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Study Of Impact Of Diet And Lifestyle Factors On Prevalence Of Gastric Cancer

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Abstract

The second most frequent cancer worldwide and the second greatest cause of cancer-related death is gastric cancer. However, the incidence rates in various geographical areas vary significantly. While the prevalence of stomach cancer has been declining, there are reports that in some areas, the prevalence of gastric cardia cancer is increasing. Infection with *Helicobacter pylori* (*H. pylori*) is a significant contributor to the development of non-cardia gastric cancer, and evidence has been gathered supporting the primary prevention of gastric cancer by the elimination of H. pylori. Additionally implicated are metabolic, dietary, and behavioral variables. Although addressing these additional factors may improve health, it is uncertain how doing so will actually affect cancer prevention. Anatomically, gastric adenocarcinomas and gastro-esophageal junction adenocarcinomas are distinguished, and histologically, different usage and intestine kinds are distinguished. *Helicobacter pylori (H pylori)* infection and dietary variables are the primary risk factors for distal gastric cancer, but obesity and gastroesophageal reflux syndrome are significant contributors to the development of proximal stomach cancer. Eliminating H. pylori is a crucial primary prevention measure for stomach cancer. A healthy lifestyle will help avoid stomach cancer. This includes cutting back on red meat and salted and smoked foods, increasing the number of fruits and vegetables in your diet, quitting smoking, and reducing your alcohol use. The review article focuses on mainly the impact of different dietary factors on the incidence of gastric cancer.

Keywords: gastric cancer, diet, lifestyle, prevalence, world, association

INTRODUCTION:

One of the main causes of death worldwide is cancer, and its prevalence is rising in the United States (Asombang and Kelly 2012; Zali *et al*., 2011). Evidence indicates that in 2012, six types of cancer—lung, breast, colorectal, prostate, and gastric—accounted for 55% of the worldwide burden of cancer include liver cancer (Ferlay *et al*. 2015). The most typical kind of upper gastrointestinal malignancies among them is gastric cancer (Krejs *et al*. 2010). With an annual incidence of around one million new cases worldwide, this condition

ranked fifth among all cancers in GLOBOCAN 2012's analysis. This cancer's incidence rate has changed significantly in recent years, according to this data. It accounts for 723,100 fatalities (8.8% of all cancer deaths) and is the third most prevalent reason for cancer mortality in both sexes globally (Ferlay *et al*. 2015). Gastric cancer is more common in certain civilizations than others; East Asia, Central and South America, and Eastern Europe have the highest rates, while Africa and North America have the lowest rates (Nagini *et al*. 2012). The proper identification of the risk factors and underlying causes of this illness, as well as the management of these variables, are necessary for prevention of stomach cancer due to the multifaceted character of this disease (Yoon and Kim 2015). The diffuse subtype exhibits weakly cohesive single cells without gland development (Bosman *et al*. 2010). In addition, despite prior meta-analyses examining the links between red or processed meat and stomach cancer, none have looked at the data on the associations between white meat and stomach cancer. Because most previous research largely concentrated on the effect of red or processed meat, it is crucial to explore the specific information of white meat as a source of protein consumption. (Zhou *et al*. 2017). Comparatively speaking, the proximal gastric cancer rate is greater than the distal one. This trend may be explained by improved food preservation, higher intakes of fresh fruits and vegetables, and the elimination of Helicobacter pylori (Sitarz *et al*. 2018). This complex illness, which is influenced by both environmental and hereditary factors, is frequently detected in its advanced stages (Carcas 2014; Karimi *et al*. 2014) and Additionally, high-fat, high-salt, and high-nitrogen diets, a history of Helicobacter pylori infection, the EBV virus, genetic factors, pre-malignant stomach lesions, and cigarette use have all been identified as risk factors for gastric cancer (Gonzalez and Agudo 2012, Dikshit *et al*. 2011, Matsuo *et al*. 2011, Nabizadeh *et al*., 2011, Hu *et al*. 2011, Babaei *et al*. 2010, Long *et al*. 2010).

DISCUSSION:

Globally, roughly 990,000 people are diagnosed with stomach cancer each year, and of those, about 738,000 will pass away from it (Ferlay *et al*. 2010) and making it the second most prevalent cause of cancer-related mortality and the fourth most common cancer occurrence (Jemal *et al. 2010).* A country-by-country analysis of the data was done using the United Nations' geoschema. The regions and individual nations of the United Nations geoscheme were used to analyse the data. Based on estimates of regional prevalence, it is estimated that 4.4 billion people worldwide—more than half the world's population—had H. pylori infections in 2015. However, there was significant geographical variance. Africa (almost 70%) and Oceania (about 24%) had the greatest and lowest combined prevalences of H. pylori infection, respectively. Nigeria (87.7%) and Switzerland (18.9%) had the lowest and highest rates of H. pylori infection, respectively. The H. pylori prevalence trend through time was examined for two time periods (1970-1999 and 2000-2016). After 2000, the prevalence of H. pylori decreased in Europe (from 48.8% to 39.8%), Northern America (from 42.7% to 26.6%), and Oceania (from 26.6% to 18.7%) (Hooi *et al.*2017). Numerous risk factors for gastric cancer were found and divided into nine main categories, including diet, lifestyle, genetic predisposition, family history, treatments and medical conditions, infections, demographic characteristics, occupational exposure, and ionizing radiation.

