



GASTROINTESTINAL INFLAMMATION AND NEOPLASIA

April 24-25, 2026

Symposium 243
WARSAW, POLAND



10,5
CME
CREDITS

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10,5 credit hours (CME) have been awarded by the European Union of Medical Specialists (UEMS).

PREFACE

With great pleasure and anticipation, we invite you to the international Falk Foundation Symposium on “**Gastrointestinal Inflammation and Neoplasia**” which will take place in Warsaw.

This meeting will bring together scientists and clinicians from across the field of gastroenterology to examine the epidemiological foundations, pathophysiological mechanisms, and clinical manifestations of a broad spectrum of gastrointestinal diseases - including eosinophilic esophagitis (EoE), inflammatory bowel disease (IBD), microscopic colitis, celiac disease, and selective gastrointestinal neoplasias. With the participation of leading experts in our field, we will engage in discussions on the latest insights into the pathophysiology, epidemiology, approved and emerging diagnostic approaches, and evidence-based and emerging medical, endoscopic and surgical treatment strategies—placing particular emphasis on the practical implications of recent scientific breakthroughs.

We are confident that this symposium will not only foster scientific advancement but also strengthen professional connections among those committed to improving patient care in gastrointestinal health.

The setting in Warsaw - a dynamic city where cultural heritage meets modern scientific progress - offers an inspiring backdrop for collaboration and forward-thinking dialogue.

We look forward to welcoming you to what promises to be a stimulating and impactful meeting with ample time to interact with a well-recognized international faculty and lots of opportunities for networking. Meet old and make new friends.

Axel Dignass, Michał F. Kamiński, Julia Mayerle and Edyta Zagórowicz

GASTROINTESTINAL INFLAMMATION AND NEOPLASIA

April 24-25, 2026

Scientific Organization:

Axel Dignass, Frankfurt, Germany
Michał F. Kamiński, Warsaw, Poland
Julia Mayerle, Munich, Germany
Edyta Zagórowicz, Warsaw, Poland

Start of Registration:

Thursday, April 23, 2026
16:00 – 20:00 h
at the congress office

Congress Venue:

Hilton Warsaw City
Grzybowska 63
00-844 Warsaw
Poland

For admission to scientific events your name badge should be clearly visible.

Accompanying persons are not permitted during the conference at any time.

FRIDAY, APRIL 24, 2026

9:00 Welcome
Axel Dignass, Frankfurt; Edyta Zagórowicz, Warsaw

SESSION I

Epidemiology and pathophysiology of immune-mediated gastrointestinal disorders

Chairs *Marcin Dziekiewicz, Warsaw; Gerhard Rogler, Zurich*

09:10 EoE
Christopher Ma, Calgary

09:30 Celiac disease
Jonas Ludvigsson, Stockholm

09:50 Microscopic colitis
Darrell S. Pardi, Rochester

10:10 IBD
Britta Siegmund, Berlin

10:30 **Coffee break with ePoster session**

SESSION II

Current diagnostic and monitoring strategies of immune-mediated gastrointestinal disorders

Chairs *Axel Dignass, Frankfurt; Jarosław Reguła, Warsaw*

11:00 EoE
Ulrike von Arnim, Magdeburg

11:20 Celiac disease
Carolina Ciacci, Salerno

11:40 Microscopic colitis
Piotr Eder, Poznan

12:00 IBD
Ana Gutierrez Casbas, Alicante

12:20 **Lunch break with ePoster session**

SESSION III

Current treatment strategies in immune-mediated gastrointestinal disorders

Chairs *Grażyna Rydzewska, Warsaw; Britta Siegmund, Berlin*

14:00 EoE: Diet, steroids or more?
Edoardo V. Savarino, Padova

14:20 Celiac disease: Gluten free diet and more
Detlef Schuppan, Mainz

14:40 Microscopic colitis: Current strategies for uncomplicated and refractory disease
Andreas Münch, Linköping

15:00 IBD: Treatment goals in 2026
Alessandro Armuzzi, Milan

15:20 **Coffee break with ePoster session**

SESSION IV

Diet and microbiome based approaches in immune-mediated gastrointestinal disorders

Chairs *Milan Lukáš, Prague; Harry Sokol, Paris*

16:00 EoE: Elimination diet or more?
Alfredo J. Lucendo, Tormelloso

16:20 Celiac disease: Modern strategies to improve and monitor adherence to lifelong diet
Luca Elli, Milan

16:40 IBD: Is nutrition an underutilized therapeutic option?
Iris Dotan, Petah Tikva

17:00 IBD: The relevance of the microbiome in pathophysiology and treatment
Harry Sokol, Paris

SATURDAY, APRIL 25, 2026

SESSION V

Special situations

Chairs *Anita Gąsiorowska, Lodz; Małgorzata Zwolińska-Wcisło, Krakow*

09:00 Refractory celiac disease and differentials
Michael Schumann, Berlin

09:20 Acute severe colitis
David Laharie, Bordeaux

09:40 Immune-mediated gastrointestinal disorders during pregnancy and lactation
Uma Mahadevan, San Francisco

10:00 Management of complicated fibrostenosing EoE
Alain Schoepfer, Lausanne

10:20 **Coffee break with ePoster session**

SESSION VI

Gastrointestinal tract neoplasia and differentials

Chairs *Cesare Hassan, Milan; Michał F. Kamiński, Warsaw*

10:50 Colorectal cancer screening and surveillance by primary colonoscopy: Nordic and beyond
Cesare Hassan, Milan

11:10 Colorectal cancer screening and surveillance by FIT
Antoni Castells, Barcelona

11:30 Endoscopic resection of colonic and rectal lesions: On the oncological edge
Nastazja Pilonis, Warsaw

11:50 Early onset colorectal cancer
Willem Bemelman, Amsterdam

12:10 **Presentation of poster awards**
Axel Dignass, Frankfurt; Edyta Zagórowicz, Warsaw

12:30 **Lunch break with ePoster session**

SESSION VII

Tips and tricks for excellent gastroenterologists

Chairs *Axel Dignass, Frankfurt; Andreas Sturm, Berlin*

13:30 Impaired coagulation: What gastroenterologists need to know
Jeanin van Hooft, Leiden

13:50 Pitfalls in sampling: We need to consult our pathologist
Rafal Peksa, Gdansk

14:10 FMT: Pitfalls and tips
Jarosław Biliński, Gdansk

14:30 State of the art: Management of antibiotic-associated colitis
Christoph Högenauer, Graz

14:50 **Coffee break with ePoster session**

SESSION VIII

Treatment decisions: Pro and cons

Chairs *Ailsa Hart, London; Edyta Tulewicz-Marti, Warsaw*

15:20 Strictureing Crohn's disease: Medical, endoscopic or surgical treatment?
Pro conservative treatment *Florian Rieder, Cleveland*
Pro surgery *Willem Bemelman, Amsterdam*

15:50 De-escalation of medication for IBD: Continue forever or time to scale down?
Ailsa Hart, London

16:10 Top-down or step-up approach in IBD: Still unresolved?
Pro top down *Nurulamin Noor, Cambridge*
Pro step-up *Andreas Sturm, Berlin*

16:40 Cancer surveillance and prevention in IBD: Universal or individualized approaches?
Fernando Velayos, San Francisco

17:00 Closing remarks

LIST OF SPEAKERS, MODERATORS AND SCIENTIFIC ORGANIZERS

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REGISTRATION



You can register for the event via our homepage:

www.falkfoundation.org

Registration is only possible online.

You will receive an automatic confirmation of registration by e-mail.

Please transfer the congress fee to the bank account listed in the e-mail within two weeks.

CONGRESS FEES

Scientific Program of Symposium 243 EUR 300

Students (copy of student ID required) EUR 150

The congress fees include:

- Pre-Opening and Welcome on Thursday, April 23, 2026
- Refreshments during coffee breaks
- Lunch on Friday, April 24 and on Saturday, April 25, 2026
- A copy of the final program

CONGRESS OFFICE AND REGISTRATION

Opening Hours:

Thursday, April 23: 16:00 – 20:00 h

Friday, April 24: 08:00 – 17:00 h

Saturday, April 25: 08:00 – 17:00 h

The Falk Foundation will take pictures during the meeting. Additionally, parts of the meeting might be recorded. By participating all attendees consent and agree with the recording and the photo shoots.

ARRIVAL

Hilton Warsaw City

Grzybowska 63
00-844 Warsaw
Poland

By car

There is an underground car park in the hotel at approx. 180,-- PLN (42,-- EUR) per 24 hours.

By plane

The international Warsaw Frederic Chopin Airport is 20 min (8km) away by taxi. By bus and tram it takes approx 40 min (exit Muzeum Powstania Warszawskiego).

By train

Warsaw Central Railway Station is 5 min away by taxi or 17 min by public transport.

CONFLICTS OF INTEREST

Members of the scientific committee declare the following potential conflicts of interest:

Axel Dignass: Abbvie, Ferring, Roche, Takeda, Vifor, Dr. Falk Pharma, Johnson & Johnson, Pfizer, Sandoz, BMS, Tillotts, Fresenius Kabi, Alfasigma, Pharmacosmos, Gilead, Celltrion, Lilly, Amgen, Abivax, MSD, Stada, Böhringer Ingelheim, Janssen, High5MD, Materia Prima, Streamed-Up, MedToday, Biogen, CED Service GmbH, Thieme

Michał F. Kamiński: no potential conflict of interest to report

Julia Mayerle: no potential conflict of interest to report

Edyta Zagórowicz: no potential conflict of interest to report

POSTER ABSTRACTS

1. Colorectal dysplasia risk in inflammatory bowel disease patients with small-duct versus large-duct primary sclerosing cholangitis: A retrospective cohort analysis
N. Ahangama Arachchige, S. Jarnathanan, S. Subramanian, M. Thoufeeq, C. Fu, O. Baker, A. Temple-Marsh, E. Mohamed, M. Afsana, B. Darshana, K. Mustafa, A. Hadjinicolaou (Barnsley, Cambridge, Sheffield, GB)
2. Clinical and endoscopic features associated with missed eosinophilic oesophagitis
S. Batal, H. Haboubi (Cardiff, GB)
3. Differentiating psychosocial profiles in Crohn's disease and ulcerative colitis using the Millon Behavioral Medicine Diagnostic
Belen Galipienso, P. Bernabeu, R. Amrani, G. Garcia Del Castillo, M. Garcia Sepulcre, M. Rodriguez Aguilar, E. Gomez, F. Gregori, C. Van der Hofstadt, A. Abad, P. Zapater, A. Gutierrez, L. Madero Velazquez, R. Munoz, B. Herreros, V. Moreno, A. Garcia, C. Mira, R. Jover, R. Sempere (Alicante, Alcoy, San Juan de Alicante, Elche, ES)
4. Risk of colorectal cancer in microscopic colitis compared with colonoscopy-verified normal mucosa
D. Bergman, S. Rabin Bozorg, J. Ludvigsson (Solna, SE)
5. Risk factors of fatigue in patients with inflammatory bowel disease
O. Daboussi, M. Luwawu, A. Herber (Le Coudray, FR)
6. Joint involvement in patients with celiac disease and ulcerative colitis
C. Deliu, O. Chirea, A. Genunche, D. Neagoe (Bals, Craiova, RO)
7. Real-world effectiveness and safety of Jorveza® in adult eosinophilic oesophagitis: A UK tertiary-centre cohort
W. Fong, G. Villarejo (Nottingham, GB)
8. The relationship between stress indicators and the severity of inflammation in celiac disease
A. Furgala, M. Przybylska-Felus, K. Gil, M. Zwolinska-Wcislo (Krakow, PL)
9. Wild thyme extract improves gastrointestinal comfort through gut microbiota modulation and inflammation-related pathways: A clinical study in adults
J. Galvez, J. Garcia-Garcia, L. Lopez-Escanez, T. Vezza, R. Lopez-Zambrano, L. Gbati, R. Palacios-Lopez, B. Martin-Castano, G. Vargas-Castillo, M. Martinez-Zaldivar, A. Caballero, F. Garcia, E. Redondo-Cerezo, E. Fernandez-Varon, R. Moron, I. Pischel, C. Suarez, A. Rodriguez-Nogales, M. Rodriguez-Cabezas (Granada, ES, London, GB; Andernach, DE)
10. Low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 attenuate tumor progression and modulate gut microbiota in AOM/DSS-induced colorectal cancer
M. Garcia-Mansilla, L. Gbati, J. Molina Tijeras, M. Rodriguez Sojo, L. Hidalgo Garcia, J. Garcia Garcia, L. Lopez Escanez, A. Ruiz Malagon, T. Vezza, M. Rodriguez-Sanchez, A. Ho Plagaro, J. Galvez Peralta, M. Rodriguez Cabezas, A. Rodriguez Nogales (Granada, ES)
11. Low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 ameliorate DSS-induced colitis in mice
J. Garcia, L. Gbati, J. Molina, M. Rodriguez, L. Hidalgo, L. Lopez, T. Vezza, A. Ruiz, A. Ho, J. Galvez, A. Rodriguez, M. Rodriguez (Granada, ES)

