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Asian Pacific Association of Gastroenterology task force recommendations on surveillance for *Helicobacter pylori* associated gastric premalignant conditions

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ABSTRACT

Background The burden of gastric cancer remains substantial in Asia. Gastric premalignant conditions, including chronic atrophic gastritis, intestinal metaplasia and dysplasia, are important intermediate stages in the gastric carcinogenesis cascade. The sojourn time allows endoscopic surveillance to have a pivotal role in early detection and timely intervention.

Objective This task force was commissioned by the Asian Pacific Association of Gastroenterology to formulate recommendations for the surveillance and management of *Helicobacter pylori* associated gastric premalignant conditions in the region.

Design A systematic literature review was conducted across multiple databases, including PubMed, Cochrane Library and Embase, focusing on studies related to gastric premalignant conditions and their surveillance, particularly from Asia. The evidence quality and strength of recommendations were appraised using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system.

Results These recommendations address four key aspects of gastric premalignant condition surveillance: (1) epidemiology and risk factors; (2) endoscopic and histopathological diagnosis; (3) risk stratification and endoscopic surveillance strategies; and (4) management strategies. 28 statements were made after multiple rounds of voting by experts. The statements offer a comprehensive, evidence based framework designed to assist clinicians in the Asia Pacific region on the early detection and management of gastric premalignant conditions.

Conclusion These statements aim to provide a structured, evidence based surveillance framework for clinical practice in the Asia Pacific region, while also identifying priority areas for future research.

INTRODUCTION

Gastric cancer remains the fifth most common malignancy and the fourth leading cause of cancer related death worldwide. According to the latest GLOBOCAN 2022 data,¹ there were 968 784 new

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ Asia accounts for 70% of the global gastric cancer burden, due to its massive population and high prevalence of *Helicobacter pylori* infection.
- ⇒ There are currently no regional guidelines or recommendations for the Asia Pacific region on the endoscopic surveillance and management of these conditions.

WHAT THIS STUDY ADDS

- ⇒ 28 consensus statements addressed the epidemiology, endoscopic and histopathological diagnosis, risk based surveillance interval and management of gastric premalignant conditions in Asia.
- ⇒ For diagnosis, we emphasised the importance of detailed and high quality endoscopic examination and standardised gastric biopsy protocols, together with proper pathological reporting for better risk stratification.
- ⇒ For most individuals with mild changes (operative link on gastritis assessment (OLGA) or operative link on gastric intestinal metaplasia (OLGIM) stage I) and in the absence of other risk factors, surveillance is not mandatory.
- ⇒ In the highest risk patients (OLGA or OLGIM stages III–IV), biennial endoscopic surveillance is suggested as a standard recommendation.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ This consensus report helps to standardise the approach to endoscopic diagnosis, surveillance and management of gastric premalignant conditions in Asia, particularly in most Asian countries without a nationwide gastric cancer screening programme.
- ⇒ The knowledge gap and priority area identified in this report will guide future research on surveillance and prevention of gastric cancer in Asia, where gastric cancer is prevalent.



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cases and 659 853 deaths globally. However, there is marked geographic variation in the gastric cancer burden, with >70% of new cases and deaths occurring in the Asia Pacific region. The highest incidence rates are reported in East Asia, particularly in China, Japan and Korea.²

Early stage gastric cancer often has no symptoms. Early detection and timely intervention of early stage gastric cancer can improve 5 year survival rates to >90%.³ With the advancement of gastrointestinal endoscopy, including high definition endoscopy and image enhanced endoscopy, endoscopy has emerged as the most direct and widely used method for early detection of gastric cancer, as well as enhanced detection and staging of gastric premalignant conditions.

Chronic atrophic gastritis (CAG), gastric intestinal metaplasia (GIM) and dysplasia (DYS) are well recognised premalignant conditions in Correa's gastric carcinogenesis cascade, usually triggered by chronic *Helicobacter pylori* infection.^{4,5} In a recent meta-analysis, the global prevalence estimates of CAG, GIM and DYS were 25.4%, 16.2% and 2.0%, respectively.⁶ The prevalence rates were generally higher in countries with high or medium gastric cancer incidence, and with a higher prevalence among *H pylori* infected individuals. The pooled global estimates of the progression rate per 1000 person years were 2.09 (95% CI 1.46 to 2.99) for CAG, 2.89 (2.03 to 4.11) for GIM and 10.09 (5.23 to 19.49) for DYS, and progression rates seemed to be similar between countries with low and medium or high gastric cancer incidences.⁷

In recent years, both European and North American societies have issued guidelines and recommendations on the management of gastric premalignant conditions.^{8–10} The updated European guideline (MAPS III)¹¹ recommends structured surveillance intervals based on histopathological risk stratification (eg, the operative link on gastritis assessment (OLGA) and the operative link on gastric intestinal metaplasia (OLGIM) system) and offers a detailed follow-up algorithm. In contrast, the American Gastroenterological Association (AGA)^{4,12} and the American College of Gastroenterology¹⁰ provide broader surveillance recommendations, emphasising individual risk assessment and shared decision making without a unified interval schedule. While some individual Asian countries have developed country specific guidelines,^{13–15} a regional evidence based guideline of recommendations is lacking, despite the high incidence of gastric cancer and high prevalence of gastric premalignant conditions. Hence there is a pressing need for a unified regional recommendation on endoscopic surveillance and management of patients with gastric premalignant conditions or neoplastic lesions in the Asia Pacific region.

This task force was commissioned by the Asian Pacific Association of Gastroenterology (APAGE) to develop guidelines and recommendations on the surveillance of gastric premalignant conditions for the Asia Pacific region. The aims of the task force were to standardise the surveillance approach and management in the region as well as highlight areas for future research.

METHODS

Data sources and search strategy

The task force was formed in January 2024. Working group members were invited from various member countries of APAGE, based on local gastric cancer incidences and individual expertise on gastric cancer screening and prevention. Members included 18 gastroenterologists, endoscopists, pathologists and epidemiologists from nine Asia Pacific countries and regions. Moreover, two international experts

were invited from Europe (MD-R) and North America (SS) to comment and vote on the drafted statements.

A comprehensive literature search was conducted across multiple databases (PubMed, Cochrane Library and Embase) to identify relevant studies for each topic under consideration, particularly those from Asia. The search was supplemented by manual checking of reference lists from identified studies and relevant systematic reviews.

This consensus used the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system as the standard for evaluating the evidence level and recommendation strength (online supplemental table S1).¹⁶ The strength of each recommendation was assessed based on several key factors, including the quality of evidence, alignment between the target population and the study population, extent of clinical benefit and cost considerations, among other factors. The recommendations are categorised into two levels: strong and conditional (weak).

A core group (WKL, YL, TLA, Y-CL and KSC) was first formed to develop the clinical questions pertinent to the focus of surveillance and management of gastric premalignant conditions and neoplastic lesions. Four subgroups were then formed to address four different areas: (1) epidemiology of gastric premalignant conditions and neoplastic lesions, (2) histopathological and endoscopic diagnosis, (3) risk stratification for endoscopic surveillance and (4) management of gastric premalignant conditions. Internal discussion among each subgroup was through online meetings and emails.

The modified Delphi process was adopted in developing the consensus statements. The first round of voting was performed anonymously via a web based platform in early November 2024. Voting was made on the strength of recommendation in five point Likert scales, from strongly agree, agree, neutral, disagree to strongly disagree. Consensus was achieved for all statements under a preset threshold of 80% agreement (strongly agree and agree). After the first round of voting, a hybrid meeting was conducted in November 2024 in Bali, Indonesia, during the Asia Pacific Digestive Week to provide feedback and discuss the statements. It was then followed by two more rounds of online voting in December 2024 and January 2025 after incorporation of suggestions and feedback from all members.

This consensus primarily focuses on gastric premalignant conditions that arise through the *H pylori* associated Correa cascade, namely CAG, GIM and DYS. Conditions arising from hereditary cancer syndromes, gastric neoplasms arising from *H pylori* infection naïve gastric mucosa,^{17,18} gastric polyps or autoimmune gastritis are not discussed in this consensus report. While we acknowledge that these conditions are important, they require distinct diagnostic, surveillance and management strategies, which are covered in other dedicated guidelines.^{19–21} Specifically, the management of gastric polyps (eg, hyperplastic polyps) centred on resection rather than surveillance. Autoimmune gastritis has a distinct pathophysiology and risk profile, including risk of neuroendocrine tumours, which necessitates different management and surveillance strategies.

CONSENSUS STATEMENTS

A summary of all 28 consensus statements is provided in online supplemental table S2.

Part 1. Epidemiology

Statement 1

The term 'gastric premalignant conditions' refers to mucosal environments characterised by chronic inflammation, leading to specific mucosal changes associated with an increased risk of gastric cancer.

Quality of evidence: low

Strongly agree 80%, agree 20%

The Correa gastric carcinogenesis cascade outlines a sequential process beginning with chronic non-atrophic gastritis, progressing through CAG, GIM and DYS, and culminating in gastric adenocarcinoma.²² Gastric premalignant conditions represent an extensive pathological state predisposing the tissue environment to carcinogenesis, incrementally elevating the long term risk of gastric cancer development without necessarily implying an immediate or direct progression to malignancy. Specifically, CAG is characterised by the loss of gastric glandular cells and their replacement with fibrosis or intestinal-type cells. GIM refers to the transformation of gastric mucosal epithelial cells into structures resembling those found in the small intestine (complete type) or colon (incomplete type), marked by the appearance of goblet and absorptive cells.²³ The extensive distribution of GIM in the stomach, such as involvement of the fundus and body of the stomach, also carries a higher risk of malignant transformation than localised GIM.²⁴ The presence of incomplete GIM indicates a further increased risk of malignancy.^{12 25} However, it remains controversial whether adenocarcinoma mainly develops from GIM.^{25 26} Recent studies have provided evidence that certain subsets of GIM may act as direct precursors of gastric cancer.^{27–29} For DYS, a detailed histopathological diagnosis is provided in statement 16.

In this report, we would also like to highlight the practical distinction between 'gastric premalignant conditions' and 'neoplastic lesions'. Neoplastic lesions usually refer to endoscopically visible and discrete lesions, which are either dysplastic lesions (that can progress to cancer) or early gastric cancer (EGC), both of which require endoscopic or surgical intervention. On the other hand, premalignant conditions typically require monitoring and surveillance only.

Statement 2

Individuals with advanced chronic atrophic gastritis (OLGA III/IV), advanced gastric intestinal metaplasia (OLGIM III/IV or incomplete-type) and gastric dysplasia are at increased risk for the development of gastric cancer.