REGION	BEFORE (2000 *H. pylori* infection percentage)	AFTER (2000 *H. pylori* infection percentage)
EUROPE	48.8 %	39.8%
NORTHERN AMERICA	42.7%	26.6%
OCEANIA	26.6%	18.7%

Table. 1. Tabular representation of decrease in *H. pylori* infection in different places of world

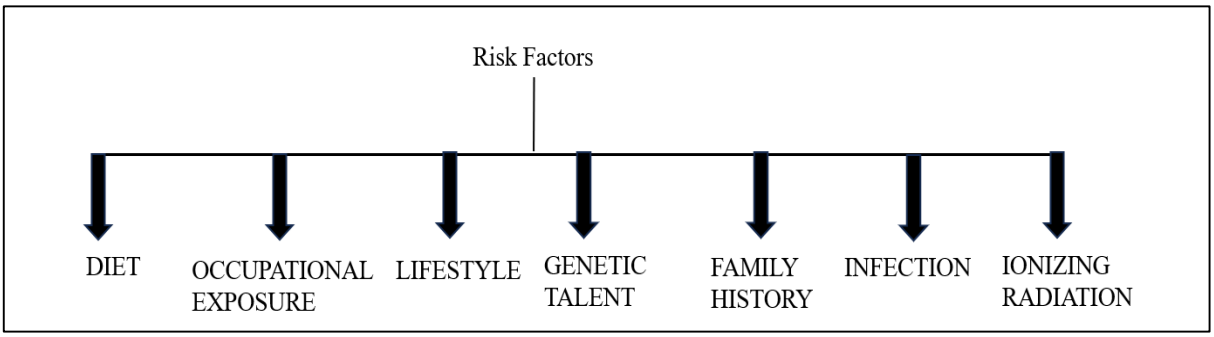

Figure1: Risk Factors related to Gastric Cancer

DIET: According to several studies (Amin *et al*. 2015, Daniyal *et al*. 2015, Bertuccio *et al*. 2013, Sadhier *et al*. 2013, Wu *et al*. 2013, Nemati *et al*. 2012, Compare *et al*. 2010, Krejs 2010, Shen *et al*. 2009), food and eating habits are one of the most significant determinants in the incidence of gastric cancer. The World Cancer Research Fund and American Institute for Cancer Research (WCRF/AICR) have designated salt as one of the most significant risk factors for stomach cancer. Adequate quantities of salt If a gastric bypass was required, excessive eating mucosa stimulant, causing atrophic gastritis, increased DNA synthesis and cell growth provide the causes of stomach cancer (Fang *et al. 2015).*

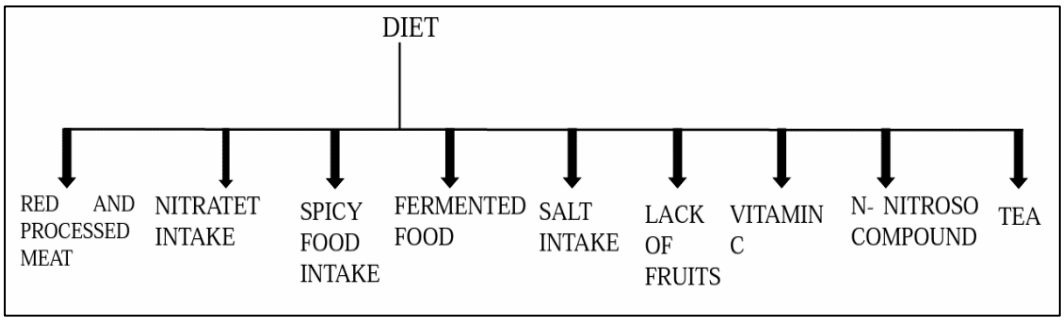

Figure 2: Dietary Factors Related to Gastric Cancer

RED MEAT AND PROCESSED MEAT: Several speculative processes might account for the links between red and processed meat intake and cancer outcomes. First, the processing, preservation, and high-temperature cooking of meat may produce carcinogens such HCAs, PAHs, nitrate, and NOCs, which are thought to be crucial in the development of several cancers. (Cross *et al*. 2011, Ferguson 2002, Skog Johansson & Jagerstad 1998, Tricker & Preussmann 1991, Wang & Jiang 2012). Meat is also abundant in several chemicals, including nitrate, heterocyclic amines (HCAs), polycyclic aromatic hydrocarbons (PAHs), and N-nitroso compounds (NOCs), which have been linked to an elevated risk of some cancers (Mirvish 1995, Moorthy *et al.* 2015, Sugimura *et al.* 2004). According to Kim *et al*. (2019), consuming more red meat was linked to an increased risk of stomach cancer.

