Nutrition and gastrointestinal disorders

Shao K. Lin, John R. Lambert and Mark Wahlqvist

Monash University, Department of Medicine, Monash Medical Centre, Melbourne, Australia.

The gastrointestinal tract, including the liver and pancreas, is a complex system whose function is to process a wide range of nutrient and other products enabling their absorption as well as detoxification and excretion. During the process, food is converted into energy and into other substances that are used by cells throughout the entire body. Many diseases can affect the various organs of the gastrointestinal (GI) system and diet plays a relatively minor role in the onset of such GI diseases.

Recently it has become clear that glutamine, a 'non-essential' amino acid, is important in the maintenance of intestinal mucosal metabolism, structure and function. Dietary fibre has complicated properties including trophic effects on intestinal mucosa, volatile fatty acid production, alteration of bacterial flora and faecal bacterial mass and change in faecal bile acids.

Gastrointestinal disease many result from deficiency or excess of specific nutrients in normal individuals. In allergic or susceptible subjects, diseases such as food allergy, disaccharidase intolerance and gluten sensitive enteropathy may occur with intake of normal daily requirements. In genetically susceptible individuals, specific nutrients have been linked, based on epidemiological studies and animal experimentation, to carcinoma of the stomach (high starch, high nitrate foods and smoked meats) and colon (low fibre, high fat, low vitamin A). A recent Australian multi-centre polyp prevention project has recruited subjects with adenomatous polyps cleared at colonoscopy. Subjects were randomized to receive high fibre, low fat, β-carotene or a combination of these and compared to an unchanged control group at 2-yearly follow up colonoscopy. Low fat and high fibre were not protective against polyp development; however, β-carotene ingestion was associated with an increased risk.

Duodenal ulcer disease is multifactorial with gastric acid and *H. pylori* induced gastroduodenitis playing important aetiological roles. Protection is afforded to individuals with a higher unsaturated fatty acid and lower refined sugar intakes.

Treatment of gastrointestinal disease may require dietary modifications or, if the gut is not functioning adequately, nutritional support via the parenteral route. In subjects with inflammatory bowel disease and short gut syndrome replacement of specific nutrients may be required particularly calcium, magnesium, zinc, iron, and vitamins B_{12}, folate, D and A. Controversy still exists as to the role of parenteral and enteral nutrition as primary therapy for inflammatory bowel disease.

Introduction

The gastrointestinal tract is a complex organ involved in the processing, absorption and detoxification of foods as well as excretion of waste products. Macro and micronutrients are important for the integrity of this system. This paper will review several aspects of nutrition and the gastrointestinal tract including the important role of glutamine and fibre in the normal physiological function of the gut. The role of nutrition in the aetiology and treatment of gastrointestinal disease will be discussed.

Nutrition and gastrointestinal physiology

Glutamine

Glutamine is a non-essential amino acid that has the highest concentration in whole blood 500–900 μM of all amino acids[4,15,48,68] and it accounts for more than 60% of the free amino acid pool of the body[5]. The majority of the 'free' glutamine is in skeletal muscle where its concentration is 30 times greater than the circulating level[5,50,60].

Recent studies have suggested that glutamine plays an important role in the maintenance of intestinal metabolism, structure, and function. These studies indicate that glutamine supplementation of total parenteral nutrition (TPN) solutions results in an increase in jejunal mucosal weight and DNA content and significantly decreases the villous atrophy associated with standard TPN[31] as well as an increase in villous height and nitrogen content[19]. Glutamine-supplemented elemental diets also are able to stimulate intestinal mucosal growth following starvation[64].

In addition glutamine prevents bacterial translocation from the gut[1,8,67]. Thus in patients with systemic gut insults (shock, sepsis, multiple trauma, malnutrition) and occasionally local gut injury (chemotherapy,

Dr J R Lambert, Monash University, Department of Medicine, Monash Medical Centre, Clayton Road, Clayton 3168 Victoria, Australia.

enterocolitis) that are associated with bacterial translocation, the provision of diets that are glutamine supplemented may reduce the incidence of bacterial translocation, promote 'bowel rescue', and possibly improve overall survival. Further studies are needed to confirm a beneficial role of glutamine in these subjects.

Dietary fibre

Dietary fibre is a group of endogenous components of plant materials in the diet that are resistant to human digestive enzymes[56]. Dietary fibre can be divided into seven major categories based on chemical structure: cellulose, hemicelluloses, pectins, gums, mucilages, algal polysaccharides and lignin. Dietary fibre has also been classified according to its solubility in water (gums, pective and other hemicelliuloses). Soluble fibre sources include psyllium, oat bran, and beans, whereas insoluble fibre sources include cereals and whole grains.