Quality of evidence: high

Strongly agree 85%, agree 15%

Overall, the incidence of gastric cancer in patients with CAG and GIM is relatively low. A previous study reported a pooled gastric cancer incidence rate of 1.24 per 1000 person years for CAG (95% CI 0.80 to 1.76) and 3.38 per 1000 person years for GIM (95% CI 2.13 to 4.85).³⁰ In a 12 year follow-up study, Rugge *et al* showed that high risk CAG (OLGA stages III–IV) was consistently associated with invasive or intra-epithelial gastric neoplasia, whereas mild atrophy (OLGA stages I–II) was associated with a lower risk.³¹ Additionally, a prospective, multi-centre cohort study from Singapore showed that participants with OLGIM stages III–IV were at the highest risk (adjusted hazard ratio (HR) 20.7; 95% CI 5.04 to 85.60) of developing early gastric neoplasia.³² Moreover, a meta-analysis by Akbari *et al* demonstrated that incomplete GIM (6.60, 95% CI 2.73 to 11.75) was associated with a higher estimated gastric cancer incidence compared with complete GIM (3.24, 95% CI 0.12 to

9.01).³⁰ The details of OLGA and OLGIM staging are discussed in subsequent statements.

Statement 3

The prevalence of chronic atrophic gastritis and gastric intestinal metaplasia in the Asia Pacific region varies across different Asian countries and populations, with chronic atrophic gastritis more common than gastric intestinal metaplasia.

Quality of evidence: moderate

Strongly agree 60%, agree 35%, neutral 5%

The prevalence of CAG and GIM varies widely across Asia, which is attributed to differences in population characteristics, *H pylori* prevalence, local risk factors and even study methodologies. Most existing data are from small scale, hospital based or regional studies that used heterogeneous diagnostic criteria and age groups. Due to these uncertainties, it is challenging to provide a single Asia Pacific prevalence rate. Reported rates in the literature typically range from 10% to 30% for CAG and from 8% to 25% for GIM,^{33 34} but these figures should be interpreted with caution due to potential sampling bias and variability. In general, Southern Asia has a higher CAG prevalence and Eastern Asia has a higher GIM prevalence.³⁵ Data from Oceania countries remain sparse.

Statement 4

The prevalence of chronic atrophic gastritis and gastric intestinal metaplasia in Asia is declining overall. However, the rate of decline varies, due to differences in socioeconomic conditions and access to healthcare.

Quality of evidence: moderate

Strongly agree 65%, agree 25%, neutral 10%

The prevalence of CAG and GIM in the Asia Pacific region has declined in recent decades, likely due to improved public health, socioeconomic development and reduced *H pylori* infection in younger birth cohorts. A pooled analysis showed that the prevalence of GIM fell from 30% (1991–2000) to 21.1% (2011–22), with little change in CAG.³⁵ This decline was more evident in high income countries with better healthcare systems, widespread screening and national screening programmes (eg, Japan and South Korea). However, higher endoscopy rates may also lead to increased detection.^{36 37} In contrast, data from low resource countries remain limited³⁸, and the true prevalence may be underestimated due to various diagnostic constraints.

Statement 5

There is usually a strong positive correlation between *Helicobacter pylori* infection and gastric premalignant conditions in Asia; however, there are exceptions in some regions which may be modulated by host, bacterial and environmental factors.

Quality of evidence: high

Strongly agree 65%, agree 30%, neutral 5%

While *H pylori* infection has been classified as a group 1 carcinogen, it is usually closely correlated with CAG and GIM, with a previous meta-analysis showing an OR of 2.16 (95% CI 2.09 to 2.22) for CAG and 1.64 (95% CI 1.57 to 1.72) for GIM in Asia.³⁵ East Asian countries, such as China, Japan and South Korea, have a higher prevalence of CAG/GIM than other Asian countries.^{35 38}

However, there are also 'Asian enigma'³⁹ and 'Indian enigma',⁴⁰ where regions with a high *H pylori* prevalence have low gastric cancer rates, suggesting that other factors, such as difference in *H pylori* virulent strains, host genetic polymorphisms and dietary habits may mitigate the risk of progression to malignancy.⁴¹

Statement 6

Apart from *Helicobacter pylori* infection, dietary habits, smoking, genetic factors and lower socioeconomic status are all associated with the development of gastric premalignant conditions

Quality of evidence: moderate

Strongly agree 40%, agree 55%, neutral 5%

Several environmental and genetic factors beyond *H pylori* infection could also influence the development of gastric premalignant conditions and neoplastic lesions. The International Agency for Research on Cancer (IARC) of WHO has classified diet related factors, including high salt intake, pickled and preserved food, and smoked and grilled foods, as group 1 carcinogens for gastric cancer.^{42,43} Despite the lack of definitive guidelines or consensus regarding the impact of dietary factors on the development of CAG or GIM, a growing body of evidence suggests that poor dietary habits in some Asian countries are significant risk factors for gastric premalignant conditions.⁴³ An endoscopy based study in South Korea showed that high levels of salt intake were associated with CAG and GIM (OR 2.87, 95% CI 1.34 to 6.14).⁴⁴ Other dietary risk factors, such as deficiencies in fresh fruits and vegetables, are associated with increased oxidative stress and diminished antioxidant defence mechanisms (eg, flavonoids), resulting in chronic inflammation and damage to the gastric mucosa and causing premalignant conditions and neoplastic lesions.⁴⁴

Cigarette smoke is rich in free radicals and reactive oxygen species, which penetrate the gastric epithelium and induce oxidative stress. Additionally, nicotine present in cigarette smoke induces vasoconstriction, leading to reduced blood flow to the gastric mucosa, impaired nutrient delivery and diminished mucosal healing capacity.⁴⁵ Genetic predispositions also has a

role, with polymorphisms in genes associated with inflammatory and immune responses (such as tumour necrosis factor (TNF) α , interleukin 22 (IL-22)^{46,47} and IL-1B⁴⁸) correlating with heightened susceptibility to premalignant conditions. The prevalence of CAG and GIM decreased significantly with higher income or college education levels in a US population based study,⁴⁹ and individuals with lower educational attainment or socioeconomic status were at a higher risk of developing gastric premalignant conditions and gastric cancer in the Chinese population.⁵⁰

Part 2. Endoscopic and histopathological diagnosis**Statement 7**

APAGE recommends implementing measures to ensure optimal mucosal visualisation and adequate inspection time, along with adequate photographic documentation, to enhance the quality of endoscopic examination of the stomach.

Quality of evidence: Moderate

Strength of recommendation: Strong

Strongly agree 85%, agree 15%

The endoscopic appearance of gastric premalignant conditions, particularly CAG and GIM, can be inconspicuous, requiring a systematic and high quality examination to maximise diagnostic yield^{51,52} (figure 1). Endoscopists should ensure excellent mucosal visualisation by aspirating luminal contents and adequately insufflating the stomach to expand mucosal folds. Pre-procedural consumption or irrigation with mucolytic agents like simethicone, 1% N-acetylcysteine and proteolytic enzymes (pronase) may improve mucosal visualisation versus water irrigation alone.^{53–55} However, theoretical concerns regarding the risk of intraprocedural aspiration may limit the routine adoption of this practice.⁵⁶

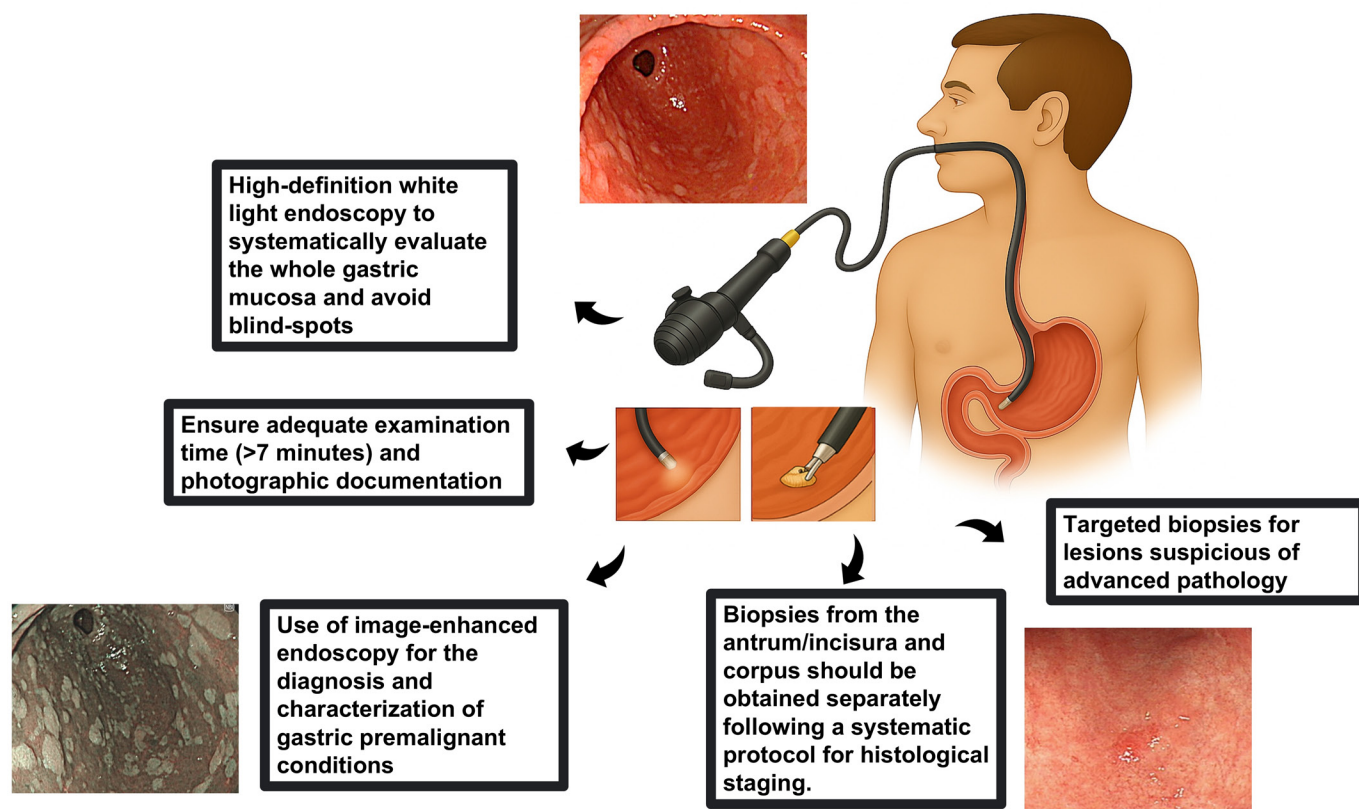


Figure 1 Systematic endoscopic evaluation and biopsy protocols.

