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European
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Group

HELICOBACTER PYLORI

Consensus Report Maastricht VI - Florence

27th – 28th
September 2021

The VI Edition

Chairman Prof. Peter Malfertheiner

UNDER THE AUSPICES OF



PRESIDENT OF THE MEETING

Prof. P. Malfertheiner

OVGU, University Medical faculty, Magdeburg
LMU, Universitätsklinikum München, Med. Klinik II, DE

CO-ORGANIZED BY

European Helicobacter & Microbiota Study Group
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We are here to finalize the 6th *H. pylori* consensus conference (Maastricht VI//Florence) in a face-to-face meeting and we are truly sorry that some of our team members from outside Europe will have to be connected via Video. We will sincerely miss their presence on site.

This event will bring the dedicated process of researching on the latest developments in the clinical field of *H. pylori*, the writing of comments according to best available scientific evidence and expressing votes on the elaborated statements to a successful end.

To all delegates, clinical experts, and scientists for their invaluable contributions a big Thank-you!

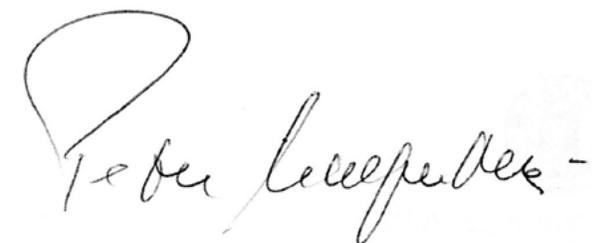
The structure of the meeting is designed to provide time slots for all 5 Working Groups to present each individual statement by the endorsed delegate for general final voting. Statements already accepted in the Delphi round are not intended for further discussion of their contents.

Statements left with uncertainty because of controversial opinions however will be repropose for clarification, debate, and final voting.

We believe that in the context of the unique event of the *H. pylori* Consensus development we will appreciate and benefit in receiving short presentations of the latest News on basic and translational science, diagnostic procedures, therapeutic strategies, preventive measures, and the interaction of *H. pylori* with gut microbiota.

We look forward to a challenging and rewarding meeting with on site and Video assisted get together for the successful conclusion of Maastricht VI/ Florence *H. pylori* consensus 2021.

Thank you again for all your enthusiasm and efforts to contribute.

A handwritten signature in black ink, appearing to read 'Peter Malfertheiner', with a large, stylized initial 'P'.

Prof. P. Malfertheiner
On Behalf of EHMSG
President of the Meeting

H. PYLORI CONSENSUS REPORT MAASTRICHT VI FLORENCE

Monday, 27th September 2021 - Morning

08.45 a.m. 09.00 a.m.	<p>Welcome and introduction</p> <p>P. Malfertheiner (München, DE) – President of the meeting</p> <p>G. Caracciolo (Florence, IT) – Fondazione Internazionale Menarini</p>
Session I – Progress reports since Maastricht V/Florence	
Moderators:	<p>F. Megraud (Bordeaux, FR)</p> <p>C. O`Morain (Dublin, IRL)</p>
09.00 a.m. 09.20 a.m.	<p>Basic science</p> <p>S. Suerbaum (München, DE)</p>
09.20 a.m. 09.40 a.m.	<p>Translational science</p> <p>E. El Omar (Sydney, AUS)</p>
09.40 a.m. 10.00 a.m.	<p>General Update on the consensus development</p> <p>P. Malfertheiner (München, DE)</p>
Session II – WG 1 Indications	
Moderators:	<p>T. Rokkas (Athens, GR)</p> <p>E. El Omar (Sydney, AUS)</p>
10.00 a.m. 10.15 a.m.	<p>What is new compared to Maastricht V Florence (MV/FL)</p> <p>M. Leja (Riga, LV)</p>
10.15 a.m. 11.15 a.m.	<p>Working group 1</p> <p>Pathogenesis and Clinical Manifestations/Indications for Therapy</p> <p>Presentation of individual statements with voting results by delegates. Please see the list of statements in the appendix. Revoting of each statement</p>
11.15 a.m.	<p><i>Coffee break</i></p>

Session III – WG 2 Diagnosis

Moderators:	E. Kuipers (Rotterdam, NL) M. Rugge (Padua, IT)
11.30 a.m. 11.45 a.m.	Novelties in diagnostic tests since MV/FL F. Megraud (Bordeaux, FR)
11.45 a.m. 12.45 p.m.	Working group 2 Diagnostic Assessment of the Infection and Gastritis Presentation of individual statements with voting results by delegates. Please see the list of statements in the appendix. Revoting of each statement
12.45 p.m.	<i>Lunch</i>

Monday, 27th September 2021 – Afternoon

Session IV – WG 3 Therapy

Moderators:	R. Hunt (London, UK) C. O`Morain (Dublin, IRL)
02.00 p.m. 02.15 p.m.	What is new compared to Maastricht V Florence (MV/FL) J. Gisbert (Madrid, ES)
02.15 p.m. 05.00 p.m.	Working group 3 Treatment/Antibiotic Stewardship Presentation of individual statements with voting results by delegates. Please see the list of statements in the appendix. Revoting of each statement
05.00 p.m.	<i>End of the day 1</i>

