Review on

Dietary impact on esophageal & stomach cancer & its possible management

[In the partial fulfillment of the requirements for the degree of Bachelor of Pharmacy]

Submitted To

The Department of Pharmacy,

Faculty of Allied Health Sciences,

Daffodil International University

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November 2022

APPROVAL

This project paper, Review on “Dietary impact on esophageal & stomach cancer & its possible management”, submitted to the Department of Pharmacy, Faculty of Allied Health Sciences, Daffodil International University, has been accepted as satisfactory for the partial fulfillment of the requirements for the degree of Bachelor of Pharmacy and approved as to its style and contents.

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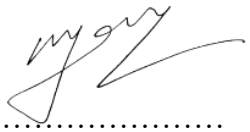
This is to certify that the results of the investigation that are embodied in this project works are original and have not been submitted before in substance for any degree or diploma of this university. The entire present work submitted as a project work for the partial fulfillment of the degree of Bachelor of Pharmacy.



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DECLARATION

I hereby declare that this project report, “Dietary impact on esophageal & stomach cancer & its possible management”, is done by me under the supervision of Md. Mizanur Rahman, Assistant Professor, Department of Pharmacy, Faculty of Allied Health Sciences, Daffodil International University. I am declaring that this Project is my original work. I also declare that neither this project nor any part thereof has been submitted elsewhere for the award of Bachelor or any degree.

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ACKNOWLEDGEMENT

I might want to communicate my profound applause to the All-powerful Allah who has given me the capacity to finish my undertaking work and the chance to concentrate in this subject.

I'm a lot of thankful to my honorable project supervisor of Md. Mizanur Rahman

Assistant professor, Department of Pharmacy, Daffodil International University.

I would like to express my humble regards to Dr. Muniruddin Ahmed, Professor and

Head, Department of Pharmacy, Daffodil International University.

I also wish to offer my respect to all of the teachers of Pharmacy Department, Daffodil International University and thankful to other members for their excellent cooperation with us.

Finally, I would like to express my gratitude towards my parents and other family

members for their kind cooperation and encouragement which helped me in completion of this project

……..

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My Parents

The persons who always encourage me in every sphere of my life.

Abstract

Cancer is an aberrant cell development that has a propensity to multiply uncontrollably. There are 32.6 million people worldwide who have cancer of some kind, and 456 000 of them have esophageal cancer. Approximately 80% of instances were found in less developed parts of the world. Gender discrepancy was shown by an incidence ratio of 2.4:1 in males and women. This study revealed that dietary acrylamide consumption was not linked to an elevated incidence of esophageal, gastric, or colorectal cancer. One of the fundamental meals in the human diet, cereals are primarily ingested as refined grains. The risk of various gastrointestinal cancers is inversely correlated with WG ingestion, most consistently with the risk of colorectal tumor. The only available treatment for regionalized gastric and esophageal cancer is surgical resection (R0). Due to the short life expectancy (20–50% at 5 years) following surgery alone, efforts must be made to improve health satisfaction with perioperative chemotherapy or postoperative (adjuvant) chemoradiotherapy. Approved Drugs for gastric cancer Cyramza (Ramucirumab), Docetaxel, Doxorubicin Hydrochloride, Enhertu (Fam-Trastuzumab Deruxtecan-nxki), 5-FU (Fluorouracil Injection), Fam-Trastuzumab Deruxtecan-nxki, Fluorouracil Injection. Approved Drugs for esophageal cancer Keytruda (Pembrolizumab), Nivolumab, Opdivo (Nivolumab), Pembrolizumab. The mortality rates for individuals with stomach cancer have improved as a result of advancements and the availability of numerous additional reference chemotherapy regimens.

