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DIETS/DIETARY HABITS AND CERTAIN GASTROINTESTINAL DISORDERS IN THE TROPICS: A REVIEW

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Summary: Against the background that what one eats affects the gastrointestinal tract (G.I T), the role of diet and dietary habits including fibres, food additives and preservatives on the aetiology of gastric cancers, colorectal cancers and other G.I disorders in the tropics are herein reviewed. Carcinomas of the gut believed to be on the decline in the developed countries have plateaued and increasing cases are being reported in the tropics. Africa and Nigeria in particular, with little or no cases previously are currently experiencing patterns of incidence similar to those of the Western Hemisphere. All these developments are premeditated by the nature of diets and dietary factors contained therein. Some of these factors contain chemical carcinogens, irritants as additives or preservatives, high cholesterol, highly spiced foods, alcohol, nicotine, xanthines, caffeine, most of which provoke gastric acid secretions dyspepsia and heartburn, and they lack vegetables and dietary fibres known to protect the G.I tract against various diseases. The roles of dietary hygiene implicating certain microorganisms associated with G.I diseases like *Helicobacter Pylori* are also discussed. It presupposes that well articulated diet and proper dietary manipulations remain the cure for all diet induced G.I disorders while avoidance of such habits that predispose to them must be encouraged to ensure proper and healthy G.I.T.

Key Words*: Cancers, Ulcers, G.I disorders, Diet/Dietary fibres, Additives, Dietary Habits.*

Introduction

Diet\Dietary Habits and G.I. Disorders

Food is an integral part of the daily life but we spend less time choosing, preparing and eating food. Consequently many of us are not eating properly which may result to major G.I dysfunctions (Okike, 1997) or as a note of warning was sounded "abuse your stomach and be sick, control your stomach (what you eat) and be well" (Amure, 1990). Practicing good dietary habit requires a little more time and planning than we can afford in this fast changing world. However, its benefits will pay off at the long run because of the sufficient scientific evidence that link most G.I disorders to diets\dietary habits (Hyams, 1983). Despite the above findings, some diets also have beneficial effects on the G.I tract (Howe *et al*, 1992; Alberts *et al*, 1992).

In this review, we tried to highlight the pathophysiological basis for some of these diets\dietary habits as they relate to G.I disorders in the tropics.

Role Of Diet/Dietary Fibres, Food Preservatives/Additives and Helicobacter Pylori on the Aetiology of Gastric Cancers and Colorectal Cancers

Gastric and colorectal cancers

Gastric cancer used to be one of the commonest G.I tumors / malignancy in the world and one of the principal causes of death (Bailey and Love, 1995) mostly in the Caucasians (Badoe *et al*, 1994). Carcinoma of the stomach was once reported to be on the decline in some advanced countries of the western hemisphere (Remine and Priestly, 1966). Other reports however suggest that this decline has plateaued and that the number of new cases is actually increasing(Williams and Williams, 1972). It has become relatively uncommon in the United States (Pisani *et al*, 1999) whereas in Japan, gastric cancer remained the leading cause of cancer mortality and in United States, it maintained a 60 – year decline

incidence of the disease (Parker *et al*, 1997). The range of conditions associated with an increased risk of gastric cancer include: atrophic gastritis, *Helicobacter Pylori* infection, pernicious anaemia, subtotal gastrectomy, gastric polyps and certain immune deficiency states (Brown and Shaheen, 2004). There is increased risk associated with smoking and the ingestion of salted or pickled foods (Haluszki *et al*, 2005). In areas with high soil and water nitrates high gastric cancer rates abound because the rich content of nitrates in foods, vegetables grown from such places are converted to nitrites and the use of nitrites in food preservatives allow for interaction with secondary amines and formation of N - nitrosoamine, a known carcinogen. Some foods contain N – nitroso compounds (Brunt *et al*, 1988). Gastric carcinoma was previously thought to be rare in Africa and Nigeria (Jos, Plateau State) (Obafunwa, 1990). However, earlier reports from Nigeria (Edington and Maclean, 1963; Olurin and Itayemi, 1971; Grillo *et al*, 1971 reported a 14.4% incidence; Onuigbo, 1975; Ajao, 1979) and in other parts of Africa, Oettle (1964); Badoe (1965) while later reports (Sule *et al*, 1994, 2001; Attah 2000) have shown that the disease is not rare after all and that its presentation closely resembles what obtained in the developed Western Nations.