08.30 a.m. 09.00 a.m.	Our discovery of <i>H. pylori</i> Honorary Lecture B. Marshall (Perth, AUS)
Session V – WG 4 Prevention, gastric cancer	
Moderators:	J. M. Liou (Lyon, FR) P. Malfertheiner (München, DE)
09.00 a.m. 09.15 a.m.	Prevention strategies any progress since MV/FL K. Sugano (Tokyo, JP)
09.15 a.m. 11.00 a.m.	Working group 4 Pathogenesis and Clinical Manifestations/Indications for Therapy Presentation of individual statements with voting results by delegates. Please see the list of statements in the appendix. Revoting of each statement
11.00 a.m.	<i>Coffee break</i>
Session VI – WG 5 <i>H. pylori</i> and gut microbiota	
Moderators:	A. Gasbarrini (Rome, IT) H. Tilg (Innsbruck, AT)
11.15 a.m. 11.30 a.m.	What is the contribution of other microbiota to <i>H. pylori</i> related pathologies any progress since MV/FL C. Schulz (München, DE)
11.30 a.m. 01.00 p.m.	Working group 5 Pathogenesis and Clinical Manifestations/Indications for Therapy Presentation of individual statements with voting results by delegates. Please see the list of statements in the appendix. Revoting of each statement
01.00 p.m.	<i>Lunch</i>

Tuesday, 28th September 2021 – Afternoon

Session VII – Two outstanding careers

Moderator:	R. Hunt (London, UK)
02.00 p.m. 02.30 p.m.	The contribution of <i>H. pylori</i> to my career and advice for the next generations G. Gasbarrini (Rome, IT) D. Y. Graham (Houston, TX, US)

Session VII – Interactive session

Moderators:	C. Fallone (Montreal, CA) S. Moss (Providence, RI, US))
02.30 p.m. 03.15 p.m.	Ready for <i>H. pylori</i> eradication on the population level? What would be my strategy? D. Bordin (Moscow, RU) M. Leja (Riga, LV) B. Tepeš (Rogaška, SI) J. Y. Park (Lyon, FR)
03.15 p.m. 04.00 p.m.	General Discussion Proceedings, publication, dissemination
04.00 p.m.	Concluding remarks P. Malfertheiner (München, DE)

WORKING GROUP 1

	STATEMENT	DELEGATE/COORDINATOR
1	<p><i>Does H. pylori always cause gastritis?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> infection always causes gastritis, irrespective of symptoms or complications. 	Varocha Mahachai
2	<p><i>Is H. pylori a pathogen or a commensal?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> is a gastric pathogen. Therefore, <i>H. pylori</i> gastritis is an infectious disease. 	Varocha Mahachai and Agreus Lars
3	<p><i>Is test-and-treat an appropriate strategy for uninvestigated dyspepsia?</i></p> <ul style="list-style-type: none"> ▪ Test-and-treat is an appropriate strategy for uninvestigated dyspepsia. 	Agreus Lars and Goh Khean-Lee
4	<p><i>Should an endoscopy-based strategy be considered in patients with dyspeptic symptoms, particularly in populations with low prevalence of H. pylori?</i></p> <ul style="list-style-type: none"> ▪ Endoscopy is not necessary in the initial investigation of dyspepsia in low <i>H. pylori</i> prevalence areas. 	Goh Khean-Lee
5	<p><i>Can H. pylori gastritis increase or decrease acid secretion?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> gastritis is associated with increased, decreased or no overall change in acid secretion in the stomach. 	Moss Steven
6	<p><i>Is H. pylori eradication superior to placebo or acid suppressive therapy for long-term relief of dyspepsia?</i></p> <ul style="list-style-type: none"> ▪ Overall, <i>H. pylori</i> eradication is superior to placebo or acid suppressive therapy for long-term relief of dyspepsia but the magnitude of the benefit is small. 	Moss Steven and Bordin Dmitry
7	<p><i>Does H. pylori gastritis have to be excluded before a reliable diagnosis of functional dyspepsia can be made?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> gastritis has to be excluded before a reliable diagnosis of functional dyspepsia can be made (Level of evidence: high Grade of recommendation: high) 	Lanas Angel
8	<p><i>Does the use of aspirin and NSAIDs increase the risk of ulcer disease in H. pylori infected subjects?</i></p> <ul style="list-style-type: none"> ▪ The use of either aspirin or NSAIDs increases the risk of peptic ulcer disease and its complications in <i>H. pylori</i> infected subjects. 	Lanas Angel

