0.84$, is adequate to explain the observed relationship between sucrose and CHD without recourse to the idea that sucrose was somehow involved in the etiology of CHD. Figs. 1, 2, and 3 include data on the Japanese men of Tanushimaru but if the analysis is limited to the ten occidental cohorts the findings are essentially the same: for sucrose *vs.* CHD, $r = 0.75$; for saturated fatty acids *vs.* CHD, $r = 0.84$; for sucrose *vs.* saturated fatty acids, $r = 0.88$.

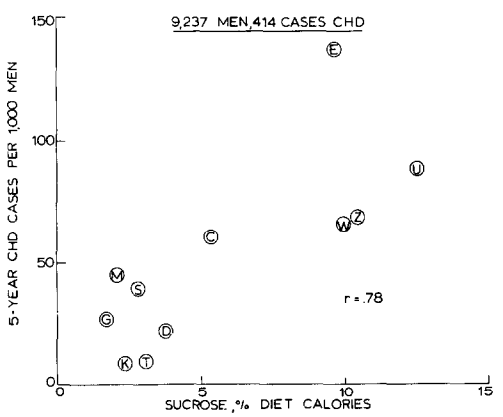

Fig. 1. Correlation between 5-year incidence rate of CHD, men aged 40–59 and CHD-free at outset, and average percentage of calories supplied by sucrose in the diet. C = Crevalcore (Italy), D = Dalmatia (Yugoslavia), E = East Finland, G = Corfu (Greece), K = Crete, M = Montegiorgio (Italy), S = Slavonia (Yugoslavia), T = Tanushimaru (Japan), U = U.S. railroad men, W = West Finland, Z = Zutphen (Netherlands).

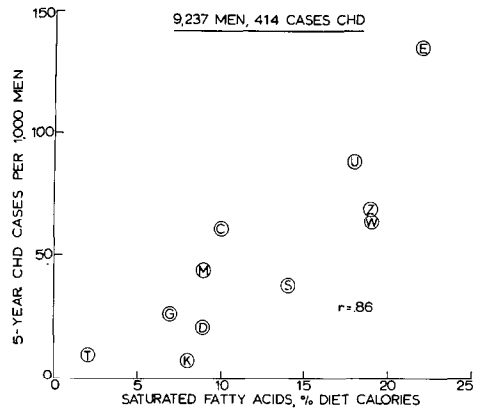

Fig. 2. Correlation between 5-year incidence rates of CHD and average percentage of calories provided by saturated fatty acids in the diet.

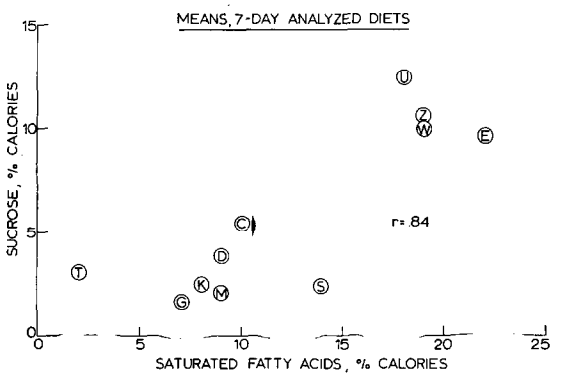

Fig. 3. Correlation between percentage of calories from sucrose and from saturated fatty acid in the diet.

YUDKIN's second argument for a major role of dietary sucrose in the etiology of CHD is that over the years there was an enormous increase in the incidence of CHD which was coincident with a huge rise in the per capita use of sugar. One hundred and fifty years ago the per capita consumption of sugar in Great Britain was perhaps only a tenth of the figure today; coronary heart disease then was certainly not considered to be a major scourge — or should we say that it was unknown as a clinical entity?

But more than half a century ago the per capita consumption of sugar in Great Britain was not grossly less than the current level and CHD deaths were then so rarely reported that they were not even listed as such in the mortality rolls. The situation was similar in the United States. In the United Kingdom, the per capita use of sucrose was over 40 kg per year prior to the first World War; by the mid 1960s the figure was a trifle over 50 kg[13]. If account were taken of the far more equitable economic distribution of goods in the U.K., it is possible that the median intake of

sucrose in that country declined over the last forty years. For the United States, the figures of the Department of Agriculture show yearly per capita averages of 51.7 kg for the 1920s and only 50.2 kg in the 1960s[14]. U.S. Vital Statistics show a tremendous rise in the CHD death rate from the 1920s to the 1960s; this deplorable change was associated with a *decrease* in the per capita use of sugar.