Recently, the WHO classified the bacterium, *Helicobacter Pylori* reported to be the primary cause of peptic ulcer as a definite human carcinogen, estimated to account for up to 60 per cent of stomach cancer cases worldwide (Rangwani, 2006). Epidemiologic evidence had linked *H. Pylori* infection with the increased risk of gastric adenocarcinoma and primary gastric lymphoma and hence, the prevalence of stomach cancer in developing countries like India where 80 per cent of the children are infected by the bug by age of five (Rangwani, 2006). Gastrointestinal ulcers, which number among the most common serious digestive system complaints in developing countries like India, have for long been linked to dietary intake and unremitting stress: 'To what you are eating and to what's eating you!" The discovery of *Helicobacter Pylori* in human stomach and its association with peptic ulcer disease (PUD) in 1983 by Australian Physician threw more light into the pathogenesis of the disease and influenced its management (Marshall and Warren, 1984). Some other studies have linked *H. Pylori* infection to normal non – dyspepsia, chronic type B gastritis, peptic ulcer, mucosa associated lymphoma tissue (MALT) and gastric cancers (Marshall *et al*, 1985; Liabenz and Bosch, 1994; Correa, 1995). *Helicobacter Pylori* infection has been found in association with duodenal ulcer in over 90 per cent patients and with gastric ulcer in approximately 70 – 80 per cent patients (Ndububa *et al*, 2001). A casual role however, has been postulated for *H. Pylori* infection in the sequence of atrophic gastritis, intestinal metaplasia and dysplesia that could result in gastric carcinoma (Ndububa *et al*, 2001). According to Rangwani (2006), the most likely route of *H. Pylori* transmission to an individual include oral, with other sources as saliva, dental plaque, faeces, vomitus and drinking water. He also reported that poor sanitation especially in crowded urban areas contributes to primary infection. Thus, upper GI endoscopic study would be incomplete without the investigation of *H. Pylori* infection. Various incidence rates of *H. Pylori* had been obtained in West Africa (Magrand *et al*, 1989; Olusanya, 1990; Baako and Darke, 1996). In Nigeria, a handful of upper gastrointestinal series that linked endoscopic findngs to *H. Pylori* infection have been published (Coker and Akande, 1989; Holcombe *et al*, 1990; Smith *et al*, 1990; Oluwasola *et al*, 2002). Studies by Ndububa *et al* (2001) revealed that DU with 38.7% incidence remained the predominant pathology among Nigerians patients with upper GI disorders while GU with an incidence of 4.07% was still relatively uncommon and gastric carcinoma with incidence of 6.2%. These workers reported that *H. Pylori* infection played a major role in the development of peptic ulcer in Nigeria and that DUs, pyloric channel ulcers and gastroesophageal polyps disorders could be due to *H. Pylori* infection and a 73% incidence of *H. Pylori* infection was observed among the study population at Obafemi Awolowo University Teaching Hospital, Ile - Ife.

Dietary factors have been observed to play a major role in the aetiology of gastrointestinal disorders (Levin, 1995) in addition to genetic and environmental factors especially chemical carcinogens (Wright, 1980). Pickled and fermented vegetables have been reported to be high risk factors because of their high level of nitrates and salts with low level of antioxidant (Tricho *et al*, 1985). Red chilli pepper, an additive is the most commonly used spice throughout the world (Sirsatunuk and Khanolkar, 1960). Consumers of chilli pepper have also been found to be at higher risk due to the hot tasting component or pungent agent in it called capsaicin (Lopez *et al*, 1994; Lillie and Ramirez,1935).Other risk factors for gastric cancer include meat, animal fats in the diet, lack of dietary fibres, refined carbohydrates and anaerobic bacteria in the large intestine (colon) (Brunt *et al*,1988;Contran,1989).The cancers of