9	<p><i>Should testing for H. pylori be performed on all patients started on long-term aspirin and NSAIDs?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> testing and treatment are advisable for high-risk patients who are already on long-term aspirin, and for naïve patients started on long-term NSAID therapy. Those at high-risk may need additional PPI therapy. 	Lanas Angel
10	<p><i>Do anticoagulants (coumarins, new oral, anticoagulants) increase the risk of bleeding in patients with H. pylori infection?</i></p> <ul style="list-style-type: none"> ▪ There is no evidence to suggest that anticoagulants (coumarins, new oral and VKA) increase the risk of bleeding in patients with <i>H. pylori</i> infection. 	Moss Steven
11	<p><i>Does long-term treatment with PPIs alter the topography of H. pylori gastritis?</i></p> <ul style="list-style-type: none"> ▪ Long-term treatment with PPIs alters the topography of <i>H. pylori</i> gastritis. 	Wu Chun-Ying
12	<p><i>Does eradication of H. pylori heal gastritis in long-term PPI users?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication improves gastritis in long-term PPI users. 	Wu Chun-Ying
13	<p><i>Should H. pylori eradication be given to all patients with unexplained iron deficiency anaemia (IDA), idiopathic thrombocytopenic purpura (ITP), and vitamin B12 deficiency?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication is recommended for patients with unexplained iron deficiency anaemia (IDA), idiopathic thrombocytopenic purpura (ITP) and Vitamin B12 deficiency. 	Moss Steven
14	<p><i>Is H. pylori eradication the first-line treatment for localized low grade gastric MALT lymphoma?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication is the first-line treatment for localized low grade gastric MALT lymphoma. <i>H. pylori</i> eradication therapy is also recommended for cases without evidence of <i>H. pylori</i> infection and may provide benefit even for more advanced staged disease. 	Goh Khean-Lee
15	<p><i>Are there any associations between H. pylori infection and extra-gastrointestinal disorders?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> has been positively and negatively associated with some extra-gastrointestinal disorders. However, the causality of these associations has not been definitively proven. 	Agréus Lars and Bordin Dmitry
16	<p><i>Has the COVID-19 pandemic impacted the management of H. pylori –related diseases?</i></p> <ul style="list-style-type: none"> ▪ The COVID-19 pandemic has negatively impacted management of <i>H. pylori</i> –related diseases. 	Agréus Lars

WORKING GROUP 2

	STATEMENT	DELEGATE/COORDINATOR
1	<p><i>Are there patient groups in whom <i>H. pylori</i> infection can be diagnosed by non-invasive tests alone?</i></p> <ul style="list-style-type: none"> ▪ Non- invasive testing for <i>H. pylori</i> infection is recommended in young dyspeptic patients (age below 50) with no specific risk and no alarm symptoms. 	Di Mario Francesco
2	<p><i>What is the recommended diagnostic workup in dyspeptic patients older than 50 years?</i></p> <ul style="list-style-type: none"> ▪ In dyspeptic patients older than 50 years, upper GI endoscopy is required. Functional serology may be considered as a complementary diagnostic tool. 	Di Mario Francesco
3	<p><i>Is citric acid meal useful for increasing the accuracy and sensitivity of Urea Breath Test (UBT)?</i></p> <ul style="list-style-type: none"> ▪ Citric acid is an essential component of the UBT protocol as it increases its accuracy and sensitivity. 	Smith Stella
4	<p><i>Can stool antigen tests be used to diagnose <i>H. pylori</i> infection?</i></p> <ul style="list-style-type: none"> ▪ Monoclonal stool antigen tests, including rapid tests, are useful and can be used for <i>H. pylori</i> diagnosis. 	Suerbaum Sebastian
5	<p><i>What is the role of <i>H. pylori</i> serological test in the diagnostic work up?</i></p> <ul style="list-style-type: none"> ▪ Tests for serum IgG antibodies against <i>H. pylori</i> can achieve high accuracy (specificity and sensitivity). When locally validated, these remain an important tool for <i>H. pylori</i> diagnosis, especially in those clinical conditions when a low bacterial density may lead to false negative results in other tests. 	Suerbaum Sebastian
6	<p><i>What is the optimal use of upper endoscopy in the management of <i>H. pylori</i> infection?</i></p> <ul style="list-style-type: none"> ▪ When endoscopy is indicated, it should apply the best available technologies and include biopsy sampling. Biopsy samples should be obtained in accordance with validated protocols and classifications both for diagnosis and staging of precancerous conditions and for <i>H. pylori</i> diagnosis. Any focal lesions should be additionally sampled. 	Dinis Ribeiro Mario
7	<p><i>Can molecular methods detect/predict resistance of <i>H. pylori</i> to antibiotics?</i></p> <ul style="list-style-type: none"> ▪ Molecular methods (in particular, PCR, whole genome sequencing, and digital PCR) allow detection of <i>H. pylori</i> mutations associated with resistance to clarithromycin, levofloxacin, tetracycline and rifampicin. 	Suerbaum Sebastian

8	<p><i>Can gastric biopsies previously recovered from Rapid Urease Test (RUT) be reused for molecular testing by PCR with accuracy?</i></p> <ul style="list-style-type: none"> ▪ Gastric biopsies recovered from RUT can be reused for molecular testing by PCR. 	Smith Stella
9	<p><i>Is clarithromycin susceptibility testing (molecular techniques or after culture) recommended before prescribing any clarithromycin containing therapy?</i></p> <ul style="list-style-type: none"> ▪ Clarithromycin susceptibility testing (molecular techniques, or after culture) is recommended before prescribing any clarithromycin containing therapy. 	Megraud Francis
10	<p><i>What treatment can be used in the short-term post-eradication (4-6 weeks) follow-up to permit optimum testing for H. pylori?</i> <i>Alternative wording: What is the optimal time and circumstances to test for H. pylori after eradication therapy?</i></p> <ul style="list-style-type: none"> ▪ The optimal time to test for <i>H. pylori</i> eradication is 4-6 weeks after cessation of treatment. No PPI, antibiotics or bismuth should be used for at least 4 weeks prior to testing. 	Megraud Francis
11	<p><i>Do non-invasive tests potentially provide clinically valuable information on the likelihood of gastric atrophy and its aetiology?</i></p> <ul style="list-style-type: none"> ▪ Gastric functional serology (pepsinogen I-II, gastrin), anti-<i>H. pylori</i> antibodies, anti-intrinsic factor and anti-parietal cell auto-antibodies, may provide clinically valuable information on the likelihood of gastric mucosa atrophy, including its aetiology 	Megraud Francis
12	<p><i>What is the definition of atrophic gastritis and why is it important to report?</i></p> <ul style="list-style-type: none"> ▪ Gastric mucosal atrophy is defined as “loss of native glands”. Atrophy is the major determinant of non-hereditary gastric cancer risk and it may be (complementarily) assessed by gastric serology, endoscopy, and histology. 	Kuipers Ernst J.
13	<p><i>How should gastric atrophy be histologically assessed and reported?</i></p> <ul style="list-style-type: none"> ▪ The histological assessment of atrophy should result in a conclusive gastritis staging (OLGA/OLGIM), which consistently ranks the patient-specific cancer risk. Histological staging makes IM subtyping clinically redundant. 	Ristimaki Ari

