Review

Impact of Culture and Traditional Dietary Habits on Gastric Cancer Carcinogenesis in East Asia

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Gastric cancer is one of the most common and deadly malignant neoplasms. Its incidence significantly varies by region. Most cases of newly diagnosed gastric cancer were reported in East Asian countries where there are unique cultures and traditional dietary habits. Culture affects eating habits and influences dietary choices. People in East Asia traditionally have high salt consumption rates and eat many salt-preserved foods/pickled foods, which have been well described as important risk factors in the carcinogenesis of gastric cancer. High dietary salt intake directly damages gastric tissue, facilitates colonization by Helicobacter pylori (H. pylori) infection, and amplifies the activity of bacterial virulence factors such as CagA, thereby accelerating carcinogenesis. In East Asian countries, the incidence of H. pylori infection is significantly higher. Studies demonstrated that dietary habits correlated significantly with H. pylori infection. Recent molecular evidence suggests that H. pylori plays a significant role in the initiation and development of gastric cancer. The roles of miRNA dysregulation and signaling pathway alterations has also played a role in the carcinogenesis of gastric cancer. The interplay of diet, culture, and microbial infection underscores the importance of modifiable risk factors in shaping gastric caner outcomes. Recognizing these patterns provides a foundation for prevention strategies and public health policies tailored to the particularly high-risk populations of East Asia. *[N A J Med Sci. 2025;18(1):032-037. DOI: 10.7156/najms.2025.1801032]*

Key Words: *Culture, Tradition, Dietary habit, East Asia, Gastric cancer*

INTRODUCTION

Gastric cancer (stomach cancer) (GC) remains one of the leading causes of cancer worldwide, with over 1.08 million new cases each year. It is the fifth most diagnosed cancer and the fourth leading cause of cancer-related deaths globally.[1,3] While rates of GC have declined in some regions, the burden is still especially high in East Asia. Approximately 50% of the patients had advanced disease when they were first presented and diagnosed. The total cost of the disease was up to $1.82 billion and the total cost of care was about $2.31 billion in 2020.[4,5]

Although researchers have studied the carcinogenesis of GC for decades, its development is still not fully understood. However, growing evidence points to the role of cultural and dietary habits as key factors.[1,2,3] These lifestyle choices, particularly the frequent consumption of high-salt foods, preserved vegetables, and pickled dishes, are common in East Asian countries and are now recognized as modifiable risk factors. Because long-standing cultural traditions influence these habits, it's important to understand their impact not only from a biological perspective but also through the lens of culture and history. This review highlights recent findings on how traditional dietary habits may contribute to GC, focusing on the potential carcinogenic mechanisms of both traditional diets and Helicobacter pylori (H. pylori) infection.

EPIDEMIOLOGY OF GASTRIC CANCER

Over the past sixty years, the total number of new GC cases in the world has slowly declined. However, the incidence of GC is comparatively higher in Asian countries and for Asian Americans.[1,2] In 2023, Yang reported that the highest age-standardized incidence rate occurred in Asia (14.3/100000), followed by Latin America, the Caribbean, Europe, and Oceania. The lowest incidence rate occurred in Africa and North America. Most of the newly diagnosed GC cases were reported in East Asian countries with 31.6/100000 in Japan, 27.9/100000 in South Korea, and 20.6/100000 in China.[1]

HISTOLOGY AND TYPE OF GASTRIC CANCER

GC is histologically classified into four categories: adenocarcinomas, lymphomas, gastrointestinal stromal tumors (GISTs), and other cancers.[6,7,8] Adenocarcinoma is the most common type, making up 90% of all gastric cancers (**Figure 1**).[2]