Another crucial aspect of a high quality endoscopic examination is to ensure an adequate inspection time. A longer inspection time has been strongly associated with increased detection of gastric lesions.^{57–59} In a retrospective cohort study involving 837 Singaporean patients, inspection time >7 min was associated with increased detection of CAG and GIM, with an OR of 2.5 (95% CI 1.52 to 4.12).⁵⁷ In another Japanese retrospective cohort of 55 786 patients, inspection time >7 min was associated with increased detection of gastric neoplastic lesions. Compared with that in the fast group, OR for the neoplastic lesion detection rate in the moderate and slow groups were 1.90 (95% CI 1.06 to 3.40) and 1.89 (95% CI 0.98 to 3.64), respectively.⁵⁹ Both the AGA and European Society of Gastrointestinal Endoscopy (ESGE) have recommended that the whole inspection time for upper endoscopy should be at least 7 min.^{56 60} Furthermore, we advocate for standardised training programmes to ensure that endoscopists are adequately equipped to visually identify gastric premalignant conditions or neoplastic lesions.

Statement 8

APAGE recommends using high definition white light endoscopy to systematically evaluate the whole gastric mucosa and avoid blind spots

Quality of evidence: low

Strength of recommendation: strong

Strongly agree 70%, agree 30%

A systematic review reported that the rate of missing gastric cancer during upper endoscopy was 9.4% (95% CI 5.7 to 13.1%). Missed cancers were mainly located in the gastric body and were presumably due to blind spots that were not examined or lesions that were not recognised despite being in the field of examination.⁶¹ A systematic approach to ensure complete examination of the stomach has been proposed.⁶² The ESGE recommends precise photographic documentation that encompasses at least one illustrative image of the following anatomical landmarks: duodenum, major papilla, antrum, incisura, corpus, retroflexed view of the fundus, diaphragmatic indentation, upper aspect of the gastric folds, squamocolumnar junction, and distal and proximal oesophagus.⁶⁰ The AGA and World Endoscopy Organisation (WEO) have similar recommendations.^{56 63}

There is indirect evidence from a multicentre study using an artificial intelligence (AI) system to monitor photographic documentation that reduces the blind spots during upper endoscopy examination.⁶⁴ An AI assisted upper GI endoscopic system has been launched commercially and, if available, may be used to complement the systematic examination. Early data concerning the use of AI assistance for gastric cancer diagnosis are also promising.^{65 66}

Another study showed that training an endoscopist to perform standardised stomach examination improved the detection rate of gastric neoplasm from 0.2% to 2.3%.⁶⁷ Additionally, the impact of dedicated teaching on the optimal technique of examination and recognition of gastric lesions using video based platforms has been demonstrated in randomised controlled trials (RCTs) to improve the endoscopic diagnosis of EGC.⁶⁸

Statement 9

APAGE recommends use of image enhanced endoscopy for the diagnosis of gastric premalignant conditions and neoplastic lesions.

Quality of evidence: moderate

Strength of recommendation: Strong

Strongly agree 70%, agree 30%

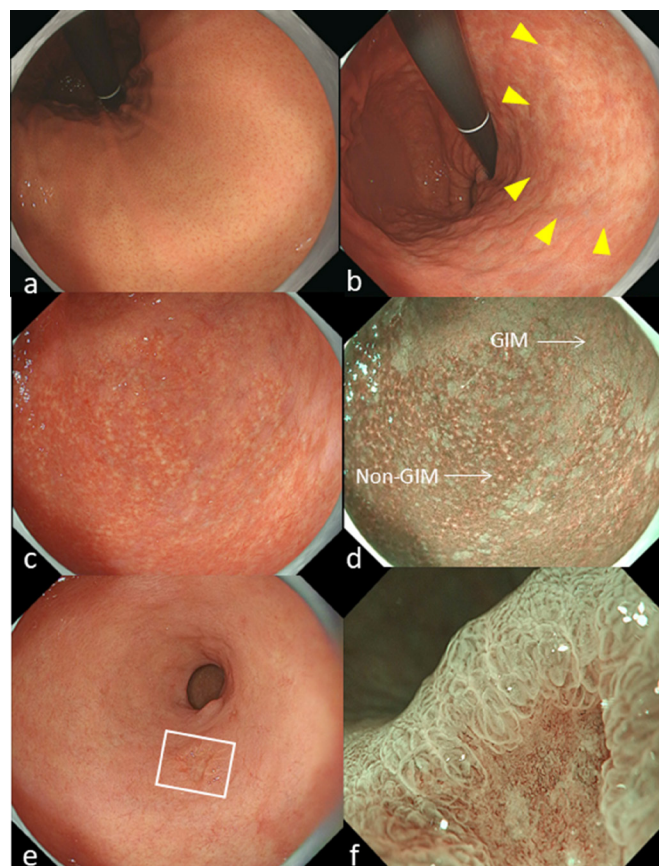


Figure 2 Endoscopic progression of gastric premalignant and neoplastic lesions. (A) *Helicobacter pylori* infection naïve stomach. The gastric folds are straight and preserved circumferentially, with regular arrangement of collecting venules in the corpus along lesser curvature. (B) In patients with chronic atrophic gastritis, endoscopic atrophic mucosa, characterised by loss of gastric folds, pale colour and increased vessel visibility, is seen in the corpus of lesser curvature (yellow arrowheads). (C) In patients with gastric intestinal metaplasia (GIM), areas with metaplasia are seen as slightly whitish areas. (D) The colour difference becomes prominent in narrow band imaging. (E) A small depressed lesion in the antrum. (F) Magnification narrow band imaging shows clear demarcation line and irregular microvessel pattern inside (white box in (E)). The lesion is resected by endoscopic submucosal dissection, and the resected specimen revealed it to be moderate to poorly differentiated intramucosal adenocarcinoma.

Unlike statement 8 that refers to systematic examination of the stomach to detect lesions, this statement refers to the evaluation of detected pathologies. High definition white light endoscopy (WLE) could be complemented by image enhanced endoscopy to improve visualisation of mucosal details (figure 2a–2c). A cross sectional study from Greece showed that high definition WLE had a sensitivity of 75% and specificity of 94% for detection of GIM.⁶⁹ However, high definition WLE alone seems to be inferior to image enhanced endoscopy. A meta-analysis including 10 studies confirmed that narrow band imaging (NBI) increased the detection of GIM by 32% compared with WLE.⁷⁰ Blue light imaging (BLI, Fujifilm, Tokyo, Japan) is another form of narrow band width imaging that is similar to NBI.⁷¹ There are also data suggesting that newer technologies based on white light, such as linked colour imaging (LCI; Fujifilm) and texture and colour enhancement imaging (TXI; Olympus, Japan) may improve visibility of gastric lesions,^{72–74} not limited to GIM. LCI has been

shown to increase the detection rate of gastric neoplastic lesions in RCTs.^{72 73 75} A prospective RCT in Singapore involving 90 patients showed that LCI significantly increased the absolute detection rate of CAG by 1.6% and GIM by 12.1% compared with WLE.⁷⁶ Another multicentre prospective RCT of 1924 patients from China also showed that LCI had better accuracy (78.8% vs 68.4%) and shorter examination time in diagnosing EGC than WLE.⁷⁵ Some studies also suggested that the use of magnifying endoscopy can predict the presence of CAG and GIM with an accuracy of up to 90%.⁷⁷ Nevertheless, it requires a special magnifying endoscope and training, which may not be available in most centres.^{78 79}

Statement 10

APAGE recommends that the extent of chronic atrophic gastritis and gastric intestinal metaplasia detected during endoscopy be objectively documented.

Quality of evidence: moderate

Strength of recommendation: Strong

Strongly agree 70%, agree 30%

The risk of development of gastric cancer depends on both the severity and extent of CAG and GIM. The estimated 5 year incidence for gastric cancer was 0.7% for mild CAG but up to 10% for severe CAG.⁸⁰ For GIM, the risk was reported as 5.3% for antral GIM but 9.8% for GIM involving the antrum and corpus.⁸¹ Hence the extent of CAG and GIM detected during endoscopy should be objectively documented. The OLGA and OLGIM scoring systems were developed as histopathological reporting frameworks for CAG and GIM that could be easily understood and prognosticate the risk of developing gastric cancer. They are based on a systematic biopsy protocol for documentation of disease extent and use a visual analogue scale for severity assessment (more details in statement 15).

Three endoscopic classification systems have been validated against the gold standard of histology, including the Kimura–Takemoto classification of CAG,⁸² the Kyoto classification system⁸³ and the endoscopic grading of gastric intestinal

metaplasia (EGGIM) (table 1 and figure 2A–2D).⁸⁴ In particular, the Kyoto classification and EGGIM have been validated against OLGA and OLGIM systems.⁸⁵ EGGIM is based on image enhanced endoscopy, whereas the other two are based on WLE. A systematic review reported that a Kyoto classification score of ≥ 4 might indicate an increased risk of developing gastric cancer (OR 7.3, 95% CI 3.6 to 14.7).⁸⁶ EGGIM can be used to assess a patient's risk by the endoscopic assessment of GIM in the antrum incisura, and in the corpus with the use of high definition endoscopes with NBI (or by extension, BLI) function. For the diagnosis of OLGIM stages III–IV, sensitivity, specificity and positive likelihood ratio were 9%, 95% and 16.5, respectively, by using a cut-off value of >4 .⁸⁴ In a multicentre Japanese observational study, OLGIM stages III–IV (OR 2.8, 95% CI 1.5 to 5.3), high EGGIM score (OR 1.8, 95% CI 1.0 to 3.1) and open-type Kimura–Takemoto (OR 2.5, 95% CI 1.4 to 4.5) had significant associations with gastric cancer risk.⁸⁵ Apart from NBI, recent data have also supported the use of an LCI based EGGIM system,^{87 88} which may have some advantages (eg, reducing the number of biopsy pieces) over NBI based systems for risk stratification. However, it must be acknowledged that these endoscopic scoring systems may not work as well in non-expert hands and there are a lack of data regarding validation in real world practice.

Statement 11

APAGE recommends that if biopsies are to be considered for histopathological diagnosis of chronic atrophic gastritis and gastric intestinal metaplasia, biopsies from the antrum/incisura and corpus should be obtained separately following a systematic protocol to allow histopathological staging, while targeted biopsies should be obtained separately for lesions suspicious for advanced pathology.