14	<p><i>In patients with H. pylori-negative gastritis (naïve or after H. pylori-eradicated), how can a diagnosis of autoimmune gastritis be confirmed?</i></p> <ul style="list-style-type: none"> ▪ Patients who have a suspected diagnosis of auto-immune gastritis based on <i>H. pylori</i>-negative gastritis (naïve or after <i>H. pylori</i>-eradicated), require testing for gastrin, pepsinogens ratio, and auto-antibodies to intrinsic factor and parietal cells. Clinical context and functional serology may provide the rationale for any further need of endoscopy/biopsy assessment. 	Rugge Massimo
15	<p><i>Are molecular biomarkers available to predict the risk of non-hereditary (i.e., sporadic) gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ Currently, no large-scale trials have provided evidence that molecular biomarkers can reliably predict the risk of non-hereditary (i.e., non-syndromic) gastric cancer. 	Dinis Ribeiro Mario
16	<p><i>Do patients with low-stage gastritis after successful H. pylori eradication require any follow up?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i>-eradicated patients who have low-stage gastritis, as properly assessed by endoscopy/histology, do not require any specialist follow up and may be referred back to their general practitioner. 	Dinis Ribeiro Mario
17	<p><i>What is the risk of gastric cancer after successful eradication of H. pylori in cases of high-stage gastritis?</i></p> <ul style="list-style-type: none"> ▪ After successful <i>H. pylori</i> eradication, patients with high-stage (III-IV) gastritis, and/or extensive atrophy on endoscopy, are still at risk of gastric cancer. The timing of the endoscopic/biopsy surveillance is based on the gastritis stage as assessed at the last check-up. 	Di Mario Francesco
18	<p><i>What is the optimal management of intra-epithelial neoplasia (IEN)?</i></p> <ul style="list-style-type: none"> ▪ A diagnosis of IEN (low- and high-grade) requires gastric mapping by high resolution endoscopy, confirmatory histological assessment and targeted EMR or SBD (particularly high grade) in tertiary endoscopy centres. As ablation does not abolish metachronous cancer risk, <i>H. pylori</i> eradication and post-ablation surveillance are both mandatory. 	Kuipers Ernst J.

WORKING GROUP 3

	STATEMENT	DELEGATE/COORDINATOR
1	<p><i>Should susceptibility tests be performed routinely before prescribing 1st line treatment?</i></p> <ul style="list-style-type: none"> It is reasonable to recommend that susceptibility tests (culture or molecular) are routinely performed, even before prescribing first-line treatment, in specialized centres with interest in <i>H. pylori</i> management. However, the evidence is too limited to support the generalized use of such a susceptibility-guided strategy in routine clinical practice. 	Gisbert Javier
2	<p><i>If susceptibility testing is not available, what are the recommended 1st line treatments in areas of high clarithromycin resistance?</i></p> <ul style="list-style-type: none"> If individual susceptibility testing is not available, the first line recommended treatments in areas of high (>15%) or unknown clarithromycin resistance are bismuth quadruple therapy or non- bismuth concomitant quadruple therapy. In areas of high dual clarithromycin/metronidazole resistance (>15% dual), first line recommended treatment is bismuth quadruple therapy. High-dose PPI-amoxicillin dual therapy can be considered as an alternative. 	Fallone Carlo
3	<p><i>What is the optimal duration of bismuth quadruple therapy?</i></p> <ul style="list-style-type: none"> The treatment duration of bismuth quadruple therapy should be 14 days, unless 10-day therapies are proven effective locally. 	Coelho Luiz
4	<p><i>Which is the most effective non-bismuth quadruple regimen (sequential, concomitant, hybrid)?</i></p> <ul style="list-style-type: none"> In choosing a non-bismuth quadruple therapy, concomitant therapy (PPI, amoxicillin, clarithromycin, and a nitroimidazole administered concurrently) should be the preferred choice given its proven reproducible effectiveness and less complexity compared to sequential and hybrid therapies. 	Fallone Carlo
5	<p><i>What is the optimal duration of non-bismuth quadruple therapy?</i></p> <ul style="list-style-type: none"> The recommended treatment duration of non- bismuth quadruple therapy (concomitant) is 14 days. 	Coelho Luiz
6	<p><i>If susceptibility testing is not available, what are the recommended 1st line treatments in areas of low clarithromycin resistance?</i></p> <ul style="list-style-type: none"> In areas of low clarithromycin resistance, bismuth quadruple therapy or clarithromycin-containing triple therapy may be recommended as first-line empirical treatment, if proven effective locally. 	Graham David Yates
7	<p><i>What is the optimal duration of PPI-based triple therapies?</i></p> <ul style="list-style-type: none"> The recommended treatment duration of PPI- clarithromycin-based triple therapy is 14 days. 	Coelho Luiz

