

Dietary sugar and Crohn's disease

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ABSTRACT: Epidemiologically, Crohn's is a disease of modern Western civilization. The diet of the Western world is highly processed. Many surveys have shown that patients with Crohn's disease habitually eat more than the average amount of some processed foods, namely those rich in added sugars. Sugar rich meals lead to increased intestinal permeability which is a feature of Crohn's disease patients and of their relatives. Replacement of added sugars and fibre depleted cereals by whole or unrefined foods is well tolerated by most patients with Crohn's disease and it may reduce their need for surgery and hospital treatment. Until the pathogenesis of the disease is clarified the role of dietary factors remains debatable but the evidence incriminating a sugar rich highly processed diet in the etiology of Crohn's disease, albeit indirect, is enough to justify further research in this area. *Can J Gastroenterol* 1988; 2(1): 41-44

Key Words: *Crohn's disease, Diet, Sugar*

CROHN'S DISEASE IS A DISEASE OF Westernized countries (1) and is more common in northern Europe than southern Europe (2). Its prevalence in Europe and USA increased in the middle part of this century, at least until the mid-1970s (3). These epidemiological facts imply that environmental factors are important in causing Crohn's disease. Because

these factors are unknown the possibility exists that the disease is linked in some way to the eating habits characteristic of the mid-20th century.

CASE CONTROL STUDIES

Case control studies are an accepted way of looking for dietary causes of a disease. Patients with the disease are matched with healthy controls for

known determinants of the disease (such as age and race) and both groups are then questioned about their habitual diet in the hope that present eating habits reflect pre-illness habits. If the disease itself could modify eating habits the study should be limited to recently diagnosed patients.

The only reported case control study of Crohn's disease in which all the patients were newly diagnosed and in which dietary intakes were comprehensively analyzed is that of Thornton and colleagues (4). Two differences emerged between the patients and controls. The patients ate more 'refined' or added sugars, indeed, nearly twice as much; they also ate less fruit and vegetables so their intake of dietary fibre was slightly lower. There were no differences in the intake of starch, fat, protein or alcohol. The excess sugar was taken not only as table sugar added to foods and drinks but also as sugars consumed in prepared food-stuffs. This invisible sugar is, nowadays, responsible for the majority of the intake of refined or added sugars (5).

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Interest in the possible dietary causes of Crohn's disease began in 1976 with two reports from Germany of high intakes of sugar, based on self-administered postal questionnaires (6,7). Since then, no less than 13 case control studies of sugar intake have been reported, using several different methodologies (4,8-18). All 15 studies have found high sugar intakes in patients with Crohn's disease (Table 1).

The only other consistent finding in these studies is that, when looked at, the intake of fresh fruit has been lower in the patients than in the controls (4,11,18). In the present author's study (4), the median intake of raw fruit and vegetables in patients was only 26% of that in the controls.

Sceptics can point out the imprecision of dietary methods and the impossibility of getting hard data on past eating habits. They cannot, however, deny the unanimity of the findings, which is quite remarkable and cannot be explained by inaccurate methods.

WHY ARE EATING HABITS DIFFERENT IN PATIENTS WITH CROHN'S DISEASE?

Three explanations have been offered as to why eating habits differ between Crohn's patients and controls. First, patients may eat more sugar and sugary foods because they have a high taste threshold for sweetness; this in turn could be due to zinc deficiency which is known to occur in Crohn's disease and which impairs taste. This hypothesis has been thoroughly tested and disproved. Patients with Crohn's disease have similar taste thresholds to healthy controls (17,19). Also there is no correlation between sugar intake and the plasma zinc concentration (17).