Received: 07/31/2025; Revised: 09/18/2025; Accepted: 09/21/2025
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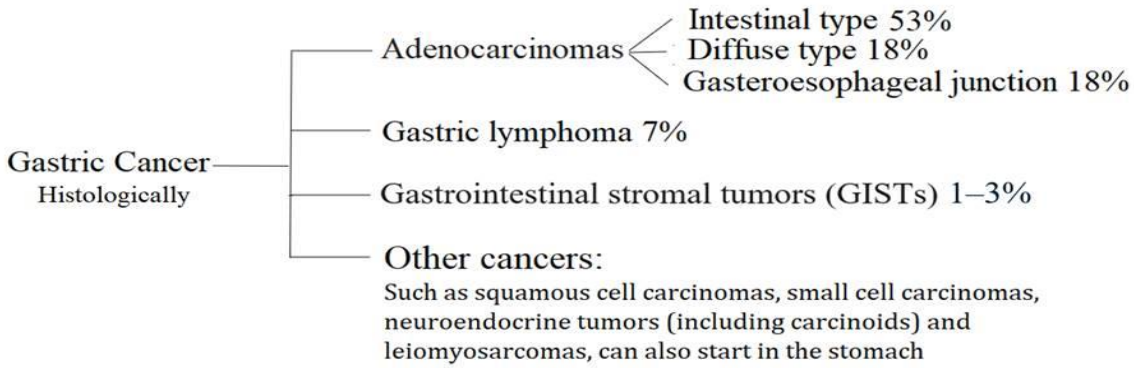

Figure 1. Classification of gastric cancer by histology.

Classifying GC by location in the stomach can be challenging. The boundary between the stomach and the esophagus (the gastric cardia) often shows overlapping tissue features, making it difficult to clearly separate cancers that occur there from those slightly above or below it. Furthermore, we have not yet reached a universal definition of the anatomic gastric cardia, making it even more unclear.[7] As a result, most clinicians group gastric cancers into two broader categories: cardia GC and noncardia GC. Noncardia gastric cancer is more common, but cardia cancer is typically more aggressive and is associated with worse outcomes because it tends to invade more deeply and spread earlier.[2,9]

The International Gastric Cancer Association supports the classification of gastric cancers into type I, type II, and type III, representing the tumor located at the distal esophagus, at the cardia, and at the stomach distal to the cardia, respectively.[10] Yet this classification still does not give clear definitions for each of these different anatomic locations.[7]

In an effort to improve diagnosis and treatment, researchers have moved beyond anatomy to molecular classification. The Cancer Genome Atlas (TCGA) identified four main molecular subtypes (**Table 1**).[11,12] Similarly, based on p53 (TP53) status, the Asian Cancer Research Group (ACRG) also proposed a classification including these four subtypes (**Table 2**).[12,13]

Table 1. Four distinct molecular subtypes of GC by the TCGA.

Epstein-Barr virus (EBV)-positive	Microsatellite instability (MSI)	Genomically stable (GS)	Chromosomal instability (CIN)

Table 2. Four distinct molecular subtypes (percentage of GC by the ACRG).

MSI subtype (22.7%)	mesenchymal group microsatellite stable (MSS)/EMT (15.3%)	microsatellite stable TP53-positive subtype MSS/TP53+ (26.3%)	microsatellite stable TP53-negative subtype MSS/TP53− (35.7%)

These molecular subtypes help researchers better understand how GC progresses and which patients are most likely to respond to certain treatments. For example, according to the ACRG, patients with MSI or TP53-negative tumors are often diagnosed earlier and may have better overall survival. On the other hand, tumors classified as MSS/EMT tend to be more aggressive. Interestingly, EBV infection was more frequently found in tumors with active TP53 pathways, suggesting a possible interaction between viral and genetic factors in the development of cancer.[13,14]

ETIOLOGY OF GASTRIC CANCER AND CULTURE AND TRADITIONAL DIETARY HABITS

GC is a multifactorial malignant disease and many factors are involved in the carcinogenesis of GC.[1,2,3,15] While genetics and infections play a role, lifestyle and diet are equally important in shaping an individual's risk. In East Asia, people have their own unique cultures and dietary habits which directly or indirectly result in the population's higher rate of exposure to carcinogen(s) that lead to GC. These unique cultural variations in East Asian countries allow for higher rates of GC compared to other parts of the world.

In **Table 3**, researchers have outlined various dietary and lifestyle factors that may raise GC risk, including high salt intake, alcohol consumption, smoking, obesity, and low intake of protective nutrients like vitamin A. Many of these risks are amplified when combined - for example, *H. pylori* infection and high salt consumption together are far more damaging than either on its own.