NITRATE INTAKE: The risk of developing stomach cancer and dietary consumption of nitrates, nitrites, and nitrosamines are examined in this study for the first time. We discovered that eating foods high in nitrates was associated with a lower risk of developing gastric cancer but eating foods high in nitrites and NDMA was associated with a higher risk of developing cancer. Similar findings were seen in the case-control studies when using stratifying analysis for study design, and the cohort studies also showed the effects of this trend. The chemical symbols for nitrate and nitrite are NO3 and NO2, respectively. Nitrates and nitrites are two categories of inorganic compounds that consist of a single nitrogen atom (N) and a number of oxygen atoms (O). Prior to being converted to nitrites, it is thought that nitrates are a rather innocuous substance. In the mouth, bacteria may convert nitrates into nitrites, which can subsequently be ingested. Nitrites are transformed into nitrous acid when they come into contact with the stomach's extremely acidic secretions. Nitrous acid then combines with amines to create nitrosamines (Kobayashi *et al*. 2015*)*. Additionally, it has been demonstrated that nitrites can cause cancer in animals when combined with amines or amides. The majority of nitrosamines can cause DNA adductions and gene mutations in animals, which can lead to carcinogenesis (Bryan *et al.* 2012).

SPICY FOOD INTAKE: Spicy food intake refers to the direct ingestion of fresh chilies, the addition of fresh or dried chilies, chilly oil/sauce/paste, curry, or other 'hot' spices when cooking, or the addition of chilly oil/sauce/paste to food while eating. Participants were questioned about their intake of spicy foods in the previous month at both the baseline and follow-up questionnaires. Response options included: never/almost never, sometimes, 1-2 days per week, 3-5 days per week, or 6-7 days per week. The primary biologically active ingredient in spicy foods is capsaicin has demonstrated a number of carcinogenic effects in studies on animals, including causing mucosal damage (Mann *et al*. 1997). Capsaicin, on the other hand, has also shown anti-carcinogenic effects via affecting GI cancer risk factors, such as preventing *Helicobacter pylori (H. pylori)* from growing and reducing the body fat (Jones *et al.* 2006).

FERMENTED FOOD: Foods or drinks created by managed microbial growth and enzymatic conversion of food components are referred to as fermented foods (Marco *et al.* 2017). Historically, a wide variety of foods have been fermented, including meat and fish, dairy, vegetables, soybeans, other legumes, grains, and fruits.

There are countless varieties of fermented foods due to the numerous variables in the fermentation process, including the microbes, dietary components, and environmental factors. Because the production of antimicrobial metabolites (such as organic acids, ethanol, and bacteriocins) lowers the danger of infection with harmful bacteria, food fermentation has historically been used as a technique of preservation. Some foods, like olives, are inedible without fermentation that eliminates the bitter phenolic molecule, hence fermentation is also employed to improve the organoleptic features (e.g., taste and texture). Foods are fermented using one of two major techniques. First, foods can naturally ferment; these processes are sometimes referred to as "wild ferments" or "spontaneous ferments," and they involve using microorganisms that are already present in the raw food or processing environment. Examples of such foods include sauerkraut, kimchi, and several fermented soy products. Second, by adding starter cultures, sometimes referred to as "culture-dependent ferments," to foods, such as kefir, kombucha, and natto, meals can be fermented (Rezac *et al*. 2018).