12. Extracellular vesicles derived from *Limosilactobacillus fermentum* CECT5716 attenuate tumor development and inflammation in an AOM/DSS-induced colorectal cancer mouse model
L. Gbati, J. Molina Tijeras, M. Rodriguez Sojo, L. Hidalgo Garcia, J. Garcia Garcia, L. Lopez Escanez, T. Vezza, A. Ruiz Malagon, A. Ho Plagaro, J. Galvez Peralta, A. Rodriguez Nogales, M. Rrodriguez Cabezas (Granada, ES)
13. Retrospective assessment of the association of the budesonide with gluten-free diet in the treatment of the celiac disease in autoimmune hepatitis
A. Genunche-Dumitrescu, C. Badea, C. Neagoie, R. Surugiu, A. Badea (Craiova, Bucharest, RO)
14. Why upper GI pathology matters to the otolaryngologist
R. Ghosh, R. Srivastava (Nottingham, GB)
15. Evolution of histologic and endoscopic assessment of eosinophilic esophagitis in Europe and its effects on diagnostic delay: Data from the EUREOS EoE CONNECT registry
E. Gueso-Navarro, E. Dilaghi, J. Fernandez-Pacheco, E. Amorena, E. Savarino, L. Blas-Jhon, I. Perez-Martinez, A. Granja-Navacerrada, D. Guagnozzi, L. Martin-Asenjo, G. Pellegata G, M. Colleta, C. Garcia-Suarez, S. De la Riva, L. Lapena Negro, E. Betore, A. Krarup, J. Barrio, M. Votto, L. Bujanda, S. Fernandez, J. Naves, A. Gonzalez Almeida, R. Honrubia Lopez, S. Carrion, s. Oliva, C. Cano Cerdan, M. Masiques Mas, S. Espina Cadena, J. Gonzalez Cervera, S. Casabona Frances, J. Nicolay Maneru, V. Ubeda Vargas, M. Ghisa, P. Romo, C. Donado, A. Benagues, V. Martin Dominguez, O. Nantes Castillejo, L. Arias Gonzalez, D. Maniero, H. Moreira Gonzalez, I. Maray, J. Barrio Torres, M. Perez Fernandez, M. Ostiz Llanos, L. Rodriguez Alcolado, A. Cabrera Martinez, E. Laserna Mendieta, C. Santander, A. Lucendo (Tomelloso, Madrid, Navarra, Oviedo, Fuenlabrada, Barcelona, Alava, Gijon, Zaragoza, Valladolid, San Sebastian, Leganes, Barcelona, Gran Canaria, Mataro, Marbella, Grannollers, Toledo, Pamplona, ES; Rome, Padova, Rozzano, Milan, Pavia, IT; Aalborg, DK)
16. Colonic mucosal biofilm microbiota alterations in IBD
I. Hryhorchuk, L. Sydorчук, A. Sydorчук, A. Vakarchuk, R. Sydorчук, I. Plehutsa, I. Sydorчук, P. Kyfiak (Chernivtsi, Storozhynets, UA; Neu-Ulm, Siegen, DE)
17. Strategy for intestinal ultrasonography: A new goal in the follow-up of patients with inflammatory bowel disease undergoing treatment with biological agents and small molecules
V. Ilieva, A. Petrova, L. Grudeva (Varna, BG)
18. The efficacy and safety of vedolizumab as first-line advanced therapy for active ulcerative colitis in patients after liver transplantation
P. Kucha, M. Zaborowska, J. Wypych, M. Augustyn, T. Brodowski, P. Eder, R. Filip, M. Gawron-Kiszka, M. Kaniewska, J. Koza, K. Kloskowska-Kapica, K. Maciejewska, E. Zagórowicz (Warsaw, Gdansk, Krakow, Końskie, Poznań, Rzeszów, Katowice, Bydgoszcz, Lublin, PL)
19. Beyond dysphagia: Prospective evidence that fatigue is an under-recognised burden in eosinophilic oesophagitis
R. Kumar, J. Brooks-Warburton, D. Morris (Stevenage, GB)
20. Can capsule sponge sampling replace gastroscopy for monitoring eosinophilic oesophagitis? A single-centre prospective study
R. Kumar, A. Rai, H. McDonald, P. Baker, K. Shaw, F. Cole, E. Santa, D. Morris (Stevenage, GB)
21. Therapeutic potential of *Limosilactobacillus fermentum* CECT5716 extracellular vesicles in promoting gut barrier repair through TLR4-dependent macrophage polarization
A. Lista (Granada, ES)

22. Gut microbiota-associated modulation of intestinal mesenchymal stromal cells in colorectal cancer
L. Lopez Escanez, L. Hidalgo Garcia, J. Molina Tijeras, A. Ruiz-Malagon, T. Vezza, M. Rodriguez-Sojo, L. Gbati, J. Garcia Garcia, D. Palacios Vicente, A. Lista, C. Ayala Mosqueda, A. Rodriguez Nogales, J. Galvez, P. Anderson, M. Rodriguez Cabezas (Roquetas De Mar, Granada, ES)
23. Listening to patients – Real world evaluation of capsule sponge monitoring in eosinophilic oesophagitis
H. McDonald (Hertfordshire, GB)
24. Impact of HLA-DQA1*05 genotype in immunogenicity and response to treatment with tumour necrosis factor-alpha antagonists in inflammatory bowel disease patients
V. Moreno, C. Mira, L. Madero Velazquez, R. Munoz, O. Belen Galipienso, J. Cameo, L. Bernal, A. Garcia Trueba, L. Sempere, A. Gutierrez (Alicante, ES)
25. Female sexual dysfunction in inflammatory bowel disease: Utility of a novel specific scale
R. Munoz (Alicante, ES)
26. Intestinal stem cell adaptation and temporal response to inflammatory stress
S. Patel, P. Ordonez Moran, B. Balasubramanian, G. Moran (Nottingham, GB)
27. Baseline duodenal epithelial fitness and interferon tone stratify gluten-challenge mucosal injury in celiac disease
A. Pesi, V. Dotsenko, R. Mohrbacher, K. Viiri, D. Schuppan (Mainz, Freiburg, DE; Helsinki, Oulu, FI)
28. Pathogen-driven selection of HLA-DQ2 and HLA-DQ8: In silico evidence for predominant bacterial peptide presentation relevant to celiac disease
A. Pesi, D. Schuppan (Mainz, DE)
29. *Cereus jamacaru* DC attenuates intestinal inflammation and modulates the immune response in a DSS-induced colitis model
M. Rodriguez-Sanchez, D. Fernandes de Souza Araujo, A. Lista, N. Da Costa Melo, L. Gbati, M. Rodriguez Sojo, G. Bernardo Guerra, S. Zucolotto Langassner, J. Galvez, A. Rodriguez Nogales (Granada, ES, Natal, BR)
30. Mucosa-associated microbiota signatures linked to postoperative recurrence in Crohn's disease
A. Rodriguez Nogales (Granada, ES)
31. Targeting gut inflammation to limit colorectal cancer progression: Immunomodulatory and anti-proliferative effects of propyl-propane thiosulfonate (PTSO)
M. Rodriguez Sojo, A. Ruiz-Malagon, L. Hidalgo-Garcia, J. Molina-Tijeras, P. Diez-Echave, L. Lopez- Escanez, J. Garcia Garcia, T. Vezza, L. Gbati, M. Rodriguez-Sanchez, A. Banos, M. Rodriguez-Cabezas, J. Galvez, A. Rodriguez-Nogales (Granada, ES)
32. *Parabacteroides goldsteinii* mediates the antitumor effects of tigecycline in colorectal cancer under an obesity-associated microenvironment
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33. Development validation of a novel patient-reported outcome for microscopic colitis – Microscopic Colitis Score (MCS)
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1. Colorectal dysplasia risk in inflammatory bowel disease patients with small-duct versus large-duct primary sclerosing cholangitis: A retrospective cohort analysis

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Introduction: Primary sclerosing cholangitis (PSC) markedly increases the risk of colorectal dysplasia in patients with inflammatory bowel disease (IBD). While this association is well established in large-duct PSC (ldPSC), the neoplastic risk profile of small-duct PSC (sdPSC) remains poorly defined. Therefore, we wanted to evaluate differences in dysplasia risk between sdPSC and ldPSC among IBD patients and to determine clinical factors associated with dysplasia development.

Methods: We conducted a retrospective cohort study of patients with concurrent IBD and PSC followed at Cambridge University Hospitals NHS Foundation Trust. Demographic, clinical, endoscopic, and histopathological data were obtained from institutional registries and electronic medical records. Dysplasia incidence was examined according to PSC subtype, IBD phenotype, and patient characteristics. Time to dysplasia was analyzed using Kaplan-Meier survival methods.

Results: The cohort comprised 117 IBD-PSC patients, including 16 with sdPSC (14%) and 101 with ldPSC (86%). Underlying IBD included ulcerative colitis (72%), Crohn's disease (20%), and IBD-unclassified (8%). Over a mean follow-up of 10 years, dysplasia was identified in 9 patients (9%), all within the ldPSC group; no sdPSC patients developed dysplasia ($p = 0.3$). All dysplasia cases occurred in individuals with ulcerative colitis and pancolitis. Patients who developed dysplasia were significantly older (mean age 58 vs. 44 years, $p = 0.02$) and had longer IBD duration (25 vs. 15 years, $p = 0.03$), whereas PSC duration did not differ between groups. Dysplasia-free survival did not significantly differ between PSC subtypes on Kaplan-Meier analysis (log-rank $p = 0.42$). No cases of high-grade dysplasia or colorectal carcinoma were observed.