The fact is, of course, that only in relatively recent years is it possible to estimate the actual frequency of CHD from vital statistics. Probably the incidence of this disease has actually increased over the past century but there is no way to estimate the failures to diagnose and report CHD in the past. It must be emphasized, then, that there is no basis in fact for the claim that the trend of CHD mortality reports reflects changes in the consumption of sugar — or of any other item in the diet, for that matter.

YUDKIN's third claim, the one most often repeated as supposedly clinching the argument, is that men with coronary heart disease commonly prove to be unusually heavy consumers of sugar. In the first such claim, in 1964, from responses to a brief questionnaire, it was reported that 20 men in hospital with a diagnosis of myocardial infarction were thought to have consumed an average of roughly twice as much sugar as 25 men with no health problems or patients who were in hospital because of orthopedic problems[15]. Patients with peripheral vascular disease were said also to consume an undue amount of sugar. Three years later, from the results of similar questioning of 20 patients with CHD, 20 "healthy controls", and 13 orthopedic patients, the average daily sugar intakes were estimated to be 148, 78, and 90 g, respectively[16].

YUDKIN provided no validation for his method of estimating dietary intake with a brief questionnaire. It is interesting that, according to his report, one-fourth of the CHD patients were supposed to be consuming over 230 g of sugar daily, around a thousand or more calories of sucrose daily. Is that credible? It is interesting, too, that the CHD patients were said to be getting more than three-quarters of their sugar in their average of over 8 cups of coffee or tea every day.

YUDKIN's reports indicate no attention to the most rudimentary requirements of sampling. There is no evidence that the small groups of men queried about their eating habits were comparable in social and economic class, in occupation, family status, physical activity, obesity, place of origin and ethnic background. Even if methods of demonstrated validity had been used, any reported differences in sugar use could well be attributable to differences in one or more of the variables listed above. YUDKIN's reports on the use of sugar by CHD patients clearly will not bear scrutiny. Worse still for his case, convincing proof that his generalizations are simply erroneous is provided by the unvarying reports of 6 other groups of investigators.

In Toronto, Canada, a questionnaire survey on dietary habits covered 170 military veterans, 86 men with CHD and 84 age-matched "healthy" controls from the same population of local veterans[17]. The finding was that the CHD patients consumed slightly *less* sucrose than the controls. In Montreal, Canada, 20 CHD patients and 20 age-matched controls were studied in detail with the 7-day dietary recall method[18].

The average sucrose intake of the CHD patients was estimated to be 121 g daily; the corresponding figure for the controls was 117 g, with nothing approaching statistical significance in the trivial difference. In Dublin, Eire, trained interviewers used carefully structured interview methods with 100 CHD patients and 50 controls matched in respects other than the presence of CHD[19]. The average sucrose intake of the CHD patients was 66 g daily; the average for the controls was 69 g daily. Two studies in England, both technically and statistically more acceptable than YUDKIN's, found no significant difference in sucrose intake between CHD patients and suitably matched "healthy" controls. In one of the studies, 170 men who had CHD were compared with 1158 age-matched controls[20]. In another study, each of 80 men recovering from myocardial infarction was compared with two healthy men matched in age and in place of birth[21].

All of the 5 studies noted above concerned the comparison of CHD patients with "control". Such cross-sectional comparisons raise serious questions about relevance to etiology, of course, even when an apparent association is sound; findings from prospective studies would be much more interesting. Only one prospective study has been reported. PAUL *et al.* included a detailed dietary interview as part of the entry examination in their continuing study of men employed in a large industry in Chicago[22]. On the average, men who later developed CHD reported a little higher sucrose consumption than their fellows who stayed well but in detailed statistical analysis of CHD and the three variables of sucrose use, coffee drinking, and cigarette smoking, only cigarettes were indicated to be significantly associated with later development of CHD. The authors concluded that in regard to CHD "there is no simple link with sucrose consumption"[22].

The record is clear enough; the public is grossly misled by repetitions in print of the claim that victims of CHD tend to be persons who habitually consume more sugar than the average for the population. It is *not* true but even if it were it would not justify the claim that sugar causes CHD. Attention must be given to other variables that may be confounding. For example, sometimes sugar and smoking may be confounded as indicated by recent surveys in England involving electrocardiograms and questionnaires on personal habits[23]. No relationship was found between the use of sugar and indications of CHD but there was a significant correlation between the use of sugar and the use of cigarettes. The non-smokers averaged 83 g of sugar daily while the average for the pack-a-day smokers was 103 g.