Fibre is necessary to maintain the normal function of the gastrointestinal tract[34,65]. The physical properties of fibre that contribute to the gut function include an increase in stool weight and defecation frequency as well as a decreased gastrointestinal transit time. Soluble fibre may have its effect by forming gels which trap noxious agents. Soluble fibre can also be fermented by colonic bacteria thus producing short chain fatty acids (SCFA) which reduce colonic pH. SCFA are rapidly cleared from the colon and appear to enhance Na^+ absorption. Both soluble and insoluble fibre bind bile acids and other mutagens.

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Diet and peptic ulcer disease

Peptic ulcer is one of the most prevalent gastrointestinal disorders and still is a common, chronic and recurrent disease world-wide. Changes in the incidence of peptic ulcer disease have been noted in recent years[73]. Peptic ulcers are local defects of the mucosa that result from excessive production of gastric acid and pepsin along with a decrease in mucosal resistance to these and other substances.

Recent advances have led to improved understanding of peptic ulcer pathophysiology. Endogenous risk factors, such as acid output, pepsin secretion and serum pepsinogen, genetic predisposition, and the use of anti-inflammatory drugs are involved in causation. Stress, the use of alcohol, tobacco and caffeine, and other diseases (eg chronic lung disease, rheumatoid arthritis, and hepatic cirrhosis) have been implicated as less important aetiologic factors. In addition, infection with *Helicobacter pylori (H. pylori)* is an important associated factor in the pathogenesis of gastritis and duodenal ulcer disease[42]. Differences exist in the type and prevalence of peptic ulcers in different countries which would suggest cultural as well as environmental factors, including social class distribution, diet and *H. pylori* infection rates as important factors.

Renewed attention has been given to the role of diet in peptic ulcer disease. Alcohol can cause superficial mucosal erosions, and bleeding in both experimental animals and human volunteers[27], however it has no role in ulcer healing and relapse[66]. Cigarette smoking has been associated with initiation and delayed healing of peptic ulcer and to the relapse of duodenal ulcer in both men and women[2,37,41,49]. This effect appears to be dose-dependent and the mechanism is most likely via increased acid secretion particularly at night[37]. Coffee use is often limited by physicians in patients with peptic ulcer disease. Caffeine-containing foods have strong acid stimulatory effects, however in coffee other factors contribute to this effect. A recent report described patients with nonulcer dyspepsia as more likely to experience dyspeptic symptoms after coffee ingestion, but this did not occur in duodenal ulcer patients[13]. Foods or spices such as 'hot' pepper that are severe irritants or acid stimulants have no proven effect on human ulcer pathogenesis.

The decrease in ulcer incidence in the United States has been hypothesized to be due to a 200% increase in the use of dietary essential fatty acids since the beginning of the 20th century[26]. Hollander and colleagues have shown that dietary essential fatty acid can be rapidly converted to prostaglandins E (PgE) by the gastrointestinal mucosa. This PgE will then protect the gastrointestinal mucosa from injury by agents such as alcohol, aspirin and bile acids[26]. A previous study of dietary linoleic acid has confirmed an increased gastric prostaglandin secretion with suppression of acid output[17]. The mean dietary linoleic acid intake is lower in male duodenal ulcer patients than in control subjects[18]. These findings are consistent with the hypothesis that increased intake of polyunsaturated essential fatty acids is a potential explanation for the decreasing incidence of peptic ulcer disease[26]. Hansky and Soveny[20] have shown that fish and fish oil protects the rat from cysteamine induced duodenal ulcer through an unknown mechanism. Depletion of duodenal PGE_2 levels were found in this study suggesting that the protective mechanism is either unrelated to prostaglandins or that there is a rapid turnover of prostaglandins[20].

The role of dietary fibre in peptic ulcer disease is a controversial one. Dietary fibres have the potential to buffer gastric bile acid concentrations and decrease total gut transit time resulting in less distension and acid output. The geographic distribution of duodenal ulcer is not consistently associated with fibre consumption[70]. Previous studies suggest a high fibre, high complex carbohydrate, low saturated fat diet is helpful in the prevention of the peptic ulcer disease or reduces duodenal ulcer relapse[47,61]. No differences in healing rates between patients treated with diets of different fibre contents have been reported[62,63]. A higher incidence of peptic ulcer has been reported in rice eating population[71]. In animal experiments fresh rice oil protects against gastric ulceration, where as stored oil is ulcerogenic[33]. If the hypothesis that a high fibre intake reduces the incidence of duodenal ulceration is true, there has to be either qualitative differences between various types of dietary fibre or alternatively protective factors may be present in selected high fibre foods affecting mucosal resistance[62]. Katschinski and colleagues in a case-control study assessed the relationship between the intake of refined foods and the risk of duodenal ulceration. No protection by a high total fibre or cereal fibre intake was observed. However, they suggested that a low refined sugar intake may be a protective factor. Larger studies are needed to confirm and clarify this relationship[35].