A second suggestion is that, as the disease develops, patients slowly and unconsciously change their food intake to counter symptoms such as weight loss, diarrhea and abdominal pain. This seems plausible, but the weight of evidence is against it. If it were true, patients with ulcerative colitis, who have similar symptoms, should also have high sugar intakes. But when such patients were investi-

TABLE 1
Published data on sucrose (added sugar) intake in groups of patients with Crohn's disease and in healthy controls

City	Number of patients	Mean sucrose intake Crohn's vs controls	Method	Reference
Marburg	63	57% more*	Postal Q	6
Düsseldorf	34	109% more*	Postal Q	7
Würzburg	20	'Heavy consumers' 35% vs 5%	7-day record	8
Cardiff	100	57% more in drinks† 87% more on cereals	Dietary history	9
Manchester	68	43% more†	Dietary history	10
Bristol	30‡	88% more	Dietary history	4
Würzburg	35	71% more	7-day recall	11
Tel Aviv	27	119% more*†	7-day recall and dietary history	12
Cardiff	120	77% more*	Postal Q	13
Cardiff	16	220% more*†	5-day weighed record	14
Birmingham & Cardiff	32	150% more†	Postal Q	14
Tübingen	NS	'Much more'	NS	15
Örebro	30	41% more	Dietary history	16
Cardiff	70	124% more†	Dietary history	17
Milan	109	'Higher'§	NS	18

*During illness; even more sugar eaten before illness; †Visible sugar only; ‡Newly diagnosed patients; Q Questionnaire; NS Not stated; §Relative risk in high versus low consumers of packet sugar and of sweets 3.41 and 3.34, respectively

gated, no difference was found between their sugar intakes and those of controls in two out of three studies (18,20,21). Also, when patients with Crohn's disease have been asked whether their eating habits have changed since they became ill they have, in four out of five reports, stated that they have cut down on sugar, not increased it (6,7,12-14). To quote Kasper and Sommer (11), "the excessive consumption of sweets, pastries and sugar sweetened beverages had become a habit in all many years before onset of symptoms". There are, in fact, no grounds for assuming that symptoms are relieved by replacing fruit with sugary foods. On the contrary, fruit is usually well tolerated (22) and a high sugar intake may even worsen symptoms (23).

The third explanation is that a sugar rich diet increases the risk of developing Crohn's disease. This idea is also consistent with the epidemiology of the disease.

HOW COULD A SUGAR RICH DIET FAVOUR THE DEVELOPMENT OF CROHN'S DISEASE?

Since the pathogenesis of Crohn's disease is unknown one can only

speculate as to how a sugar rich diet could encourage it to develop. There are, however, three possibilities consistent with current limited knowledge.

First, sugars are osmotically active. Sucrose is the only osmotically active substance ingested by human beings in concentrated form and in large quantities (more than 10 g daily). A candy bar or a slice of cake may well contain 30 to 40 g sucrose. Hypertonic solutions increase the permeability of the intestinal mucosa (24-26). Indeed, normal mucosa becomes as permeable as the damaged mucosa of celiac disease patients when it is exposed to the stress of a hypertonic drink (26). Increased permeability of the small intestine is a well known feature of patients with Crohn's disease (27). It has also been reported in their relatives (28), which suggests that increased permeability may pre-date the disease. The precise significance of increased permeability remains to be established but, if it allows repeated absorption of antigens from the gut lumen, this could perhaps lead to an inflammatory response in the sensitized gut wall.

Secondly, a high sucrose intake might induce changes in the intestinal flora or their metabolism, for example

increased bile acid degradation, with the formation of toxic metabolites. Addition of 120 g sucrose daily to the diet has been reported to increase the excretion of secondary, ie, degraded, bile acids and to enhance intestinal fermentation in normal volunteers (29). Whole gut transit time was also lengthened.

Thus, a high intake of sugar can alter gut physiology in at least two ways which are potentially harmful. Much more research is needed, however. Among other things we need to know what a sugar rich diet does to the gut's defences against microbial invasion. Because a sugar rich diet tends to be low in fruit and other 'whole' foods it is relatively deficient in vitamins and minerals, including trace elements (30), and low intakes of vitamin C, pyridoxine, zinc and linoleic acid may lead to impaired function of T lymphocytes (31). This is of interest because T lymphocyte function tends to be abnormal in Crohn's disease patients (32).