the alimentary tract probably have been linked to something ingested. The initiating factor or carcinogen has not been known but one promoting factor, a co-carcinogen was identified as cholesterol (Clark *et al*, 1979). Dietary cholesterol is a factor determining the rate of development of experimental colon cancer (Cruise *et al*, 1978). A report on animal experiments had shown that when an animal cancer model was studied, the progress of the cancer was considerably influenced by the nature of the diet (Clark *et al*, 1979). These workers showed that when cholesterol was removed from the diet, the cancers were slower to appear and also progressed slowly. Thus, this had implication on the aetiology of cancer in man with a possible therapeutic. In the 1970s, colorectal cancer was the most common internal malignancy in the United States (Cutlers and Young, 1975), while it was reported later to be second only to bronchogenic carcinoma (Brunt *et al*, 1988), and western dietary factors are likely to be implicated and high fats diets correlate well with the geographical incidence of colon cancer (Weisburger *et al*, 1977). There were indications that the incidence of colorectal carcinoma had surpassed that of gastric carcinoma, with colorectal carcinoma becoming one of the main targets of cancer management in many centers in Japan (Ilinima *et al*, 1981; Hirayama, 1981 Tommaga *et al*, 1989). Many epidemiologic studies included immigrants to some foreign countries and they have found colorectal carcinoma to be closely related to environmental factors including dietary habits or change in dietary styles as carcinogenic factors than in previous decades (Staszewshi *et al*, 1971, Newman and Slengler, 1984; Lee *et al*, 1988; Martha *et al*, 1996; Trapido *et al*, 1999). Harris and Go (2004) reported that colorectal cancer was the third most commonly occurring cancer in the USA and it accounted for 11% of cancer deaths, and second to lung cancer as the leading cause of cancer related deaths in United States and had become relatively uncommon in USA but the leading cause of cancer deaths in Japan (Pisani *et al*, 1999; Parker *et al*, 1990). Lung cancer is currently the leading cancer killer in both men and women in the United States. In 1987, it surpassed breast cancer to become the leading cause of cancer deaths in women (American Cancer Society, 2006). It noted that lung cancer causes more deaths than the next three common cancers combined (colon, breast, and prostate) as an estimated 162,460 deaths from lung cancer would have occurred in the United States during 2006 (American Cancer Society, 2006). Between 1979 and 2003, lung cancer deaths increased by 60% with age – adjusted deaths rate. In males, it was 74% greater than females with studies on black populations showing an incidence of 12% greater than in the white population (Alberg and Samet, 2003). Also, lung cancer incidence rates have been decreasing among men while the rates have been stable since 1998 in women after a long period of increases (American Cancer Society, 2006). In a 10 – year retrospective study at University of Nigeria Teaching Hospital (UNTH) Enugu, Ngokere *et al*, (2003) reported on different types of colorectal cancers of which adenocarcinoma had 62.7% incidence. Other reports from Nigeria on colorectal cancers observed the occurrence of more rectal cancers than colon cancers as against a higher colon incidence in the Caucasians, thus they opined that both types should not be combined to prevent masking effects (Ajao *et al*, 1994; Adesanya and da-Rocha- Afodu, 2000; Sule *et al*, 1994, Tade, 2006). Slattery *et al*, (2000) evaluated the association between dietary alpha-carotene, beta-carotene, lycopene, lutein, zeaxanthin and beta-cryptoxanthin and the risk of colon cancers. This epidemiologic study reported that lutein was inversely associated with colon cancers in both males and females while other carotenoids gave no significant effects. The major dietary sources of lutein in the study were spinach, broccoli, lettuce, tomatoes, oranges and orange juice, carrots, celery, and greens. They suggested the incorporation of these foods into the diets to reduce the risk of developing colon cancers. Many protective factors against G.I cancers have been documented. These include green vegetables and fresh fruits reported to have a high level of antioxidant and carotenoids that are known anti cancerous agents (Potter, 1992).

Dietary fibre intake had also been shown to have an inverse relationship with the risk of colonic cancer (Howe *et al*, 1992). The pathway of its action includes the inhibition of DNA synthesis and epithelial cell proliferation within the rectal mucosa (Alberts *et al*, 1992), activation of diacylglycerol, a second messenger, that is, released by cellular membrane phosphatidyl inositol breakdown (Reddy *et al*, 1994). This in turn activates cellular protein kinase C that has an effect on growth control and signal transduction (Reddy et al, 1994). The role of dietary fibre in cancer control in the intestine is very interesting. Thus, some additives and colouring flavours influence the transit time, hence, the longer the transit time of food in the body, the greater the chances of carcinogens getting in contact with the mucosa membrane linking the gastrointestinal tract (GIT) (Durosami-Etti, 1997). The presence of increased fibre in diet causes a decreased