Discussion/Conclusion: In this single-center cohort, colonic dysplasia occurred exclusively in patients with ldPSC, with no events observed among those with sdPSC. Advanced age and prolonged IBD duration were key risk factors for dysplasia. These findings suggest a potentially lower colorectal neoplasia risk in sdPSC, supporting consideration of risk-stratified surveillance approaches. Larger, multicenter studies are needed to confirm these observations.

2. Clinical and endoscopic features associated with missed eosinophilic oesophagitis

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Introduction: Eosinophilic oesophagitis (EOE) is a chronic, immune-mediated condition of the oesophagus, characterised by eosinophilic inflammation and presenting with symptoms of oesophageal dysfunction. Despite growing awareness, EOE is commonly missed or delayed in diagnosis, resulting in prolonged patient morbidity. Evidence from recent literature suggests that the average time to diagnosis can be up to four years, with nearly one-third of patients experiencing delays of greater than 10-years. These findings highlight the need for improved recognition and timely intervention to prevent complications such as strictures and persistent dysphagia.

Methods: We conducted a retrospective study to identify factors associated with missed diagnosis of EOE between 2022 and 2023. Patient records were reviewed for prior endoscopies, demographic characteristics, and endoscopic features. Univariate and multivariate analyses were performed using SPSS to identify predictors of a positive diagnosis and potential risk factors for missed recognition.

Results: A total of 79 patients were included, of whom 46 (58%) had undergone at least one prior oesophagogastroduodenoscopy (OGD). Detailed data sufficient for analysis were available for 32 of these patients. Male gender was significant on univariate analysis ($p = 0.05$) but did not retain significance on multivariate analysis ($p = 0.195$). Multivariate analysis also identified food bolus obstruction ($p < 0.001$) and mucosal oedema ($p = 0.016$) as significant predictors of EOE. These findings indicate that specific clinical and endoscopic features can reliably predict EOE, even in patients with previous negative investigations.

Discussion/Conclusion: Food bolus obstruction and mucosal oedema are strong predictors of missed diagnosis in EOE. Patients presenting with these features should be considered for early treatment, even if initial biopsies or other endoscopic findings are negative, highlighting the need to explore initiating therapy based on clinical grounds. Recognition of these clinical and endoscopic signs may help reduce missed diagnoses, improve patient outcomes, and prevent long-term complications associated with untreated disease.

3. Differentiating psychosocial profiles in Crohn's disease and ulcerative colitis using the Millon Behavioral Medicine Diagnostic

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ES), Cristina Mira (Alicante, ES), Rodrigo Jover (Alicante, ES), Robles Sempere (Alicante, ES)

Introduction: Psychosocial factors are determinants of health outcomes in Inflammatory Bowel Disease (IBD). This study aims to characterize psychosocial and behavioral profiles using the Millon Behavioral Medicine Diagnostic (MBMD).

Methods: A multicentre observational prospective study was conducted including patients with a confirmed diagnosis of IBD. Clinical and socio-demographic variables were collected.

The psychometric evaluation was performed using the MBMD, a self-report inventory designed to assess a wide array of psychosocial and behavioral factors that impact adjustment to illness and medical treatment outcomes. The MBMD contains 165 true/false items and takes approximately 20–30 min to complete. We have focused on psychiatric indications, coping styles, stress moderators, and treatment prognostics.

Results: A total of 185 subjects were included: 94 (50.8%) Crohn's disease (CD), 88 (47.6%) Ulcerative Colitis (UC) and 3 (1.6%) IBD Unclassified. 99 (53.5%) women, 86 (46.5%) men. Mean age 50.23 (\pm 13.68). There were no significant differences in sex or age between CD and UC. The patient cohort included 53 (28.6%) never smokers, 93 (50.3%) former smokers, and 39 (21.1%) current smokers.

The MBMD profile highlighted a clinically significant alteration in Illness Adaptation (76.35 ± 14.79), exceeding the pathological threshold (> 75) and suggesting poor coping.

Statistically significant differences were observed in the mean scores of different scales between CD and UC (Figure 1). Patients with CD presented significantly higher scores than those with UC in Anxiety-Tension (60.57 vs. 52.84; $p < 0.05$), Depression (52.71 vs. 41.51; $p < 0.01$), and Pain Sensitivity (59.85 vs. 48.28; $p < 0.001$). Likewise, this group showed greater Functional Deficits (59.64 vs. 49.40; $p < 0.01$) and a greater need for Psychological or Psychiatric Referral (58.44 vs. 53.19; $p = 0.05$).

Conversely, Ulcerative Colitis (UC) was characterized by high-risk health management behaviors, with significantly higher scores in problematic compliance (66.62 vs. 57.61; $p < 0.01$) and information discomfort (58.69 vs. 46.87; $p < 0.01$).

Discussion/Conclusion: CD is characterized by a higher prevalence of anxiety, depression, and difficulty in adapting to the illness, whereas UC patients exhibit a high-risk health management profile. These findings suggest prioritizing psychological support in CD and psychoeducational adherence strategies in UC.

4. Risk of colorectal cancer in microscopic colitis compared with colonoscopy-verified normal mucosa

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Introduction: Microscopic colitis (MC), encompassing collagenous colitis (CC) and lymphocytic colitis (LC), is associated with a reduced risk of colorectal cancer (CRC), although this may reflect selection bias (MC is rarely diagnosed in the presence of concomitant CRC) and the protective effect of colonoscopy. Whether an inverse association persists also compared to individuals with histopathologically normal mucosa at colonoscopy remains unknown.

Methods: Using the nationwide Swedish histopathology cohort, ESPRESSO, we conducted a nationwide matched cohort study to estimate CRC risk in patients with MC, diagnosed between 1990 and 2023. Each patient with MC was matched, on age, sex, county of residence, and calendar year, to up to five reference individuals who had undergone colonoscopy with histopathologically normal mucosa. Individuals with prior malignancy were excluded. Incident CRC was ascertained through the Swedish Cancer Register.

Results: Between 1990 and 2023, 9,933 patients with a first-time diagnosis of MC met the eligibility criteria. These patients were matched to 32,238 reference individuals. The median age at MC diagnosis was 58.7 years, and 67% were women.

During a median follow-up of 8.4 years, 82 patients with MC (0.8%) were diagnosed with CRC, compared with 363 reference individuals (1.1%). This corresponded to incidence rates of 8.7 (95% CI: 7.0-10.8) and 11.5 (95% CI: 10.4-12.8) per 10,000 person-years, respectively.

After adjustment for the matching variables, educational attainment, chronic obstructive pulmonary disease, type 1 diabetes, celiac disease, and inflammatory bowel disease, MC was associated with a reduced risk of CRC (adjusted hazard ratio [aHR] = 0.65 [95% CI: 0.51-0.83]).

When stratified by subtype, the aHR was 0.74 (95% CI: 0.55-0.98) for LC and 0.49 (95% CI: 0.31-0.79) for CC.

Discussion/Conclusion: The lower risk of CRC observed in MC persisted despite comparison with colonoscopy-verified normal mucosa, warranting further mechanistic studies to explore a potential protective effect of MC.

5. Risk factors of fatigue in patients with inflammatory bowel disease

Oussama Daboussi (Le Coudray, FR), **Murphy Luwawu** (Le Coudray, FR), **Anne Herber** (Le Coudray, FR)

Introduction: Fatigue is a common reported symptom, in IBD patients, present in 86 % of patients with active disease, and between 20% and 48% in those in remission. It could impact negatively the quality of life of our patients. The aim of this study was to determine the degree of fatigue and to identify possible risk factors associated to its presence.

Methods: Consecutive patients presented at our department from 2024 to 2025 were included. Disease activity scores, endoscopic scores, disease duration, localization, laboratory data, were collected for each patient. Total Mayo score (TMS) and Crohn's disease activity index (CDAI) were used to assess disease activity in

patients with ulcerative colitis (UC) and Crohn's disease (CD) respectively. Clinical remission was defined as TMS \leq 2 points, or CDAI $<$ 150 points. Presence of anxiety and depression was evaluated using Hospital Anxiety and Depression Scale (HADS) and fatigue was assessed with self-administered questionnaire FACIT Fatigue Scale. Based on this scale, clinically significant fatigue is defined by a score $<$ 30.

Results: A total of 60 patients were included; mean age was 39.4 ± 13.0 years, 60% females; 66% Crohn's disease. Mean disease duration was 7 ± 3 years. Of UC patients, 40% were in clinical remission, 20% had mild disease, 30% moderate disease, and 10% severe disease. Of CD patients, 60% were in clinical remission and 40% had active disease.

Clinically significant fatigue was found in 12/60 (20%) of patients, and was significantly higher in those with active disease, and anxiety. The mean fatigue score was 32 points with no statistical difference between CD and UC. Higher fatigue scores were observed among patients with presence of active disease; among patients with higher TMS scores; in patients with anxiety, and in patients with depression.

Discussion/Conclusion: Our study findings confirm that the most important factors associated with fatigue during IBD course, are disease activity and presence of anxiety and/or depression.

6. Joint involvement in patients with celiac disease and ulcerative colitis

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Introduction: Intestinal diseases are immune-mediated disorder with a multiform presentation and therefore a challenging diagnosis. Our purpose is to evaluate the prevalence and severity of joint involvement in young patients with celiac disease compared to patients with ulcerative colitis using musculoskeletal ultrasound

Methods: In this study we examined musculoskeletal ultrasound at level of knees and ankles patients with celiac disease and ulcerative colitis. The presence of synovial hypertrophy, joint effusion, power Doppler signal and structural damage lesions was registered. Inflammatory abnormalities were scored on a semi-quantitative scale (0-3), and structural damage lesions on a dichotomous scale (0-1).

Results: Thirty-six young patients with celiac disease (mean age 19.6 years) and thirty-two with ulcerative colitis (mean age 18.4 years) was enrolled. In celiac disease ultrasound showed the presence of abnormalities in 13 patients in total (33.1%); joint effusion was the most frequently observed change (10/13). Ultrasound abnormalities were observed in 8 patients (22.2%). Interestingly, 7/13 (53.8%) patients with changes detected by ultrasound were asymptomatic. In ulcerative colitis ultrasound showed the presence of abnormalities in 18 patients in total (56.2%); also joint effusion was the most frequently observed change (17/18). Ultrasound abnormalities were observed in 13 patients (40.6%), and 10/18 (55.5%) patients with changes detected by ultrasound were asymptomatic. The arthritis was most often oligoarticular and asymmetric in both disease.

Discussion/Conclusion: Joint effusion, the most common manifestation, was present in both celiac disease and colitis patients and also arthritis is a common symptom that occurs in most intestinal diseases and is not always associated with intestinal symptoms.

7. Real-world effectiveness and safety of Jorveza® in adult eosinophilic oesophagitis: A UK tertiary-centre cohort

William Fong (Nottingham, GB), **Guiomar Villarejo** (Nottingham, GB)

Introduction: Eosinophilic oesophagitis (EoE) is a chronic, immune-mediated condition characterised by oesophageal dysfunction and eosinophil-predominant inflammation. Jorveza®, an orodispersible formulation of budesonide, was licensed in 2018 as a targeted topical therapy and is increasingly used following the 2022 BSG EoE guideline recommendations; however, real-world data remain limited. This study evaluated the effectiveness and tolerability of Jorveza® in adults with EoE managed at a UK tertiary centre.

