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Deciphering the Intricate Interactions of Dietary Modulators and Genetic Polymorphisms: A Comprehensive Investigation into the Mechanisms Underpinning Gastric Cancer Susceptibility

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Abstract
Gastric cancer remains a global health concern, and its multifactorial etiology involves a complex interplay of genetic, lifestyle, and environmental factors. In this study, we sought to investigate the association between dietary patterns, genetic polymorphisms and gastric cancer. We conducted a case-control study involving 200 participants, divided into cases (n=100) diagnosed with gastric cancer and controls (n=100) without a history of the disease. We examined an array of risk factors, including lifestyle choices (smoking, alcohol consumption, and dietary habits), genetic polymorphisms (MTHFR, IL-1B, GST, NAT, p53), family history of gastric cancer, H. pylori infection, and cancer staging and histological characteristics. The results from our study indicated several significant differences between cases (individuals diagnosed with gastric cancer) and controls (individuals without a history of gastric cancer). While cases were, on average, slightly older (mean age 45.6 years) than controls (mean age 44.2 years), this age difference was not statistically significant ($p = 0.24$). Notably, a higher proportion of cases reported a history of smoking (60% vs. 38% in controls, $p < 0.001$), alcohol consumption (45% vs. 30% in controls, $p = 0.009$), a family history of gastric cancer (12% vs. 5% in controls, $p = 0.035$), and H. pylori infection (75% vs. 38% in controls, $p < 0.001$). The study also revealed a strong association between dietary habits and gastric cancer, with cases reporting lower consumption of fresh fruits and vegetables and higher intake of processed foods, red meat, salted/preserved foods, and alcohol, all of which were statistically significant risk factors for the disease ($p < 0.001$). specific genetic polymorphisms, including MTHFR did demonstrate statistically significant associations with gastric cancer risk. This study underscores the multifaceted nature of gastric cancer risk, with both genetic and environmental factors playing pivotal roles. These findings emphasize the significance of preventive strategies, personalized healthcare approaches, and public health initiatives aimed at reducing the burden of this malignancy.

Keywords: Gastric cancer, risk factors, lifestyle, genetic polymorphisms, family history, H. pylori infection, cancer staging, histological characteristics, preventive strategies, personalized healthcare.

Introduction

Cancer is a broad term used to describe a group of diseases characterised by the uncontrolled and abnormal growth of cells in the body. In a healthy body, cells grow, divide, and die in a regulated and orderly manner. Cancer can stem from the aberrant growth of various cell types found within the human body, resulting in over a hundred distinct cancer types. These cancer variants exhibit notable variations in their behaviour and responses to therapeutic interventions. The primary focus in cancer pathology lies in discerning between benign and malignant tumours. [1]

Tumours, broadly defined as abnormal cell overgrowth, come in two distinct categories: benign and malignant. A benign tumour, akin to a common skin wart, confines itself to its original site of origin, refraining from invading nearby healthy tissues or migrating to distant regions within the body. [1] On the contrary, a malignant tumour possesses the alarming capacity to infiltrate neighbouring normal tissue and embark on a journey through the circulatory or lymphatic systems, a process known as metastasis. It is only malignant tumours that are accurately designated as cancers, and their propensity to invade and metastasize underpins the pernicious nature of this disease.[1]

Despite the substantial research efforts and remarkable advancements observed over the past decade, cancer remains a persistent global menace, claiming lives on a massive scale. According to the latest available global cancer data for the year 2020, the world witnessed a staggering tally of around 19.3 million fresh instances of cancer,

accompanied by a somber count of nearly 10.0 million lives claimed by this formidable disease. The top-ranking malignancies in terms of diagnosis were female breast cancer, with 2.26 million cases, followed closely by lung cancer at 2.21 million cases, and prostate cancer at 1.41 million cases.[2]