transit time and time of contact of food with bowels (Burkitt and Trowel, 1975). Americans have been reported to have a higher incidence of colonic cancer because their diet is low in fibre, hence leading to a longer transit time (4-5 days) (Poll *et al*, 1989). Also, in communities living on a high – residue diet, constipation and colonic diverticulosis do not occur whereas both conditions are common frequently in older people taking a low – residue diet (Burkitt *et al*, 1972; Painter and Burkitt, 1971). According to Cumming (1973), dietary fibre's chief importance is that it increases the bulk of the colonic contents partly by its own volume and partly by uptake of water and this in turn stimulates bowel movement so that defecation is facilitated. This is in sharp contrast to Africans with a transit time of 30 - 35hours (Badoe, 1965; Contran *et al*, 1994). This low transit time as seen in Africans is a consequence of their high fibre diet, and a probably environmental factor protecting them from the development of colorectal cancer. Most recent study in the United States revealed that low-fat diet pattern made up of increased fruit, vegetable fibre intake did not reduce the risk of colorectal cancer in postmenopausal women during a 8.1 year of follow up (Shirley *et al*, 2006). Also, another large pooled analysis showed that high dietary fibre intake was inversely related with the risk of colorectal cancer in an age-adjusted analysis but after accounting for other dietary risk factors, they concluded that high dietary fibre intake was not associated with a reduced risk of colorectal cancer (Park *et al*, 2005). Increased fat ingestion leads to increased release of bile salts which may be degraded by bacteria to carcinogens or promoters and increased faecal bile acids have been found both in people in high risk communities and in individuals with colonic cancers (Brunt *et al*, 1988). The American Cancer Society (2006) has explained that conventionally smoked foods such as hams, bacons; some varieties of sausages and fish as well as fruits dried with the risk of smoke absorbed /some of the tars that arise from incomplete combustion. These tars contain numerous carcinogens that are similar to the carcinogenic tars in tobacco smokers. Some salted or smoked fish and meats contain the known carcinogen, 3,4 –benzpyrene (Brunt *at al*, 1988) as well as pickled or preserved foods have frequently been associated with an increased risk of gastric cancer (Palli, 1994). As a result, the understanding between food like high meat diets and the development of tumours will give more clues to the prevention of more cancers (Cumming and Bingham, 1998; Bingham, 1999; 2006). With regards to oral cancers, alcohol consumption has been implicated as an aetiological factor than tobacco in Nigeria (Adewole, 2002).

Role of Diet/Dietary Fibres, Food Preservatives and Additives on the Aetiology of Peptic Ulcers and Gastritis

Most substances that promote gastric acid secretion have been implicated in the damage of the gastric mucosa and the release of pepsin before superficial ulceration occurs. Also, the accumulation of acid in the tissues during damage may activate pepsin to digest the mucosa from which the enzymes originate. In gastroenterology, this adage "NO ACID, NO ULCER" remains very significant since peptic ulcer formation does occur not in patients whose stomachs are achlorhydric. In the same way, "peptic ulcer does not occur in the absence of pepsin" (Schwartz, 1910). It has been shown that the physical presence of food in the stomach through its distensible effect and the chemical factor in food affect the gastric mucosa and cells (Walsh and Grossman, 1975). Peptic ulcers therefore are caused most significantly by increased gastric acid secretion, impaired mucosal circulation and damages to the mucosa (Wienbeck and Lubke. 1987). In the contrast, motility plays only a minor role, and the following motility disturbances in peptic ulcers are obvious: 1. Impaired gastric emptying may favour the development of gastric ulceration, if it is grossly delayed, and that of duodenal ulceration, if it is inappropriately accelerated. 2. Disturbances of duodenal motility in the fasted state may disturb bicarbonate secretion and, thus, become theoretically ulcerogenic. 3. Duodenogastric reflux has been known to cause gastric ulceration for a long time. 4. Gastric ulcer was frequently accompanied by morphological and functional changes in the antrum (Wienbeck and Lubke, 1987). Urbanization has been currently fashionable with diets deficient in fibres and rich in refined carbohydrates (Tovey and Tunstal (1979), and this has been linked to ulceration because the diets contain low residue and non-masticating residue as well as being low in buffering proteins (Cleave, 1974; Burkitt and Trowel, 1975). Such diets have contributed to the worldwide increase in peptic ulcer cases in the last one hundred years (Baron, 1969). Brunt *et al* (1988) reported that high fibre could protect against ulceration by reducing the transit time (and therefore, the time colonic mucosa would be exposed to potential carcinogen) by altering bowel flora, and by diluting or binding the carcinogens. A flare up in a person who has ulcer by highly spiced foods that caused it remains plausible until it is proven as in certain habits like alcoholism (Grossman, 1981).