Methods: A retrospective review was conducted of adults with EoE who initiated Jorveza® between October 2023 and October 2025 and subsequently underwent follow-up oesophagogastroduodenoscopy (OGD) with biopsies. Demographic characteristics, atopic comorbidities, prior treatments, and age at diagnosis were recorded. Primary outcomes were histological remission (<15 eos/hpf), endoscopic improvement based on EREFS, and symptomatic response. Secondary outcomes included adverse effects and treatment adherence. Associations between baseline characteristics and treatment outcomes were analysed using Chi-square/Fisher's exact tests and logistic regression.

Results: Fifty-six patients were included (median age at diagnosis 34.5 years; 64.3% male). Atopy was common (62.5%), most frequently asthma. Prior to Jorveza®, 78.6% had trialled PPIs, 63.7% were steroid-naive, and 24.1% had attempted food elimination diets. Adequate biopsy sampling at follow-up OGD was achieved in 82.2%. The median Jorveza® treatment duration was 12 weeks. Deep histological remission occurred in 66.6%, endoscopic remission in 64.1%, and symptomatic improvement in 79.6%. Histological and endoscopic remission were significantly associated with previous steroid exposure ($p = 0.01$), pre-treatment dilatation ($p = 0.04$), and fibrostenotic phenotype. Symptomatic improvement was significantly associated with prior PPI exposure ($p = 0.02$). Adherence was high (88.5%), and treatment was well tolerated. Abdominal symptoms were the most common adverse effect (10.5%), and one case of oral candidiasis was identified.

Discussion/Conclusion: Jorveza® achieved high rates of histological, endoscopic, and symptomatic remission in this real-world cohort. Prior steroid exposure, fibrostenotic phenotype, and pre-treatment dilatation were associated with improved objective outcomes, while previous PPI use was linked only to symptomatic improvement. Jorveza® was safe, well tolerated, and demonstrated excellent adherence, supporting its effectiveness in routine clinical practice.

8. The relationship between stress indicators and the severity of inflammation in celiac disease

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Introduction: The activity of the autonomic nervous system (ANS) plays a crucial role in the regulation of gastrointestinal (GI) function and the inflammatory response. Increased stress levels may lead to an overactive sympathetic nervous system response and may be associated with the intensification of inflammation. Celiac disease (CD) is a complex immune-mediated disorder characterized by chronic inflammation of the small intestinal mucosa triggered by gluten intolerance in genetically susceptible individuals, resulting in subsequent malabsorption. The aim of this study was to evaluate the correlation between stress indicators and the advanced stage of celiac disease (CD).

Methods: The study comprised 53 patients with CD, with a mean age of 38.2 ± 14 years. The severity of stress was assessed using stress indices calculated based on hemodynamic parameters and indicators from linear and nonlinear heart rate variability (HRV) analysis using the HRV Kubios Premium software.

Results: A high level of stress, indicated by a Stress Index value above 12, was found in 18% of patients with CD. Positive correlations were observed between the Stress Index, SNS index (stress), and the Marsh scale ($R = 0.325$; $p = 0.017$), as well as with sympathetic ANS activity indices ($R = 0.295$; $p = 0.031$). Negative correlations were identified with indicators of parasympathetic nervous system activity and the Marsh scale ($R = -0.225$; $p = 0.06$).

Discussion/Conclusion: Approximately 18% of CD patients experienced significant levels of stress. The stress indicators, sympathetic ANS activation, and/or impaired function of the parasympathetic system were positively correlated with the Marsh scale (stage of CD). Assessing the severity of stress and reducing it will enable targeted therapy to improve the advanced stage of the disease.

9. Wild thyme extract improves gastrointestinal comfort through gut microbiota modulation and inflammation-related pathways: A clinical study in adults

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Introduction: Functional gastrointestinal discomfort affects approximately 40% of the global population and negatively impacts quality of life. Its multifactorial origin – including diet, lifestyle, and gut microbiota imbalance, most likely associated with a chronic inflammatory state – makes its management challenging. Nutritional supplements targeting the gut microbiota, such as polyphenol-rich botanical extracts with anti-inflammatory properties, represent promising strategies to improve digestive comfort and well-being. Wild thyme (*Thymus serpyllum* L.) contains bioactive polyphenols and prebiotic fibers that may influence gastrointestinal function and the gut-brain axis. This study evaluated the efficacy of a wild thyme extract on digestive comfort, quality of life, and gut microbiota composition.

Methods: In this multicenter, double-blind, randomized, placebo-controlled study, healthy adults with self-reported gastrointestinal discomfort received either a wild thyme extract (36OGUT®, Finzelberg) or placebo. The extract (70% native extract, 30% soluble fiber; 11–12% polyphenols; essential oil-free) was obtained through a patented water-based extraction process. Efficacy was assessed using a gastrointestinal symptom severity rating scale (GSSR) and a quality-of-life (QoL) questionnaire. Gut microbiota composition was analyzed by shotgun sequencing to explore associations with clinical outcomes. (ClinicalTrials.gov no: NCT06639126)

Results: Participants receiving the wild thyme extract showed significant reductions in gastrointestinal symptoms, particularly indigestion, constipation, and diarrhea. QoL scores improved, reflecting better overall health perception, reduced impact of discomfort on daily life, and greater emotional resilience compared with placebo. These benefits were associated with modulation of the intestinal microbiota, promoting a balanced and metabolically active bacterial community enriched in taxa linked to short-chain fatty acid production and intestinal anti-inflammatory properties, thereby supporting normal gastrointestinal function and well-being.

Discussion/Conclusion: Supplementation with 36OGUT® alleviates gastrointestinal imbalances and enhances physical and emotional well-being, likely through prebiotic effects and modulation of the gut-brain axis. These findings suggest that this botanical extract represents a safe and effective natural approach to improving digestive comfort and quality of life through gut microbiota modulation and the downregulation of inflammation-associated processes involved in these conditions.

10. Low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 attenuate tumor progression and modulate gut microbiota in AOM/DSS-induced colorectal cancer

Maria Jose Garcia-Mansilla (Granada, ES), Luckman Gbati (Granada, ES), Jose Alberto Molina Tijeras (Granada, ES), Maria Jesus Rodriguez Sojo (Granada, ES), Laura Hidalgo Garcia (Granada, ES), Jorge Garcia Garcia (Granada, ES), Laura Lopez Escanez (Granada, ES), Antonio Jesus Ruiz Malagon (Granada, ES), Teresa

Veza (Granada, ES), Maria Jose Rodriguez-Sanchez (Granada, ES), Ailec Ho Plagaro (Granada, ES), Julio Galvez Peralta (Granada, ES), Maria Elena Rodriguez Cabezas (Granada, ES), Alba Rodriguez Nogales (Granada, ES)

Introduction: Bioactive low-molecular-weight fractions derived from probiotic bacteria have been shown to exert anti-inflammatory and anti-proliferative effects, which may be beneficial in inflammation-associated colorectal cancer. This study evaluated the effects of low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 on tumor development, epithelial proliferation, and gut microbiota modulation in an AOM/DSS-induced colorectal cancer mouse model.

Methods: Colorectal carcinogenesis was induced using AOM/DSS, followed by administration of low-molecular-weight bacterial fractions. Tumor number and volume were quantified macroscopically. Histological and immunohistochemical analyses were conducted to assess tumor morphology and cellular proliferation (Ki-67). Inflammatory and immune parameters were evaluated using molecular and cytometric approaches, and gut microbiota composition was analyzed.

Results: Low-molecular-weight fraction treatment significantly reduced both tumor incidence and tumor volume in AOM/DSS-treated mice. Histological evaluation demonstrated reduced dysplasia and improved tissue organization. A marked reduction in epithelial proliferation was observed, as indicated by decreased Ki-67 expression. In addition, treatment modulated inflammatory responses and restored gut microbiota balance, suggesting a protective role against inflammation-driven tumorigenesis.

Discussion/Conclusion: Low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 effectively attenuate colorectal tumor progression and modulate host inflammatory and microbial environments in AOM/DSS-induced cancer. These findings highlight the therapeutic potential of postbiotic molecular fractions in colorectal cancer.

11. Low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 ameliorate DSS-induced colitis in mice

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Introduction: Inflammatory bowel diseases (IBD) are chronic disorders characterized by excessive intestinal inflammation, disruption of epithelial barrier integrity, and gut microbiota dysbiosis. Postbiotics derived from probiotic strains represent a promising therapeutic strategy for restoring intestinal homeostasis. This study aimed to evaluate the protective effects of low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 on intestinal inflammation in a DSS-induced colitis mouse model.

Methods: Experimental colitis was induced in mice using dextran sulfate sodium (DSS), followed by treatment with low-molecular-weight fractions derived from *L. fermentum* CECT5716. Disease severity was assessed using the Disease Activity Index (DAI) and macroscopic evaluation of colonic damage. Histopathological analysis was performed to assess tissue architecture and inflammatory infiltration. Inflammation and intestinal barrier integrity were evaluated by RT-qPCR and fluorescence-based techniques. Cellular proliferation and inflammatory activity were assessed by Ki-67 immunostaining, and gut microbiota modulation was analyzed.

Results: Treatment with low-molecular-weight fractions from *L. fermentum* CECT5716 significantly ameliorated DSS-induced colitis, as evidenced by a marked improvement in DAI scores and reduced macroscopic colonic damage. Pro-inflammatory cytokine IL6 and TNFalpha levels were significantly decreased, indicating a strong anti-inflammatory effect. Histological analysis revealed preservation of colonic architecture, reduced inflammatory cell infiltration, and improved mucosal integrity. Fluorescence permeability assays confirmed a significant restoration of epithelial barrier function. In addition, treatment reduced inflammation-associated epithelial hyperproliferation, as demonstrated by decreased Ki-67 expression, and favorably modulated gut microbiota composition.

Discussion/Conclusion: These findings demonstrate that low-molecular-weight fractions derived from *Limosilactobacillus fermentum* CECT5716 effectively promote gut barrier repair, reduce intestinal inflammation, and modulate gut microbiota in DSS-induced colitis. This postbiotic-based approach represents a promising therapeutic strategy for the management of inflammatory bowel diseases.

12. Extracellular vesicles derived from *Limosilactobacillus fermentum* CECT5716 attenuate tumor development and inflammation in an AOM/DSS-induced colorectal cancer mouse model

Luckman Gbati (Granada, ES), Jose Alberto Molina Tijeras (Granada, ES), Maria Jesus Rodriguez Sojo (Granada, ES), Laura Hidalgo Garcia (Granada, ES), Jorge Garcia Garcia (Granada, ES), Laura Lopez Escanez (Granada, ES), Teresa Veza (Granada, ES), Antonio Jesus Ruiz Malagon (Granada, ES), Ailec Ho Plagaro (Granada, ES), Julio Juan Galvez Peralta (Granada, ES), Alba Rodriguez Nogales (Granada, ES), Maria Elena Rodriguez Cabezas (Granada, ES)

Introduction: Chronic intestinal inflammation is a major driver of colorectal cancer development. Extracellular vesicles derived from probiotic bacteria have emerged as bioactive mediators capable of modulating host immune responses and tumor-associated pathways. This study aimed to investigate the anti-tumoral and immunomodulatory effects of extracellular vesicles derived from *Limosilactobacillus fermentum* CECT5716 LEVs in a mouse model of colorectal cancer induced by azoxymethane (AOM) and dextran sulfate sodium (DSS).