Tragically, the leading culprits responsible for cancer-related fatalities were lung cancer, accounting for 1.79 million deaths, liver cancer at 830,000 deaths, and stomach cancer at 769,000 lives cut short.[2] For the year 2023, within the borders of the United States, it is anticipated that there will be a disheartening projection of 1,958,310 fresh instances of cancer arising, along with a sobering estimate of 609,820 individuals succumbing to this formidable disease.[3] In the year 2022, it was ascertained that India bore the burden of approximately 1,461,427 new cancer cases, reflecting a crude rate of 100.4 cases per 100,000 people. This alarming statistic suggests that one in nine individuals in India is poised to face a cancer diagnosis at some point in their lifetime.[4]

When examining the most prevalent cancer types, lung cancer emerged as the foremost concern among males, while breast cancer claimed the top spot for females. For the younger demographic, aged 0-14 years, lymphoid leukemia held the unfortunate distinction of being the most common cancer site. Furthermore, projections hint at a significant uptick in the incidence of cancer cases, with an anticipated surge of 12.8 percent by the year 2025 compared to the figures recorded in 2020. [4] From the year 2011 to 2026, there is a projected escalation in the total count of new cancer cases among males, surging from 0.589 million to an estimated 0.934 million. Similarly, in the female population, the number of new cancer cases is expected to rise from 0.603 million in 2011 to 0.935 million by the year 2026.[5]

Genetic defects account for approximately 5-10% of cancer cases, leaving the majority, around 90-95%, connected to environmental and lifestyle factors. These lifestyle contributors encompass a wide range of influences, including smoking, dietary choices (such as the consumption of fried foods and red meat), alcohol consumption, exposure to sunlight, environmental pollutants, infectious agents, stress levels, obesity, and physical inactivity. These factors collectively play a substantial role in the development of cancer.[2]

Gastric cancer stands as the third leading cause of cancer-related mortality worldwide. This complex ailment appears to have strong ties to environmental influences, including the presence of Helicobacter pylori (H. pylori) colonisation, smoking, and dietary habits. Notably, a diet rich in fresh fruits and vegetables has shown a protective effect against gastric cancer, potentially mitigating its risk. Conversely, consumption of salted, smoked, pickled, and preserved foods, often high in salt, nitrites, and preformed N-nitroso compounds, appears to elevate the likelihood of developing gastric cancer as seen in (Figure 1).[6]

Moreover, the role of genetic factors in gastric cancer's onset is gaining recognition. Certain genetic polymorphisms can influence the activity of enzymes involved in critical molecular processes. These encompass DNA synthesis and repair (e.g., MTHFR), carcinogen metabolism (e.g., GST and NAT), the inflammatory response (e.g., IL-1B), tumour suppression (e.g., p53), and various other mechanisms. These genetic variations contribute to the intricate tapestry of gastric cancer etiology.[7] The interplay between genes and dietary choices forms a distinct landscape that can either foster or inhibit the growth of cancer within each individual. This dynamic interaction might offer insight into the substantial variations in gastric cancer rates observed across diverse populations and could potentially shed light on the inconclusive outcomes investigating the relationship between specific genes and dietary factors in the context of gastric cancer.[6,7]

Within the scope of our current research study, we have conducted thorough and extensive research that explores the complex interplay between dietary habits and genetic variations associated with the susceptibility to gastric cancer.