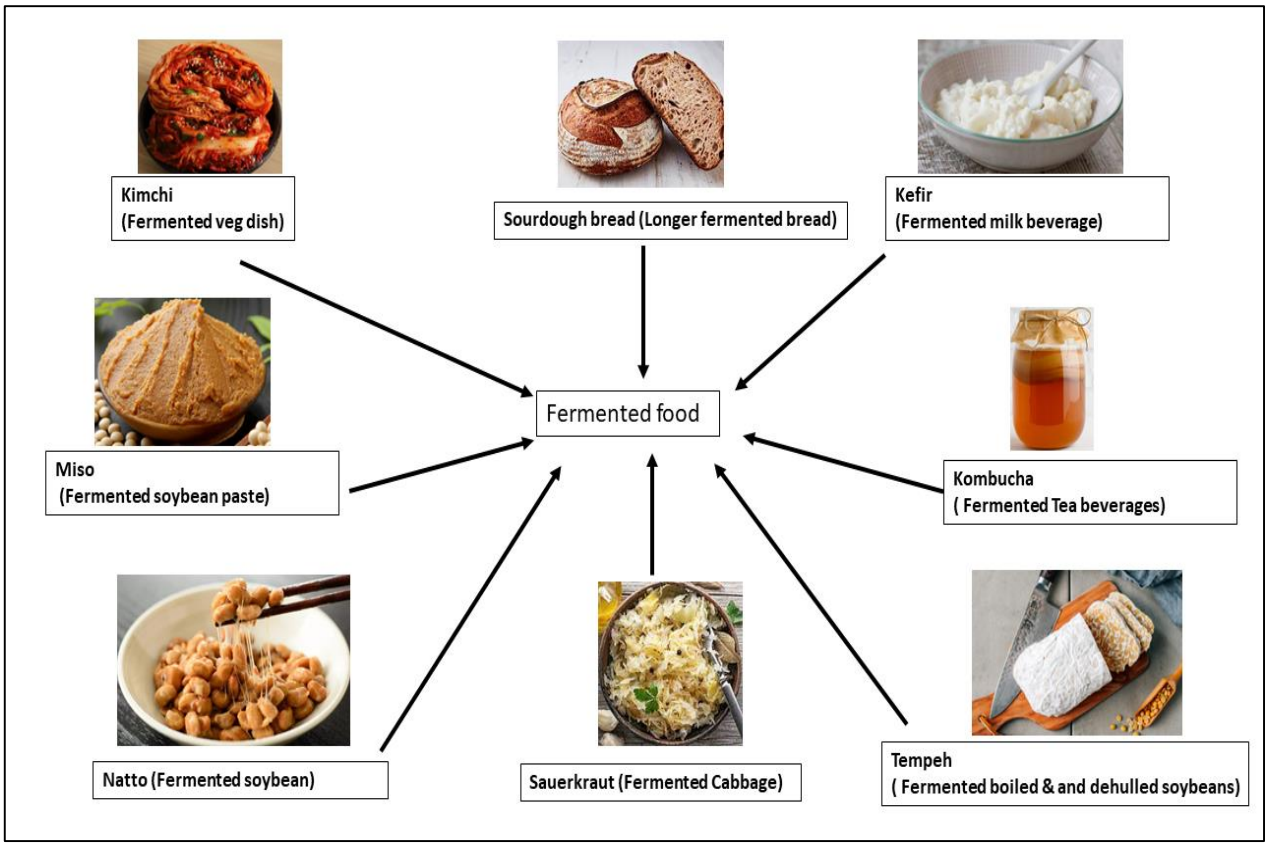

Figure 3: Figure showing the fermented foods which are reported to contribute to gastric carcinoma

SALT INTAKE: One of the most significant risk factors for stomach cancer, according to the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR), is salt consumption. Although adequate amounts of salt were required, Fang et al. suggested that excessive consumption might act as a gastric mucosa stimulant, causing atrophic gastritis, increased DNA synthesis, and cell proliferation, and thus serving as the reason for the prevalence of gastric cancer (Fang *et al*. 2015). Excessive salt consumption was linked to an increased risk of stomach cancer, according to data from earlier research. More than 6 grammes of salt per day was thought to be linked to 24% of stomach cancer incidences in the UK (31% of males and 12% of women) (Parkin *et al.* 2011).

LACK OF FRUITS: According to six studies, a further risk factor for stomach cancer is a lack of or insufficient consumption of fresh fruit and vegetables. Researchers found that this was the case in their study (Nemati *et al*. 2012). In this context, several studies have demonstrated that increasing physical activity might lower the incidence of stomach cancer by around 66-75% intake of vegetables and fruits as part of a healthy diet concurrently reduced intake of salt-rich foods (Sendler *et al*. 2001). An increased intake of fresh fruit and vegetables and a restriction on the use of salt and salt-preserved foods may be effective dietary interventions for prevention. The chance of developing the condition may also be decreased by making lifestyle changes, such as increasing physical activity and quitting smoking. Rich in folate, carotenoids, vitamin C, and phytochemicals, fruits and vegetables may act as a barrier against the development of cancer (Elingarami *et*

al. 2014). Vegetable eating was shown to have a preventative effect, mostly for GC of the intestinal type. Consumption of citrus fruits may help to prevent stomach cancer. In a recent analysis, the International Agency for Research on Cancer (IARC) said that eating more fruit and vegetables "probably" and "possibly" lowers the incidence of GCs (International Agency for Research on Cancer, 2003).