Methods: Colorectal cancer was induced using the AOM/DSS protocol, followed by treatment with bacterial extracellular vesicles. Tumor burden was assessed by measuring tumor number and volume. Histopathological analysis was performed to evaluate tissue architecture and tumor progression. Cellular proliferation was assessed by Ki-67 immunostaining, while immune and inflammatory responses, gut barrier were analyzed using cytometric and molecular approaches. Gut microbiota composition was also evaluated

Results: Treatment with *L. fermentum* derived extracellular vesicles significantly reduced tumor number and tumor volume compared to untreated AOM/DSS mice. Histological analysis revealed reduced tumor invasiveness and improved tissue organization. LEVs treatment markedly decreased epithelial proliferation, as evidenced by reduced Ki-67 expression, and modulated inflammatory and immune responses. Additionally, extracellular vesicles induced favorable changes in gut microbiota composition, consistent with a tumor-protective intestinal environment.

Discussion/Conclusion: These results demonstrate that extracellular vesicles derived from *Limosilactobacillus fermentum* CECT5716 exert significant anti-tumoral and immunomodulatory effects in inflammation-driven colorectal cancer. LEVs based postbiotics represent a promising strategy for colorectal cancer prevention and therapy.

13. Retrospective assessment of the association of the budesonide with gluten-free diet in the treatment of the celiac disease in autoimmune hepatitis

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Introduction: The aim of this retrospective study was the assessment the efficacy of association of gluten-free diet with budesonide-azathioprine combined therapy in patients with both autoimmune hepatitis (AIH) and celiac disease (CD).

Methods: We studied 32 patients with associate CD and AIH, were treated with combined therapy with prednisone (40 mg/day and tapered to 10 mg/day) and azathioprine (1-2 mg/kg/day) or with budesonide (3 mg, oral doses three times daily) in association with azathioprine (1-2 mg/kg/day). Six of these patients had pre-existent AIH (6 cases, 10.9%) and then were tested positive for anti-IgA tissue transglutaminase and were subsequently confirmed to be affected with CD by small-bowel biopsy findings. After CD detection, patients with CD and AIH continued current therapy for AIH and associated this treatment with a gluten-free diet (GFD). We monitoring, for a 12 months period, the activity disease and evaluated response of therapy.

Results: Structure of the lot of patients indicate the predominance cases of AIH type I (23 cases, 71.87%) comparative with type II AIH (9 cases, 28.13%). At baseline, most patients (25 cases) were included in the Marsh 3 stage of celiac disease, 5

in Marsh 4 and two in the Marsh 2 stage. In two cases (6.25%) celiac disease was asymptomatic. GFD was associated in all cases with AIH therapy: in 20 cases with budesonide-azathioprine combined therapy (the A group) and in 12 cases with prednisone-azathioprine (B group). After 6 months, the clinical symptoms were improved in both groups but tissue transglutaminase IgA antibody (tTG-IgA) and liver enzymes decreased more in the A group: celiac serology normalized in 70.0% of patients that received budesonide and in 50.0% of B groups patients. At 12 months, intestinal biopsies were reported to be normal in 15 patients who associated GFD with budesonide-azathioprine therapy (75.0%) and in 6 patients who received prednisone-azathioprine therapy (50%). Also, disappearance of clinical symptoms of CD was observed in 90% of the patients from the A group and in 75% of the B group patients. We have not found a correlation between GFD and rate of remission of AIH, but after 12 months on a GFD, normal liver biochemistry was observed in most patients with CD and AIH (23 cases, 71.87%).

Discussion/Conclusion: The gluten-free diet associated with budesonide-azathioprine combined therapy, is effective in induces and maintains remission in patients diagnosed with both CD and AIH. Long term gluten-free diet may have a beneficial effect in reversing autoimmune liver disease in patients with CD.

14. Why upper GI pathology matters to the otolaryngologist

Rukmini Ghosh (Nottingham, GB), **Rishi Srivastava** (Nottingham, GB)

Introduction: Gastrointestinal inflammation and neoplasia are of increasing relevance to otolaryngology-head and neck surgery, reflecting the continuity of the aerodigestive tract and the central role of gastrointestinal pathology in upper airway disease. Inflammatory disorders of the oesophagus and stomach, can exert effects on the pharyngeal and laryngeal mucosa. Exposure to gastric acid, bile salts, enzymes and inflammatory mediators can disrupt epithelial integrity, alter local microbiota, and promote chronic inflammation in the upper aerodigestive tract, contributing to symptoms and pathology frequently encountered by ENT surgeons.

We know the vagus nerve innervates the pharynx, larynx, and oesophagus. Therefore, it is reasonable to assume that symptoms at the level of the pharynx can be due to referred symptomatology from the lower oesophagus.

In the ENT setting, patients can present with a multitude of throat symptoms, including pharynx-localised dysphagia. It is disputed whether the ENT examination should finish at the cricopharyngeus or in the upper oesophagus.

Methods: This abstract represents single-surgeon data in a tertiary otolaryngological setting, (prospectively collected and retrospectively analysed) undertaking the transnasal oesophagoscopy technique to examine the ENT and upper GI systems.

Results: This case series of 491 patients, over a 4 year period, presenting primarily with pharynx localised dysphagia, has revealed 182 patients with conditions that belong to the realm of gastroenterology and the upper GI surgeon. These include reflux oesophagitis, gastritis, inlet patches, Barrett's oesophagus, hiatus hernia,

eosinophilic oesophagitis, achalasia, and malignancy. This represents approximately 37% of patients presenting to the ENT surgeon.

Discussion/Conclusion: This shows that we need to rethink the boundaries of ENT and gastroenterology, consider all-in-one assessments for better patient care pathways that could help reduce the financial burden currently facing the NHS.

15. Evolution of histologic and endoscopic assessment of eosinophilic esophagitis in Europe and its effects on diagnostic delay: Data from the EUREOS EoE CONNECT registry

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Introduction: Endoscopy with histopathological assessment of esophageal biopsies is the cornerstone for the diagnosis and phenotypic characterization of eosinophilic esophagitis (EoE).

Here, we aim to evaluate temporal trends in endoscopic assessment and biopsy sampling practices for EoE in a large European cohort and their impact on diagnostic delay (DD).

Methods: A cross-sectional analysis was conducted using multicenter data from the EoE CONNECT registry. Endoscopic findings and biopsy data at diagnosis were analyzed. Temporal trends in biopsy strategies, EREFS implementation, and DD were assessed. Diagnostic sensitivity of individual esophageal segments and their combinations was evaluated in patients undergoing multi-segment sampling. Associations between guideline publication and biopsy practices were analyzed using logistic regression models.

Results: A total of 3,298 patients were included. Biopsies were obtained from one esophageal segment in 12.6% of patients, two segments in 63.6%, and three segments in 23.8%. Adherence to guideline-recommended biopsy strategies improved over time, with reduced single-segment sampling and increased use of ≥ 2 segments following the 2017 guideline release. Distal esophageal biopsies provided the highest diagnostic sensitivity, and sampling that included the distal segment achieved sensitivity above 97%, particularly when combined with middle esophageal biopsies. EREFS reporting increased from 88-90% to over 98% in recent years. DD decreased significantly over time (approximately 0.7 years per calendar year), accompanied by a shift from mixed/stricturing to inflammatory phenotypes.

Discussion/Conclusion: Increased alignment with international guidelines, reflected by standardized endoscopic reporting and multi-segment biopsy protocols, was associated with improved diagnostic sensitivity, reduced DD, and fewer fibrostricturing presentations.

16. Colonic mucosal biofilm microbiota alterations in IBD

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Introduction: The mechanisms explaining complex relationship between the commensal colonic microbiota and IBD have a common outcome, a violation of bacterial antigens exposure to effector T-cells and innate immune cells residing in the intestinal mucosa and/or alteration of the host immune response to bacteria. Application of microbiome knowledge is traditionally on treatment and prevention of dysbiosis. The discovery of dominant members of microbial communities serving beneficial functions including immunomodulation is likely to bridge gap of lacking complete understanding of microbiome engineering will widen this scope to suit preventive, therapeutic, and diagnostic needs in IBD. While the role of gut microbiota and respective immune changes has become more evident in recent years there is no sufficient database explaining the character of microbiota changes in IBD. The aim of this study is to find associations between changes of colonic microbiome and IBD.

Methods: Totally 104 individuals participate in the study. Among them 34 had clinically and endoscopically proven IBD, (12 – CD, 22 – UC), others with at least three risk factors of IBD (family history, smoking, antibiotics, travel history, immune, etc). Colonic resistance studied in multiple mucosal bioplates. Standard aerobic and anaerobic microbiology techniques with nosology identification and quantity composition of microbiota were used. Diversity and microecological indices were used to assess the structures of colonic microbial populations. All isolated taxons were analyzed by antibiotic resistance analysis for each sampling event for all individuals. RT-PCR used for assessment of microbial genotypes.

Results: Major autochthonic species (14 in total) were present in all samples: among them Lactobacteria, Bifidobacteria, E. coli, several other anaerobic species were dominating. However, Lacto- and Bifidobacteria were found in significantly lower levels compared to healthy subjects ($p = 0.02-0.0031$). The general tendency for colonic resistance in IBD was decrease of autochthonic anaerobes (Bifido-, Lactobacteria, Bacteroides spp, Clostridia spp, Bacillae spp.) and significant growth of allochthonic aerobes and facultative anaerobes (E. coli Hly+, Pseudomonas, Serratia, Hafniae, P. mirrabilis and other conditionally pathogenic Enterobacteriaceae). Enterococci were present in 60.0% of control and 7.14–20.69% of study group. Staphylococci were present only in study group (17.24–31.58%). Group and individual biodiversity indices were significantly lower in IBD, especially in UC group.

Discussion/Conclusion: Our data suggest that morbid changes of colonic mucosal microbiota, e.g. abnormal ratio of autochthonic and allochthonic species, may be considered as a strong characteristic feature of IBD. Meanwhile, there is no exact IBD “pathogen” found. This study gives possible objectives for modulating therapies – restoration of microbiome’s biodiversity and additional population by Bifido- and Lactobacteria with use of adjuvants or diets for creating adequate conditions for microflora development.

17. Strategy for intestinal ultrasonography: A new goal in the follow-up of patients with inflammatory bowel disease undergoing treatment with biological agents and small molecules

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Introduction: Inflammatory bowel diseases (IBD), which include ulcerative colitis and Crohn’s disease, are chronic immune-mediated conditions affecting between 6 and 8 million people worldwide. They are a challenge to manage because of their complex pathophysiology and tendency to relapse. At the same time, with a wider range of treatment options and ways to optimize and personalize the therapy, there’s a need for reliable monitoring tools. The optimal choice is for those tools to be non-invasive, safe for the patient, low-cost and convenient for repeated use. This is the case with the intestinal ultrasonography.

Methods: A systematic review of the literature in PubMed, Scopus, Web of Science, and Google Scholar was conducted, examining articles published in English up to July 2025.