Contents

List of figure

Chapter one
Introduction

1. Introduction

The poor prognosis and high mortality associated with cancer make it a major health risk. [1] The world is home to 32.6 million sufferers of this dreadful illness who have been dealing with it for an average of five years, thus according figures from Globocan (2012). 8.2 million cancer-related fatalities and 14.1 million new cancer cases were recorded globally in the same year. [2] In less advanced countries, 15.6 million (48%) of these cancer patients died within five years after diagnosis, 8 million (57%) had new cases, and 5.3 million (65%) were newly diagnosed. [3] Esophageal cancer, the eighth commonest prevalent malignancy, was diagnosed in 456,000 (3.2%) of newly diagnosed cancer patients. Long-term sensitivity to acrylamide was once believed to be mostly caused by certain vocations and smoking (Food Safety Commission of Japan Evaluation paper of dietary acrylamide produced by heating). [4] Researchers from Sweden found that acrylamide is produced when regularly ingested starchy foods are cooked at high temperatures, indicating that meals are yet another significant source of acrylamide [5]. Numerous studies have examined the link among acrylamide consumption in the workplace and the chance of developing cancer; nevertheless, the findings do not support the claim that acrylamide is an occupational carcinogen. Around 370,000 (80%) instances of esophageal cancer are seen in less developed nations. [6] While 323,000 male and 133,000 female cases of EC (2.4:1) were documented in 2012, the prevalence of the disease among men and women differed up to 20 times across different locations. [7] There were 400,000 EC casualties (4.9%), of which 281,000 were men and 119,000 were women. It ranks as the sixth most frequent reason for melanoma fatalities. The cause of EC is unknown to the medical establishment because it differs from location to location. [8] It is growing, albeit at varying rates according on the area. The tissues that line the inner surface, or epithelium, of the esophagus, are where esophageal cancer develops. [9] There are two main subtypes of EC. Esophageal squamous cell carcinoma is one that develops from the epithelial cell the top layer of the esophagus (ESCC). The other is esophageal adenocarcinoma, which develops from the glandular cells found at the junction of the esophagus and stomach. Just about 0.5% of esophageal tumors are benign; the majority are malignant. [10] Less than 10% of all tumors are smooth muscle tumors (leiomyomas) or gastrointestinal stromal tumors (GIST). An uncommon non-epithelial tumor that develops in the esophagus is leiomyosarcoma. [11] Additionally, the esophagus can sometimes develop small cell

cancer. This resembled small cell lung cancer and responds to chemotherapy more readily than other varieties of EC. [12] The pathophysiology of ESCC and EADC can be used to distinguish them from one another. The medical industry has advanced dramatically over the years, yet the outlook is bleak for patients with EC in terms of their chances of surviving. [13] After a major esophagectomy, the 5-year survival rate is less than 20% in China and 15% in the US. This is due to the sad fact that most instances only receive stage III or IV diagnoses. [14] Regular esophageal examinations should be carried out because EC is curable if discovered early. 90% of EC cases in the past were ESCCs, but currently even EADC incidence is rising, and the ratio ultimately demonstrates relatively less discrepancy. [15]

1.2 Diagnosis of Esophageal Cancer

Your doctor will study your symptoms, medical history, and physical exam results to make the diagnosis of esophageal cancer. Additionally, they could request specific X-rays and blood testing. Among the tests for esophageal cancer are: [16] When having a barium swallow X-ray, you ingest a liquid that coats your esophagus. This allows your doctor to spot specific issues on the X-ray because the esophagus stands out on it. Endoscopy: [17] The procedure involves the doctor looking into your esophagus through your throat with a narrow, illuminated tube called an endoscope. Sound waves are used in endoscopic ultrasound to provide more details regarding the amount of tumor development in adjacent tissues. Biopsy: [23] A doctor doing an endoscopy may remove tissue or cells from your esophagus. A microscope is used to check the cells for signs of malignancy. [18]