8	<p><i>Does the use of high-dose PPI increase H. pylori cure rates?</i></p> <ul style="list-style-type: none"> ▪ The use of high dose PPI twice daily increases the efficacy of triple therapy. It remains unclear whether high dose PPI twice daily can improve the efficacy of quadruple therapies. 	Zhou Lyia
9	<p><i>What is the role of Potassium Channel Acid Blocking Drugs (P-CABs: Vonoprazan, Tegoprazan) in the treatment of H. pylori infection?</i></p> <ul style="list-style-type: none"> ▪ P-CAB combination treatments are superior, or not inferior, to conventional PPI-based triple therapies for first- and second-line treatment, and superior in patients with evidence of antimicrobial resistant infections. 	Hunt Richard
10	<p><i>Should susceptibility tests be performed routinely after H. pylori eradication failure (for prescribing second line or third line therapy)?</i></p> <ul style="list-style-type: none"> ▪ Empiric second line and rescue therapies should be guided by local resistance patterns and eradication rates in order to optimise treatment success. Susceptibility guided therapy should be performed in local laboratory reference centres with specialist expertise in H. pylori infection. 	O'Morain Colm
11	<p><i>If susceptibility testing is not available, what are the recommended empirical 2nd line therapies after failure of bismuth quadruple therapy?</i></p> <ul style="list-style-type: none"> ▪ After failure of bismuth-containing quadruple therapy, a fluoroquinolone-containing quadruple (or triple) therapy, or the high-dose PPI-amoxicillin dual therapy may be recommended. In cases of high quinolone resistance, the combination of bismuth with other antibiotics, or rifabutin, may be an option. 	Bazzoli Franco
12	<p><i>If susceptibility testing is not available, what are the recommended empirical 2nd line therapies after failure of PPI-clarithromycin- amoxicillin triple therapy?</i></p> <ul style="list-style-type: none"> ▪ After failure of PPI-clarithromycin-amoxicillin triple therapy, a bismuth-containing quadruple therapy, a fluoroquinolone-containing quadruple (or triple) therapy, or a PPI-amoxicillin high-dose dual therapy are recommended as a second-line treatment. 	Bazzoli Franco
13	<p><i>If susceptibility testing is not available, what are the recommended empirical 2nd line therapies after failure of non-bismuth quadruple therapy?</i></p> <ul style="list-style-type: none"> ▪ After failure of a non-bismuth quadruple therapy, either a bismuth quadruple therapy or a fluoroquinolone-containing quadruple (or triple) therapy is recommended. PPI-amoxicillin high- dose dual therapy might also be considered. 	Bazzoli Franco

<p>14</p>	<p><i>If susceptibility testing is not available, what are the recommended empirical 3rd line therapies after failure of 1st line with clarithromycin-containing (triple or non- bismuth quadruple) and 2nd line with bismuth quadruple therapy.</i></p> <ul style="list-style-type: none"> ▪ After failure of the first-line treatment with clarithromycin-containing triple or non-bismuth quadruple therapies and second-line with bismuth quadruple therapy, it is recommended to use a fluoroquinolone-containing regimen. In regions with a known high fluoroquinolones resistance, a bismuth quadruple therapy with different antibiotics, rifabutin-containing rescue therapy, or a high dose PPI-amoxicillin dual therapy, should be considered. 	<p>Niv Yaron</p>
<p>15</p>	<p><i>If susceptibility testing is not available, what are the recommended empirical 3rd line therapies after failure of 1st line with clarithromycin-containing therapy (triple or non-bismuth quadruple) and 2nd line with levofloxacin- containing therapy.</i></p> <ul style="list-style-type: none"> ▪ After failure of the first-line treatment with clarithromycin-containing triple or non-bismuth quadruple therapies, and second-line treatment with fluoroquinolone-containing therapy, it is recommended to use the bismuth-based quadruple therapy. If bismuth is not available, high-dose PPI-amoxicillin dual or a rifabutin-containing regimen could be considered. 	<p>Niv Yaron</p>
<p>16</p>	<p><i>If susceptibility testing is not available, what are the recommended empirical 3rd line therapies after failure of 1st line with bismuth quadruple therapy and 2nd line with levofloxacin-containing therapy.</i></p> <ul style="list-style-type: none"> ▪ After failure of first-line treatment with bismuth quadruple and second-line treatment with fluoroquinolone-containing therapy, it is recommended to use a clarithromycin-based triple or quadruple therapy only if from an area of low (<15%) clarithromycin resistance. Otherwise, a high-dose PPI-amoxicillin dual therapy, a rifabutin- containing regimen or a combination of bismuth with different antibiotics should be used. 	<p>Niv Yaron</p>
<p>17</p>	<p><i>What are the recommended treatments in patients with penicillin allergy (1st and 2nd line)?</i></p> <ul style="list-style-type: none"> ▪ In patients with penicillin allergy, for a first-line treatment, bismuth quadruple therapy (PPI-bismuth-tetracycline-metronidazole) should be recommended. As second line therapy, bismuth quadruple therapy (if not previously prescribed) and fluoroquinolone-containing regimen may represent empirical second-line rescue options. 	<p>Zhou Lyia</p>