Results: Intestinal ultrasonography is a fast, reliable and non-invasive imaging method. According to existing data from clinical trials, it can be used in the diagnosis of IBD, determination of severity and localization of the inflammation and the presence of complications. Intestinal ultrasonography is used for precise monitoring of the therapeutic response in IBD and plays an important role in certain patient groups, such as children and pregnant women. The parameters for assessment in intestinal ultrasonography include bowel wall thickness (BWT), submucosal layer echogenicity, colour Doppler, loss of haustration, stratification

and the presence of lymph nodes. There are also the following limitations to performing intestinal ultrasonography: increased abdominal adipose tissue, high BMI and more complex intestinal anatomy, which can be difficult to assess.

Discussion/Conclusion: Nowadays, intestinal ultrasonography is becoming a popular and valuable way to keep an eye on patients with IBD who are being treated with biologics and small molecules. If this strategy proves successful, it could not only contribute to improving patients' quality of life, but also optimize healthcare resources by reducing hospitalizations, surgical interventions, and disability.

18. The efficacy and safety of vedolizumab as first-line advanced therapy for active ulcerative colitis in patients after liver transplantation

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Introduction: Treating active ulcerative colitis (UC) in patients who have undergone solid organ transplantation remains challenging due to the complexity of multidrug regimens and the increased risk of infectious and non-infectious complications compared with non-transplant patients. We aimed to evaluate the efficacy and safety of vedolizumab in UC patients who previously underwent liver transplantation (LT).

Methods: This was a multicenter retrospective study. Data were collected on adult post-LT patients with moderately to severely active UC (Mayo score ≥ 6) who were naïve to advanced therapies and received induction treatment with intravenous vedolizumab 300 mg at weeks 0, 2, and 6. Treatment response assessment at week 14 required for reimbursement of therapy was defined as a decrease in total Mayo score of $\geq 30\%$ and ≥ 3 points from baseline while remission as a total Mayo score ≤ 2 . Endoscopic improvement was defined as a Mayo endoscopic subscore of 0 or 1, with a decrease of ≥ 1 point from baseline. Data on maintenance therapy in patients who responded to induction, as well as complications during treatment, are also reported. The study was approved by the local Bioethics Committee.

Results: Twenty-nine eligible patients treated across 10 Polish gastroenterology centers between 2021 and 2025 were identified (20 males, 9 females; median age 33 years, range 19–54). Indications for liver transplantation included primary sclerosing cholangitis (PSC; 19 patients, with recurrent PSC diagnosed in 9), overlapping autoimmune hepatitis/PSC (AIH/PSC; 7 patients), and AIH (3 patients). The median interval since LT was 6 years (range 1–14), and the median duration of UC was 10 years (range 3–22); extensive UC was present in 23 patients. The median follow-up period was 9 months (range 1–47).

At week 14, clinical response was observed in 24 patients (83%), who next received intravenous or subcutaneous maintenance therapy. Clinical remission at week 14 in

occurred 6 patients (21%), and endoscopic improvement in 13 (45%). At the time the data was analysed, with vedolizumab was being continued in 14 patients (48%).

Severe complications were the primary reasons for discontinuation among 10 patients who responded to induction therapy. These included intestinal lymphoma (n = 1), colonic dysplasia (n = 1), cholangitis (n = 2), Epstein-Barr virus infection (n = 1), and *Clostridioides difficile* infection (n = 1). Secondary loss of response occurred in one patient; in two others, infusion intervals were shortened due to exacerbation of symptoms. Two patients discontinued treatment for personal reasons. One patient died due to complications following a second LT. An uncomplicated pregnancy occurred in one female patient who remains on subcutaneous vedolizumab.

Discussion/Conclusion: The 14-week clinical response rate to vedolizumab in UC patients after LT was high, and nearly half of the patients achieved endoscopic improvement. The relatively low remission rate following the induction therapy suggests that more time may be required to reach the desired treatment targets in this specific patient population. Close surveillance is essential to enable early detection of UC/PSC- and treatment-related complications.

19. Beyond dysphagia: Prospective evidence that fatigue is an under-recognised burden in eosinophilic oesophagitis

Rohith Kumar (Stevenage, GB), Jo Brooks-Warburton (Stevenage, GB), Danielle Morris (Stevenage, GB)

Introduction: Fatigue in eosinophilic oesophagitis (EoE) remains under-characterised. Prior work relies largely on generic HRQoL vitality domains or indirect quality-of-life measures, which consistently demonstrate reduced energy yet correlate poorly with histologic or endoscopic disease activity [1]. Qualitative studies further highlight fatigue explicitly, with adults describing exhaustion, constant vigilance around food, and feeling drained by chronic symptoms and dietary restrictions [2–3]. Despite these observations, fatigue has not been prospectively evaluated using a dedicated validated assessment tool alongside validated EoE activity measures such as EREFS, eosinophil counts, or the Dysphagia Symptom Questionnaire (DSQ).

Aim: To prospectively assess fatigue in adults with EoE using the Fatigue Assessment Scale (FAS) and examine its relationship with endoscopic phenotype, eosinophilic activity, and symptom burden.

Methods: We conducted a fatigue audit for a clinical quality improvement project over 3 months (Sept 2025–Nov 2025) of adults with confirmed EoE. Patients completed FAS at the time of clinical visit, and paired with the most recent gastroscopy, including those performed on the same day. FAS categories: no fatigue (10–21), fatigue (22–34), severe fatigue (≥ 35). Endoscopic phenotype was classified by EREFS scoring as inflammatory or fibrostenotic. Histologic active disease was defined as ≥ 15 eosinophils/high powered field. DSQ was available in a subset.

Results: 63 patients had FAS and gastroscopy data (median age 47.5 years; IQR, 36.8–58.0; 67% male). All gastroscopies occurred within 2.5 years (median gastroscopy-to-FAS interval 38 days; IQR, 0–107). Fatigue severity was similar between inflammatory and fibrostenotic phenotypes (any fatigue 37.5% vs. 35.9%; severe fatigue 12.5% vs. 5.1%), with no statistical association ($\chi^2 = 1.20$, $p = 0.55$). Fatigue tended to be more common in active histologic disease (any fatigue 47.8% vs. 30.0%; severe fatigue 13.0% vs. 5.0%), though not significant ($\chi^2 = 2.41$, $p = 0.30$; FAS ≥ 22 $p \approx 0.16$). In the DSQ subgroup ($n = 31$), 12 were in clinical remission (DSQ = 0) and 19 were symptomatic (DSQ ≥ 1). Fatigue was more frequent in symptomatic patients (68.4% vs. 41.7%), and all severe fatigue occurred in this group. DSQ correlated modestly with FAS ($r = 0.28$, $p = 0.13$).

Table 1. Fatigue severity in inflammatory vs. fibrostenotic EoE

Fatigue Category	Inflammatory (n = 24)	Fibrostenotic (n = 39)	Total (n = 63)
No fatigue (FAS < 22)	15 (62.5%)	25 (64.1%)	40
Fatigue (22–34)	6 (25.0%)	12 (30.8%)	18
Severe fatigue (≥ 35)	3 (12.5%)	2 (5.1%)	5
All fatigue (≥ 22)	9 (37.5%)	14 (35.9%)	23
Total	24	39	63

$\chi^2 = 1.20$, $p = 0.548$

Table 2. Fatigue severity in inactive (< 15 eos/hpf) vs. active EoE (≥ 15 eos/hpf)

Fatigue Category	Inactive EoE (n = 40)	Active EoE (n = 23)	Total (n = 63)
No fatigue (FAS < 22)	28 (70.0%)	12 (52.2%)	40
Fatigue (22–34)	10 (25.0%)	8 (34.8%)	18
Severe fatigue (≥ 35)	2 (5.0%)	3 (13.0%)	5
All fatigue (≥ 22)	12 (30.0%)	11 (47.8%)	23
Total	40	23	63

$\chi^2 = 2.41$, $p = 0.3$

Discussion/Conclusion: In this prospective cohort, fatigue was prevalent but did not correspond closely with endoscopic phenotype or eosinophilic activity. Symptom burden showed the strongest directional association but lacked statistical significance in this early dataset. These findings reinforce that fatigue represents a distinct and incompletely captured aspect of EoE burden not reflected in current validated measures of EoE. Incorporating dedicated fatigue measures into future EoE studies and core outcome sets is warranted, and further recruitment at our centre is ongoing.

20. Can capsule sponge sampling replace gastroscopy for monitoring eosinophilic oesophagitis? A single-centre prospective study

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Introduction: Eosinophilic oesophagitis (EoE) is characterised by dysphagia and food impaction with oesophageal histology demonstrating ≥ 15 eosinophils/high power field (eos/HPF). Current UK and European guidelines recommend repeat endoscopic biopsies to assess treatment response, which can be burdensome for patients, costly, and resource intensive. Capsule sponge devices offer a minimally invasive alternative but have not been extensively evaluated in EoE using contemporary cytological processing methods.

Methods: Adults attending EoE clinics between July 2023 and December 2025 at a district general hospital were prospectively enrolled. Patients with strictures or contraindications to capsule sponge sampling were excluded. Participants underwent capsule sponge sampling (Endosign) and same-day gastroscopy ≥ 8 weeks after treatment initiation or change. Dysphagia Symptom Questionnaire (DSQ), capsule sponge eosinophil counts, endoscopic histology, and Endoscopic Reference Score (EREFS) were recorded. Active EoE was defined as ≥ 15 eos/HPF. Analyses were performed per paired visit. Fisher's exact test and Mann-Whitney U testing were used as appropriate.

Results: A total of 173 patients had paired same-day capsule sponge and gastroscopy data available; 68.8% were male, with a median age of 48 years (IQR, 36–57). Median DSQ score was 7.5 (IQR, 0–22.9). Significant diagnostic concordance was observed between capsule sponge cytology and endoscopic histology for active EoE ($R \approx 0.60$). Forty-one patients were identified as having active disease by both capsule sponge and OGD histology, and 100 as inactive by both modalities (Fisher's exact $p = 3.6 \times 10^{-15}$). Capsule sponge cytology demonstrated a sensitivity of 63.1%, specificity of 92.6%, and PPV of 83.7%. Eight patients had active disease detected by capsule sponge alone, while 24 had active disease detected only on endoscopic biopsy. In patients with histologically active disease, DSQ scores and EREFS were significantly higher than in those in histologic remission (Mann-Whitney U, $p < 0.05$).

Discussion/Conclusion: Capsule sponge sampling is feasible, safe, and highly specific for detecting active EoE in non-stenotic patients in routine clinical practice. While sensitivity is moderate, the strong concordance with histology and the advantage of pan-oesophageal sampling support its role as a complementary monitoring tool. Capsule sponge sampling may reduce the need for routine gastroscopy in selected patients, particularly those in clinical remission, and provides a platform for future biomarker development beyond eosinophil counts alone.

21. Therapeutic potential of *Limosilactobacillus fermentum* CECT5716 extracellular vesicles in promoting gut barrier repair through TLR4-dependent macrophage polarization

Andreea Roxana Lista (Granada, ES)

Introduction: Extracellular vesicles released by commensal bacteria have emerged as key mediators of host-microbe interactions, with increasing interest in their

therapeutic potential for inflammatory diseases. This study investigated extracellular vesicles derived from *Limosilactobacillus fermentum* CECT5716 (LEVs) and their role in modulating epithelial barrier function and immune responses during intestinal inflammation.