Helicobacter pylori has been implicated as strongly associated with peptic ulcer disease. In most studies there is no documentation of dietary intake in infected individuals. Studies have suggested that chopstick use is associated with an increased sero-prevalence of *H. pylori* supporting the importance of food culture in *H. pylori* transmission[44]. Hopkins et al. recently compared *H. pylori* seroprevalence between Seventh-Day Adventists (who are vegetarians abstaining from alcohol and caffeine) and two other populations. Neither meat consumption nor age-related dietary factors, such as alcohol and caffeine, were a significant risk factor for *H. pylori* infection[28].

Nutrition and the aetiology of gastric cancer

The incidence and mortality rate statistics of gastric carcinoma indicate a clear decline in almost all countries and continents. This trend is particularly noted in the USA and Western Europe[30], with the incidence in Latin America and Asia remaining very high[51,53]. The reasons for this decline are not known, however epidemiologic data suggest that exposure to environmental factors, especially eating habits, is important in the pathogenesis of gastric carcinoma[30]. Differences in the eating habits of populations of various continents having a similar risk of developing the disease clearly exist. Recent studies have suggested that persons infected with *Helicobacter pylcri* are at an increased risk of gastric carcinoma[52,54].

Previous epidemiologic studies have suggested that dietary risk factors for gastric carcinoma include low consumption of animal fat and protein, salad, vegetables, fruits and a high intake of poorly digested carbohydrates, salted foods (especially salted meats and fish), smoked meats and fish and fava beans. A case-control study in China noted that the daily consumption of sour pancakes, a fermented indigenous staple, was associated with a 30% increase in risk of gastric cancer[76].

Protective factors include a diet rich in vitamin C and A, high consumption of fresh vegetables/fruit, calcium, proteins and fat. The 'protective' vegetables include dark green, cruciferous and allium (onions, garlic, leeks) vegetables[76]. Further studies are required to identify the specific vegetables and their components which may be protective agents. The nitration of drinking water may also be an important factor in the risk of gastric carcinoma[9,29].

Consumption of alcohol and nicotine is also considered a risk factor for the development of gastric carcinoma[11,12,76]. Of the main tobacco variables, a prolonged exposure seems more important than the amount smoked per day as a risk factor for developing gastric carcinoma. Both wine and liquor ingestion are associated with an increased risk of gastric carcinoma[12].

Dietary fibre, colon cancer and polyps

Colorectal cancer has a lifetime cumulative incidence of 5% in the population of Western countries, and follows lung cancer as the most frequent cause of death from cancer in the USA and Australia[3,14]. In Australia, approximately 7000 new cases of colorectal cancer arise per annum, resulting in 3700 deaths annually[3]. Although genetic and inflammatory disorders clearly predispose to large intestinal cancer, case-control studies in Australia have indicated that patients higher intake of alcohol, beef, animal fat and total energy are associated with a higher risk of colon cancer[39,40,57]. In experimental models fat promotes colon cancer development and fibre is protective against this change. Fat is thought to promote colon cancer by increasing the amount of secondary bile acids in the colon[36,58]. Fatty acid intake in rodents, results in a compensatory increase in the proliferation of colonic epithelial cells. Other factors are clearly important as in rural Finland subjects consume a high-fat diet and yet have a low distal colon cancer risk[43]. A high fibre, particularly wheat bran intake, may theoretically lower cancer risk. Fibres and especially specific lignin-type compounds appear to decrease the risk of experimental colorectal cancer, owing to effects on decreased intestinal transit time, dilution of carcinogens, reduction in faecal pH, chemical binding, alteration in faecal bacterial metabolism and production of SCFA[7,32,72,75]. Fibre also increases faecal bulk resulting in reduced transit time and less contact with the colonic mucosa.

High consumption of refined sugar is associated with the development of colorectal cancer[6]. Recent studies by[38] Kruis et al. compared high refined and low refined sugar diets and found that the wet and dry weight, pH and water content of the stools were similar on the two diets. On the high sugar diet mouth-to-anus transit time was significantly prolonged, despite a shortened mouth-to-caecum transit time as well as an increased faecal concentration of total and secondary bile acids[38].

Most large bowel cancers arise in adenomatous polyps. Subjects with polyps have a higher consumption of fat, sugar, and confectionery and a lower consumption of fibre and cruciferous vegetables[24,46]. It has been suggested that cigarette smoking is associated with colonic polyps[10,25]. Another study has indicated that patients with colonic polyps had no significant differences in diet or smoking history, but drank more alcohol compared with controls[69]. Cope and colleagues have confirmed an association with current alcohol and cigarette consumption. The risk of adenomatous polyps was increased three times in drinkers who did not smoke and two times in smokers who did not drink, while those who both drank and smoked had 12 times the risk of total abstainers. This adds further support to the view that alcohol consumption may be an important factor in the pathogenesis of colorectal neoplasia[10].