However, epidemiological studies in Africa and India, for example showed a high incidence of duodenal disease in rural areas, characterized by a high rate of duodenal ulcer compared to gastric ulcer; a high male to female ratio, a peak age incidence 10 years earlier than in western countries (Watt and Jones, 1993). The likelihood of dietary indiscretion has been widely accepted to be a cause of dyspepsia and peptic ulceration with a few studies existing that implicated dietary factors in causing or re – activating peptic ulceration (Jones, 1993). He suggested that differences in diets could be responsible for some of the regional and international differences in the incidence. For example, in the rice –eating belts of Southern India, duodenal ulcer was more common than in the wheat – eating areas in the North. In a Norwegian study, duodenal ulcer recurrence rates were found to be higher in people on a low – fibre diet, compared with people eating a normal or high – fibre diets (Jones. 1993). In Nigeria where diverse dietary dispositions exist, for example: the people of South West eat a lot of pepper, the South East and South South eat a lot vegetables, high fibre diets and the North subsist on mixed dietary habits with the consumption of fresh goat, cow milk (*fura denonu*), there is a paucity in the literature on clinical and epidemiologic studies apart from the early ones. Nigeria has been categorized by earlier studies as one of the high prevalent area for duodenal ulcers (DUs) in Africa (Amure, 1967, 1970). In a clinical study, 300 cases were reported and the presentations were similar to those in the western world, except that nausea and vomiting were more common and accounted for about 5% cases (Amure, 1967). Amure and Elegbe, (1975) reported that in Ibadan, gastric ulcers was largely a disease of associated with lower socioeconomic strata. It has also been shown that peptic ulcer was common among Nigerians (Adesola, 1971). In Nigeria, a high incidence of duodenal ulcers was reported in Western Nigeria (Kolawole and Solanke, 1973; Solanke, 1973, 1976). It has also been documented that there was a very low prevalence of gastric ulcer when compared to duodenal ulcers or gastritis in Nigerians (Cook, 1980).

In his experience, Cook (1980) reported the existing view that lower cases of GUs than DUs occurred in developing countries like Nigeria, Uganda, Zambia and Saudi Arabia. Arigbabu and Adekunle (1985) reported a 3.3 % incidence of gastric ulcer, 37.8 % of duodenal ulcers and majority of these gastric bleeding (58.8 %) resulted from acute gastric mucosal lesions in a 4 – year endoscopic study in Ife, a Semi – Urban area.

The aetiology of peptic ulcer disease among Nigerians had implicated local environmental factors and customs that are diet-dependent (Amure, 1965). It was in response to the earlier observation by Amure (1965, 1967, 1970) that our diets and dietary habits could provide a clue to the prevailing incidence of peptic ulcers that led to the increased clinical and experimental studies in the late 1970s to early 2000s by various Nigerian Physiologists and Clinicians as is reviewed in this article. A prevalence ratio of male: female of 3:2 for southern Nigeria and 3:1 for northern Nigeria have been reported (Amure, 1990) and that if aggressive factors exceeds defensive factors, it leads to ulceration but if defensive factors exceed aggressive factors, no ulcers occur (Amure, 1990). In Nigeria, the people are fond of highly spiced foods and are particularly liberal with their use of pepper. In addition, many Nigerians subsist on one or two small meals a day which apart from containing spices and pepper are composed principally of carbohydrates and small quantities of vegetables (Amure, 1965), except for the Efiks and Ibibios. Another local custom that may contribute to the aetiology of peptic ulcer disease was the universal use of powdered tobacco by women or the aged who chew it and men who smoked or snuffed it even in empty stomach (Amure, 1965). The consumption of palm wine and locally made illicit native gin were other major contributors to the incidence of ulcer (Amure, 1965). It has been implicated in acute pancreatitis where high alcoholism existed (Oyama *et al*, 1994; The National Institute on Alcohol Abuse and Alcoholism and the Office of Rare Diseases of National Institute of Health, 2003). It has been shown that two factors operated in Kenya, Ethiopia, Burundi and South India as aetiologic factors for DUs namely: that highly spiced diets with pepper have been shown to provoke copious acid secretion, and that diet which is bulky by its sheer weight tended to sag down the stomach putting tension on the hepto-duodenal ligament and the accompanying supra-duodenal vessels, thus creating ischaemic blanching at the point of attachment of the ligament, the so-called “critical area” for ulceration (Kolawole and Solanke,1973). This has also been confirmed in guinea pepper (*Piper Guinensis*) (Enyikwola, 1976). Pepper, a major spice common in hot appetizer during ill – and good-health in the form of “pepper soup” (Onokpite *et al*, 1990), usually taken hot may provide a good clue to ulcers and upper epigastric disorders. The relation of peptic incidence to a protein-diet has been emphasized (Cleave, 1962). Other commonly consumed substances shown to