Quality of evidence: low

Strength of recommendation: strong

Strongly agree 85%, agree 15%

Table 1 Descriptions of severity of gastritis with different endoscopic classification systems for gastritis

Kimura–Takemoto classification of chronic atrophic gastritis ⁸²	Kyoto classification System ⁸³	Endoscopic grading of gastric intestinal metaplasia ⁸⁴	LCI-EGGIM ^{87 88}
Extent of atrophic gastritis described in terms of C1, C2, C3, O1, O2 and O3, with C1 being limited to antrum and O3 being very extensive involving antrum and all walls of corpus.	Atrophy ► None, C1 (0) ► C2 and C3 (1) ► O1–O3 (2) Intestinal metaplasia ► None (0) ► Antrum (1) ► Corpus and antrum (2) Enlarged folds ► Absence (0) ► Presence (1) Nodularity ► Absence (0) ► Presence (1) Diffuse redness ► None (0) ► Mild (with regular arrangement of collecting venules) (1) ► Severe (2) Kyoto score (0–8): score >4 may indicate increased risk for gastric cancer	Sites assessed: Antrum ► Lesser curve ► Greater curve Incisura Corpus ► Lesser curve ► Greater curve Scoring: ► No GIM: 0 ► $\leq 30\%$ GIM: 1 ► $>30\%$ GIM: 2 Range of score: 0–10 Cut-off score for diagnosis of OLGIM III/IV: >4	0, no GIM (ie, PIM change observed under LCI); 1, focal GIM (ie, $\leq 30\%$ PIM range observed under gastroscopy); 2, extensive IM (ie, $>30\%$ PIM change observed under gastroscopy). The PIM range in the lesser and greater curvature of the gastric antrum, incisura and the lesser and greater curvature of the gastric corpus was evaluated; so the total score is 10 points
EGGIM, endoscopic grading of gastric intestinal metaplasia; GIM, gastric intestinal metaplasia; LCI, linked colour imaging; OLGIM, operative link on gastric intestinal metaplasia; PIM, purple in mist.			

We acknowledge that there are differences in practice within Asia, as well as differences in expertise and resources available, in terms of obtaining biopsies for histology. It must be remembered that although the data on EGGIM are promising, there remains a need for further real world validation. Under most circumstances, there is still a need for histopathological assessment and confirmation of premalignant conditions, particularly in the absence of endoscopic expertise or diagnostic certainty.^{89 90} In addition, objective histopathological confirmation of focal DYS or EGC is required as part of pretreatment confirmation.⁹⁰ The Updated Sydney System facilitates a consistent approach to map out the extent of CAG and GIM.⁹¹ A minimum of five biopsies should be obtained with samples from antrum/incisura and corpus placed in separately labelled containers. Any suspicious areas should also be described and biopsied separately.^{91 92} Incisura biopsy is suggested because it has been shown to increase the number of cases diagnosed in more advanced stages of CAG and GIM,^{93 94} although admittedly this observation is not universal.⁹⁵ A real world retrospective study demonstrated that routine adherence to a systematic protocol increased the detection rates of premalignant conditions, including CAG and GIM.⁹⁶ Such data have prompted published international guidelines and reviews to consistently advocate for such a systematic approach.^{4 8 9 97}

Statement 12

APAGE recommends that the presence of chronic atrophic gastritis and gastric intestinal metaplasia should prompt a diligent examination for the concomitant presence of neoplastic lesions.

Quality of evidence: moderate

Strength of recommendation: strong

Strongly agree 85%, agree 15%

While CAG and GIM are associated with an increased risk of gastric neoplasia,⁹⁷ the presence of extensive GIM may partially obscure focal neoplastic lesions. It is important to be cognisant that at the index endoscopy, there may already be concomitant gastric neoplasia (including DYS and gastric cancer) and hence a careful endoscopic examination of the gastric mucosa is crucial.

Statement 13

The presence of any suspicious focal neoplastic lesion on white light endoscopy should be described using the updated Paris classification and further characterised with high definition, image enhanced endoscopy, preferably with magnifying/near focus observation and targeted biopsies obtained for histopathological confirmation.

Quality of evidence: moderate

Strength of recommendation: Strong

Strongly agree 45%, agree 45%, neutral 10%

The updated Paris classification serves as an important means of documenting the morphology of detected focal neoplasia in a consistent manner.⁹⁸ This will facilitate communication between healthcare providers and, importantly, it also provides an estimate of the risk of submucosal invasion of cancer.⁹⁸ High resolution image enhanced endoscopy has been shown to be useful in characterising focal lesions and differentiating between EGC and non-cancer (figure 2E, 2F).

For EGC, the diagnostic criteria for the vessel plus surface classification system using magnifying endoscopy with NBI (ME-NBI) are irregular microvascular patterns with a demarcation line or irregular microsurface pattern with a demarcation line.⁹⁹ A meta-analysis that examined 20 studies involving 7770 patients and 7917 lesions reported a pooled sensitivity for ME-NBI for

diagnosing EGC of 0.86 (95% CI 0.80 to 0.90) and specificity of 0.92 (95% CI 0.86 to 0.96).¹⁰⁰ The use of BLI has demonstrated comparable performance with NBI.^{101 102} The overall diagnostic performance for EGC using near focus NBI (NF-NBI) was an accuracy of 87.7%, sensitivity 60.7%, specificity 93.1%, positive predictive value 63.8% and negative predictive value 92.2%.¹⁰³ A recent phase II RCT compared the third generation NBI with the newer TXI, and high definition WLE. Gastric neoplasia detection rates in the NBI, TXI and WLE groups were 7.3%, 5.0% and 5.6%, respectively.¹⁰⁴ The Magnifying Endoscopy Simple Diagnostic Algorithm for Gastric Cancer (MESDA-G) was jointly devised by the Japan Gastroenterological Endoscopy Society, the Japanese Gastric Cancer Association and the WEO as a unified international algorithm for magnifying endoscopy diagnosis of EGC.¹⁰⁵ After a focal lesion is detected by WLE, the presence of a demarcation line is first assessed. The lesion is then examined in greater detail with narrow band width imaging, combined with magnification.

It is, however, important to note that many of these diagnostic criteria were developed and validated based on the Japanese classification system, where lesions pathologically defined as high grade dysplasia in the west are typically classified as mucosal adenocarcinoma, regardless of invasion. This distinction and its implications for practice in the wider Asia Pacific region are discussed further in statement 16.

Statement 14

APAGE recommends that pathology reports standardise the description of intestinal metaplasia: to be quantified for each site as mild, moderate and severe. The presence of gastric atrophy in oxyntic mucosa should be assessed where feasible.

Quality of evidence: low

Strength of recommendation: Strong

Strongly agree 45%, agree 45%, neutral 10%

The presence of CAG and GIM (including complete or incomplete subtypes) are important and should be reported in all pathology reports of gastric biopsies. Quantifying the severity is also of prognostic relevance as the extent and severity of the findings have an impact on the risk of developing gastric neoplasia.¹¹ By convention, the grading of severity for CAG and GIM is defined as none (0%), mild (1–30%), moderate (31–60%) and severe (>60% involvement of the biopsy specimen).¹⁰⁶ Subclassification of GIM into complete and incomplete is encouraged when feasible, given its higher association with gastric cancer risk.²⁴ Pseudopyloric metaplasia, which marks chronic injury and a reparative response in the stomach, typically arises in the oxyntic mucosa of the gastric body,¹⁰⁷ may also be present and should be documented when identified. It must be noted that while the interobserver agreement for GIM is high, it is more variable and can be low for CAG.^{91 108 109}

Statement 15

APAGE recommends that if gastric biopsies are obtained by a mapping protocol, histopathological staging by OLGA/OLGIM can be used to stratify the risk for gastric cancer progression.

Quality of evidence: moderate

Strength of recommendation: Strong

Strongly agree 40%, agree 55%, neutral 5%

The OLGA system was developed to establish a reporting framework for gastric atrophy that is easily understood by clinicians and provides prognostic information.⁹² The OLGA system uses the biopsy sampling protocol and visual analogue scale recommended in the updated Sydney system.⁹¹ Atrophy

severity for antrum/incisura and corpus is scored as 0 (no atrophy), 1 (mild), 2 (moderate) or 3 (severe), within the corresponding anatomic locations (antrum/incisura and corpus). The overall antrum/incisura score and overall corpus score are then combined to provide the OLGA stage (stages I–IV), with stages III and IV shown to be predictive for the development of gastric neoplastic lesions.⁹²

Similarly, the OLGIM system was developed to evaluate the severity and extent of GIM on gastric mapping biopsies from the antrum/incisura and corpus in a similar fashion. Stages III and IV were found to be predictive for gastric neoplastic lesions.¹¹⁰ The prognostic values of both OLGA^{31 111 112} and OLGIM³² have since been validated in longitudinal studies and a meta-analysis. A Singapore study (n=2980) showed increased early gastric neoplasia risk in patients with GIM (adjusted HR 5.36; 95% CI 1.51 to 19.0), particularly in OLGIM stages III–IV (HR 20.7; 95% CI 5.04 to 85.6).³² In a Japanese multicentre observational study, OLGIM stages III–IV were also associated with a higher cancer risk (OR 2.8; 95% CI 1.5 to 5.3).⁸⁵ OLGIM is favoured over OLGA due to better interobserver agreement^{9 110} and is endorsed by ESGE (MAPS II–III), Academy of Medicine, Singapore (AMS) and British Society of Gastroenterology.^{8 9 11 13}

However, we recognise that OLGA/OLGIM may not be feasible in all Asian countries due to lack of availability of expertise or resources, or a predominant focus on endoscopic diagnosis. Hence this statement does not mandate histology for diagnosis, but rather recommends that if histopathological assessment is performed, it should follow validated systems, such as OLGA/OLGIM.

Statement 16

APAGE recommends that dysplasia be graded on a two tiered system as low grade and high grade dysplasia, as recommended in the latest WHO classification system. However, APAGE also accepts the Japanese Gastric Cancer Association classification system for the classification of high-grade dysplasia in appropriate settings.

Quality of evidence: low

Strength of recommendation: strong

Strongly agree 40%, agree 55%, neutral 5%

According to the WHO classification system, gastric DYS is defined as an unequivocal neoplastic lesion without evidence of stromal invasion. Currently, DYS is graded dichotomously as low or high grade.¹¹³ In a large cohort study from the Netherlands with 92250 patients, de Vries *et al* reported that the annual incidence of gastric cancer was 0.1% for patients with CAG, 0.25% for GIM, 0.6% for low grade dysplasia (LGD) and 6% for high grade dysplasia (HGD) within 5 years after diagnosis.¹¹⁴ Notably, within 1, 5 and 10 years of follow-up after initial diagnosis, gastric cancer was diagnosed in 0.3%, 0.6% and 0.8% of patients with CAG; 0.7%, 1.2% and 1.8% of patients with GIM; 2.1%, 3.1% and 3.9% of patients with LGD; and 24.9%, 29.5% and 32.7% of patients with HGD.¹¹⁴ It is worth noting that the study did not exclude gastric cancers diagnosed within the first year of identifying CAG, GIM or DYS. Thus the percentages reported in the study included some prevalent gastric cancers in addition to incident cases.