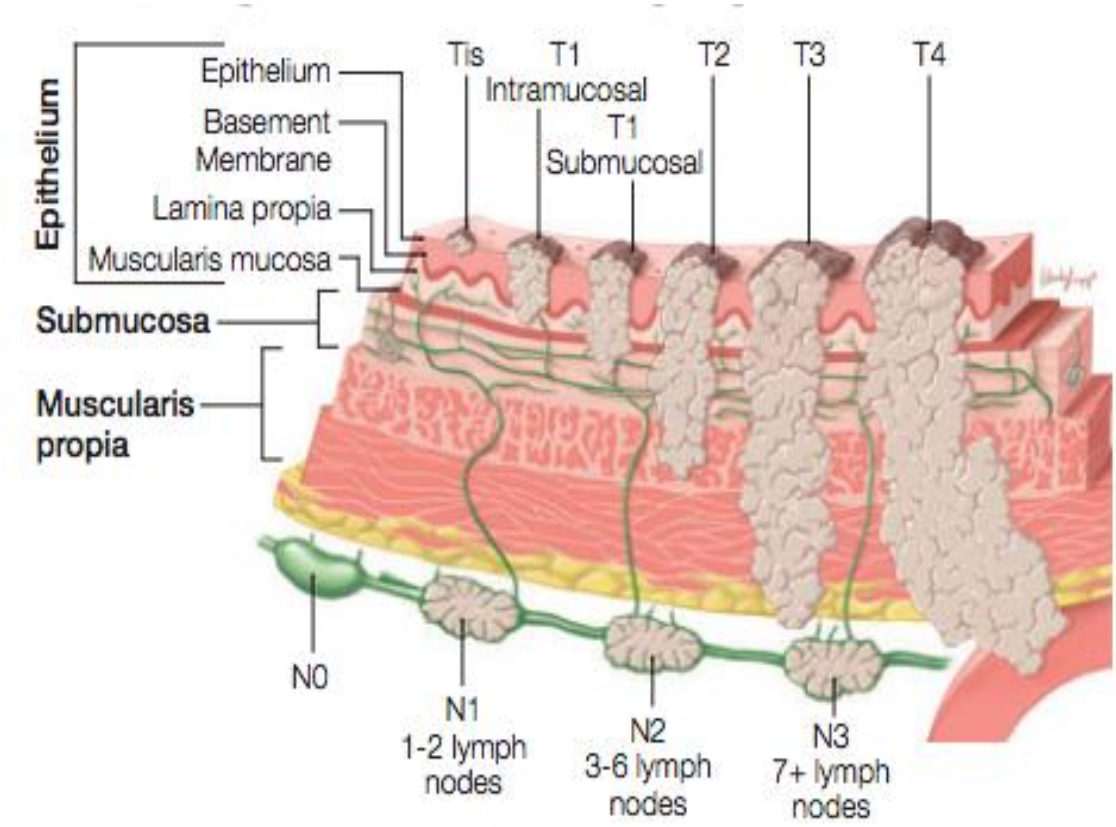

Figure 1: Pathology of Esophageal Cancer [19]

1.3 Determining the stage of stomach cancer

If you are diagnosed with stomach cancer, you may have additional testing to determine whether the disease has spread. [22] The cancer is staged using this information. The stage provides information to your doctor regarding the prognosis and how far along your cancer is. [20] The following tests and methods are used to determine the stage of stomach cancer: **Blood test.** [21] Stomach cancer cannot be identified by a blood test. Blood testing can offer your doctor with information about your health. [27] For instance, tests to assess the condition of your liver may reveal issues brought on by stomach cancer that has moved to the liver. [24] A different kind of blood test looks for cancer cell fragments in the blood. A test for circulating tumor DNA is what this is. For those with stomach cancer, it is only applied under specific circumstances. [26] For instance, if you have advanced cancer and are unable to get a biopsy, this test may be used. Blood sample collection might provide your medical team with information to assist them plan your therapy. [25]

Abdominal ultrasound: an imaging exam called an ultrasound uses sound waves to produce images. Pictures of stomach cancer can demonstrate how far the disease has spread into the stomach wall. [32] A small tube with a camera on the tip is inserted into the stomach and down the throat to take the photographs. [28] It is possible to utilize ultrasound to examine the lymph nodes close to the stomach. To extract tissue from the lymph nodes, a needle can be guided by the images. In a lab, the tissue is examined to check for cancerous cells. [33]

Imaging test: Your care team can use images created by imaging studies to seek for evidence that stomach cancer has spread. [34] The images might reveal cancerous cells in neighboring lymph nodes or other bodily regions. CT and positron emission tomography are two possible test types (PET). [35]

Surgery: when imaging tests cannot accurately depict your malignancy, surgery may be required to gain access to the internal organs. The term "metastasized cancer" refers to cancer that has spread and can be detected surgically. [36]

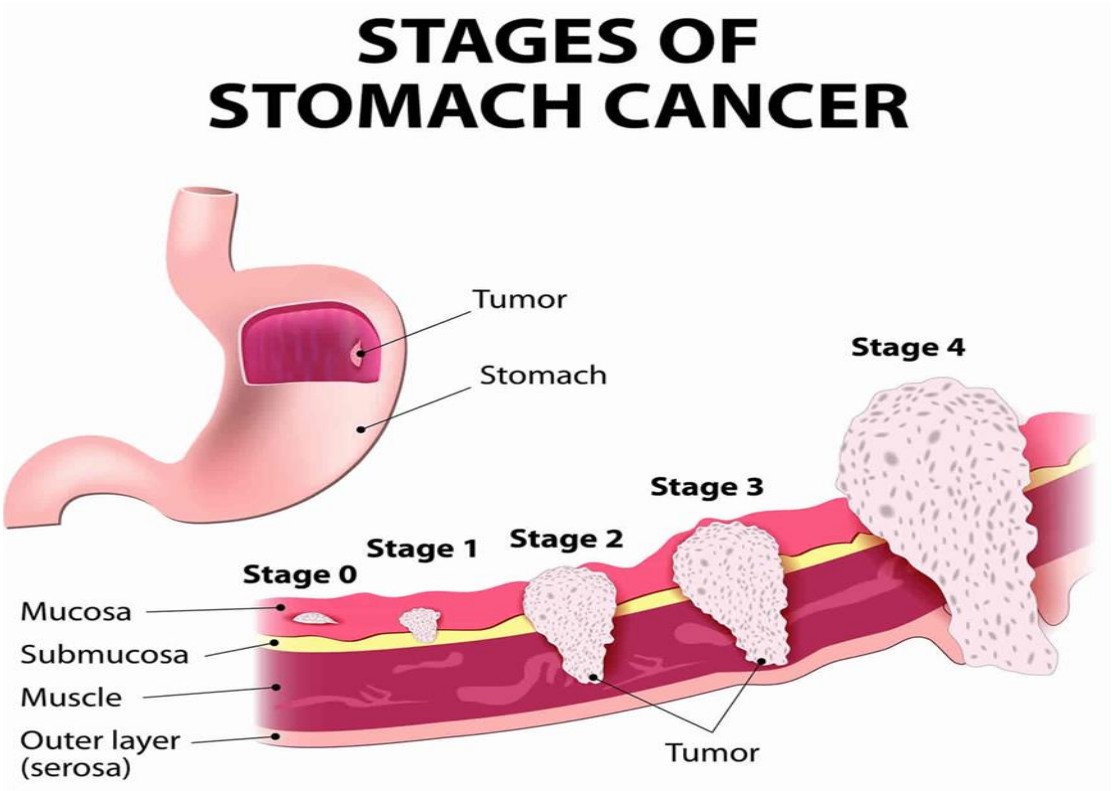

Figure 2: Stage of stomach cancer [37]