provoke acid secretion and probably have possible role in the aetiology of peptic ulceration agree with postulation that high acidity caused the release of histamine and gastrin in the stomach (Konturek, 1974), either directly or indirectly. These substances include various types of kola nuts commonly eaten including *garcinia conrauana* (Ibu *et al*, 1986a, b, c; Okoi and Nwafor, 1989; Osim *et al*, 1991a), crude plantain extract (Osim *et al*, 1991b), aqueous natron, a tenderizer and preservative (Nneli and Nwafia, 2002, Nneli, 2000), fresh palm wine not standardized(Ibu *et al*,1986d, Osim *et al*, 1986, 1991), theophylline (cola nuts)(Osim, 1976), caffeine (coffee) (Debas *et al*, 1971; Cohen *et al*, 1972), Roselle(*Hibiscus Subariffa*)(*Yakura*) (Enyikwola *et al*, 1993). Natron has been linked to dyspepsia and heartburn (Nneli, 1991; Cohn – Jones, 1988). Also, a similar report using other substances have been documented (Gjesing, 1980; Babka and Castell, 1973). Plantain meal aggravated gastric wounds (Nneli and Osim, 1990, 2000). Other diets/dietary habits have been found to be ameliorative hence effective as antiulcerogenic agents. Cattle milk (*fura denonu*) have been reported to be a powerful defensive factor to peptic ulcers due to its high protein contents (*fura*-millet) and *denonu*(fresh milk)(Amure,1990). Dried unripe plantains-bananas have been employed as an anti - ulcerogen (Best *et al*, 1984) while a combined usage of alligator pepper and kola nut were reported to decrease acid secretion (Nwafor *et al*, 1990). Garri (grated and fried cassava) tapioca, potatoes, rice, yam and bread have been identified as commonest causes of ulcer in an epidemiological survey that confirmed the prevalence of ulcers in the Southern Nigeria involving Private Medical Practitioners (Songonuga and Elegbe, 1993). In India, doctors have used musapep to treat ulcers (Rangwani, 2006). Studies in Nigeria and other African countries showed that peptic ulcer has the highest incidence in the fourth and fifth decades and that gastric cancer also occurred in an age group at least 10 years younger than in the Western World, they suggested that endoscopy should be considered seriously for any person in the late 30s and above if they have even no 'alarm' symptoms (Obakpite *et al*, 1994; Malu and Sobala, 1999). Ndububa *et al* (2001) observed that duodenal ulcer remained the predominant pathology among Nigerian patients with upper gastrointestinal disorders while gastric ulcer was still relatively uncommon, and that *Helicobacter Pylori* infection played a major role in the development of peptic ulcer in Nigeria and duodenal ulcers, pyloric channel ulcers and gastroesophageal polyp disorders could be due to massive *H. Pylori* infection. The upper gastrointestinal series relating to endoscopic findings linked to *H. Pylori* incidence in Nigeria, Ghana have been published (Magrad *et al*, 1989; Olusanya, 1990; Coker and Akande, 1989; Holcombe *et al*, 1990; Smith *et al*, 1990). Mbagwu and Adeyemi (2006) reported that the mortality and morbidity rates of peptic ulcer disease (PUD) were quite high in Nigeria and other developing countries. Other observed rare causative factors of PUD include: cigarette smoking, certain foods and beverages such as caffeine containing foods, milk due to lack of enzymes in the stomach for it digestion, alcohol, spicy foods and genetic prediliction (Berardi, 1992; Soll, 1990). Some studies on admissions into medical wards gave clue to the prevalence when medical cases were categorized. Onwubere and Ike (1999) reported that GI diseases ranked third in a 5 –year survey at the University of Nigeria Teaching Hospital, Enugu with an incidence rate of 12.6% (males – 13.3%; females – 11.45%) while Osuafor and Ele, (2004) reported on a 1990 – 1992 data which ranked peptic ulcer 14th with preponderance rate of 2.1% though the total population was lower than 200 patients.