VITAMIN C: The decrease of oxidative stress at the stomach mucosal/luminal surface is facilitated by vitamin Through the regeneration of urate, glutathione, beta-carotene, and -tocopherol (vitamin E), it also supports the antioxidant defense system. In order to repair oxidised vitamin E (tocopheryl radical) or vitamin C (dehydroascorbic acid; DHA), glutathione must first produce a thionyl radical (GS) (Pasupathil *et al*. 2009). Since then, it has been clear how important vitamin C is for the production of collagen. A lack of vitamin C prevents the Fe2+/2-oxoglutarate (2-OG)-dependent dioxygenase prolyl hydroxylase from properly hydroxylating the amino acid proline into hydroxyproline. Vitamin C is the primary and most important water-soluble antioxidant in human blood and tissues as a result of its redox capacity. By using the ascorbyl radical as an intermediary, vitamin C can act as an electron donor to create DHA. Thus, vitamin C may neutralize hypochlorous acid (HOCl), scavenge superoxide anion radicals (O2), singlet oxygen (1O2), and hydroxyl radicals (OH), and prevent lipid peroxidation. Hydrogen peroxide (H2O2) cannot be scavenged or neutralized by vitamin C. Instead, by preventing catalase action, vitamin C could increase its toxicity. Myeloperoxidase (MPO), an enzyme found in neutrophils, converts H2O2 and Cl into HOCl (Blaszczak *et al.* 2019). In situations when hydroxyl radicals are produced via Fenton chemistry, such as at supraphysiological levels or in the presence of transition metal ions like iron or copper, vitamin C may have a pro-oxidant effect. For instance, consuming large amounts of ascorbic acid orally might have a pro-oxidant impact on eating red meat (heme). Lipid peroxidation happens in the stomach due to iron toxicity. The combination of ascorbic acid, however they have synergistic antioxidant properties and reduce lipid peroxidation when combined with polyphenols (such as catechins).is avoided. The development of ALEs in the body can be prevented by polyphenols found in sage or rosemary by up to 100% the stomach from red meat.

N-NITROSO COMPOUND: Studies conducted at the period in Europe revealed that the countries with the greatest incidence of gastric cancer were Finland and Iceland, where the consumption of smoked fish and meat was particularly high. This prompted researchers to further investigate the role of smoked foods' polycyclic aromatic hydrocarbon (PAH) content in gastric carcinogenesis. Since then, benzo[a]pyrene and other PAHs produced in smoked food have been linked to many regions of the world with high prevalence of stomach cancer. Additionally, some cooking techniques may be linked to an elevated risk of gastric cancer. These include sun drying, curing, and pickling, all of which promote the synthesis of N-nitroso compound, roasting, grilling, baking, and deep frying of meats in open furnaces, as well as roasting, grilling, curing, and roasting of vegetables (Rubenstein *et al*. 2010)

TEA: Green tea is well recognized for having catechins, which are polyphenols. Green tea has been shown to contain antioxidants, and both in vitro and in vivo investigations have demonstrated that these antioxidants can prevent polyphenols from becoming nitrosated (Stewart *et al*. 2003). Additionally, recent research proposed additional potential mechanisms for green tea's anti-cancer effects, including modulation of signal transduction pathways that inhibit cell proliferation and transformation, induction of apoptosis and cell cycle arrest, inhibition of tumour invasion, and angiogenesis (Su Y *et al*. 2007).

The use of black tea or coffee does not, however, appear to have any impact on the chance of developing stomach cancer. The most significant vitamins and derivatives found in vegetables and fruit, such as vitamin C and carotenoids, appear to lower the risk of stomach cancer in this comparison, although there is still some uncertainty surrounding vitamin E (Tsubono *et al. 2000)*.