WORKING GROUP 4

	STATEMENT	DELEGATE/COORDINATOR
1	<p><i>Is H. pylori the principal aetiological factor for gastric cancer at all locations?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> infection is the primary aetiological factor for gastric adenocarcinoma at all locations, including proximal gastric cancer. 	Sugano Kentaro
2	<p><i>Does H. pylori contribute to the aetiology of a subset of adenocarcinomas at the gastroesophageal junction (GEJ)?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> infection plays an aetiological role in a subset of adenocarcinomas at the gastroesophageal junctional zone (GEJ) 	Sugano Kentaro
3	<p><i>Is there a role for non-Helicobacter environmental factors in non-cardia gastric carcinoma?</i></p> <ul style="list-style-type: none"> ▪ The influence of environmental factors is subordinate to the effect of <i>H. pylori</i> infection. 	Machado José Carlos
4	<p><i>Does H. pylori modify the risk of developing hereditary gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ Hereditary gastric cancer is a distinct entity. The role of <i>H. pylori</i> infection in the clinical course of the disease remains to be elucidated. 	Machado José Carlos
5	<p><i>Does H. pylori gastritis with atrophy carry a higher risk for gastric cancer development than autoimmune gastritis?</i></p> <ul style="list-style-type: none"> ▪ Severe atrophy (OLGA 3/4) in the context of <i>H. pylori</i> gastritis carries a much higher risk for gastric cancer development as compared to atrophy in the context of autoimmune gastritis. 	Vieth Michael
6	<p><i>Does coinfection of H. pylori with EBV, or other viral infections, modify the risk of gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> infection and EBV are independent risk factors of gastric cancer. Whether coinfection of <i>H. pylori</i> and EBV is associated with higher risk of gastric cancer than either one alone remains uncertain. 	Liou Jyh-Ming
7	<p><i>Does H. pylori eradication prevent progression to atrophic gastritis, apart from curing the inflammatory response?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication abolishes the inflammatory response in chronic active non-atrophic gastritis and prevents further progression to atrophy and intestinal metaplasia. 	Vieth Michael

8	<p><i>Does H. pylori eradication lead to regression of gastric atrophy and prevent from progression to gastric neoplasia?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication may reverse gastric atrophy and intestinal metaplasia to some extent and may hold the progression from chronic atrophic gastritis to neoplastic lesions in a subset of patients. 	Vieth Michael
9	<p><i>Does H. pylori eradication prevent gastric cancer only if offered at younger age?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication offers the chance for gastric cancer prevention at any age in adulthood, but the magnitude of the benefit decreases with age. 	Malfertheiner Peter
10	<p><i>Is there a “point of no return” when H. pylori eradication can no longer be expected to prevent the progression of chronic gastritis to gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication is most effective for gastric cancer prevention before severe chronic atrophic gastritis develops. 	Malfertheiner Peter
11	<p><i>Which tests can be used to screen H. pylori for gastric cancer prevention?</i></p> <ul style="list-style-type: none"> ▪ Diagnostic tests used to screen <i>H. pylori</i> infection for the purpose of gastric cancer prevention should preferably be non-invasive. 	Liou Jyh-Ming
12	<p><i>Can therapy decision for H. pylori eradication be solely based on a positive serological test?</i></p> <ul style="list-style-type: none"> ▪ If a serological method is used for <i>H. pylori</i> detection a further test (UBT, FAT) confirming current infection is required for therapy decision. 	Liou Jyh-Ming
13	<p><i>Is endoscopy with biopsies an effective approach for early detection of gastric cancer in individuals with a positive family history of gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ Endoscopy with biopsies is required in asymptomatic individuals (aged >45 years) with a family history of gastric cancer (excluding hereditary gastric cancer). 	Machado José Carlos
14	<p><i>Which age group should be considered as a high-risk population for gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ Asymptomatic individuals aged above 50 years are considered at increased risk of gastric cancer compared to younger individuals. 	Liou Jyh-Ming
15	<p><i>Should serological screening include Pepsinogens to identify preneoplastic lesions?</i></p> <ul style="list-style-type: none"> ▪ In individuals aged over 40 years, <i>H. pylori</i> serological population-based screening should include Pepsinogens to identify preneoplastic lesions. in case of abnormal pepsinogens, an endoscopic examination is recommended. 	Milosavljevic Tomica