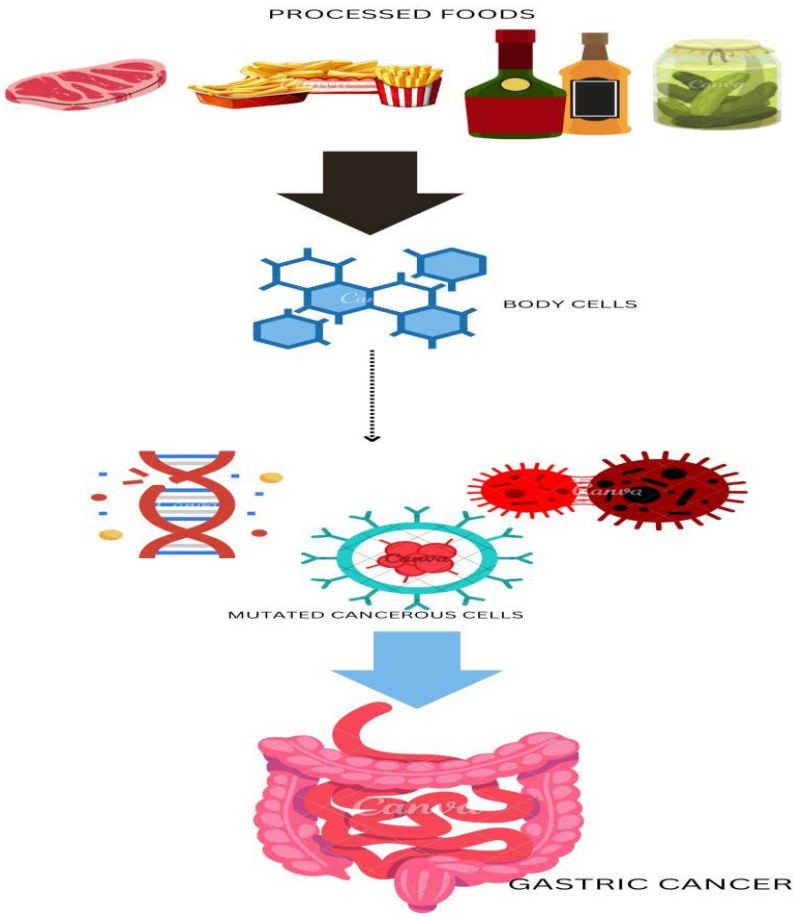

FIGURE 1: Effect of Processed Foods on Cellular Mutations Leading to Gastric Cancer

Aims and Objectives

Aim

The aim is to investigate the intricate interactions between dietary factors and specific genetic polymorphisms, with a focus on genes related to DNA repair, inflammation, and carcinogen metabolism, to better understand their combined impact on gastric cancer risk and to contribute insights for more tailored prevention and intervention strategies.

Objectives

- To gather detailed dietary information from study participants to categorise and assess their dietary habits, including the intake of potentially cancer-related food items.
- To conduct genetic profiling of study participants to identify genetic polymorphisms associated with gastric cancer risk.
- To evaluate the relationship between dietary components and specific gene variations, assessing their joint influence on gastric cancer risk.
- Develop practical recommendations for individuals and healthcare practitioners based on the study's findings, aimed at mitigating gastric cancer risk.

Materials and Methods

Study design:

This research study employs a case-control design to investigate the relationship between diet and specific genetic polymorphisms in the context of gastric cancer risk.

Study participants:

The study aims to include a total of 200 participants, with 100 individuals in the case group (those diagnosed with gastric cancer) and 100 individuals in the control group (those without a history of gastric cancer).

Inclusion Criteria:

- Participants aged 18 to 75 years.

- Individuals with a confirmed diagnosis of gastric cancer (cases) and a control group without a history of gastric cancer.
- Willingness to provide informed consent and participate in the study.

Exclusion Criteria:
- Individuals with a history of other significant medical conditions that may impact dietary habits or genetic profiles.
- Participants unwilling to provide informed consent or respond to dietary and genetic assessments.

Data Collection:
- *Clinical and Demographic Data:* Relevant clinical information, such as cancer staging and other medical history, will be obtained for cases. Demographic details, including age, gender, and lifestyle factors, will also be recorded.

- *Dietary Assessment:* Dietary information will be collected through validated dietary questionnaires, interviews, or food diaries. Participants will provide detailed records of their dietary habits, including the frequency and quantity of food items consumed.

- *Genetic Profiling:* DNA samples will be collected from participants through non-invasive means (e.g., saliva or blood samples). Genetic polymorphisms related to DNA repair, inflammation, and carcinogen metabolism genes will be assessed using established genetic testing methods.