Notably, there are differences in the diagnostic criteria of superficial gastric neoplastic lesions between the Japanese and western pathologists. Lesions diagnosed as HGD by western pathologists are considered adenocarcinoma by the Japanese Gastric Cancer Association classification based on nuclear and structural atypia, regardless of the presence of

invasion.^{115 116} The pathological criteria adopted by Japan (and South Korea) allow them to correlate better with endoscopically identifiable neoplastic lesions. However, most Asia Pacific countries adhere to their own or international guidelines (such as the WHO criteria) for pathological reporting standards which have implications for training, laboratory accreditation and oncological management. For instance, a single centre observational study from China reported their long term experience with a three tier classification system for gastric dysplasia (mild, moderate or severe), which was based on architectural distortion, nuclear atypia and glandular arrangement. This histopathological framework was also shown to correlate with increasing risk of malignant transformation across the severity spectrum.¹¹⁷ While differences exist, both lamina propria and muscularis mucosae invasion are diagnosed as invasive adenocarcinoma and staged pathologically as pT1a cancer. Irrespective of the differences in diagnostic categories, the approach to treatment is the same, in that resection is indicated and endoscopic resection is preferred when feasible.¹¹⁷

Statement 17

In the absence of formal OLGA and OLGIM staging, advanced stage chronic atrophic gastritis/gastric intestinal metaplasia may be indicated by the presence of marked and extensive chronic atrophic gastritis/gastric intestinal metaplasia in multiple biopsies, or incomplete subtype gastric intestinal metaplasia.

Quality of evidence: moderate

Strength of recommendation: Strong

Strongly agree 70%, agree 25%, neutral 5%

Although OLGA and OLGIM staging systems are preferred for comprehensive risk assessment,^{92 110} routine implementation is limited by the need for multiple biopsies and expert pathology. Other easily assessable risk factors include the extent/severity of CAG and GIM, incomplete GIM subtype, smoking (>20 pack years), persistent *H pylori* infection and a family history of gastric cancer.¹¹⁸ A systematic review of 12 cohort studies (four in Asia) found that incomplete GIM carried a significantly higher cancer risk than complete GIM (relative risk (RR) 5.16; 95% CI 3.28 to 8.12).¹¹⁹ However, this subtype based classification does not reflect the extent/severity of GIM.

As an alternative, the endoscopic grading system EGGIM evaluates GIM across five gastric regions using NBI. Studies from Portugal, Italy, Japan and China showed that high EGGIM scores correlated with increased gastric cancer risk,^{84 85 87 120} and LCI-EGGIM may offer an advantage for detection.⁸⁷ However, the routine application of EGGIM by less experienced endoscopists requires further evaluation.

Part 3. Endoscopic surveillance

Statement 18

Endoscopic surveillance is recommended for patients with moderate to advanced stages of chronic atrophic gastritis and gastric intestinal metaplasia, as well as patients with non-advanced chronic atrophic gastritis and gastric intestinal metaplasia who have additional risk factors for gastric cancer.

Quality of evidence: low

Strength of recommendation: weak (Conditional)

Strongly agree 75%, agree 25%

Endoscopic screening and surveillance are widely implemented in Asia due to high *H pylori* prevalence and elevated gastric cancer risk. Although non-invasive tests

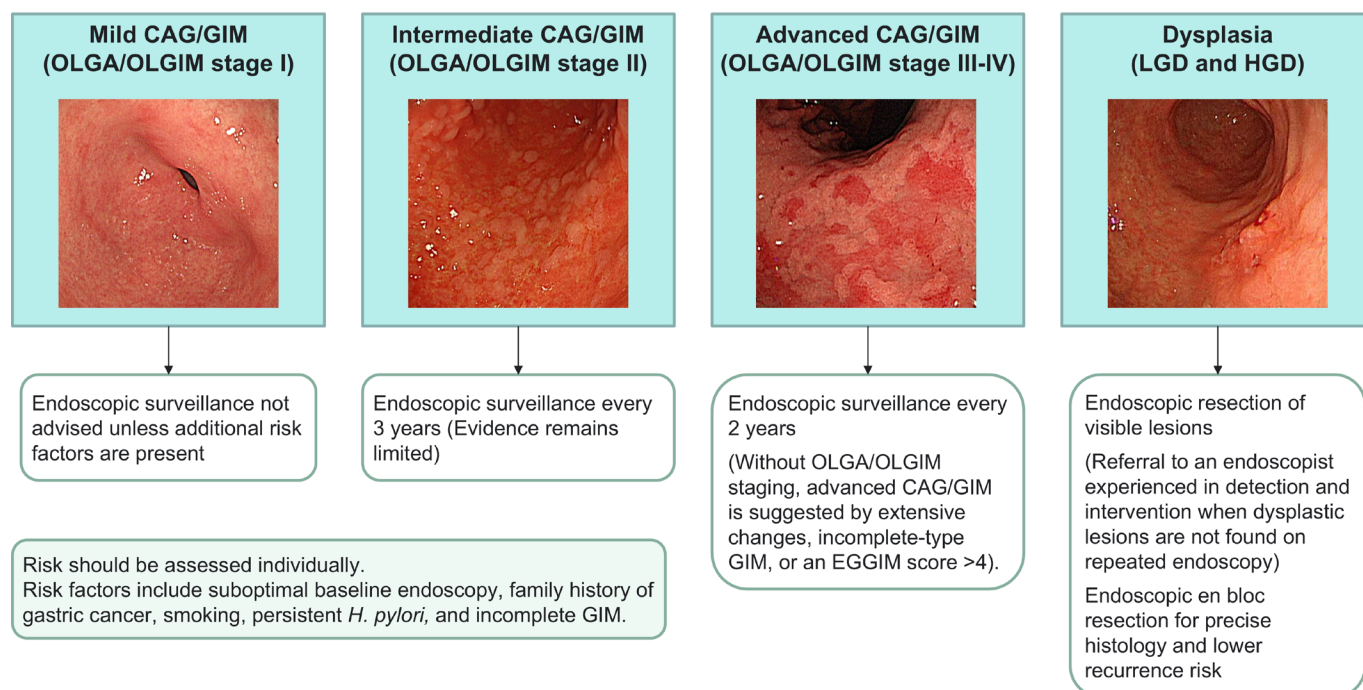


Figure 3 Recommendations for endoscopic surveillance according to the staging of gastric premalignant conditions. CAG, chronic atrophic gastritis; EGGIM, endoscopic grading of gastric intestinal metaplasia; GIM, gastric intestinal metaplasia; HGD, high grade dysplasia; LGD, low grade dysplasia; OLGA, operative link on gastritis assessment; OLGIM, operative link on gastric intestinal metaplasia.

(eg, pepsinogen) are preferred for risk identification, they lack accuracy in predicting EGC, particularly after *H. pylori* eradication, as irreversible mucosal damage may persist and affect pepsinogen reliability.^{121–124} Direct endoscopic and histopathologic evaluations remain the most reliable for risk stratification. Surveillance is not routinely recommended for non-advanced CAG/GIM unless risk factors exist, such as poor baseline endoscopy, first degree family history, smoking, persistent *H. pylori* or incomplete GIM (figure 3).^{12,13,125} Despite the lack of RCTs comparing surveillance versus no surveillance, simulation studies suggest surveillance is cost effective in high risk populations.¹²⁶ Even in US populations, 5 year surveillance for incidentally detected GIM reduced gastric cancer incidence/mortality and was cost effective.¹²⁷ Nonetheless, due to assumptions and variability in parameter inputs within cost effectiveness simulation models, the strength of the recommendation may vary by region.

Statement 19

For patients with advanced stage chronic atrophic gastritis and gastric intestinal metaplasia (OLGA/OLGIM III/IV), endoscopic surveillance every 2 years is recommended

Quality of evidence: low

Strength of recommendation: weak (Conditional)

Strongly agree 55%, agree 40%, neutral 5%

The primary goal of endoscopic surveillance is to detect EGC or DYS at a curable stage. In Japan and South Korea, national policies recommend upper endoscopic screening starting at age 40–50 years, regardless of *H. pylori* status or premalignant conditions.^{128,129} The screening interval is generally based on an estimated 2 year dwelling time, which reflects the window of opportunity to detect EGC before it progresses to an advanced stage.¹²⁹ This dwelling time, derived from the prevalence-to-incidence ratio, is consistent

with observations from routine clinical practice in Asia. Moreover, geographic location does not appear to influence the progression rate from advanced premalignant conditions to gastric cancer, or from EGC to advanced disease.

Patients with OLGA/OLGIM stages III–IV are at increased risk of gastric cancer, with similar progression rates reported globally.¹⁰¹ Although RCTs are lacking, we recommend a 2 year surveillance interval for these high risk individuals, primarily based on cost effectiveness analyses and in alignment with policies implemented in high incidence Asian countries.^{13,15,129} This interval, which is more intensive than the 3 year interval recommended by other international guidelines for endoscopic surveillance,^{8–11} is intended to balance the increased likelihood of early detection with resource efficiency. In South Korea, universal biennial endoscopic screening was found to be cost effective, partly due to lower procedure costs.¹³⁰ In Japan, biennial surveillance following *H. pylori* eradication was also cost effective, taking into account the residual risk of gastric cancer.¹³¹ In the US, a cost effectiveness analysis stratified by race and ethnicity found that endoscopy with mapping biopsies and biennial surveillance for advanced premalignant conditions remained cost effective among Asians.¹³² However, we acknowledge that if future evidence shows a longer interval provides comparable effectiveness in reducing gastric cancer mortality, the surveillance interval could be lengthened to improve cost-efficiency.

Statement 20

For patients with intermediate stage chronic atrophic gastritis and gastric intestinal metaplasia (OLGA/OLGIM stage II), endoscopic surveillance every 3 years is suggested. Different surveillance intervals may be considered depending on the population risk and available resources.