Table 3. Dietary factors that increase the risk of GC.

Authors	Factors
Yang et al[1]	Gastric ulcer or previous gastric surgery
Nouraie et al[16]	α-tocopherol and β-tocopherol
Mccullough et al[17]	Obesity, hot beverages, and coffee
Duell et al[18]	Heavy alcohol consumption (≥60 compared with 0.1-4.9 g/d)
González et al[19]	Total meat, red meat, and processed meat
Jakszyn et al[20]	Nitrosodimethylamine (NDMA) and endogenous formation of nitroso compounds
Miyazaki et al[21]	Dietary vitamin A
Shikata et al[22]	High dietary salt intake only; combination of high dietary salt, H. pylori, and atrophic gastritis has synergistic effects on carcinogenesis of GC
Murata et al[23]	High salt intake
O'Doherty et al[24]	Obesity ≥35 kg/m² and waist circumference
Moy et al[25]	Smoking cigarettes and heavily drinking alcohol
WCRF[26]	Salt-preserved foods
WCRF/AICR[26]	H. pylori infection
Shah et al[27]	Pickled foods
Rawla et al[28]	Gastrointestinal microbiota
Maddineni et al[29]	Processed foods

CULTURE AND TRADITIONAL DIETARY HABITS IN EASTERN ASIAN POPULATIONS

In East Asian countries such as China, Japan, and South Korea, food is deeply tied to culture, family, and identity. Meals are often built around traditional dishes that include salt-preserved vegetables, pickled foods, and fermented pastes like jiang.[30] Many of these centuries-old preservation techniques were created out of necessity. But while these practices are culturally meaningful, they also tend to involve high levels of salt, which studies have shown can increase the risk of GC.

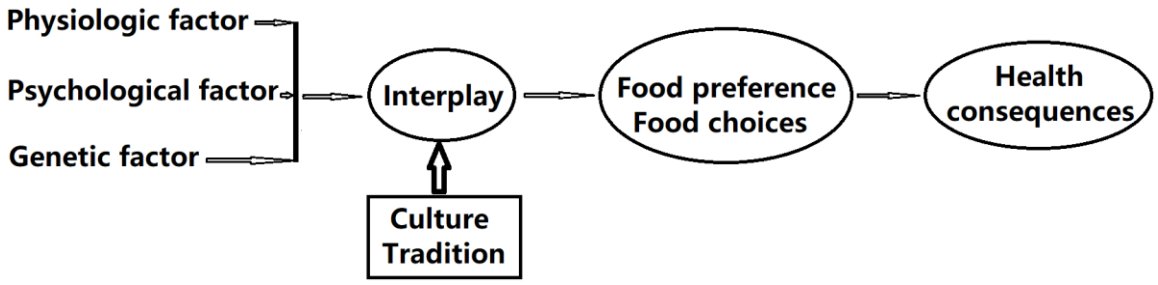

Figure 2. What you are eating results in health consequences.

Researchers like Adela Jamorabo Ruiz have emphasized how culture influences eating behaviors and, in turn, health consequences. People tend to make food choices not just based on taste or nutrition, but also on tradition and belonging.[31] Similarly, Laurie Demeritt found that cultural ties to food remain strong across generations. For example, Asian and Hispanic communities often maintain their traditional diets more consistently than other groups, not only in daily meals but especially during holidays and family gatherings (**Figure 1**).[32]

TUMORIGENESIS MECHANISMS OF SOME TRADITIONAL FOODS AND H. PYLORI INFECTION IN THE PATHOGENESIS OF GASTRIC CANCER

In East Asian communities, many people still closely follow traditional diets, which often include high-salt dishes, pickled vegetables, and fermented foods. While these are culturally important and commonly enjoyed, research shows that several components of these foods may contribute to the development of gastric cancer. In this review, we will only focus on three factors: high salt intake, salt-preserved and pickled foods, and H. pylori infection.