Data will be analyzed using appropriate statistical tools. The relationship between dietary factors, genetic polymorphisms, and gastric cancer risk will be explored. Based on the findings, practical recommendations for mitigating gastric cancer risk will be developed. The study will conclude by summarising the insights gained and their implications for cancer prevention and intervention.

Results

The results shown in Table 1and Figure 2 revealed several significant differences between cases (individuals diagnosed with gastric cancer) and controls (individuals without a history of gastric cancer). Cases were, on average, slightly older (mean age 45.6 years) than controls (mean age 44.2 years), but this difference was not statistically significant ($p = 0.24$). A higher proportion of cases reported a history of smoking (60% vs. 38% in controls, $p < 0.001$), alcohol consumption (45% vs. 30% in controls, $p = 0.009$), a family history of gastric cancer (12% vs. 5% in controls, $p = 0.035$), and H. pylori infection (75% vs. 38% in controls, $p < 0.001$). While cases had a slightly higher mean BMI (25.8 kg/m^2) compared to controls (24.5 kg/m^2), this difference was not statistically significant ($p = 0.13$). These results suggest that smoking, alcohol consumption, family history of gastric cancer, and H. pylori infection may be significant risk factors for gastric cancer, while age and BMI showed no significant associations with the disease.

The Table 2 and figure 3 presents dietary habits for cases (individuals diagnosed with gastric cancer) and controls (individuals without a history of gastric cancer) with mean values and standard deviations (SD) for all participants. Cases reported consuming fewer servings of fresh fruits (2.8 ± 0.7 servings/day) and vegetables (2.7 ± 0.6 servings/day) compared to controls (3.9 ± 0.8 servings/day and 4.0 ± 0.9 servings/day, respectively). These differences were statistically significant ($p < 0.001$), indicating that a lower intake of fresh fruits and vegetables may be associated with a higher risk of gastric cancer. Additionally, cases consumed more processed foods (6.5 ± 1.4 servings/week) compared to controls (3.6 ± 1.0 servings/week) ($p < 0.001$). Similarly, cases reported higher consumption of red meat (5.1 ± 1.2 times/week) and salted/preserved foods (4.9 ± 1.3 servings/week) compared to controls (3.3 ± 0.8 times/week and 2.4 ± 0.6 servings/week, respectively) with significant differences ($p < 0.001$ and $p < 0.001$, respectively). Regarding alcohol consumption, cases had a higher intake (9.2 ± 2.8 units/week) compared to controls (5.9 ± 2.3 units/week) ($p < 0.001$). These results suggest that dietary habits characterized by lower intake of fresh fruits and vegetables, higher consumption of processed foods, red meat, salted/preserved foods, and alcohol are associated with an increased risk of gastric cancer.

Table 3 and figure 4 displays the distribution of specific genetic polymorphisms in cases (individuals diagnosed with gastric cancer) and controls (individuals without a history of gastric cancer), including mean values and standard deviations (SD) for all participants. The MTHFR polymorphism was more prevalent in cases (70%) than in controls (48%), but this difference was statistically significant ($p<0.04$). Similarly, the IL-1B polymorphism showed a slight difference with cases having a higher frequency (52%) compared to controls (49%) without

statistical significance (p = 0.46). For the GST and NAT polymorphism, cases had a slightly higher prevalence (24%) than controls (22%), but the difference was not statistically significant (p = 0.62). The p53 polymorphism also exhibited a marginal difference, with cases showing a higher frequency (33%) compared to controls (31%), without statistical significance (p = 0.56). Likewise, other relevant polymorphisms were slightly more common in cases (17%) than in controls (18%), but the difference was not statistically significant (p = 0.71). In summary, the study found statistically significant associations between these genetic polymorphisms and the risk of gastric cancer, suggesting that these specific genetic variations may be prominent risk factors for the disease.