Two studies have prospectively assessed the effects of fibre on the recurrence of adenomatous polyps in humans. In an Australian study, the effects of reduced fat intake, wheat bran fibre supplement, and a betacarotene supplement have been examined in a 2x2x2 randomized factorial design involving 424 subjects[45]. Increased fibre and reduced fat intake had no effect on decreasing polyp recurrence; however, β-carotene appeared to increase the rate.

Dietary calcium appears to be protective against tumour growth and polyp formation in humans and in animal experiments[16,59,74]. The mechanism is unknown.

Nutritional consequences and therapy in inflammatory bowel disease

Nonspecific inflammatory bowel disease includes two diseases of the gastrointestinal tract: ulcerative colitis

and Crohn's disease. Their aetiology and pathogenesis are unknown. Smoking, refined sugar intake and lower fibre have been postulated as aetiological factors, in the absence however of conclusive proof.

It is notable that a high refined sugar intake has been consistently found to be associated with Crohn's disease[23,55]. A diet containing unrefined carbohydrate and extra fibre, such as fruits and vegetables, is a useful adjunct in the management of patients after surgery and does not precipitate intestinal obstruction[22].

Nutrition support is an important aspect of the regimen of bowel rest that may be initiated to induce a remission of the disease or to treat its complications. Controversy exists as to the role of TPN and enteral feeding as primary treatment for Crohn's disease. Nutritional support has no role in primary therapy of ulcerative colitis.

Irritable bowel syndrome

In irritable bowel syndrome (IBS), the two major triggering factors are psychological, largely stress-related and alimentary via the ingestion of food. IBS has no specific aetiology but rather is a clinical syndrome with a group of specific symptoms. Food intake, particularly high fat and low fibre, may play a role in triggering or perpetuating symptoms in patients with IBS. Dietary fibre has been used to treat IBS particularly in subjects with constipation[21]. Addition of complex carbohydrates or soluble fibre known to reduce proximal small bowel motor activity, may alleviate symptoms that originate in the small bowel. The dose of dietary fibre must be individually adjusted to each patient's response, allowing enough time to evaluate changes in stool patterns and relief of pain.

Summary

In summary, the role of nutrition in aetiology, treatment and prevention of gastrointestinal disease is complex. As our knowledge of basic food science and the effect of luminal contents on mucosal growth and repair improve a greater understanding of the effects of nutrients will be possible.

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胃腸道包括肝臟和胰臟是一個復染的系統，它們的功能是處現和吸收各種營養物質，同時還具有消毒和排泄功能。在現過程中，食物被轉化成能量和其他物質，供給全身細胞利用。很多疾病能影響胃腸系統的多個器官，在消化道疾病的發病時，膳食因素起一個相對次要的作用。

谷氨酸胺，一種非必需氨基酸，最近已明確了它在維持胃腸道粘膜的代謝，結構及功能上的重要性。膳食中的纖維素具有復染的特性，包括腸道粘膜的營養作用，擇發性脂肪酸的生成。細菌菌蟲的改變及糞便細菌質量和膽酸的變化。

在正常人中，特殊營養素的缺乏或過量可引起消化道疾病。對于過敏或敏感体質的人，在正常需求的飲食中可能出現例如食物過敏，不耐雙糖 ，谷蛋白過敏性腸病等。對于遺傳性敏感的個体。統計病學的研究和動物實驗顯示，特殊營養素與胃癌（高澱粉、多硝酸鹽食物和煙勳肉類）和結腸癌（低纖維、高脂肪、低維生素A）有關。最近澳大利亞多中心息肉預防計劃對腺瘤性息肉的病人隨机抽樣分成三組。試驗組進食高纖維、低脂肪、beta - 胡蘿蔔素膳食，對照組保持原來膳食，二年後結腸鏡復查，結果發現低脂肪和高纖維不能防止息肉復發，而beta - 胡蘿蔔素的攝入會增加發生率。

十二指腸潰瘍是多因素引起。胃酸和幽門螺杆菌誘發胃十二指腸炎起重要的病因學作用。高度不飽和脂肪和低精制糖類攝取對個体有保持作用。

消化系統疾病的治療可能需要飲食限制，或當腸道功能不全時，需要通過腸外營養支持。患炎症性腸病或短腸綜合症的病人，需要特殊營養素，特別是鈣、鎂、鋅、鐵和維生素B12、葉酸維生素A和D。在炎症性腸病的早期，腸外和腸內的營養治療的效用仍存在爭議。