Quality of evidence: low

Strength of recommendation: weak (Conditional)

Strongly agree 65%, agree 30%, neutral 5%

Evidence regarding the increased risk of gastric cancer in individuals with intermediate stage CAG and GIM (OLGA/OLGIM stage II) is limited.^{8 9 12} For patients in this category, surveillance every 3 years may be considered, but supporting evidence remains insufficient, and risk assessment should be individualised, particularly when OLGA/OLGIM is not available (figure 3). A cohort study from Singapore which involved mainly Chinese individuals aged ≥ 50 years demonstrated a stepwise increase in the risk of early gastric neoplasia (ie, HGD and gastric adenocarcinoma), with incidence rates of 12.5 per 1000 person years in the no GIM group, 21.5 in OLGIM I, 108.8 in OLGIM II and 543.8 in OLGIM III–IV. Notably, the intermediate category (OLGIM II) accounted for approximately a quarter of subsequent cases of early gastric neoplasia.³² These results implied that patients with intermediate stage CAG and GIM within a high risk population may benefit from endoscopic surveillance after considering individual factors, such as poor quality baseline endoscopy, first degree family history of gastric cancer, smoking, persistent *H pylori* infection or the presence of incomplete-type GIM. However, it may not apply to regions with lower cancer incidences. A long term follow-up study in Italy found an incidence of gastric cancer of 1.48 (95% CI 0.48 to 4.58) per 1000 person years for OLGA II patients.¹³³ It is important to note that this recommendation also may not apply to patients with autoimmune gastritis, that are at least OLGA/OLGIM stage II¹³⁴ and could have distinct malignant potential, including increased risk of neuroendocrine tumours, but is outside the scope of our recommendation.

Statement 21

For patients with mild chronic atrophic gastritis and gastric intestinal metaplasia (OLGA/OLGIM stage I), endoscopic surveillance may not be justified but can be considered based on individual and population risks, as well as resources available.

Quality of evidence: low

Strength of recommendation: weak (Conditional)

Strongly agree 60%, agree 40%

In patients with mild CAG or GIM (OLGA/OLGIM stage I), routine surveillance is generally not recommended unless additional risk factors are present. This is primarily because mild CAG or GIM is commonly observed, the risk of developing gastric cancer is low and routine endoscopic surveillance is not cost effective in most countries unless the population's gastric cancer risk is high⁶ or the individual has an elevated risk (eg, family history of gastric cancer). Additionally, the progression rate is relatively slower, and risk factors associated with histopathological progression,³² such as persistent *H pylori* infection and lifestyle risk factors,^{124 135} can often be treated or modified after the endoscopic examination through interventions like *H pylori* eradication and lifestyle changes.^{121 136 137} However, this should not be interpreted as there being no gastric cancer risk for these patients.¹³⁸ Histopathological grading can also be influenced by factors such as biopsy location, limited number of samples and the patchy distribution of premalignant conditions, as well as variability in histopathological interpretations.^{90 101} With many uncertainties, clinical decisions should therefore consider both individual and population level risks, as well as the available resources.

Part 4. Management**Statement 22A**

APAGE recommends endoscopic resection of visible lesions concerning for, or histopathologically confirmed to be, dysplasia, with the goal being en bloc R0 resection.

Quality of evidence: low

Strength of recommendation: Strong

Strongly agree 80%, agree 20%

Statement 22B

In cases where the biopsy report shows dysplasia but no focal lesions are identified on the repeat high quality endoscopy (so-called non-visible dysplasia), referral to an endoscopist with experience of endoscopic detection and intervention is recommended. Surveillance endoscopy should be performed every 6 months for patients with resected high grade dysplasia and annually for low grade dysplasia, for a minimum duration of 5 years, with relaxed intervals but ongoing surveillance thereafter.

Quality of Evidence: low

Strength of recommendation: weak (Conditional)

Strongly agree 80%, agree 20%

DYS is clinically significant, being associated with synchronous gastric cancer in nearly 10% of cases and an increased risk of progression to gastric cancer.^{139–141} For visible focal gastric lesions with DYS, endoscopic en bloc resection is recommended, as it also allows for proper histopathological assessment. Therefore, en bloc resection is necessary even for LGD to ensure thorough evaluation and management. For lesions ≤ 15 mm, cap assisted endoscopic mucosal resection is generally effective and straightforward, while for lesions >15 mm, endoscopic submucosal dissection is preferred due to its superior en bloc resection and lower recurrence rates, despite its greater complexity and higher risks related to the procedure, such as bleeding and perforation.^{142–144} Specifically for endoscopic submucosal dissection, the MAPS III guidelines recommend endoscopic submucosal dissection for differentiated lesions staged as dysplastic or intramucosal carcinoma (of any size if not ulcerated and <30 mm if ulcerated),¹¹ which follows the latest Japanese recommendation.¹⁴⁵

In cases where HGD or LGD is detected incidentally from random biopsies, but no focal lesions are found on repeat high quality endoscopy, ongoing surveillance is needed.¹⁴⁶ A single centre study from China showed that during a median follow-up of 4.2 years, 51.4% of patients with baseline mild-to-moderate DYS showed no detectable DYS.¹⁴⁷ Within 5 years of diagnosis, the annual incidence rate of advanced neoplasia and gastric cancer was 0.43% and 0.26%, respectively, which was much lower than previous reports.⁷ While the results of this single centre study should be interpreted with caution, it also highlights the role of repeated examination, preferably by expert endoscopists and pathologists. Advanced endoscopic techniques, such as image enhanced endoscopy, chromoendoscopy or AI assistance, have been shown to improve detection rates, revealing lesions missed in the initial examinations.^{64 148} Undetected and unresected DYS is associated with a substantially higher risk of progression to gastric cancer, highlighting the need for careful follow-up.¹¹⁴ Specifically, patients with resected HGD should undergo surveillance endoscopy every 6 months, while those with resected low grade DYS could be monitored annually. This frequent surveillance should continue for at least 5 years.¹⁴⁷ In patients with indefinite for dysplasia, where the extent of cellular abnormality is unclear or insufficient to classify as LGD or HGD, a similar strategy of close surveillance and repeated histopathological assessment is recommended.^{8 9}

Statement 23

APAGE recommends test-and-treat for *Helicobacter pylori* in individuals with gastric premalignant conditions to reduce the risk of histopathological progression and gastric cancer

Quality of evidence: moderate

Strength of recommendation: strong

Strongly agree 85%, agree 15%

A recent meta-analysis of 11 RCTs¹⁴⁹ showed that *H pylori* eradication therapy reduced the risk of gastric cancer and gastric cancer related death by 36% and 22% among *H pylori* infected individuals without gastric neoplasia, respectively. The pooled results from some observational studies also showed consistent results.^{150 151} However, for patients with GIM, there was no significant benefit on gastric cancer risk (RR 0.7 6; 95% CI 0.36 to 1.61),¹⁵² which is consistent with other systematic reviews and meta-analyses.^{153 154} In contrast, gastric cancer risk was still reduced by 48% after *H pylori* eradication therapy among those with gastric neoplasia undergoing endoscopic mucosal resection who were likely having concurrent premalignant changes.¹⁴⁹

Histopathological progression of GIM is defined as (1) progression to DYS or non-cardia gastric cancer or (2) increase in histopathological score in terms of Correa's cascade, OLGA or OLGIM. Correa's precancerous cascade can be categorised into six stages, from normal to non-atrophic gastritis, multifocal atrophic gastritis, GIM, DYS and gastric cancer.^{5 22 155 156} Mera *et al* reported that the histopathological score increased by an average of 0.20 units per year (95% CI 0.12 to 0.28) in individuals with persistent *H pylori* infection.¹⁵⁶ In a meta-analysis conducted within AGA technical review, the RR of histopathological progression after *H pylori* eradication for all patients in terms of Correa's histopathological score was 0.91 (95% CI 0.83 to 1.00).¹⁵² However, for those with GIM, the RR was 1.17 (95% CI 1.01 to 1.36).¹⁵²

On the other hand, histopathological regression of GIM is defined as (1) regression to normal mucosa, chronic gastritis or CAG or (2) decrease in histopathological score stage.¹⁵⁷ According to the study by Mera *et al*, *H pylori* eradication was associated with a progressive decline in the score. At the 6 year follow-up, the score for those subjects was 0.13 less than baseline (95% CI 0.11 to 0.15) while at 12 years the score was 0.59 less than baseline (95% CI 0.51 to 0.67).¹⁵⁵ The Shandong Interventional Trial^{158 159} reported the outcome in patients with GIM, showing an RR of 1.56 (95% CI 1.03 to 2.33). Another meta-analysis showed that in terms of regression in CAG and GIM, the pooled OR from four RCTs was 2.61 (95% CI 1.41 to 4.81) and 2.61 (95% CI 1.66 to 4.11), respectively.¹⁵³ Notably, some meta-analyses^{153 154} only observed the regression of premalignant conditions in studies with follow-up periods longer than 5 years.

In view of the rising prevalence of antibiotic resistance of *H pylori*,¹⁶⁰ successful *H pylori* eradication should be confirmed by preferably a non-invasive test (either ¹³C urea breath test or faecal antigen test). Endoscopy for biopsy can be considered when there is a concomitant indication (eg, follow-up for gastric ulcer).

Statement 24

APAGE recommends test-and-treat for *Helicobacter pylori* infection for individuals with first degree relatives diagnosed with gastric cancer. Endoscopic screening for detecting both *H pylori* infection and gastric premalignant conditions and/or cancer can be considered.

Quality of evidence: high

Strength of recommendation: Strong

Strongly agree 70%, agree 30%

Germline pathogenic variants are closely associated with the risk of gastric cancer¹⁶¹ and familial aggregation of gastric cancer occurs in approximately 10% of cases.¹⁶² A meta-analysis showed that among first degree relatives of patients with gastric cancer, the OR of *H pylori* infection, CAG and GIM were 1.93 (95% CI 1.42 to 2.61), 2.20 (95% CI 1.27 to 3.82) and 1.98 (95% CI 1.36 to 2.88), respectively.¹⁶³ When compared with those without a family history of gastric cancer, the pooled RR of progression from GIM to gastric cancer among those with a family history of gastric cancer was 4.53 (95% CI 1.33 to 15.46).¹⁵²

A test-and-treat strategy for *H pylori* in individuals with first degree relatives diagnosed with gastric cancer is a proactive approach to reduce the risk of developing gastric cancer. Various international and Chinese guidelines^{9 121 129 164} recommend *H pylori* screening among individuals at high gastric cancer risk (including those with a family history or first degree relatives having gastric cancer). Familial based testing may also be considered,^{125 165} particularly in regions with a high prevalence of *H pylori* infection. A meta-analysis of 12 studies from different countries (including nine from China) showed a nearly three-fold higher *H pylori* eradication success rate when comparing a whole family test-and-treatment strategy with a single patient treatment strategy, and a 70% lower rate of recurrence was noted in the former strategy.¹⁶⁶

Results from a cluster RCT found a beneficial effect of *H pylori* treatment, particularly for those aged 25–45 years, leading to a 27% decrease in the risk of developing gastric cancer.¹³⁵ A nationwide population based study showed that in patients with and without a family history of gastric cancer, early treatment of *H pylori* infection at a younger age could maximise gastric cancer prevention, the maximal benefit being observed among those who had *H pylori* eradication at age <45 years.¹⁶⁷

In addition to *H pylori* screening, individuals with a family history of gastric cancer may also need endoscopic screening to detect gastric premalignant conditions and gastric cancer.¹²¹ The recommended starting age of endoscopy screening is 45–50 years^{9 84 121} or 10 years earlier than the onset age of the first degree relative diagnosed with gastric cancer.¹⁰

Statement 25

APAGE suggests against routine empirical use of proton pump inhibitors and potassium competitive acid blockers, particularly long term use, in individuals with gastric premalignant conditions in the absence of compelling indications.