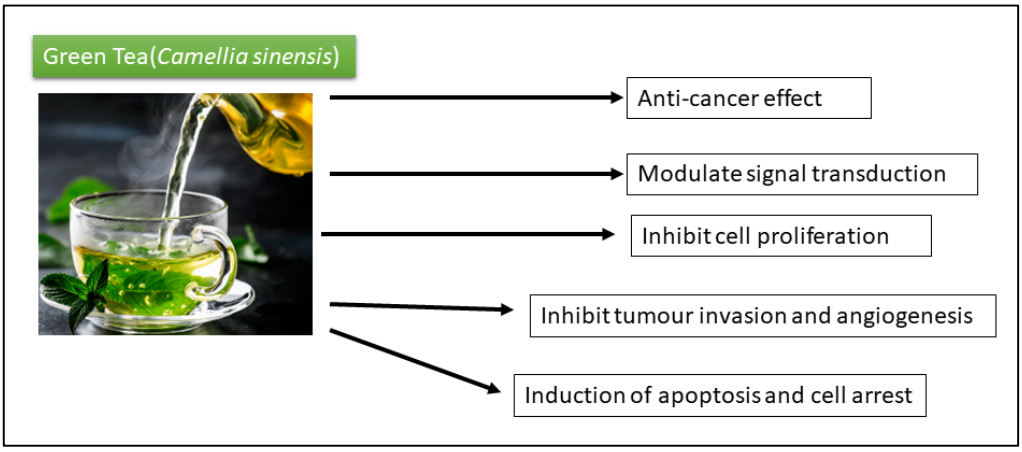

Figure 4: Figure showing the green tea extracts which are reported to exert anti cancer effects in gastric carcinoma

LIFESTYLE: A person's or a group's lifestyle really refers to their interests, attitudes, behaviours, qualities, and behavioural orientations. It also refers to their specific way of living.

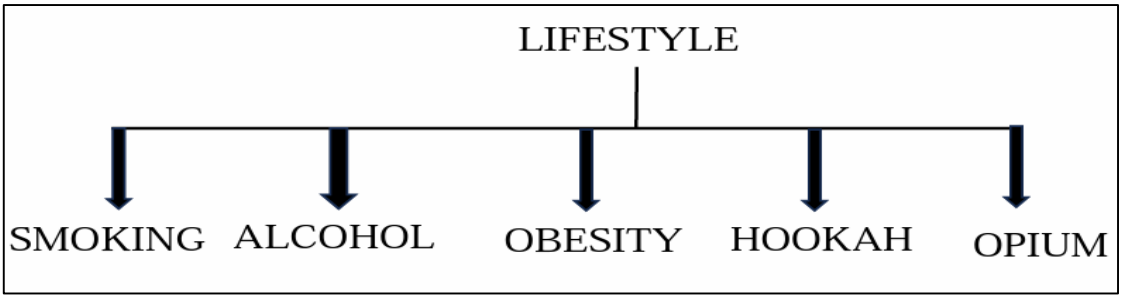

Figure 5: Figure showing factors Related to Gastric Cancer

SMOKING: Although the link between smoking and the development of various other cancers has long been known, it wasn't until 2002 that the International Agency for Research on Cancer declared that there was "sufficient" proof linking smoking to stomach cancer. Between men and women, as well as between developed and developing nations, there are significant differences in the percentage of stomach cancer cases that may be linked to smoking. Considering the variations in smoking prevalence by gender and location, as anticipated. Analysis from earlier times has revealed variance in terms of13–16% of instances of stomach cancer are believed to be caused by sex. Men are 4-7% more likely than women to smoke (Tredaniel *et al.1997).* Current smoking prevalence has certain drawbacks, and the accuracy of PAF estimations also depends on the calibre of the data gathered from each source.

Despite the fact that national surveys carried out in each country were primarily used to determine the prevalence of smoking, various methodologies may have been used in different research, particularly with regard to age restrictions and the definition of current smoking. Additionally, factors that affect the cumulative risks of smoking for malignancies, which have a relatively long latency, include the age at which smoking first started, the length of smoking, the number of cigarettes smoked daily, and the type of tobacco product used (such as cigarettes, cigars, bidis, etc.) (Peleteiro et al. 2012).

ALCOHOL CONSUMPTION: Regarding the effects of alcohol, it can be stated that alcohol triggers a cancer-stimulating process that involves a persistent inflammatory response to the cytokine and ethanol metabolite toxicity resulting in a rise in nitrosamine consumption (Bartsch and Nair 2005). Alcohol is a popular beverage. It is scientifically conceivable that drinking alcohol increases the chance of developing gastric cancer since ethanol is a fat-soluble substance that may harm the stomach mucosa. Its metabolite, acetaldehyde, may be harmful locally and be linked to the development of stomach cancer (Bao *et al*. 2001-05). Disrupting the equilibrium between external invasion and gastric mucosal defense is a factor in the pathophysiology of ethanol-induced gastric mucosal injury (Steevens *et al.* 2010).

OBESITY: Obesity is a major issue in contemporary society and has been linked to a number of illnesses, including stomach cancer. In contrast to people persons having a body mass index (BMI) of less than 25BMIs

between 30 and 35 had a 2-fold increase, while the between esophagogastric cancer risk increases thrice in those over 40gastric cancer junctional, including the cardia (Hoyo *et al. 2012).* Several mechanisms have been proposed. Abdominal fat may directly cause GERD (Gastroesophageal reflux disease), a risk factor for esophageal cancer and cardia gastric cancer.
Moreover, fat is metabolically active and produces numerous compounds that circulate in the body. These metabolic products, such as insulin-like growth factor and leptin, have been associated with malignancies, possibly through the induction of progrowth changes in the cell cycle, decreased cell death, and proneoplastic cellular changes (Aleman *et al.* 2014).