1. High Salt Intake

The World Cancer Research Fund and American Institute for Cancer Research have identified high salt intake as a major risk factor for GC. Multiple studies, including one by Yusefi et al (2018), have shown that consuming more than six grams of salt per day significantly raises the risk of developing GC.[33]

There are several proposed mechanisms to explain why high salt intake is linked to an increase in GC.[34,35]

a. High salt levels can damage the gastric mucosa and DNA, which may lead to gastric tissue hyperplasia, the turnover of gastric mucosal epithelium and eventually create a pathway for GC to develop.

b. High salt levels also seem to accelerate intestinal metaplasia which may increase the risk for dysplasia and early GC.
c. Some salty foods contain high concentrations of nitrates, which can form harmful N-nitroso compounds in the stomach (refer to 2 below).
d. High salt intake makes it easier for H. pylori to colonize in the stomach. It also appears to enhance the bacteria's ability to cause damage.[21] In particular, salt can increase the activity of the CagA gene, a known virulence factor in certain H. pylori strains. This makes the infection more aggressive and more likely to trigger cancerous changes (refer to 3 below).[36]

2. Salt-Preserved Foods/Pickled Foods

Pickled foods and salt-preserved foods are usually made in a jar spanning from weeks to months, providing conditions for fermentation and growth of fungi and yeasts. These easily yield carcinogenic compounds such as nitrate and N-nitroso compounds.[37] These foods also often contain higher amounts of salt.

The exposure to N-nitroso compounds (NOCs) is a significant risk factor for the development of GC. A study by Zhang et al (2022) found that mice exposed to these compounds developed gastric precancerous lesions linked to increased activity of the miR-194-5p gene. This gene affects cell metabolism and is connected to tumor formation.[38]

Liu et al (2023) have shown that long-term exposure to NOCs can lead to behaviors typical of cancer cells like increased migration, invasion, and uncontrolled growth. The group also found that NOCs activate the Warburg effect, a process where cancer cells use inefficient energy pathways to support rapid growth. This was linked to overexpression of the enzymes LDHA and LDHB in stomach cells, which further promoted their malignant transformation.[39]

3. H. Pylori Infection

H. pylori is a spiral-shaped, gram-negative microaerophilic bacterium. It's classified as a Group 1 carcinogen by the World Health Organization due to its strong association with gastric cancer. Once inside the stomach, H. pylori can survive for years, allowing it to interact with gastric epithelial cells and slowly deliver its harmful effects. A 2024 study by He et al showed that diet and lifestyle patterns significantly influenced infection rates across occupational groups.[40]

In China, the average H. pylori infection rate was 40.66%, with 43.45% of adults and 20.55% of children and adolescents afflicted with the infection. The data from 29 provinces out of the 34 provinces/autonomous regions/special administrative regions concluded that family-based infection rates were even higher, ranging from 50.27% to 85.06% with an average rate of 71.21%.[41]

An investigation in 2019 in South Korea from 24471 subjects showed 41.5% seroprevalence.[42]

While in Japan, rates of infection trend upwards as the age demographic increases. The male to female ratio of H. pylori was 27.5% (27.5:27.7) overall, 18.0% (18.3:16.1) in subjects in their 30s, 22.9% (22.7:24.7) in individuals in their 40s, 37.4% (37.2:38.2) in individuals in their 50s, and 46.1% (45.7:49.2) in individuals in their 60s.[43]

The ability of H. pylori to cause diseases depends on several virulence factors including proteins like BabA, SabA, and HopQ, etc. that help the bacteria survive, colonize, and damage host tissue.[44] H. pylori strains can be generally classified into two subtypes–CagA-positive and CagA-negative H. pylori strains. The most dangerous strains of H. pylori carry the cagA and vacA genes. Some different vacA genotypes, s1, s2, m1, m2, s1m1, s1m2, s2m2, and s2m1 have been identified. VacA s1m1 is usually detected in H. pylori-infected patients with chronic gastritis, whereas s1 and m1 VacA genotypes are most often found in H. pylori-induced GC.[45] High salt intake can make these infections even more dangerous. Under high-salt conditions, Loh et al (2018) found that H. pylori increased the expression of genes like sabA and hopQ, which were linked to greater inflammation and cell injury.[46] High salt also increased the activity of cagA, a gene that encodes a protein injected directly into host cells through a type IV secretion system. Once inside the cell, CagA disrupted normal cell division and signaling, leading to uncontrolled growth, DNA damage, and features typical of cancer cells.[47,48,49]