Methods: LEVs were characterized by transmission electron microscopy and dynamic light scattering to assess morphology and size, confirming their nanosized spherical morphology. Their immunomodulatory and barrier-protective effects were evaluated *in vitro* using Caco-2 intestinal epithelial cells and RAW 264.7 macrophages. An *in vivo* dextran sulfate sodium (DSS)-induced colitis mouse model was used to assess therapeutic efficacy. Colon tissues were analyzed by histology, gene expression, and immunofluorescence. Systemic immune cell populations were evaluated by flow cytometry, while gut microbiota composition and short-chain fatty acid (SCFA) levels were also analyzed.

Results: *In vitro*, LEVs enhanced epithelial barrier integrity by upregulating tight junction proteins and villin, while reducing the expression of proinflammatory cytokines IL-6, IL-8, and TNF- α in epithelial cells. In macrophages, LEVs increased CD206 expression, indicating polarization toward an M2-like regulatory phenotype.

In vivo, LEVs treatment significantly ameliorated DSS-induced colitis, as evidenced by reducing weight loss, prevention of colon shortening, and decreased mucosal damage. LEVs restored the expression of barrier-associated genes (Muc1, Muc3, villin) and normalized epithelial proliferation during colitis, reflected by restoration of Ki67 cell levels, suggesting a regulatory effect on epithelial turnover and repair. Flow cytometry revealed a shift from proinflammatory Ly6C monocytes toward regulatory Ly6C subsets. Additionally, LEVs modulated gut microbiota composition and SCFA levels.

Discussion/Conclusion: LEVs derived from *Limosilactobacillus fermentum* CECT5716 exert protective effects against intestinal inflammation by combining epithelial barrier reinforcement with immune modulation. These effects are mediated, at least in part, through context-dependent engagement of TLR4 signaling, resulting in attenuation of pro inflammatory responses and promotion of anti inflammatory and tissue repairing responses. Overall, LEVs represent a promising postbiotic strategy for the treatment of inflammatory bowel disease, by combining epithelial protection with immune regulation.

22. Gut microbiota-associated modulation of intestinal mesenchymal stromal cells in colorectal cancer

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Introduction: The gut microbiota is a key regulator of immune responses; however, the role of the intestinal stroma as a modulator of tissue integrity and homeostasis remains poorly understood. Alterations in the microbiota may influence the functional properties of intestinal mesenchymal stromal cells (iMSCs), potentially contributing to colorectal cancer-associated immune dysregulation.

Methods: iMSCs were isolated from paired non-tumoral and tumoral intestinal tissue resections obtained from colorectal cancer patients, expanded, and phenotypically characterized. Their immunomodulatory capacity was assessed by co-culture with human peripheral blood T lymphocytes using a T-cell proliferation assay. Mucosa-associated microbiota from the corresponding tissue samples was sequenced using Illumina MiSeq technology. Regenerative capacity was evaluated using a wound healing assay.

In parallel, fecal samples from colorectal cancer patients and healthy donors were used for fecal microbiota transplantation (FMT) into germ-free mice. Four experimental groups were analyzed: conventionally raised mice, germ-free mice without FMT, germ-free mice colonized with healthy donor microbiota, and germ-free mice colonized with microbiota from colorectal cancer patients. Intestinal mesenchymal stromal cell populations were evaluated by flow cytometry.

Results: Principal coordinates analysis (PCoA) identified three distinct microbial clusters correlating with iMSCs displaying high, intermediate, or low T-cell inhibitory capacity. Correlation matrix analysis revealed genus-level differences in mucosa-associated microbial composition associated with reduced iMSC immunomodulatory function, particularly in tumoral tissue-derived samples. Consistently, germ-free mice receiving FMT from colorectal cancer patients exhibited distinct alterations in intestinal mesenchymal stromal cell populations compared with mice colonized with healthy donor microbiota or control groups.

Discussion/Conclusion: These preliminary findings suggest that colorectal cancer-associated alterations in the gut microbiota are associated with changes in the phenotype and immunomodulatory properties of intestinal mesenchymal stromal cells, potentially impacting intestinal homeostasis.

23. Listening to patients – Real-world evaluation of capsule sponge monitoring in eosinophilic oesophagitis

Hyesha McDonald (Hertfordshire, GB)

Introduction: UK guidelines recommend monitoring the response to therapy in eosinophilic oesophagitis (EOE) to reduce the risk of strictures and complications. Endoscopy and biopsy are gold standard but can be uncomfortable and inconvenient for patients. A prospective cohort study (COSiE) was established to assess the feasibility, efficacy and acceptability of capsule sponge (CS) testing as an alternative. This abstract assess patient experience, tolerability and preference for CS compared with gastroscopy in EOE.

Methods: Adult patients with confirmed EOE were recruited from a dedicated EOE clinic at a UK non-specialist hospital between July 2023 and October 2025. Patients underwent CS testing using the Endosign device the same day prior to gastroscopy. Patients with known oesophageal strictures or recent food bolus obstruction were excluded. Patient feedback was collected before discharge using a single-page questionnaire assessing experience, willingness for CS, pain score using a validated Wong- Baker FACES score and preference for CS or gastroscopy and free field for comments.

Results: During this period, 134 patients with diagnosed EOE had same day CS and gastroscopy. 181 feedback forms were completed, this included patients who had 2 (n = 39) or 3 (n = 8) repeat CS. Only 1 form was left uncompleted, 10 patients failed to swallow the CS, and one withdrew consent but still completed feedback. There were 87 (65%) males and 46 (35%) female participants. 93% were white/white British background, 4% Asian, 3% Other. The median age group was 45-64 years and the median age was 54 years old for male and female patients.

For those expressing a preference, 127/159 (80%) preferred capsule sponge and 32/159 (20%) preferred gastroscopy. 10 patients expressed no preference and 12 did not complete the question.

Of 47 patients who had multiple sponges, 39 (83%) preferred CS and 8 (17%) preferred gastroscopy. Comfort score was measured using Wong-Baker FACES pain score. 57% of patients reported minimal pain score with 20% reporting a pain score of 0.

Pain score	Patient feedback
0 - no pain	20%
2	57%
4	18%
6	3%
8	1%
10 - hurts worst	1%

Free text comment included themes around speed and ease of CS procedure and mild discomfort rather than pain, a smaller group describing gagging or difficulty in swallowing CS.

Discussion/Conclusion: CS is a positive experience for patients and is a preferred option for those requiring monitoring of their EOE activity for most patients with non-stenotic EOE. These results are comparable to previous studies for CS used in investigating patients with Barrett's oesophagus and reflux which is reassuring given the underlying swallowing problems that are central to EOE patients. CS testing should be made more widely available for EOE patients across the UK: - this will also reduce unnecessary endoscopy and pressure on endoscopy services.

24. Impact of HLA-DQA1*05 genotype in immunogenicity and response to treatment with tumour necrosis factor-alpha antagonists in inflammatory bowel disease patients

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Introduction: Carrying the HLA-DQA1*05 gene has been linked to a higher risk of developing immunogenicity in patients with inflammatory bowel disease (IBD) who are treated with tumor necrosis factor-alpha inhibitors (antiTNF). However, studies have shown inconsistent evidence regarding its connection to a loss of response (LOR) or adverse events in patients with IBD.

Methods: Aim: To determine the impact of carriage of HLA-DQA105 allele on effectiveness, drug persistence and safety in patients diagnosed with IBD who had received antiTNF. Methods: Retrospective, single-center cohort study including IBD patients who had received antiTNF.

The HLA-DQA105 allele was determined from a saliva sample (kit OGD-600 de DNA Genotek Oragene) and DNA extraction with the Maxwell RSC-Stabilized Saliva DNA kits.

We evaluate drug persistence, withdrawal of antiTNF, primary non-response and secondary LOR, through antiTNF levels, development of antiTNF antibodies and adverse events in patients distributed by HLA-DQA105 allele presence.

Results: A total of 170 patients were included, 51% male, mean age 46.2, 120 (70.6%) diagnosed with Crohn's disease. The mean disease duration was 189.6 months. Fifty patients (29.4%) had extraintestinal manifestations and 30% comorbidities. HLA-DQA105 was positive in 44% patients. One hundred twenty-seven patients (75%) received infliximab and 25% adalimumab. AntiTNF was the first advanced therapy in 95% of the cohort. Seventy-five out of 170 patients (44%) received an intensified antiTNF regimen, as well as 47% were treated with combination therapy with immunosuppressants. AntiTNF was withdrawn in 52% patients during follow-up, and the rate of adverse events that led to discontinuation of the drug was 17.6%. When we compared the rate of primary non-response (12.2% vs. 7.5%, $p = 0.3$) and secondary LOR (48% vs. 44%, $p = 0.5$), withdrawal of antiTNF (48% vs. 54%, $p = 0.5$), duration of antiTNF (85.3 ± 69.1 months vs. 90.3 ± 76.4 , $p = 0.3$), need of intensified regimen (47% vs. 42%, $p = 0.4$), use of combination therapy (46% vs. 49%, $p = 0.6$) and antiTNF levels (2.6 ± 2.3 vs. 2.9 vs. 2.9 , $p = 0.3$) we did not find significant differences between patients by HLA-DQA105 status. The infusion reactions rate (8.1% vs. 11.8%, $p = 0.4$), development of anti-TNF antibodies (9.5 vs. 10.8, $p = 0.7$), overall (14.9 vs. 19.4), rate of adverse events (14.9 vs. 24.7, $p = 0.5$) or adverse events that led to drug withdrawal (14.9 vs. 19.4, $p = 0.4$) were also similar among patients regardless HLA-DQA1*05

Discussion/Conclusion: In our real-life cohort of IBD patients treated with antiTNF, being an HLA-DQA1*05 carrier did not act as a predictor of effectiveness (primary non-response or secondary LOR), immunogenicity or need of intensified regimen. The safety of antiTNF treatment has also not been influenced by the variant.

25. Female sexual dysfunction in inflammatory bowel disease: Utility of a novel specific scale

Roser Munoz (Alicante, ES)

Introduction: Reported prevalence of female sexual dysfunction (FSD) in IBD ranges from 40–90%. Although general FSD scales are available, such as FSFI, an IBD-specific scale for women has recently been developed: the IBD-FSDS, only validated in English and Danish.

Methods: Our aims were: to cross-culturally adapt IBD-FSDS into Spanish; to describe prevalence of FSD and its association with IBD-characteristics, quality of life (QoL), fatigue, and anxiety/depression (A/D) and to assess correlation between FSFI and IBD-FSDS.

This was a single-centre, cross-sectional study, including consecutive female IBD-patients aged 18–65 years, sexually active in the previous year. We assessed: FSD (with FSFI, IBD-FSDS); QoL (by SF-36, IBDQ-9); fatigue (using FSS); and anxiety/depression (A/D) (by HADS); correlation between FSD and IBD-characteristics; and correlation of FSD with QoL, fatigue, and A/D. This analysis forms part of the Spanish validation of IBD-FSDS.

Results: 181 women completed the surveys. 59.1% had CD. 6% of patients with CD and 16.2% with UC were clinically active. 21% reported a recent flare-up. The mean IBD-FSDS score was 13.01 ± 13.63 (range 0–60), and the mean FSFI score was 24.16 ± 8.86 (range 1.2–35.4). Prevalence of FSD ($FSFI \leq 26$) was 43%.