1.4 Pathophysiology of gastric cancer

Stomach tumors are often adenocarcinomas and have epithelial origins in 95% of cases. The gastric cardia (31%), antrum (26%) and gastric body (14%) are where gastric malignancies are most frequently discovered. [38] The remaining 10% of instances are adenocarcinomas called linitis plastica, which diffusely invade the stomach wall. Abdominal and widespread gastric tumors are the two main pathophysiological subtypes. [39] The cohesive neoplastic cells that make up intestinal-type malignancies are very well, form tubular forms, and often ulcerate. The linitis plastica, an invasion and thickening of the stomach wall giving it a "leather bottle" look, is a feature of the pleomorphic diffuse-type. [40] Due to delayed detection, the diffuse-type of gastric cancer does not develop a distinct mass and is associated with a poor prognosis. Women and people under 50 are more likely than men to have diffuse-type than other types of cancer. [41] Endoscopy monitoring is the most common method for detecting early gastric malignancies, where the tumor cells are restricted to the mucosa and outer layers of the stomach. High-risk nations like Japan use this method. [42] Endoscopy is a simple procedure for removing these tiny lesions, which frequently have a diameter of less than a millimeter and have a good prognosis. Early stomach cancer symptoms are nonspecific and typically include dyspepsia, which is easily misdiagnosed as acid reflux. [43] Early satiety, dysphagia from blockage, loss of weight or stamina from dietary requirements are later signs. If gastric cancer is detected, endoscopy with biopsies and brush cytology should be done. If cancer is found, a chest and abdominal computed tomography (CT) scan should indeed be done to ascertain the size of the tumor. [44] If there is proof of peritoneal involvement, distant metastases, or advanced or metastatic disease, such as infiltration of major blood arteries, a gastric tumor is deemed to be something. Palliative systemic therapy and chemotherapy and radiation therapies are the primary treatments available for individuals with severe, resectable, or metastatic illness. [45]