Quality of evidence: low

Strength of recommendation: weak (Conditional)

Strongly agree 85%, agree 15%

A meta-analysis of six RCTs (n=1623) showed that use of proton pump inhibitors (PPIs) was associated with a higher risk of premalignant conditions when used for more than 12 months (RR 2.21; 95% CI 1.47 to 3.33). The RRs for CAG, GIM and DYS with PPI use were 1.50 (95% CI 0.91 to 2.47), 1.93 (95% CI 1.03 to 3.63) and 7.73 (95% CI 0.36 to 165.7), respectively.¹⁶⁸ Another meta-analysis showed that the harmful effect of PPIs were limited to non-cardia gastric cancer (RR 2.38; 95% CI 1.90 to 2.98), but not cardia gastric cancer (RR 1.32; 95% CI 0.84 to 2.03).¹⁶⁹ A dose-response relationship was reported in a few studies. Abrahami *et al* showed that a higher dose of omeprazole equivalents was associated with a higher risk of gastric cancer.¹⁷⁰ Cheung *et al* recruited a cohort of *H pylori*

eradicated individuals, showing that at least weekly use of PPIs was associated with a 2.44-fold higher risk of gastric cancer.¹⁷¹ Furthermore, *H pylori* infection, either current or past, was a more important determinant of gastric cancer risk than PPIs, possibly due to the development of *H pylori* induced premalignant conditions.^{171 172}

While strong evidence from RCTs is still lacking, one RCT recruiting patients receiving aspirin or rivaroxaban who were assigned to either pantoprazole 40mg daily or placebo did not reveal an increase in gastric cancer risk.¹⁷³ However, aspirin is a known chemopreventive agent against many solid organ tumours, including gastric cancer,^{174 175} and the PPI associated gastric cancer risk may be negated by concomitant aspirin use.¹⁷⁶ Moreover, the study investigated multiple outcomes, posing a risk of underpower for detecting gastric cancer occurrence. Since long term use of PPIs is associated with various potential side effects,^{177 178} the indications for PPIs should be regularly reviewed and de-prescription of PPIs should be attempted when there is no indication.^{179 180}

Potassium competitive acid blockers, with a more potent gastric acid suppressive effect than PPIs, have also been shown to be associated with a nearly twofold higher gastric cancer risk in a population based cohort study of 54 055 patients, with a duration and dose–response relation.¹⁸¹ We therefore advise a similar cautious approach for use of potassium competitive acid blockers, although strong evidence supporting this is lacking.

Statement 26

APAGE recommends against the routine use of non-*Helicobacter pylori* chemopreventive agents (including aspirin or cyclooxygenase-2 inhibitors) as primary prevention of gastric cancer in individuals with gastric premalignant conditions.

Quality of evidence: moderate

Strength of recommendation: strong

Strongly agree 65%, agree 35%

Meta-analyses showed that aspirin and NSAIDs were associated with a lower gastric cancer risk in observational studies, while post hoc analysis of RCTs showed a trend favouring aspirin use.^{182 183} However, most studies did not report the status of gastric premalignant conditions and included a heterogeneous population of both *H pylori* infected and non-infected individuals.

One meta-analysis of five studies found that COX-2 inhibitors reduced gastric cancer risk by 55%.¹⁸⁴ Leung *et al* showed that rofecoxib did not regress GIM and its severity over 2 years in 213 *H pylori* eradicated individuals with GIM.¹⁸⁵ It is noteworthy that rofecoxib was subsequently withdrawn from the market due to cardiovascular adverse events.¹⁸⁶ On the other hand, another RCT showed that celecoxib use for 2 years could regress advanced gastric lesions in *H pylori* infected individuals, but not in *H pylori* eradicated subjects.¹⁸⁷ Given the uncertain benefits of NSAIDs and COX-2 inhibitors in regressing gastric premalignant conditions and cancer prevention, particularly among those with *H pylori* eradicated, and their side effects,^{188 189} we do not recommend NSAIDs or COX-2 inhibitors solely for gastric cancer prevention purposes.

We also do not recommend routine use of aspirin for chemoprevention of gastric cancer. However, given its additional cardiovascular and cancer prevention benefits, the use of low dose aspirin should be individualised if other compelling indications exist. A cohort study of *H pylori* eradicated individuals showed that aspirin users had seven fewer gastrointestinal cancer

related deaths but one more bleeding related death per 10 000 person years than non-users.¹⁷⁵

Rebamipide, a mucosal protective agent, was shown in various RCTs to reduce the risks of GIM and low grade DYS,^{190 191} to improve the histopathologic grade of CAG and GIM in the antrum and to alleviate chronic inflammation in the lesser curvature of the corpus after *H pylori* eradication.^{192 193} Moluodan, a compound prescription of Chinese herbal medicine, resulted in an 82.8% disappearance rate of DYS in a randomised, double blind, parallel controlled trial.¹⁹⁴ However, the recommendations for their routine use require further research.

Statement 27

APAGE does not recommend endoscopic treatment of gastric intestinal metaplasia, such as through endoscopic ablation or resection.

Quality of evidence: low

Strength of recommendation: Strong

Strongly agree 65%, agree 35%

Endoscopic ablation or resection is generally not recommended for eradication of GIM, especially because it is a multifocal condition. As GIM is considered a field effect suggesting a higher risk of cancer development, we emphasise the importance of careful surveillance in patients with GIM while asserting that intervention should be reserved for cases of confirmed DYS.¹⁹⁵ Furthermore, endoscopic therapies, such as ablation, have not been shown to be effective in eradicating GIM and may introduce potential risks. A study tested radiofrequency ablation for both GIM and LGD, finding that while it successfully eradicated DYS in all patients with LGD, GIM persisted in most cases.¹⁹⁶ Endoscopic resection carries potential complications, such as perforation or bleeding, which can complicate further management.¹⁹⁷

Statement 28

APAGE recommends that healthcare providers counsel patients with gastric premalignant conditions to adopt a healthy lifestyle (including smoking cessation and a healthy diet, etc), as this can lower the risk of progression towards gastric cancer.

Quality of evidence: moderate

Strength of recommendation: strong

Strongly agree 40%, agree 55%, neutral 5%

Personal lifestyle choices are closely linked to the progression of gastric cancer. A prospective cohort study including 100 220 individuals found that an unfavourable lifestyle was associated with a twofold higher risk of gastric cancer.¹⁹⁸ Even for those with a high genetic risk, a healthy lifestyle could help reduce the risk of gastric cancer by 47%.¹⁹⁸ A summary of the management strategies for gastric premalignant conditions is shown in figure 4.

Smoking and alcohol

A recent meta-analysis showed that compared with never smokers, the pooled RR of gastric cancer for current and former smokers was 1.53 (95% CI 1.44 to 1.62) and 1.30 (95% CI 1.23 to 1.37), respectively.¹⁹⁹ In the Singapore Gastric Epidemiology and Molecular Genetics Programme prospective cohort, OLGIM II–IV patients who smoked ≥ 20 pack years were associated with a 3.7-fold higher risk of early gastric neoplasia.³²

The World Cancer Research Fund International Continuous Update Project highlights strong evidence that consuming three or more alcoholic drinks per day raises the risk of gastric cancer.^{200 201} A pooled analysis of 20 epidemiological studies showed that heavy drinkers (more than four to six drinks per

Recommended	NOT Recommended
Test-and-treat for <i>H. pylori</i> infection in individuals with gastric premalignant conditions and individuals with family history of gastric cancer 	Routine empirical use proton pump inhibitors or potassium competitive acid blockers in individuals with gastric premalignant conditions in the absence of compelling indications 
Adopt healthy lifestyle such as smoking cessation and healthy diet (e.g. low salt diet, fresh vegetables and fruits) 	Routine use of non-<i>H. pylori</i> chemopreventive agents (including aspirin and anti-oxidants)  Endoscopic ablation/resection of GIM 

Figure 4 Management strategies for gastric premalignant conditions.

day) and very heavy drinkers (more than six drinks per day) had a 26% and 48% increase in gastric cancer risk, respectively.²⁰² A Korean study of 202 675 individuals showed that alcohol consumption was associated with both CAG and GIM in a dose-response manner.²⁰³

Dietary factors

The third expert report by the World Cancer Research Fund and the American Institute for Cancer Research concluded that there was strong evidence linking salt preserved foods to gastric cancer.²⁰⁴ A meta-analysis involving 2 076 498 participants demonstrated that high salt intake was associated with a twofold higher risk of gastric cancer.²⁰⁵ Another meta-analysis revealed a trend favouring association between salted/salty foods and GIM (OR 1.68; 95% CI 0.98 to 2.90).²⁰⁶

The IARC classified processed meat as a group 1 carcinogen (carcinogenic to humans) and red meat as group 2A (probably carcinogenic to humans).⁴³ A meta-analysis including 43 cohort and case-control studies found that a high intake of processed meat and red meat was associated with a 57% and 41% higher risk of gastric cancer, respectively.²⁰⁷

A meta-analysis of 18 prospective studies indicated that increased consumption of fruits alone and in combination with vegetables was associated with a 13% and 25% lower risk of gastric cancer, respectively.²⁰⁸ On the other hand, the data regarding the effects of milk and dairy products on gastric cancer risk are limited and inconclusive.^{209 210} A meta-analysis of 60 studies showed a 52% increased risk of gastric cancer with the consumption of pickled foods.²¹¹ Another meta-analysis of 18 studies revealed fried food intake was associated with a 1.5-fold higher risk of gastric cancer.²¹²

Physical activity and body mass index

A meta-analysis of 22 observational studies indicated that any form of physical activity, whether occupational, recreational or both, was linked to a 19% lower risk of gastric cancer.²¹³

A retrospective cohort study of 142 832 Korean adults showed that compared with individuals with a body mass index of 18.5–22.9 kg/m², those with a body mass index of >30 kg/m² had a 1.48-fold higher risk of GIM, while those with a body mass index of at least 23 kg/m² had at least 1.1-fold higher risk of CAG.²¹⁴

Antioxidant consumption

A meta-analysis of 20 RCTs involving the supplementation of beta-carotene, vitamin A, vitamin C, vitamin E and selenium (total n=211 818) revealed that antioxidant supplements overall did not significantly influence the risk of gastric cancer (RR 1.14; 95% CI 0.97 to 1.33).²¹⁵ While the Shandong Intervention Trial of China found no beneficial effects of either vitamins (C, E and selenium) or garlic (extract and oil) supplements for 7.3 years on gastric premalignant conditions,¹⁵⁹ a beneficial effect was noted in terms of reduction of gastric cancer and cancer related mortality after 22.3 years of follow-up.²¹⁶ Another large trial involving 29 133 male smokers in Finland, the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study, showed no significant effect of antioxidant supplementation on gastric cancer during and beyond the trial period.^{217 218} On the other hand, a meta-analysis of 13 studies suggested that a daily dose of 20–30 mg of folic acid for 3–6 months improved the pathological changes associated with gastric premalignant conditions.²¹⁹ Therefore, evidence is still conflicting to support the routine use of these antioxidant supplementations. Whether a differential effect of antioxidants exists between Asian and non-Asian populations also requires further exploration.