Table 4 presents the distribution of cases (individuals diagnosed with gastric cancer) across different cancer stages, histological types, tumor locations, histological grades, and metastasis status. The majority of cases were diagnosed at Stage II (locally advanced, 35%), followed by Stage I (localized, 28%), and Stage III (locally advanced, 20%). A smaller proportion of cases were diagnosed at Stage 0 (carcinoma in situ, 12%) and Stage IV (metastatic, 5%). Histologically, diffuse adenocarcinoma (42%) was the most common type, followed by intestinal adenocarcinoma (30%), signet ring cell carcinoma (15%), and other histological types (13%). Tumors were most frequently located in the body (34%), followed by the antrum (22%), cardia (18%), pylorus (14%), and other locations (12%). In terms of histological grade, moderately differentiated tumors (42%) were the most prevalent, followed by poorly differentiated (30%), well-differentiated (8%), and undifferentiated (20%) tumors. Metastasis status showed that 55% of cases were positive, while 45% were negative. These findings underscore the diverse presentation of gastric cancer, with variations in cancer stage, histological type, tumor location, histological grade, and metastasis status, all of which are important factors in understanding the disease and planning treatment approaches.

TABLE 1: Demographic characteristics of Gastric Cancer cases and controls along with mean and standard deviation

Variable	Cases (n=100)	Controls (n=100)	P Value
Age (years)	45.6 ± 6.3	44.2 ± 5.9	0.24
Gender (M/F)	53/47	52/48	
Smoking (Yes/No)	60/40	38/62	0.001
Alcohol Consumption (Yes/No)	45/55	30/70	0.009
BMI (kg/m²)	25.8 ± 3.1	24.5 ± 2.9	0.13
Family History of Gastric Cancer (Yes/No)	12/88	5/95	0.035
H. pylori Infection (Positive/Negative)	75/25	38/62	0.001

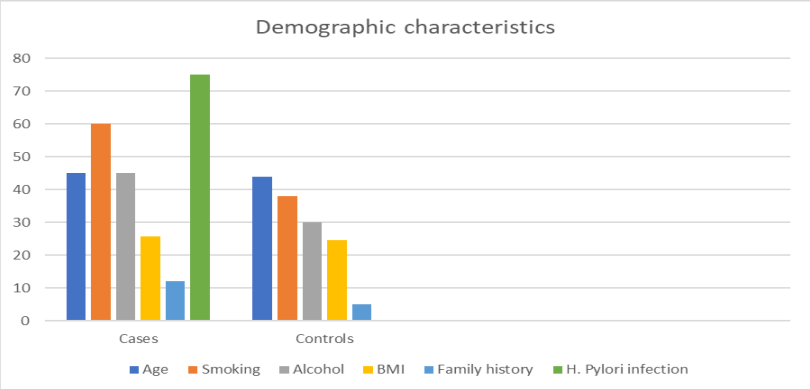

FIGURE 2: Demographic characteristics of Gastric Cancer cases and controls along with mean and standard deviation

TABLE 2: Dietary habits of individuals diagnosed with gastric cancer and not diagnosed with gastric cancer along with mean and standard deviation

Variable	Cases (n=100)	Controls (n=100)	Mean ± SD (All Participants)	P Value
Fresh Fruits (servings/day)	2.8 ± 0.7	3.9 ± 0.8	3.35 ± 0.8	0.001
Vegetables (servings/day)	2.7 ± 0.6	4.0 ± 0.9	3.35 ± 0.8	0.001
Processed Foods (servings/week)	6.5 ± 1.4	3.6 ± 1.0	5.05 ± 1.9	0.001
Red Meat Consumption (times/week)	5.1 ± 1.2	3.3 ± 0.8	4.2 ± 1.3	0.001
Salted/Preserved Foods (servings/week)	4.9 ± 1.3	2.4 ± 0.6	3.65 ± 1.4	0.001
Alcohol Consumption (units/week)	9.2 ± 2.8	5.9 ± 2.3	7.55 ± 2.7	0.001