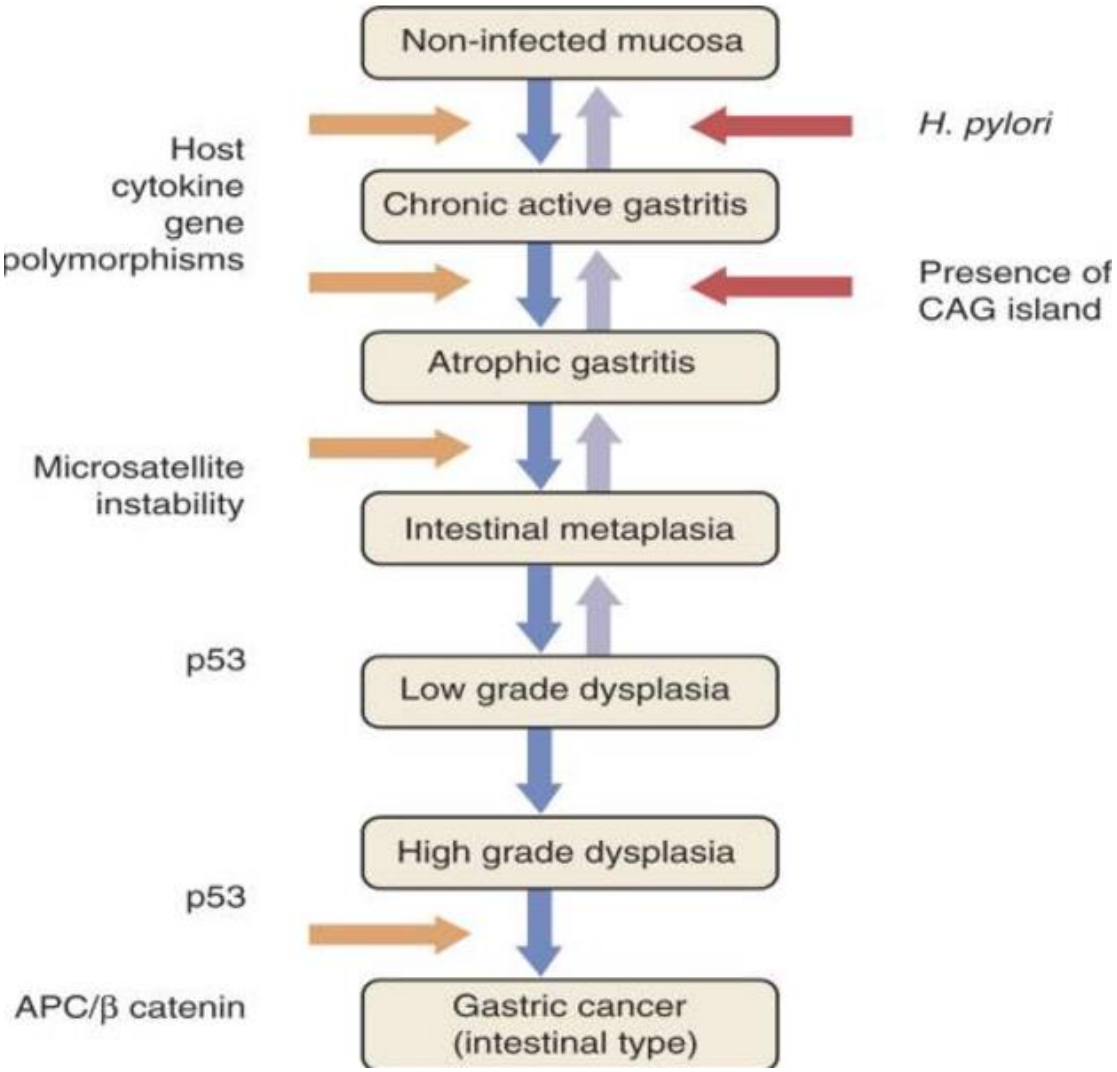

Figure 3: Pathology of Gastric Cancer [46]

Chapter two
Objective of the study

2 Objective of the study

2.1 General objective

- The goals of this project are to get a thorough understanding of the medical problem being researched. To learn more about the variables that contribute to the development of Esophageal Cancer & Stomach cancer. To have a better grasp of the many diagnostic procedures used to diagnose this ailment.

2.2 Specific objectives

- The purpose of this investigation was to understand more about Esophageal Cancer & Stomach cancer in the world.
- To find out the dietary impacts on Esophageal Cancer & Stomach cancer.
- To know the proper management system for Esophageal Cancer & Stomach cancer.
- Review the exhibition of a patient infected with Esophageal Cancer & Stomach cancer.
- Recognize common complications of Esophageal Cancer & Stomach cancer sickness preclusion and moderation measures.

Chapter three
Methodology

3.1 Methodology

A framework of exploration methodologies, as well as methods for gathering and analyzing data, is provided by a methodological review. This chapter discusses the techniques used in the investigation. Key phrases including "" " Esophageal Cancer & Stomach cancer pathogenesis," " Esophageal Cancer & Stomach cancer management, dietary impacts on Esophageal Cancer & Stomach cancer " and "diagnostic" were searched for utilizing web-based search engines, academic bibliographic databases, PubMed, Research Gate, and Medline. It gives an account of the learning environment. There are many variables to take into account, including the study sample, the study population, the investigation tools, the methodology, and the data analysis. This is a summary of earlier research on the manifestation of dietary impacts on Esophageal Cancer & Stomach cancer. All research on the causes, diagnoses, and therapies of the Esophageal Cancer & Stomach cancer sickness. A piece of the information was collected by directly reading previous research articles, while the other part came from scouring the internet for pertinent data. The activities of many managements were documented. All of the information gathered from prior study publications was numerically coded and imported.

Chapter four
Results & Discussion

4.1 Results

There were 10.3 million cancer deaths and 19.3 million new cases worldwide in 2020; more than one-third of cancer victims had gastrointestinal malignancies [54]. These malignancies comprise cancers of the upper and lower gastrointestinal tract, salivary glands, liver and bile ducts, gallbladder, and exocrine pancreas based on molecular phenotype and histological characteristics [55].

4.1.1 Whole Grains and Gastrointestinal Cancers

These malignancies comprise cancers of the upper and lower gastrointestinal tract, salivary glands, liver and bile ducts, gallbladder, and exocrine pancreas based on molecular phenotype and histological characteristics [55]. More than 60% of gastrointestinal cancer cases and deaths overall occurred in Asia, followed by Europe and North America. This is despite the regional breakdown of cancer- and sex-specific prevalence and mortality trends existing. Prognostic value of gastrointestinal malignancies continues to be a major problem for clinicians, in part because the majority of cases are discovered at a mature phase when the number of available treatments is constrained [56]. Numerous causative variables have been found; it is believed that genetic deficiencies only contribute for 5–10% of adverse outcomes while poor environments and lifestyles account for 90–95% [57]. In order to significantly reduce various risk behaviors (such as cigarette use, physical inactivity, poor diets, and alcohol misuse), primary and secondary prevention techniques, such as the promotion of healthy lifestyles, are especially relevant. The intake of WGs is highly advised for digestive health. While WG activity varies (and in some cases is missing) in various gastrointestinal organs, there is a substantial body of published data regarding WG impact on gastrointestinal cancers. To the best of our knowledge, no epidemiological studies have been conducted about the relationship between WG intake and the risk of gallbladder and bile duct carcinomas, while only one study has shown an adverse relationship between WG consumption and the risk of hepatocellular carcinoma, the most common histological type of primary liver cancer [58]. Higher consumption of WGs was similarly linked to a lower risk of pancreatic cancer, as revealed by a meta-analysis of case-control and cohort studies [59]; however, the lack of additional prospective cohorts makes it difficult to draw definite conclusions. Likewise,