YUDKIN's claims attracted so much interest that the Medical Research Council of the United Kingdom appointed a "Working-Party on the Relationship between Dietary Sugar Intake and Arterial Disease". The Working-Party has recently reported as follows[24]: "The sugar consumption of men with myocardial infarction was compared with that of matched controls in four different centres. The average sugar consumption was slightly greater in the patients with myocardial infarction than among the controls, but the differences were not statistically significant. Findings in one centre suggest that the slightly higher sugar intake in patients with myocardial

infarction were likely to have been due to an association between the consumption of sugar and the smoking of cigarettes".

It is useful to make surveys to check hypotheses and to seek new ideas but in the long run the mere finding of associations between variables must prove barren without at least a theoretical mechanistic link between them. A statistical association between two variables may be interesting but by itself is no proof of cause and effect. So, in an effort to proceed beyond mere statistical inferences, lately YUDKIN was impelled to ask, "What possible ways might sucrose be involved in the disease process?"[1] and to undertake experiments "feeding human volunteers and experimental animals diets in which the carbohydrate was either starch, sucrose or mixtures of the two". And then, with no details whatever, we are told, in a trade paper of the dairy industry, that these experiments "have revealed an impressive number of differences" including, in animals, "high levels of cholesterol and triglyceride in the blood"[1]. The implication, of course is that dietary sucrose promotes CHD in man by increasing the concentration of cholesterol and triglycerides in the blood.

But the facts are very different from the implication. Several groups of investigators have reported increases in serum triglycerides in experiments of a few days' to a few weeks' duration in which sugar was exchanged for starch to make diets in which, of total calories, sucrose provided 70%[25], or 42%[26], or 60%[27], or 44%[28], or 35%[29]. In another study[30], exchange of sugar and starch making up 40% of total calories produced no consistent changes in serum lipids.

The relevance of these short-term experiments to long-term national diets is highly questionable in view of the fact that stabilization of serum triglycerides in man, after marked change in the diet, requires many months[31,32]. It is notable, too, that the level of sugar in the experimental diets used was of the order of three or more times that in any natural diets.

The extent to which serum triglycerides, independent of cholesterol, may influence atherogenesis and the development of coronary heart disease is still controversial but there is no longer any question about the great importance of the serum cholesterol concentration. In experiments with diets extremely high in sucrose the level of cholesterol in the serum has been reported to be little affected[25,27,28,30,33,34] or to decrease[35]. In well-controlled experiments in a mental hospital the exchange of 25% of total calories from starch for equal calories provided by sucrose produced a trivial increase in serum cholesterol averaging less than 5%[36]. In the Laboratory of Physiological Hygiene Doctors J. T. ANDERSON and F. GRANDE have carried out three sets of controlled experiments on human volunteers in which sucrose and starch were exchanged isocalorically and in none of those experiments was there any significant change in the concentration of cholesterol in the blood serum[37]. Even YUDKIN, when he is not indulging in anti-sucrose propaganda, admits his inability to raise the serum cholesterol level in his subjects by feeding them vast amounts of sucrose[38]. ANTAR *et al.*[39] have suggested that sucrose and animal fats in the diet may have a synergistic effect on the lipids in the serum of man but that idea is not supported by

the results of the experiments on patients mentioned above[36]. In those experiments there was no difference in effect, or lack of it, of sucrose in combination with fats differing greatly in saturation.

The results of dietary experiments, then, do *not* suggest a significant ill effect of dietary sucrose on the concentration of cholesterol in the blood serum. As for effect on triglyceride in the serum, grossly extreme loading with sucrose has been used to produce short-time effect that would seem to be wholly irrelevant to the natural human situation and the "long pull".

It is difficult, then, to justify such implications as in YUDKIN's statement, noted above, about experimental findings. But on the subject of experiments it should be recalled that dietary experiments on animals have been going on for over 60 years in attempts to produce atherosclerosis and CHD. Thousands of successes have been reported in many species but sucrose was never shown to be contributory.

It is clear that YUDKIN has no theoretical basis or experimental evidence to support his claim for a major influence of dietary sucrose in the etiology of CHD; his claim that men who have CHD are excessive sugar eaters is nowhere confirmed but is disproved by many studies superior in methodology and/or magnitude to his own; and his "evidence" from population statistics and time trends will not bear up under the most elementary critical examination. But the propaganda keeps on reverberating and is reflected in such statements as the following: "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[40]".

None of what is said here should be taken to mean approval of the common high level of sucrose in many diets. But there are plenty of good arguments to reduce the flood of dietary sucrose without building a mountain of nonsense about coronary heart disease.

ACKNOWLEDGMENT

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