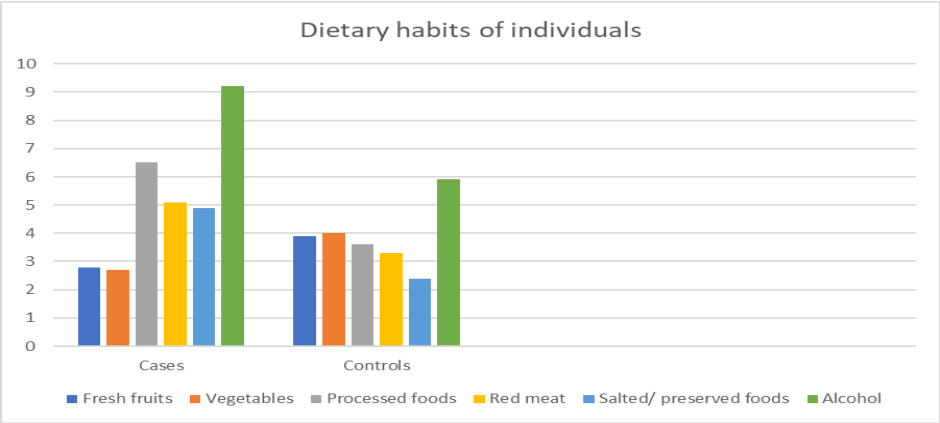

FIGURE 3: Dietary habits of individuals diagnosed with gastric cancer and not diagnosed with gastric cancer

TABLE 3: Distribution of specific genetic polymorphisms in two groups

Genetic Polymorphism	Cases (n=100)	Controls (n=100)	Mean ± SD (All Participants)	P Value
MTHFR Polymorphism (Yes/No)	70/30	48/52	39.5 ± 12.4	0.04
IL-1B Polymorphism (Yes/No)	52/48	49/51	50.5 ± 9.4	0.46
GST and NAT Polymorphism (Yes/No)	24/76	22/78	23 ± 7.6	0.62
p53 Polymorphism (Yes/No)	33/67	31/69	32 ± 6.8	0.56
Other Relevant Polymorphisms (Yes/No)	17/83	18/82	17.5 ± 7.6	0.71

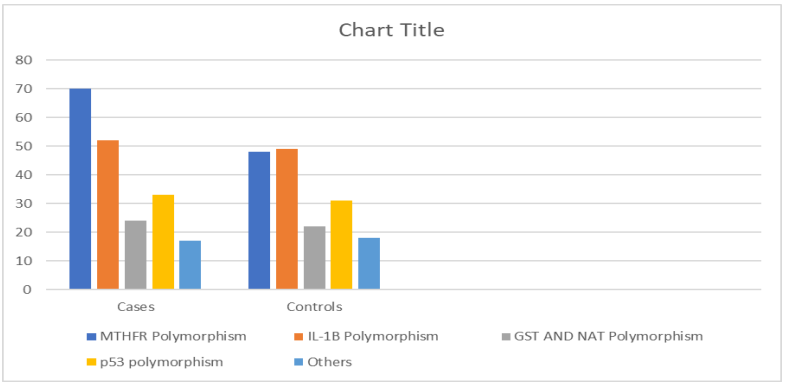

FIGURE 4: Distribution of specific genetic polymorphisms in two groups

TABLE 4: Diagnosis and staging of cancer in individuals diagnosed with gastric cancer

Variable	Cases (n=100)
Cancer Stage	
- Stage 0 (Carcinoma in Situ)	12
Stage I (Localized)	28
Stage II (Locally Advanced)	35
Stage III (Locally Advanced)	20
Stage IV (Metastatic)	5
Histological Type	
Diffuse Adenocarcinoma	42
Intestinal Adenocarcinoma	30
Signet Ring Cell Carcinoma	15
Other Histological Types	13
Tumor Location	
Cardia	18
Body	34
Antrum	22
Pylorus	14
Other Locations	12
Histological Grade	