information in the literature about the link among unrefined grains and malignancies of the oral cavity and oropharynx is scant, out-of-date, and only based on a small number of case-control and cohort studies. According to certain studies, WG consumption was positively correlated with the risk of upper aerodigestive tract malignancies [52]. Other investigations, however, found neither [54] or even favorable relationships [55]. Data on WGs and oropharyngeal cancer risk are less consistent than those for other plant-derived foods as a result of these contentious findings. Furthermore, data related to small bowel tumors are scarce and challenging to interpret, with the exception of a sizable US prospective cohort study revealing a marginally inverse link between WG food consumption and small intestinal cancer [53].

4.1.2 Whole Grains and Gastric Cancer

The fourth leading cause of tumor-related fatalities is gastric cancer. Males are more likely to develop cancer (5.6% of all instances), with 719,523 cases compared to 369,580 cases; Asia accounts for 75.3% of cases, being followed by Europe (12.5%) and Latin America and the Caribbean (6.2%) [80]. Both shapes of gastric cancer are linked to smoking and Helicobacter bacteremia, while cardiac gastric cancer is also linked to other risk factors like esophageal reflux, Barrett's esophagus, and obesity [79]. In general, gastric cancer is divided into non-cardiac gastric cancer, which arises from distal territories of the stomach, and cardiac gastric cancer, which arises near the esophageal-gastric intersection. Dietary habits are a significant influence in the development of cancer. Fruits, vegetables, and WGs are preventive factors against H. pylori infection, however salt-preserved meals and smoked meats increase its carcinogenic properties. A retrospective population-based case-control research found that diets high in WGs, but only when coupled with citrus fruit and vegetables, were associated with a slightly decreased incidence of stomach cancer in men but not in women [59]. In the many reported meta-analyses on the relationship between WG and stomach cancer, case-control studies have played a major or exclusive role, and dose-response investigations have been omitted. But according to all studies, growing WG intake was remarkable for displaying a bad connotation with stomach cancer risk (ranging from 13 to 50% lower risk for greatest WG consumers) and/or RG ingestion usually emerged to be a dose-dependent risk factor (63-65% increase in average of the risk). [65] It should be kept in mind, nevertheless, that RG-rich diets are typically low

in WGs (and other dietary fiber sources) and linked to unhealthy lifestyles. Consequently, rather than RG alone, dietary and lifestyle factors in combination with stomach cancer may be responsible for the direct connections found in the research.

4.1.3 Whole Grains in Esophageal Cancer

The sixth major source of cancer mortality, esophageal cancer, afflicted 508,585 cancer patients, or 5.3% of all cancer cases, per the Globocan 2020 [65]. Males are more likely than females to get esophageal cancer (3.1% of cases), and the Asian continent has the highest mortality (78.2%) [64]. Esophageal tumors can be divided into esophageal squamous cell carcinomas, which affect the upper layer cells lining the esophagus, and esophageal adenocarcinomas, which progress in glandular cells near the esophagus-stomach junction [66]. Esophageal squamous cell carcinomas are more common in emerging economies, whereas esophageal adenocar. Different risk factor profiles have been found: whereas overweight and gastro-esophageal reflux disease are significant risk factors for adenocarcinoma, cigarette smoking and alcohol misuse are major risk factors for esophageal squamous cell carcinoma. A much more diversified diet, both raw and cooked vegetables, vitamins, fiber, and carbohydrates are included among protective dietary factors, while red, pork, and processed meat consumption, as well as eating moldy food and roasted vegetables, are risks that can be attributed to the entire population [67]. Excessive intake of WG foods may be one of the factors influencing a lower risk of developing esophageal cancer. For instance, Levi and colleagues found that those who consumed huge quantities of WG foods (whole wheat bread and cereals) had a much lower risk of developing cancer than those who consumed RG foods (white bread and biscuits, pizza, pasta, and rice) in a small case-control study [68]. Both retrospective and prospective studies have both revealed lower likelihood for high WG consumption, however at varying ratios: for instance, the aforementioned Italian case-control study from La Vecchia's group [69] reported a 60% reduced risk for the maximum WG intake, whereas the most recent HELGA cohort study from Skeie and coworkers [63] had shown a 35–45% lowering. According to the projected 14-year Iowa Women's Health Study, which included 34,651 post-menopausal, originally cancer-free women, the occurrence of cancer was negatively proportional to both the amount of WG and total fiber consumed. The following intriguing data came to light in this frame of reference: I none of the opposite organizations witnessed for fruit fiber,

vegetable fiber, and total grain fiber were statistically significant; (ii) no barrier protection effect was discovered for fiber from RGs (according to the evidence that milling process lowers content of fiber and active components); and (iii) the connection with dietary fiber was driven by strong inverse association for WG fiber [64]. Given these results, it should be suggested to differentiate between WGs and RGs as fiber sources in order to prevent skewed data [65].