16	<p><i>How relevant is the use of antibiotics in H. pylori eradication regimens in the induction of antibiotic resistance in the community?</i></p> <ul style="list-style-type: none"> Population-based <i>H. pylori</i> search-and-treat programs for gastric cancer prevention requires caution in the selection of antibiotics to minimise antibiotic resistance development. 	Park Jin Young
17	<p><i>Does the broad use of H. pylori eradication therapy with the intention of preventing gastric cancer increase the risk of other serious diseases?</i></p> <ul style="list-style-type: none"> Broad use of <i>H. pylori</i> eradication therapies for the purpose of gastric cancer prevention does not lead to an increase in other serious pathologies. 	Malfertheiner Peter
18	<p><i>Does the screen and treat strategy provide benefit only by preventing gastric cancer?</i></p> <ul style="list-style-type: none"> Population-based <i>H. pylori</i> search and treat strategy provides additional benefits by preventing other serious gastroduodenal pathologies. 	Milosavljevic Tomica
19	<p><i>Could combining H. pylori screen and treat with colonoscopy be a good strategic opportunity for prevention of gastric cancer?</i></p> <ul style="list-style-type: none"> Screening modalities for gastric cancer prevention should be offered in combination with colorectal cancer screening. 	Tepes Bojan
20	<p><i>Is H. pylori screen and treat cost-effective for gastric cancer prevention?</i></p> <ul style="list-style-type: none"> Population-based <i>H. pylori</i> search and treat program is cost-effective in populations with intermediate or high incidence of gastric cancer. 	Liou Jyh-Ming
21	<p><i>Which endoscopic follow-up strategy is most suitable in patients with severe atrophic gastritis (OLGA 3/4)?</i></p> <ul style="list-style-type: none"> Follow up at regular intervals by use of endoscopic-biopic protocols is mandatory in patients with severe atrophic gastritis (OLGA 3/4). 	Milosavljevic Tomica
22	<p><i>Which endoscopic surveillance strategy is most appropriate for subjects with gastric premalignant mucosal changes?</i></p> <ul style="list-style-type: none"> Surveillance endoscopy every 3 years for subjects with advanced gastric atrophy or intestinal metaplasia is cost-effective and is recommended. Surveillance endoscopy every 6 months and every 12 months is recommended for those with high-grade and low-grade dysplasia, respectively. Endoscopic biopsies are insufficient for correct diagnosis of visible gastric lesions and an endoscopically visible lesion with any neoplastic change should be considered for treatment. 	Milosavljevic Tomica

23	<p><i>Is it required and still useful to eradicate <i>H. pylori</i> following the removal of early gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ Eradication of <i>H. pylori</i> is mandatory to reduce the risk of metachronous gastric cancer after curative endoscopic resection or gastric subtotal resection of early gastric cancer. 	Tepes Bojan
24	<p><i>Should patients presenting with severe atrophy/IM following successful <i>H. pylori</i> eradication receive natural substances or medications for halting progression to gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ Medicinal and special dietary chemoprevention cannot in general be recommended in patients with severe gastric atrophy or intestinal metaplasia (OLGA3/4) after <i>H. pylori</i> eradication 	Park Jin Young
25	<p><i>Are there specific requirements for <i>H. pylori</i> screen and treat strategies according to the regional public health situation?</i></p> <ul style="list-style-type: none"> ▪ Population-based <i>H. pylori</i> search- and treat programs should be targeted to special requirements at the regional level (i.e., selection of screening tool, use of eradication regimen, surveillance).. 	Park Jin Young
26	<p><i>How should health care priorities find consideration in the implementation of <i>H. pylori</i> screen and treat strategies for gastric cancer prevention?</i></p> <ul style="list-style-type: none"> ▪ Population-based <i>H. pylori</i> search- and treat programs should be integrated into healthcare priorities, especially in regions with intermediate to high gastric cancer incidence. 	Park Jin Young
27	<p><i>Are there clinically validated genetic and epigenetic markers of gastric cancer risk or progression?</i></p> <ul style="list-style-type: none"> ▪ The use of genetic and epigenetic markers for gastric cancer risk assessment and gastric cancer progression in clinical management requires further validation. 	Machado José Carlos
28	<p><i>What is the role and contribution of image enhanced endoscopy for screening of dysplasia and early gastric cancer?</i></p> <ul style="list-style-type: none"> ▪ Image-enhanced endoscopy (IEE) such as LCI should preferentially be used in the endoscopy-based screening for dysplasia and early gastric cancer. 	Sugano Kentaro
29	<p><i>Would a vaccine against <i>H. pylori</i> still be helpful in controlling the <i>H. pylori</i> infection?</i></p> <ul style="list-style-type: none"> ▪ There is still demand for a prophylactic and/or therapeutic vaccine. 	Malfertheiner Peter

WORKING GROUP 5

	STATEMENT	DELEGATE/COORDINATOR
1	<p><i>Does early-life antibiotic exposure induce long-lasting intestinal microbiota alterations?</i></p> <ul style="list-style-type: none"> ▪ Early life antibiotic exposure induces long-lasting alterations of the intestinal microbiota 	Rajilic-Stojanovic Mirjana
2	<p><i>Is the gastric niche colonised by microorganisms other than <i>H. pylori</i>?</i></p> <ul style="list-style-type: none"> ▪ The human stomach is colonized by many microorganisms other than <i>H. pylori</i>, collectively known as the gastric microbiome. 	Schulz Christian
3	<p><i>Do other microorganisms in addition to <i>H. pylori</i> have an impact on <i>H. pylori</i>-associated diseases?</i></p> <ul style="list-style-type: none"> ▪ Other gastric microbiota may also affect <i>H. pylori</i>-related changes in the stomach. 	Kupczinskas Juozas
4	<p><i>Are non-<i>pylori</i> Helicobacter species involved in gastric disease development?</i></p> <ul style="list-style-type: none"> ▪ Non-<i>pylori</i> Helicobacter species can cause human gastric disease. 	Kupczinskas Juozas
5	<p><i>Can treatment of <i>H. pylori</i> cause development of resistance among other bacteria in the gut?</i></p> <ul style="list-style-type: none"> ▪ <i>H. pylori</i> eradication therapy has the potential to select resistant strains in the gut microbiota. 	Tilg Herbert
6	<p><i>Are probiotics effective in reducing GI side effects of <i>H. pylori</i> eradication therapies?</i></p> <ul style="list-style-type: none"> ▪ Only certain probiotics have been shown to be effective in reducing GI side effects caused by <i>H. pylori</i> eradication therapies. 	Engstrand Lars
7	<p><i>Are probiotics useful in increasing <i>H. pylori</i> eradication rates, when added to antibiotic therapies?</i></p> <ul style="list-style-type: none"> ▪ Probiotics may have a beneficial effect on <i>H. pylori</i> eradication through reduction in antibiotic related side effects 	Gasbarrini Antonio
8	<p><i>Can antibiotic use for other indications than <i>H. pylori</i> eradication cause resistance in <i>H. pylori</i> strains?</i></p> <ul style="list-style-type: none"> ▪ Antibiotic treatment for other reasons might select resistant <i>H. pylori</i> strains. 	Gasbarrini Antonio
9	<p><i>Does the oral cavity harbour microbiota that can colonise distal parts of the GI tract?</i></p> <ul style="list-style-type: none"> ▪ The oral cavity harbours a rich microbiota that can contribute to colonisation of other parts of the GI tract. 	Marshall Barry