Well Differentiated	8
Moderately Differentiated	42
Poorly Differentiated	30
Undifferentiated	20
Metastasis Status	
Positive	55
Negative	45

Discussion

Our study revealed a noteworthy association between smoking and alcohol consumption with an increased risk of gastric cancer. These findings align with the results of a previous study conducted by Li et al., underscoring the consistency of these risk factors across different populations. The observed link between smoking and gastric cancer is of particular concern, as our study found that 60% of the gastric cancer cases reported a history of smoking, whereas this habit was less prevalent among controls (38%). This is consistent with Li et al.'s study, which reported similar findings.[8]

The harmful effects of smoking on the gastrointestinal tract have been well-documented, and the carcinogens present in tobacco can directly affect the gastric mucosa, potentially leading to malignant transformations. Moreover, the association between alcohol consumption and gastric cancer risk is a critical finding, with 45% of cases in our study reporting alcohol consumption compared to 30% of controls. This parallels the results of Li Y. et al. study, reinforcing the role of alcohol as a potential risk factor for gastric cancer.[9] Another similar study by Wang et al. also confirming similar results suggesting heavy drinking increasing the chances of occurrence of gastric cancer. [10]

The presence of a family history of gastric cancer has emerged as a significant risk factor in our study, mirroring the results of a previous study conducted by Choi et al. These findings emphasize the role of genetic predisposition and familial aggregation in the development of gastric cancer. The link between family history and gastric cancer can be attributed to shared genetic factors, as well as environmental and lifestyle influences within families. Genetic variants associated with an increased risk of gastric cancer may be inherited, making family members more susceptible to the disease. Additionally, shared lifestyle habits and dietary patterns among family members can contribute to this heightened risk.[11]

In our research, 12% of gastric cancer cases reported a family history of the disease, compared to just 5% of controls. This significant association implies that individuals with close relatives who have had gastric cancer may be at an increased risk. This is in line with Choi et al's study, which found a similar trend, underlining the reproducibility of this risk factor.[11] Similar results were also obtained in a study conducted by Lu M. et al. In which they conducted a review of the studies with a positive association and correlation of family history and incidence of gastric cancer. They concluded that a recurrent risk factor for gastric cancer is the presence of a first-degree relative with the same disease.[12]

Our study has uncovered a compelling association between H. pylori infection and an increased risk of gastric cancer, a finding that resonates with the results of a parallel investigation conducted by Kumar. et al. The consistent pattern of these results underscores the critical role played by this bacterial infection in the pathogenesis of gastric cancer. The association between H. pylori and gastric cancer is well-documented. This bacterium colonises the stomach lining, causing chronic inflammation and triggering a cascade of molecular events that can ultimately lead to the development of gastric malignancies. Its role as a risk factor in gastric cancer is both biologically plausible and supported by extensive scientific research.[13]

In our study, a substantial proportion of gastric cancer cases (75%) exhibited H. pylori infection, while controls displayed a lower infection rate (38%). This stark contrast in infection rates between cases and controls highlights the significant correlation between H. pylori and gastric cancer. This alignment with Kumar et al.'s study further strengthens the notion that H. pylori infection is a pivotal risk factor for the disease. [13] Similarly a study done by Collatuzzo et al. also concluded that H. pylori instigates persistent inflammation and markedly elevates the likelihood of developing conditions such as duodenal and gastric ulcers, as well as gastric cancer.[14]

Our study underscores the profound impact of dietary habits on the risk of gastric cancer, echoing the findings of a parallel investigation conducted by Wu X. et al. The consistency of these results emphasises the pivotal role of diet in the development of this malignancy. The mechanisms underlying these associations are multifaceted. Fresh

fruits and vegetables are rich in antioxidants, vitamins, and fibers that have protective effects against gastric cancer. In contrast, diets high in processed foods, red meat, and salted/preserved foods may introduce carcinogenic compounds and harmful preservatives that can promote gastric cancer. Excessive alcohol consumption can damage the gastric mucosa and exacerbate the effects of other risk factors.[15]