4.1.4 Dietary Acrylamide Intake and Risk of Esophageal, Gastric cancer

The average daily consumption of acrylamide was 6.8 3.8 mg (SD), or 0.13 0.16 mg/kg body weight, per day. foods that mostly caused Coffee (28%), green tea (22%), and alcohol (12% of total acrylamide intake) potatoes (11%), carrots (11%), and biscuits (11%). [70] According to the group (Q1) with the lowest acrylamide consumption, those in the greatest expenditure group (Q5) were younger individuals, a greater percentage of current smokers and more pack-years. Additionally, the food and beverage intake of the More coffee, green tea, fruits, vegetables, potatoes, and biscuits were found in the Q5 group, although less alcohol, meat, fish, dairy, and soy were consumed. food, energy, and diet. In the overall analysis, [78] there was no correlation among daily acrylamide consumption and esophageal cancer (P 14 0.814). Additionally, no strong associations were seen in the stratified analysis, risk assessment, or analysis that excluded carcinoma in situ. the links among routine acrylamide consumption and stomach cancer. Generally, neither total stomach cancer, cardia gastric cancer, nor non-cardia gastric cancer were linked to acrylamide consumption. Additionally, no significant relationships were found in the scenario analysis or the analysis that did not include carcinoma in situ. In the age- and area-adjusted model, daily acrylamide intake was considerably linked to a lower risk of colorectal cancer. [71] The incidence of colorectal cancer was roughly 11% lower in subjects in the highest acrylamide consumption group (Q5) than in the shortest intake group (Q1; HR 14 0.89; 95% CI, 0.78-1.01). Moreover, the importance of the reduced relationship was weakened and no meaningful association was found after further factor adjustments were included in the multivariable-adjusted model. When colorectal cancer in situ cases were omitted and the results were subjected to sensitivity analysis, the conclusions remained unchanged. Intake of acrylamide was also not linked to rectal or colon cancer. Additional presented stratified analyses by smoking status, alcohol use,

coffee consumption, and green tea consumption to show the correlations among daily dietary acrylamide ingestion and esophageal, gastric, and colorectal cancer, accordingly). In these investigations, no positive connections were often seen either.

4.2 Management of gastric & Esophageal cancer

4.2.1 Surgery

Regionalized gastric cancer can only be treated with surgical resection (R0). Consequently, the life expectancy after surgery alone is low (20–50% at 5 years), requiring attempts to enhance health satisfaction with perioperative chemotherapy 9 or postoperative (adjuvant) chemoradiotherapy [72]. In contrast to the United States, where 54% of patients undergoing basic gastrectomy receive less than a D1 lymphadenectomy, Japanese surgeons routinely do prolonged lymphadenectomy 10. The perigastric lymph nodes are removed during a D1 lymphadenectomy, and during a D2 lymphadenectomy, nodes along the left gastric, celiac, hepatic, and splenic arteries as well as those in the splenic hilum are extendedly dissected. Although there is some controversy regarding the advantages of D2 segmentation, the majority of specialists concur that comprehensive treatments, particularly in high volume centers, are optimal for treating localized gastric cancer with clinical stage >T1b 11. [73] Furthermore, it is absolutely necessary to make efforts to find trustworthy criteria in order to appropriately choose patients for multimodal psychotherapy. In an effort to not undervalue the significance of a successful operation, some pretherapeutic tumor parameters, such as tumor site, grading, Lauren's histologic subtype, and the presence of signet-ring cells, have been related with grade of response. [77] This problem still has to be resolved and provides a challenge for the future 12. We are attempting to address a multimodality strategy and examine the impact of treatment scheduling in this assessment (preoperative, postoperative, or both). Our selection criteria for cited studies call for the inclusion of significant phase 3 studies and analysis of more current trials using focused and immunotherapy medicines while avoiding older and unfavorable trials. [74]