Food Intolerance, Toxins, Allergic Substances in Diets, Food Contaminants and Pollutants and Gastrointestinal Disorders

Common gastrointestinal symptoms associated with adverse reaction to ingested food include: emesis, diarrhoea, abdominal pains/cramping and bleeding (James *et al*, 1996). These symptoms are attributable to many factors including mucosal enzymes deficiencies such as lactase, intolerance to the ingested food, e.g. sorbitol and direct toxic or chemical effect of the food (Hyams, 1983). Lactose, cow milk, soy protein and wheat are the most common examples of food not tolerated (Strobel, 1995). A common G.I. toxigenic food is gluten found in wheat. This causes toxicity of the G.I mucosa, destroying the absorptive surface of the intestinal villi. This causes the brush borders to be thickened and the lamina propria infiltrated with inflammatory cells. The final result is malabsorption (sprue) with impaired intake and transport of nutrients (Kirsner, 1995). Tropical sprue resulting from nutritional and bacterial alterations also causes inflammation of the intestinal mucosa (Edwards *et al*, 1995). A failure in the normal digestive process has been reported as a possible cause of food associated G.I disease (Ferguson, 1995) e.g., a breakdown in normally efficient bowel digestive process may help toxins and antigens from food to penetrate the intestinal surface and enter the

systemic circulation (Edwards *et al,* 1995). G.I reactions resulting from cell mediated delayed hypersensitivity processes to food and also reactions resulting from antibody dependent cytotoxicity have been implicated in specific G.I diseases. They have been found to involve natural killer cells and T- lymphocytes (Ferguson, 1995). The G.I especially the specialized gut associated lymphoid tissue is a major immunologic organ of the human body. It apparently plays an active role in the suppression of these adverse reactions to food substances (Strobel, 1995). As a solution to the above food associated diseases, diet manipulation therapy has been recommended (Strobel, 1995).

Role of Irregular Bowel Emptying on the Development of Constipation

Irregular bowel emptying that has developed through a lifetime habit of inhibition of the natural defeacation reflex has been found to be a frequent functional cause of constipation (Awasan, 1994). Clinical experience shows that if one fails to allow defeacation to occur when reflex is excited or if one overuses laxatives to take the place of natural bowel movement functions, the reflex becomes progressively less strong over a period of time and the colon becomes atonic. As a result, the establishment of regular bowel emptying in life will prevent the development of constipation. This can be best done in the morning after breakfast when gastrocolic and duodenocolic reflexes cause mass movement in the large intestine. The inhibition of normal defeacation reflex has been documented to lead to the production of toxins and excessive water reabsorption from the faeces and thus, may cause intestinal wall excoriation and bleeding during defeacation (Strocchi and Levitt, 1993).

Role of types of Food in the Production of Intestinal Gas

Gases within the G.I.T arise from only three sources namely swallowed air, intraluminal production and diffusion from blood (Strocchi and Levitt, 1993). The gases commonly passed out as flatus include: nitrogen, oxygen, carbon dioxide, hydrogen and methane (Strocchi and Levitt, 1993). Certain types of food have been found to cause greater expulsion of flatus than other types. These include: beans;cabbage, onions, corn and certain irritant food such as vinegar (Strocchi and Levitt, 1993). Most of these foods serve as suitable media for gas forming bacteria. Hydrogen gas is produced solely by bacterial fermentation of carbohydrate and proteins derived from the diet (Steggards, 1968). The quantity of carbohydrate introduced into the colon has been found to be dependent on several factors including the type and amount ingested and the completeness of digestion and absorption by the small bowel (Steggards, 1968). Poorly digested oligosaccharides found in corn, wheat, oat and potatoes may then remain inside the colon and may be associated with increased colonic hydrogen production (Levit *et al,* 1987).

In the studies on fructose and sorbitol intolerance in humans, it was observed that both could increase colonic hydrogen production, thus increasing the incidence of flatulence (Hyams, 1995; Ravich *et al,* 1985). Fructose is found in high quantities in soft drinks while sorbitol is a sugar alcohol used as sweetener in sugar free products (Hyams, 1995; Ravich *et al,* 1985). Beans has also been found to contain an indigestible sugar that passes into the colon and becomes a favorable medium for colonic bacteria (Strocchi and Levitt, 1993). Despite the above findings, excess gas has been known to result from the irritation of the large bowel. This irritation promotes rapid peristaltic expulsion of gases before they can be absorbed (Strocchi and Levitt, 1993).