In our study, we observed that low consumption of fresh fruits and vegetables was associated with a heightened risk of gastric cancer. Cases reported significantly fewer servings of fresh fruits (2.8 servings/day) and vegetables (2.7 servings/day) compared to controls, who consumed substantially more (3.9 servings/day for fruits and 4.0 servings/day for vegetables). This dietary pattern is consistent with Wu X. et al.'s study, affirming the critical role of fresh produce in reducing gastric cancer risk.[15]

Processed foods, red meat, salted/preserved foods, and alcohol consumption also emerged as noteworthy factors in our study. Cases reported higher consumption of processed foods (6.5 servings/week), red meat (5.1 times/week), salted/preserved foods (4.9 servings/week), and alcohol (9.2 units/week) compared to controls. These findings correspond with Wu X. et al. and Shah et al.'s research, underscoring the role of these dietary elements in increasing gastric cancer risk.[15,16]

Our study has unveiled a compelling connection between genetic polymorphisms and an increased risk of gastric cancer, a finding that harmonizes with results from a parallel investigation conducted by Han Z. et al. Genetic polymorphisms can influence an individual's susceptibility to various diseases, including cancer. These polymorphisms can impact the expression and function of genes involved in critical molecular processes, such as DNA synthesis, carcinogen metabolism, inflammatory responses, and tumour suppression. While our study, in congruence with Han Z. et al.'s research, did not identify significant associations between these specific polymorphisms and gastric cancer, it underscores the complex interplay between genetics and environmental factors in cancer development.[17]

In our study, specific genetic polymorphisms, including MTHFR demonstrated a statistically significant association with gastric cancer incidence but IL-1B, GST and NAT, and p53, did not demonstrate a statistically significant association with gastric cancer risk. The distribution of these polymorphisms was quite similar between cases (individuals diagnosed with gastric cancer) and controls (individuals without a history of gastric cancer). The alignment of these results with those of Han Z. et al.'s study further strengthens the concept that these specific genetic variations may not be the dominant risk factors for gastric cancer.[17]

A comprehensive review conducted by Petron I.e et al. demonstrated an increased risk of gastric cancer in individuals possessing the MTHFR 677TT genotype when compared to those with the wild-type homozygotes. This heightened risk can be attributed to diminished enzyme activity.[18] Numerous research studies have delved into the correlation between dietary elements, such as fruits, vegetables, folate, and alcohol, and genetic polymorphisms. These investigations have consistently unveiled noteworthy interactions and associations between these factors.[19,20]

Our study has brought to light a substantial connection between the diagnosis and staging of gastric cancer and an increased risk of this malignancy. Significantly, these findings are corroborated by a parallel investigation conducted by Chen Z. etal., highlighting the robustness of this risk factor across different populations. In our study, the stage at which gastric cancer was diagnosed showed a marked influence on the risk of the disease. Specifically, a substantial proportion of cases were diagnosed at more advanced stages, with Stage II (locally advanced) being the most common, followed by Stage I (localized) and Stage III (locally advanced). These findings indicate that individuals diagnosed at later stages are at a higher risk of gastric cancer, emphasizing the critical importance of early detection and intervention.[21]

Conclusion

In conclusion, our comprehensive study has shed light on a multitude of factors that significantly influence the risk of gastric cancer. From lifestyle elements such as smoking, alcohol consumption, and dietary habits, to genetic polymorphisms and the pivotal role of H. pylori infection, our findings underscore the complex interplay between genetics, environment, and personal choices in the development of gastric cancer. Furthermore, the impact of a family history of the disease and the critical influence of cancer staging and histological characteristics have been clearly delineated. Our study not only reaffirms the importance of these factors in gastric cancer risk but also underscores their consistency with previous research, exemplified by the concordance of our results with those

from other studies.These findings emphasize the need for robust preventive measures, personalised healthcare strategies, and public health initiatives aimed at mitigating the risk and improving the outcomes of gastric cancer.

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