LIMITATIONS AND FUTURE RESEARCH

We acknowledge that some of the recommendations are based on limited quality evidence and necessitate expert opinions from the region. Specifically, there are no high quality data to support the recommended surveillance intervals, particularly in those with non-advanced premalignant conditions. We recommended

Table 2 Comparison of Asian Pacific Association of Gastroenterology (APAGE) consensus with existing updated guidelines/recommendations

	2020, 2021, 2025 AGA guideline and CPUs	2025 ACG guideline ¹⁰	2025 MAPS III ¹¹	2025 APAGE consensus statement
Advanced CAG/GIM (eg, OLGA/OLGIM stages III–IV) and surveillance recommendation	2025⁴ Best practice advice 9: Individuals with severe CAG and/or multifocal/incomplete GIM are likely to benefit from endoscopic surveillance, particularly if they have other risk factors for GC (eg, family history). Endoscopic surveillance should be considered every 3 years; however, intervals are not well defined and shorter intervals may be advisable in those with multiple risk factors, such as severe GIM that is anatomically extensive. 2021¹²⁵ Providers should consider performing endoscopic surveillance every 3 years in patients with advanced CAG. However, it should be recognised that optimal surveillance intervals remain to be determined, and shorter or longer intervals may be appropriate depending on individual risk assessment 2020¹² Recommendation 2: Patients with GIM specifically at higher risk of gastric cancer include those with: ► Incomplete vs complete GIM ► Extensive vs limited GIM ► Family history of gastric cancer Recommendation 3: Patients with GIM and high risk stigmata, concerns about completeness of baseline endoscopy, and/or who are at overall increased risk for gastric cancer may reasonably elect for repeat endoscopy within 1 year for risk stratification.	Recommendation 9. In individuals with GIM who are considered high risk for GC, suggest endoscopic surveillance at 3 year intervals. High risk groups include individuals with GIM and at least one of the following criteria: 1. High risk GIM histology: ► Incomplete GIM histological subtype vs complete subtype ► Corpus extension, defined as corpus involvement also with antrum or incisura involvement Any GIM histology with one of the following risk factors for GC: ► Family history of GC in a first degree relative ► Foreign born, with emigration from a high incidence nation ► High risk race or ethnicity, including East Asian, Latino/a, Black and AIAN individuals Recommendation 10. In individuals with severe GIM or CAG in biopsies of the antrum or corpus, suggest endoscopic surveillance at 3 year intervals	Recommendation 31. Recommend that patients with extensive endoscopic changes (C3+ or EGGIM 5+) or advanced histological stages of atrophic gastritis (severe CAG or GIM and/or significant changes in both antrum and corpus, OLGA/OLGIM III/IV) should be followed up with high quality endoscopy every 3 years. Recommendation 33. Suggest that endoscopic features of extensive changes (C3+ or EGGIM 5+) or histological advanced stages of atrophic gastritis (severe atrophic changes or IM in both antrum and corpus, OLGA/OLGIM III/IV) and with a first degree relative with GC may benefit from a more intensive follow-up (eg, every 1–2 years after diagnosis)	Statement 19. For patients with advanced stage CAG and GIM (OLGA/OLGIM stages III/IV), endoscopic surveillance every 2 years is recommended
Intermediate CAG and GIM (eg, OLGA and OLGIM II)	NA	NA	Recommendation 34. Recommend no surveillance endoscopy for patients with mild to moderate CAG or GIM restricted to the antrum, in the absence of endoscopic signs of extensive lesions or other risk factors (family history, incomplete intestinal metaplasia or persistent <i>H pylori</i> infection). This group constitutes most individuals found in clinical practice	Statement 20. For patients with intermediate stage CAG and GIM (OLGA/OLGIM stage II), endoscopic surveillance every 3 years is suggested. Different surveillance intervals may be considered depending on the population risk and available resources

Continued

Table 2 Continued

	2020, 2021, 2025 AGA guideline and CPUs	2025 ACG guideline ¹⁰	2025 MAPS III ¹¹	2025 APAGE consensus statement
Mild CAG and GIM (eg, OLGA and OLGIM I)	2025⁴ Mild AG and/or focal complete type GIM with at least one of the following: 1. family history of GC 2. persistent <i>H pylori</i> infection Then OGD surveillance should be considered every 3–5 years. Otherwise, no further OGDs for gastric cancer screening/CAG-GIM surveillance advised	Recommendation 11. In individuals with low risk GIM or atrophy, ACG suggest against endoscopic surveillance. Low risk groups include 1. Complete type GIM, without evidence of incomplete GIM 2. Complete GIM of focal anatomic extent that is confined to the antrum 3. None of the high risk clinical criteria listed in Recommendation 9 above 4. CAG which is mild in severity	Recommendation 34. Same as above Recommendation 35. Suggest that in patients with GIM at a single location but with a family history of GC, or with incomplete IM, or with persistent <i>H pylori</i> gastritis, high quality endoscopic surveillance every 3 years may be considered	Statement 21. For patients with mild CAG and GIM (OLGA/OLGIM stage I), endoscopic surveillance may not be justified but can be considered based on individual and population risks, as well as resources available

ACG, American College of Gastroenterology; AGA, American Gastroenterological Association; AIAN, American Indian and Alaska Native; APAGE, Asian Pacific Association of Gastroenterology; CAG, chronic atrophic gastritis; CPU, clinical practice update; EGGIM, Endoscopic Grading of Gastric Intestinal Metaplasia; ESGE/EHMSG/ESP, European Society of Gastrointestinal Endoscopy/European Helicobacter and Microbiota Study Group/European Society of Pathology; GC, gastric cancer; GIM, gastric intestinal metaplasia; GPMC, gastric premalignant condition; HDWLE, high definition white light endoscopy; IEE, image enhanced endoscopy; MAPS, management of epithelial precancerous conditions and early neoplasia of the stomach; NA, not available; OGD, oesophagoduodenoscopy; OLGA, operative link on gastritis assessment; OLGIM, operative link on gastric intestinal metaplasia assessment.

more frequent surveillance of 2 years for patients with OLGA/OLGIM stage III or IV, and 3 years for those with stage II disease. This is to a certain extent aligned with the screening interval of upper endoscopy in some East Asian countries which practise nationwide screening endoscopy and is supported by cost effectiveness studies.^{130–132} Comparison with current international guidelines and recommendations on risk stratification and surveillance intervals are shown in table 2. Specifically, there are some differences in the recommended surveillance intervals between the APAGE and the existing recommendations from the US and Europe. Compared with the MAPS III guideline, which establishes structured surveillance intervals based on OLGA/OLGIM staging, our recommendations also adopt a risk stratified framework but incorporate local practical adjustments that reflect existing regional gastric cancer screening practices and diagnostic capabilities. Conversely, the North American guidelines focus more on individualised assessments, particularly for less advanced premalignant conditions.^{10 12} Moreover, unlike recent guidelines from Europe¹¹ and the USA,¹⁰ our consensus did not cover other premalignant conditions, such as gastric polyps and autoimmune gastritis. While we attempted to focus on *H pylori* associated gastric premalignant changes which are prevalent in this region, this omission represents a limitation of the present consensus, and future revisions and updates are needed to incorporate these different entities to achieve more comprehensive coverage.

Although we aim to harmonise surveillance strategies in Asia, we must admit that significant variations exist in endoscopic practices and healthcare delivery systems among Asian countries. For example, in Japan and South Korea, nationwide gastric cancer screening programmes use endoscopy every 2 years, often without obtaining routine biopsies. Instead, their strategy relies on repeated examinations to identify suspicious lesions and detect neoplastic changes at an early stage. In contrast, some countries in the region lack adequate endoscopic infrastructures for surveillance, a standardised biopsy protocol and even expertise on endoscopic and histopathological diagnosis. These disparities may present a considerable challenge to the

implementation of a unified surveillance strategy across Asia and may highlight the necessity of a united call for further funding support and resources allocation to this topic. We also recognise the limitation in the composition of our panel members, as experts from certain high burden Asian countries were not represented (eg, Mongolia). Members also did not include Japanese pathologists, family physicians and primary care providers, which may not have adequately addressed the wider perspectives. Future consensus efforts should strive for more inclusive representation from across the Asia Pacific region with different specialties.

Further data are needed to quantify the optimal surveillance intervals for patients with different risk profiles. In particular, the use of genetic markers together with family history and other modifiable risk factors may further help to precisely risk stratify patients for personal surveillance.¹⁶¹ Some other areas of future research are listed below.

- The role of different new image enhanced endoscopy (eg, TXI and LCI) or AI assistance on the detection of gastric neoplastic lesions remains uncertain.
- Studies are also needed to identify additional potential chemopreventive agents for individuals with premalignant conditions after *H pylori* infection, as these individuals are still at risk of developing gastric cancer.
- Identification of novel non-invasive gastric mucosal biomarkers or molecular markers for risk stratification and surveillance of patients with gastric premalignant conditions.
- A regional registry and clinical trial data to better inform the optimal surveillance intervals in patients with gastric premalignant conditions

CONCLUSIONS

This current consensus report provides the first systematic framework for the surveillance and management of gastric premalignant conditions, leveraging the best available evidence, to the Asia Pacific region. Based on current knowledge of gastric cancer risk stratification, diagnostic modalities and surveillance

strategies, these recommendations aim to standardise and enhance early detection and intervention for this common and fatal cancer in Asia. Additionally, further refinement of risk stratification models, along with the availability of novel diagnostic and surveillance tools, is essential to improve the precision of recommendations with the goal of personalised risk stratification. While we acknowledge differences within Asian countries in terms of gastric cancer incidence, endoscopy practices and healthcare resources, our current recommendation could help to identify areas for future research and inform policy to address the knowledge gaps and disparities.

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