Effect of Dietary Hygiene on the development of G.I disorders

Improper dietary hygiene is a common factor that is linked to most G.I disorders. These habits range from personal hygiene to kitchen utensils and food contamination. The above conditions commonly results to gastroenteritis. Gastroenteritis could be commonly caused by bacteria, fungi, viruses (Edwards *et al,* 1995). The commonest bacteria implicated includes *Escherichia coli (E. coli), Cholera bacillus,Salmonella species* and *Staphylococcus aureus.* They cause wide range of diseases leading to toxin production, water and electrolyte loss (Edwards *et al,* 1995). *Helicobacter pylori* formerly *Campylobacter pylori* infection is associated with a variety of clinical outcomes including the aetiology of gastric cancer and duodenal ulcers (Blaser, 1998), gastric ulcers and probably non-ulcer dyspepsia (Marshall *et al,* 1985; Edwards *et al,* 1995c; Rieggs *et al,* 1995). Convincing evidence also abound on the role of *H. pylori* in the development of the three distinct diseases namely: G.U, D.U and G.C (Graham, 1991). Socioeconomic factors rather than ethnicity has been reported to be the key to the difference between the developed and developing nations since the highest incidence of *Helicobacter Pylori* infection was highest in the poorest people (IARC, 1994). The eradication of *H. pylori* from the stomach reduced the recurrence of D.U (Rauws and Tytgat, 1990; Mohammed

et al, 1994). Earlier upper gastrointestinal endoscopic studies showed a 32.7 percent occurrence in an urban hospital in Northern Nigeria (Andrew *et al,* 1995), 35.6 percent in Zaria, Kaduna State (Malu *et al*, 1990), while in Enugu a 65.13 percent was reported (Picardo and Nwokediuko, 2001). However, a lower incidence has also been documented in children in Northern Nigeria (Holcombe *et al,* 1993).

Role of Alcohol, Nicotine and other Gastric Irritants

Alcohol, nicotine and other irritant substances have been implicated as major factors in the pathophysiology of most G.I disorders (Poll *et al,* 1989; Amure, 1967; Solanke, 1973,1976; Oyama *et al,* 1994; The National Institute of Alcohol Abuse and Alcoholism and the Office of rare Diseases of National Institute of Health (2003);Enyikwola, 1976; Ibu *et al,* 1986d; Osim *et al,* 1986, 1991b; Debas *et al,* 1971; Cohen *et al,* 1972). Cigarette smoking has been documented to be of an aetiological importance in peptic ulcer disease and also prevents its healing (Adewole, 2002; Edwards *et al,* 1995; Rangwani, 2006). Alcohol, especially the spirits is potent irritants of the G.I.T and they can worsen the already existing G.I. disorder (Amure, 1990). Rectal, but not colonic cancer is associated with high alcohol intake (Brunt *et al*, 1988) however, alcohol in moderate amounts have been noted to be non harmful (Edwards *et al,* 1995).

Conclusion

The role played by diet\dietary habits in the aetiology of most G.I disorders is inexhaustive. Overall, the high consumption of fresh fruits and vegetables coupled with a high intake of antioxidants and carotenoids have been convincingly associated with reduced risk of G.I. cancer. In contrast, a variety of food preserved by salting, smoking, prickling and irritant foods have been proved to increase the risk. The recent warning by the Nigeria National Agency For Food and Drug Administration and Control (NAFDAC) that nitrosamine has been found in our beer is an indication that we are in for another explosion of gastric cancers. Dietary manipulations are the mainstay therapy for all diet-induced GI disorders (Strobel, 1995). It has been observed that the major therapy for all food allergies and intolerance is the elimination of the offensive food from diet (Carter, 1995). This highlights the earlier suggestions that nutritionists / dieticians should be actively involved in the design and overall management of diet in order to avoid improper dietary advice. This can go a long way in helping us to avoid the risk of nutritional deficiencies and electrolyte disturbances as observed earlier (De Vizia *et al*, 1995). So, there is a call for all and sundry to be careful of what we consume for life expectancy seems to depend on the GIT just as earlier warned (Amure, 1990).

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