Recent research also showed that CagA activates other cancer-promoting pathways:

- He et al in 2024 showed that CagA increased the expression of a molecule called H19, a type of long non-coding RNA that promoted cancer cell migration and invasion. H19 appeared to disrupt the normal cell cycle by affecting YWHAZ, a regulatory protein.[50]
- Wu et al (2024) found that CagA also elevated the expression of TRIP13, a protein linked to worse outcomes in gastric cancer. When TRIP13 was blocked, cancer cells grew more slowly and the cell went into cell arrest at the G1 phase of the cell cycle.[51]

Beyond proteins, H. pylori also influences MicroRNAs (miRNAs). Dysregulation of miRNAs is crucial in the carcinogenesis of some human cancers. According to Xu et al (2024), H. pylori infection led to the abnormal production of many miRNAs, which disrupted processes like cell proliferation, apoptosis, invasion, metastasis, and even how cancer cells responded to treatment. These changes may contribute to tumor growth, spread, and drug resistance, resulting in GC development and progression.[52]

GLI1, a transcription factor in the Hedgehog signaling pathway, becomes overactive in response to H. pylori. Sun et al (2024) found that GLI1 upregulated the transcription of INHBA (Inhibin beta A), which induced the proliferation and metastasis of GC cells. This feedback loop was further enhanced by changes in m6A RNA modification, regulated by the FTO demethylase, which was also activated by H. pylori.[53]

Cheng et al (2024) showed that H. pylori increased the aggressiveness of GC cells through a regulatory axis involving HOXA-AS2, miR-509-3p/MMD2.[54]

Huang et al (2025) found that HN1 (Hematological and neurological expressed 1 protein), played a role in H. pylori-induced cancer. They demonstrated that the downregulation of HN1 significantly impaired the ability of H. pylori to promote migration of GC cells and the pathogenic processes of H. pylori-induced GC.[55]

PIEZO1 is a mechanosensitive ion channel protein that helps maintain epithelial tissue homeostasis. YAP1 is a transcription co-activator that activates genes related to cellular proliferation and suppresses apoptosis. CTGF (Connective tissue growth factor) can function as a signaling and regulatory factor involved in cell proliferation, angiogenesis, and tumorigenesis. Chen et al in 2023 identified the PIEZO1-YAP1-CTGF signaling axis as a key cancer-promoting pathway triggered by virulence factors CagA and VacA of H. pylori. They proposed that blocking PIEZO1-YAP1-CTGF might block the transformation of normal gastric epithelial cells into dysplasia and then into full-blown cancer.[56]

CONCLUSION

This review highlights the critical role of culture and traditional dietary practices in shaping gastric cancer risk in East Asia. High salt consumption, frequent intake of preserved and pickled foods, and elevated H. pylori infection rates act synergistically to promote carcinogenesis. At the molecular level, these exposures drive DNA damage, alter gene expression, and enhance bacterial virulence, ultimately accelerating tumor initiation and progression.

From a public health perspective, several preventive strategies are strongly supported by evidence. Reducing salt intake is critical to reducing the risk of developing gastric cancer, with high salt consumption increasing the risk of gastric cancer by approximately 25–55% compared to lower intake levels. Limiting traditional foods prepared with salt preservation and pickling may also lower risk, since these foods often contain carcinogenic nitrosamines and nitrates linked to gastric carcinogenesis (World Cancer Research Fund). Screening for and treating H. pylori is another key strategy. The bacterium is recognized as a Group 1 carcinogen by the World Health Organization, and eradication programs in high-risk regions have demonstrated reductions in gastric cancer incidence (NCBI). These efforts will be essential to developing targeted prevention strategies that respect cultural practices while addressing modifiable risk factors.

CONFLICT OF INTEREST DISCLOSURES
The authors have no conflict of interest to disclose.

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