4.2.2 Postoperative chemoradiotherapy

The Intergroup-0116 trial ("Macdonald protocol") for totally excised high-risk gastric or GEJ cancer provided the justification for adjuvant chemoradiotherapy by demonstrating a considerable OS advantage 10. After a median follow-up of more than ten years, this benefit in terms of OS and term pattern survival (RFS) is still present 17. [75] The preponderance of patients' insufficient lymph node dissection was the primary research drawback. More than 50% of patients had an insufficient D0 resection, and only 10% had a D2 nodal dissecting, raising the question of whether the improved OS and RFS was really due to the chemoradiotherapy or was instead the consequence of inadequate surgery. In the ARTIST study, 458 patients who underwent a R0 resection were examined for the benefits of adjuvant chemoradiotherapy against adjuvant chemotherapy (D2 dissection was a prerequisite). [76] Because there was no statistically significant distinction between the two groups for the primary objective of ARTIST, the 3-year disease-free survival (DFS) rate, the trial was deemed unsuccessful. In node-positive patients, adjuvant chemoradiotherapy enhanced DFS, according to a newly published update 18; moreover, there was no enhancement in OS despite a lengthy follow-up period. The efficacy of adjuvant chemoradiotherapy in patients with node-positive gastric cancer following successful excision is now being studied in the ARTIST-2 study. [77]

4.2.3 Postoperative chemotherapy

After a D2 nodal excision, postoperative adjuvant chemotherapy with S-1 improved OS and RFS in Japan 21. A second Asian research called the CLASSIC trial allocated randomly 1035 individuals to either surveillance for the six months following gastrectomy with D2 lymphadenectomy or capecitabine and oxaliplatin (CapeOx). Patients who received CapeOx treatment demonstrated improved DFS (at 3 years; HR = 0.56, 95% CI: 0.44-0.72, P 0.0001). In the adjuvant CapeOx arm versus 69% in the observation arm 22, the estimated 5-year OS was 78%. According to the recent review studies and meta-analyses, postoperative chemotherapy after gastrectomy with a D2 nodal dissection (Asia), postsurgical chemoradiotherapy, and perioperative chemotherapy (Europe) should all be regarded as standard of care alternative treatments for regionalized gastric cancer. [78]

4.3 Discussion

Here, we concentrated on the negative correlation among WGs, whose ingestion is becoming more and more advised, and the incidence and prognosis of gastrointestinal cancer. What has been discovered is that WGs consistently protect against gastrointestinal cancer, especially colorectal type, in contrast to polished equivalents; such variations can mostly be attributed to decrease (or loss) of essential minerals and phytochemicals during milling process. [78] In fact, WG is a complex food matrix that contains a variety of bioactive substances that work in concert to prevent chronic diseases. Because it is challenging to pinpoint which component actually protects, focus should be placed on the WG food matrix and away from specific components. For instance, fiber plays a key role in some of the stimulatory effect of WG, although fiber varies from grain to grain and is also found in other foods (vegetables, fruits, and legumes), all of which are equally ingested by high WG users. As a result, despite the fact that both fiber and WGs have positive health effects, they are not interchangeable. As a result, customers should concentrate their efforts to high-fiber goods because they occasionally contain bran or other added fiber despite having much, if any, WG. Despite all of these drawbacks, it is undeniable that WGs have positive benefits. As a result, initiatives to boost WG utilization should be carried out in a broad coalition encompassing both public (government agencies) and private (industry) stakeholders. Indeed, there are a number of obstacles to WG consumption that should be overcome by sensible initiatives. We discovered no correlation amongst dietary acrylamide consumption and overall risk of esophageal, gastric, or colorectal cancer among the Japanese population based on the large-scale representative sample of the JPHC Study. Interestingly, our findings were remarkably similar to those of earlier research. The relationship between dietary acrylamide intake and risk of esophageal cancer has been examined in two case-control studies [79] and two cohort studies The summary risk ratio (RR) for high versus low levels of acrylamide consumption was 1.14 (95% CI, 0.93- 1.38, Ptrend 14 0.41; ref. 10), indicating that there was no general connection between acrylamide intake and risk of esophageal cancer in these four investigations. This research has a number of advantages. A prospective observational study design was used. Due to the fact that the data were gathered prior to the cancer diagnosis, recall bias in exposure was avoided. The general population was the source of the

respondents, and a sizable sample was used. Furthermore, for esophageal cancer, gastric cancer, and colorectal cancer, the proportion of cases recognized solely by death certificates (DCO) was 7.1%, 4.3%, and 2.8% respectively. The cancer registrations employed in this investigation were therefore of high enough quality. The study does have certain restrictions. First off, as previously said, the FFQ has its own restrictions [80].

Chapter five
Conclusion

5.1 Conclusion

The results of the present study reveal that higher ingestion of SFA, cholesterol, concessional calories, sodium, and fat significantly increase the risk of ESCC, whereas dietary antioxidants, particularly folate, vitamin E, and selenium, could prevent esophageal damage from oxidative stress even if taken in moderation. The mortality rates for individuals with stomach cancer have improved as a result of advancements and the availability of numerous additional reference chemotherapy regimens. Moreover, by identifying the driver mutations of gastric cancer in specific patients and utilizing biologic medicines, considerable work still has to be done.

Chapter six
Reference

Reference

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