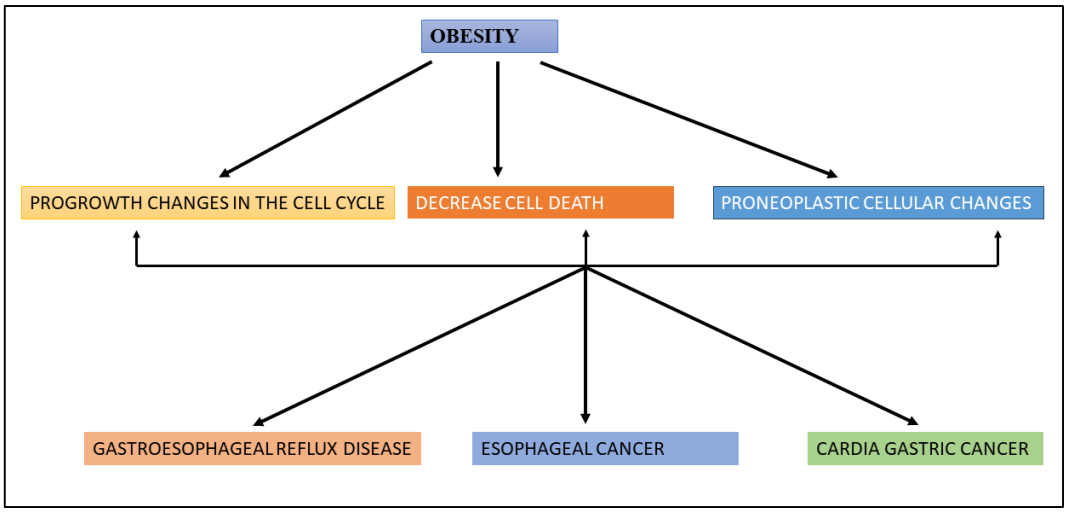

Figure 6: Figure showing the obesity factors contributing to gastric carcinoma

HOOKAH: Due in part to the widespread perception that hookah is safer than cigarettes, it is becoming more and more popular among young people. We did discover a threefold increased risk of stomach cancer, though. In another investigation, a link between hookah smoking and stomach cancer was demonstrated. Although three recent studies showed that hookah smoke contains a wide variety of carcinogenic and toxic substances, including nitrosamines, polycyclic aromatic hydrocarbons, primary aromatic amines, carbon monoxide, and various furanic compounds, such as 5-(hydroxymethyl)-2-furaldehyde, the exact mechanism by which hookah increases the risk of gastric cancer is not fully understood (Tramacere *et al*. 2011)

OPIUM: The use of opium, especially smoking, is significantly associated with a greater risk of stomach cancer. Although we were unable to gather information about opium usage in order to determine the dose-response relationship, the robust hazard ratio estimates obtained after adjusting for other smoking habits and lifestyle characteristics provide evidence that confounding effects from other factors are less likely. On the other hand, it is impossible to overlook the existence of unidentified confounders, especially those that share carcinogenic pathways with smoking (Shakeri *et al*. 2013).

OCCUPATION: The influence of occupational exposure in raising the risk of stomach cancer was highlighted in this investigation by two studies (Welling *et al*. 2015, Ji and Hemminki 2006). Ji and Hemminki (2006) found that manual laborers and farmers had a greater chance of developing stomach cancer. Cement and mineral dust were thought to be the most significant occupational risk factors for developing stomach cancer. Similarly, findings from research by Welling *et al*. (2015) revealed that persons who were exposed to chromium at work had a 27% greater chance of developing stomach cancer than those who weren't. Several possibilities have been put out; however, it is not entirely clear how specific vocations and occupational exposures are linked to stomach cancer. The highest risk groups appear to be those working in "dusty industries" (such as foundry workers, wood workers, grain farmers, coal miners, and textile machine operators), as well as occupations that require exposure to "high temperatures" (such as metal smelting/refining furnacemen, blacksmiths, railway engine drivers, boilermen, and firemen) (Raj *et al.* 2003). According to the "dust hypothesis," organic and mineral dusts are breathed, caught in the mucus layer of the airways, cleaned by the cilia, and then either expectorated or ingested. These abrasive and potentially cancer-causing substances, such as N-nitrosamines, which are widely used in the rubber, metal, agricultural, and leather sectors, come into direct contact with the gastrointestinal mucosa when eaten (Raj *et al.* 2003). Several of the relationships we found had biological plausibility since the IARC has classified rubber, nitrates/nitrites, asbestos, and lead

compounds as stomach carcinogens or likely gastric carcinogens in humans. Additionally, the IARC's classification of X- and gamma radiation as Group I gastric carcinogens is consistent with our finding that jobs involving exposure to "Radiation and Magnetic Fields" were linked to a higher risk of developing diffuse-type gastric cancer, with a 2-fold significantly high. In conjunction with the host's genetic, nutritional, microbial, and environmental variables, these substances act directly on the stomach epithelial lining, absorb radiation, or cause harm as a result of radiation exposure (Polk *et al*. 2010).