THERAPEUTIC RESPONSE TO LOW SUGAR DIETS

The fact that an agent or regimen cures a disease does not usually mean that the disease is caused by the lack of that agent or by the opposite of that regimen. However, if a chronic disease is ameliorated by abstaining from a habit which was prominent while the disease was developing, then it may be reasonable to suspect a causative relationship between the habit and the disease. Thus, it is pertinent to review the therapeutic effects of a low sugar diet in Crohn's disease.

In 1972 the author began, on empirical grounds, to advise his patients with Crohn's disease to exclude refined carbohydrates, otherwise known as fibre depleted foods, from their diet. Instead of sugar and white flour, and products made from them, patients were advised to eat fruit and wholemeal flour and products made from them. Although this regimen involved an increase in dietary fibre intake this was not the purpose of the diet, rather it was simply to replace highly processed foods with more 'nat-

ural' ones. The diet did not include bran or any other concentrated preparation of fibre. Treatment was conventional in every other respect.

In 1979, the results of using this diet in 32 consecutive patients with Crohn's disease were published (33). In comparison with a closely matched group of 32 patients from other clinics in Bristol the diet-treated patients had fared significantly better. Over a mean follow-up of 52 months they had spent only 111 days in hospital compared with 533 days in the control group and had required only one intestinal operation compared with five in the control group. This was not a randomized, prospective trial and the better progress of the diet-treated patients may have been due to some other unidentified difference in management or to a difference in the type of case referred to this clinic from the other Bristol clinics.

A very small randomized trial was reported from Brunswick in 1981 (23). When five patients with moderately active disease were given a sugar rich diet, four became worse whereas only one of five patients became worse who were allocated to a sugar free, high protein diet.

A team at St Mark's Hospital, London, England has recently reported a randomized trial involving 352 patients with mildly active or inactive Crohn's disease (34). One hundred and ninety were prescribed a diet of the type used in Bristol. The rest continued with their usual diets. At the end of a 24 month follow-up there were fewer surgical operations and hospital admissions in the group on the low sugar diet but the differences did not reach statistical significance. It seems churlish to criticize this mammoth study. In many ways it was scrupulously correct. However, its failure to show a significant difference needs close examination. The average reduction in sugar intake in the test group was only 53 g per day and there was an 11 g per day reduction in the control group. The number of operations was rather small, even in the control group (4.3% per year), suggesting that the volunteers in this study were patients

with unusually benign disease. In such patients it might be difficult or impossible to show benefit. The patients in the Bristol and Brunswick studies were unselected and had more active disease.

Thus, it remains unproven that a diet low in refined sugars is beneficial in the treatment of mildly active or inactive Crohn's disease but the author (although biased) believes it is helpful and still uses it; certainly, it does no harm.

A beneficial effect is not necessarily at variance with the proven benefit from an elemental diet (which is rich in sugars) in active Crohn's disease (35). Different phases of a disease often require different treatments.

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Clinical quiz – Answers

ESOPHAGUS

1. Indications for surgery in reflux esophagitis

Uncontrollable hemorrhage secondary to erosive esophagitis
Resistant esophageal ulcer
Stricture (nonresponding to bougienage)
Barrett's esophagus with dysplasia
Recurring aspiration-induced pulmonary disease
Intractability of symptoms (relative indication)

2. Features of typical motility pattern in patients with well established achalasia

Upper esophageal sphincter normal
Lack of primary peristaltic waves; small amplitude simultaneous waves

Resting pressure elevated in body

Lower esophageal sphincter pressure elevated, fails to fall to zero with swallowing
Hypersensitivity to cholinergics

3. Complications of endoscopic sclerotherapy of esophageal varices

Local mechanical
Ulceration
Stricture and dysphagia
Pleural effusions
Esophageal perforation
Severe chest pain due to mediastinitis
General
Transient bacteremia
Acute respiratory failure



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