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GENERAL INFORMATION

EVENT CLOSED TO THE FACULTY

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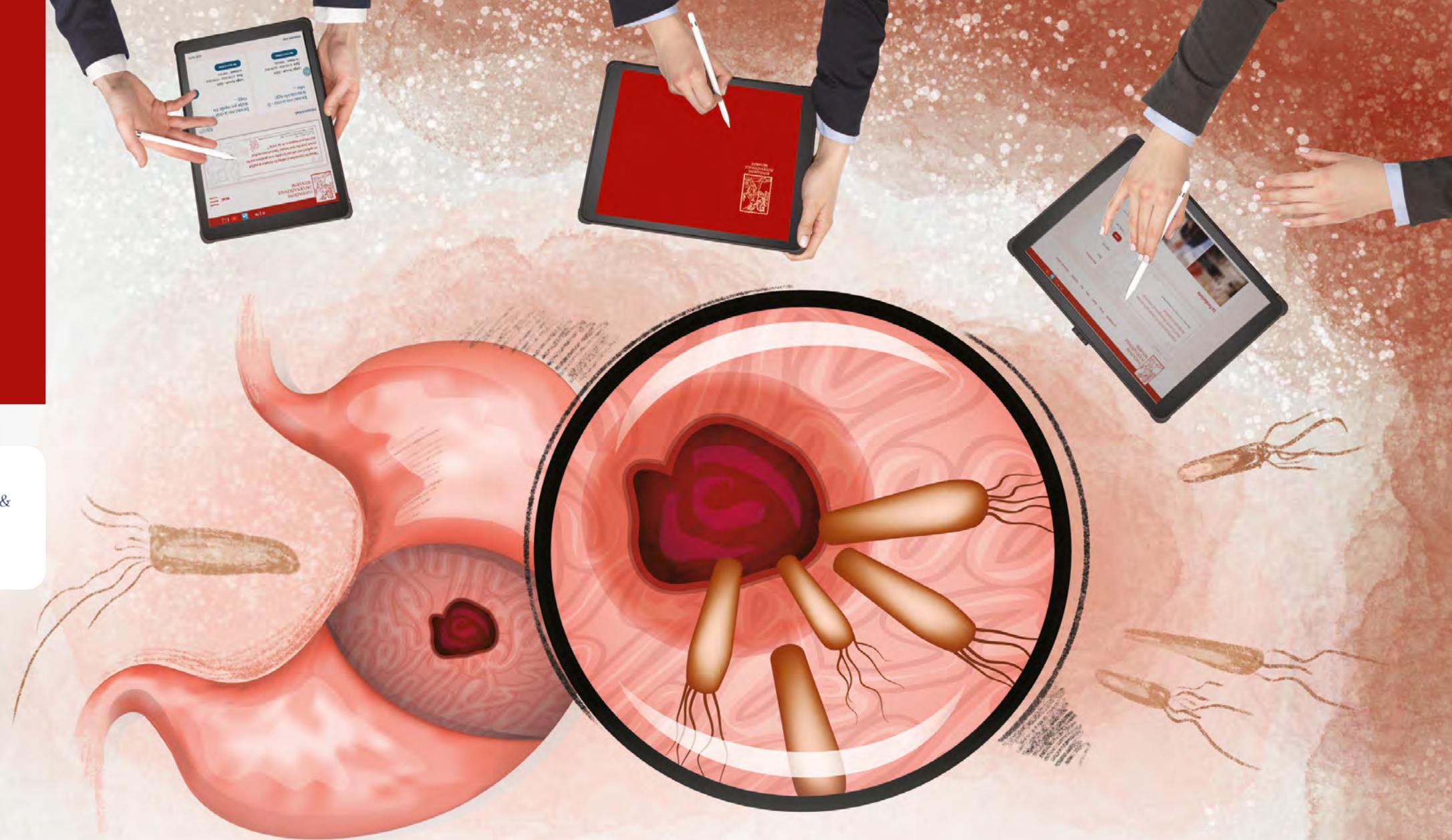
OFFICIAL LANGUAGE

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