GENETIC TALENT: One of the causes of cancer in humans is the genetic field, which describes a person's features at the genome level. The primary elements of the study of genetics are mutations and polymorphisms, which modify protein function or expression levels. There is a connection between the different cytokine gene polymorphisms, which are immune system agents, and stomach cancer. The findings of a meta-analysis on the relationship between cytokine gene polymorphisms and the risk of precancerous lesions revealed that several of them, including the IL1RNVNTR gene polymorphism, were related to the risk of these lesions (Peleteiro *et al*. 2010).

FAMILY HISTORY: A positive family history may be a risk factor due to a shared environment, such as the transmission of H. pylori from parents to children, or due to genetic characteristics that run in the family. Two patterns of risk change following migration have been identified by studies. First, immigrants' and first-generation post immigrants' risk of stomach cancer approaches that of the population of origin but does not yet reach the risk of the host population; it takes at least two generations to reach the risk levels of the adopted nation (Yaghoobi *et al*. 2009) and second, rather than present residency, location of birth may be a better predictor of the risk of developing stomach cancer (Coggon *et al*. 1990)

INFECTION: About 50% of people have *H. pylori* infection population (Fock *et al*. 2010). For non-cardiac gastric adenocarcinoma, it is the most significant causative factor. A pooled odds ratio (OR) of 2.97 (95% CI 2.34-3.77) for *H. pylori* infection for non-cardia cancer was found by the Helicobacter and Cancer Collaborative Group after analysis of 12 prospective case-control studies. In contrast, no statistically significant connection with cardia tumors was found. The OR for non-cardia cancer increased to 5.93 (95% CI 3.41-10.3) when the pooled analysis was limited to cases that occurred at least ten years after the diagnosis of H. pylori. This OR is now regarded as the best estimate of the relative risk (RR) of non-cardia cancer associated with H. pylori infection. In industrialized and developing nations, respectively, the prevalence of H. pylori is on average 35% and 85% (Helicobacter and Cancer Collaborative Group).

IONIZING RADIATION: Radiation has been linked to a higher incidence of stomach cancer among survivors of Hiroshima and Nagasaki, according to long-term follow-up studies (Preston *et al*. 2007). Gamma radiation is one type of radiation that may contribute to the development of stomach cancer (Cogliano *et al*. 2011), with radiation being linked to roughly 1% of gastric cancer cases reported in the UK (Parkin *et al*. 2011). In their study, Dong et al. came to the conclusion that enhanced gamma radiation sensitivity was related to an increased risk of gastric cancer; those who were more sensitive to gamma radiation were at a higher risk for stomach cancer (Dong *et al*. 2012).

CONCLUSION:

Gastric carcinoma is still the second most prevalent cause of cancer-related death and the fourth most common cancer worldwide, despite the fact that its total incidence rate is continuing to decline, therefore it continues to be clinically important. Therefore, a multifactorial and multistep model of gastric cancer is currently recognized under these factors. Research has shown that some diets and/or dietary chemicals may help prevent cancer, but other types of foods appear to have the opposite effect. It is still unknown how components found in fruits and vegetables contribute significantly to the prevention of gastric cancer and why cancer preventive measures are only occasionally effective in preventing the development of stomach cancer.

Naturally, a number of other variables, such as smoking, drinking, and obesity, also affect the chance of developing stomach cancer. According to all of these research findings and prospective studies, quitting smoking and making dietary changes to consume less salt and salted foods while increasing fruit intake, especially vitamin C-rich fruit, are both effective ways to avoid stomach cancer. H. pylori infection does not have a significant role in cardia cancer, in contrast to non-cardia cancer, with obesity and smoking being the key risk factors. Although nutritional, lifestyle, and metabolic risk factors have been discovered, improving

these aspects of one's lifestyle and metabolism may benefit one's health, its effect on cancer prevention is unknown. Overall, this analysis aims to present a set of facts that demonstrate how leading a healthy lifestyle beginning with good nutrition might lower your chance of developing carcinoma as well as other chronic illnesses.

AUTHOR CONTRIBUTIONS:

Acquisition and interpretation of data is done by Falguni Pal. Conception, design and revising of the article are done by Dr. Pritha Pal.

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