The IBD-FSDS score was significantly higher in patients with active CD (mean 25.33 vs. 11.05; $p = 0.005$) and in patients with IBD and a recent flare (mean 19.08 vs. 11.34; $p = 0.007$); and showed a significant negative correlation with FSFI ($p = -0.37$; $p < 0.0001$), SF-36 ($p = -0.58$; $p = 0.0001$) and IBDQ-9 ($p = -0.58$; $p = 0.0001$), and a positive correlation with FSS ($p = 0.46$; $p = 0.0001$) and HADS ($p = 0.50$; $p = 0.0001$).

Discussion/Conclusion: FSD affects 43% of women with IBD in our study. The IBD-FSDS demonstrates good correlation with FSFI, showing a positive correlation with clinical IBD activity, fatigue, A/D and poorer QoL. The IBD-FSDS may constitute a novel, useful tool for assessing FSD in Spanish women with IBD.

26. Intestinal stem cell adaptation and temporal response to inflammatory stress

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Introduction: Intestinal epithelial stem cells (ISCs) and their transit-amplifying (TA) progeny are essential for mucosal repair and regeneration. However, the impact of chronic inflammation on ISC/TA cell behaviour and differentiation remains incompletely understood in the setting of inflammatory bowel disease (IBD).

Methods: We integrated bulk RNA sequencing data from the largest IBD mucosal biopsy cohort to date ($n > 3,000$) with single-cell transcriptomic profiling of human colonic biopsies and functional assays using patient-derived intestinal organoids. Bioinformatic and experimental analyses focused on identifying transcriptional, cellular, and differentiation dynamics in ISC/TA populations across inflamed, non-inflamed, and healthy tissues.

Results: Chronic active inflammation in IBD was associated with a significant depletion of canonical LGR5 ISCs and a compensatory expansion of OLFM4-epithelial progenitors, consistent with reprogramming toward an inflammation-adapted epithelial repair phenotype. Both inflamed and adjacent non-inflamed IBD tissues exhibited persistent transcriptional changes in ISC/TA cells, distinct from healthy controls. Single-cell analysis revealed marked heterogeneity, including a novel inflammation-associated ISC/TA cluster enriched for immune signalling pathways and stress response genes. Pseudotime trajectory analysis demonstrated a shift in differentiation towards secretory (Paneth-like/deep crypt) cell lineages under inflammatory stress. Patient-derived organoid experiments recapitulated these lineage and gene expression changes, confirming the sustained impact of chronic inflammation on the epithelial regenerative compartment (Balasubramanian et al.; Patel et al.).

Discussion/Conclusion: Chronic inflammation in IBD drives dynamic and persistent reprogramming of intestinal stem and progenitor cells, promoting altered differentiation and the emergence of immune-responsive epithelial states. The IntestiDyn multi-omics dataset highlights the plasticity and adaptive mechanisms of the human intestinal epithelium in IBD, revealing targets for future therapeutic strategies aimed at promoting epithelial integrity and durable healing.

27. Baseline duodenal epithelial fitness and interferon tone stratify gluten-challenge mucosal injury in celiac disease

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Introduction: Histologic responses to controlled gluten re-exposure in celiac disease (CeD) vary widely, suggesting that baseline mucosal “setpoints” shape epithelial remodeling under inflammatory stress. We asked whether baseline duodenal transcriptomes capture epithelial fitness and interferon/antigen-processing programs that stratify subsequent gluten-challenge injury.

Methods: In a secondary analysis of the phase 2 CEC-3 trial, adults with CeD receiving placebo treatment during a standardized gluten challenge (3 g/day for 6 weeks) underwent paired duodenal biopsies ($n = 24$). Bulk RNA-seq from

PAXgene-fixed tissue was integrated with villus height-to-crypt depth ratio (VH:CrD). Participants were stratified by median post-challenge VH:CrD, and transcript-histology associations were additionally assessed across the full dataset using Spearman correlation.

Results: At baseline, individuals who preserved villus-crypt architecture after challenge showed an epithelial “resilience” program enriched for mitochondrial metabolism, lipid handling, and enterocyte differentiation (including HMGCS2, ACAA1/ACAT1, PLIN2, DGAT2). In contrast, higher baseline interferon-stimulated and antigen-processing transcripts (e.g., IRF9, GBP4, TAP1) were linked to greater architectural loss. Across all samples, a baseline resilience-module score correlated with concurrent VH:CrD ($\rho = 0.40$; $p = 9.9 \times 10^6$), with DGAT2 positively ($\rho = 0.47$; $p = 1.3 \times 10^7$) and TAP1 inversely ($\rho = -0.45$; $p = 3.9 \times 10^7$) associated. After challenge, the signature of preserved architecture was amplified with a stress-adaptation/protective module ($\rho = 0.52$; $p = 3.0 \times 10^9$), highlighting ENPP3, SELENOP, and VNN1 ($\rho = 0.48/0.53/0.56$).

Discussion/Conclusion: Baseline epithelial metabolic fitness and interferon/antigen-processing tone represent distinct transcriptomic states that precede and relate to gluten-challenge mucosal remodeling. A parsimonious DGAT2/TAP1-centered panel, complemented by stress-adaptation markers, may support risk stratification and mechanistically anchored pharmacodynamic readouts for mucosa-targeted therapies in CeD.

28. Pathogen-driven selection of HLA-DQ2 and HLA-DQ8: In silico evidence for predominant bacterial peptide presentation relevant to celiac disease

Aline Pesi (Mainz, DE), Detlef Schuppan (Mainz, DE)

Introduction: HLA-DQ2 and HLA-DQ8 are necessary but not sufficient risk alleles for celiac disease (CeD). Despite high carrier frequencies in Western populations, only a minority develops CeD, suggesting that DQ2/DQ8 may confer evolutionary advantages unrelated to gluten. We tested the hypothesis in silico that pathogen- and microbiota-driven selection favored DQ2/DQ8 variants optimized for presenting bacterial peptides, with gluten reactivity arising as a collateral effect.

Methods: We derived high-affinity 9-mer core binding motifs for HLA-DQ2.5 and HLA-DQ8.1 from NetMHCIIpan-based sequence logos and enumerated top-scoring binder-core sequences. Binder cores were queried against reference proteomes using BLASTP to identify naturally occurring exact and near-exact peptide matches. Taxonomic origin and sequence identity of hits were recorded and summarized across the highest-affinity binder sets.

Results: High-identity matches for top predicted DQ2/DQ8 binders were predominantly found in bacterial proteomes, spanning common tolerated gut commensals and opportunistic/pathogenic taxa (including Firmicutes, Escherichia coli, Pseudomonadota, Prevotella, Bacteroides, Akkermansia, and Clostridia). In contrast, only a small fraction of matches originated from fungal or helminth

proteomes. Notably, among the strongest predicted binders, we observed no 100% identity matches in the human proteome, consistent with selection against exceptionally strong self-peptide binding.

Discussion/Conclusion: These in silico data support a model in which HLA-DQ2/DQ8 evolved to efficiently present a broad bacterial peptide repertoire while minimizing high-avidity self-peptide presentation. In the intestinal context, tolerance to frequently presented bacterial antigens likely depends on antigen-presenting cell programming and regulatory CD4 T-cell responses, providing a conceptual bridge to antigen-specific tolerogenic interventions. We propose leveraging DQ2/DQ8-enriched bacterial-like binder cores as a rational starting space for DQ2/DQ8-restricted tolerogenic peptide candidates and pharmacodynamic readouts in CeD- an approach that can be tested experimentally in patient-derived T-cell systems.

29. *Cereus jamacaru* DC attenuates intestinal inflammation and modulates the immune response in a DSS-induced colitis model

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Introduction: *Cereus jamacaru* DC, known as mandacaru, is a cactus native to northeastern Brazil and traditionally used to treat inflammatory conditions such as sinusitis and bronchitis. It contains flavonoids such as isorhamnetin and kaempferol, which confer antioxidant and anti-inflammatory properties. The aim of this study was to evaluate the effect of a lyophilized hydroethanolic extract of *C. jamacaru* on dextran sodium sulfate (DSS)-induced intestinal inflammation in C57BL/6 mice

Methods: Colitis was induced by administering 3% DSS for 5 days, followed by a 5-day recovery period. Animals were treated daily with *C. jamacaru* (100 or 200 mg/kg) or left untreated (DSS group and healthy control group). Clinical progression was assessed using the Disease Activity Index (DAI), along with flow cytometry, RT-qPCR, histology (H&E), and immunofluorescence analyses.

Results: DAI progression showed a significant decrease in the groups treated with *C. jamacaru*, with reduced weight loss, diarrhea, and fecal bleeding. Scores indicated marked clinical improvement compared with the DSS group. Flow cytometry revealed that the extract reduced the proportion of Th1, Th2, Th17, Tc1, Tc2, and Tc17 cells, Ly6C monocytes, and mast cells, while increasing regulatory T cells (Tregs). Reduced expression of Cox-2, Il-17, Mip-2, Icam-1, and Il-6 was also observed, particularly at the 100 mg/kg dose, along with increased expression of Muc-1, Muc-3, and villin in both treatment groups. Histological analysis showed improved preservation of the colonic epithelium, with increased occludin expression and reduced Ki67 expression.

Discussion/Conclusion: These findings indicate that *Cereus jamacaru* exerts clinical, histological, and immunological anti-inflammatory effects and represents a promising therapeutic alternative for inflammatory bowel diseases.

30. Mucosa-associated microbiota signatures linked to postoperative recurrence in Crohn's disease

Alba Rodriguez Nogales (Granada, ES)

Introduction: Gut dysbiosis has been implicated in the pathogenesis of Crohn's disease and is thought to play a key role in the development of postoperative recurrence following ileocecal resection. This study prospectively evaluated intestinal microbiota composition and diversity in Crohn's disease patients after surgery to assess its predictive value for postoperative recurrence.

Methods: Fecal samples and biopsies from healthy and inflamed intestinal mucosa were collected from 52 patients undergoing ileocecal resection. Postoperative recurrence was defined as a Rutgeerts score \geq i2b. Microbiota composition was analyzed using 16S rRNA gene sequencing, including alpha and beta diversity assessments and correlation analyses with clinical variables.

Results: Patients who developed severe postoperative recurrence exhibited a mucosa-associated microbiota characterized by significantly reduced microbial diversity compared with patients in clinical remission. Microbial profiling revealed an enrichment of bacteria belonging to the phyla Bacillota and Pseudomonadota, along with a marked depletion of butyrate-producing bacteria and an increased presence of Fusobacteria. These alterations suggest a dysbiotic microbial signature associated with disease recurrence. Differences in microbial recolonization patterns after ileocecal resection highlight the role of specific bacterial taxa in driving postoperative recurrence.

Discussion/Conclusion: These findings support the potential use of microbiota-based biomarkers to identify patients at increased risk of recurrence and provide a rationale for targeted therapeutic strategies aimed at modulating the gut microbiota in the postoperative setting.

31. Targeting gut inflammation to limit colorectal cancer progression: Immunomodulatory and anti-proliferative effects of propyl-propane thiosulfonate (PTSO)

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Introduction: Colorectal cancer (CRC) is the third most commonly diagnosed and deadly cancer worldwide. An important factor is an exacerbated inflammatory response in the gut mucosa and it is associated with increased risk for CRC. The aim of study was to evaluate the impact of the organosulfur compound Propyl-Propane-Thiosulfonate (PTSO) from *Allium* spp., with reported anti-inflammatory properties, in the tumoral progression in a model of colitis-associated CRC (CAC).

Methods: C57Bl/6 female was pretreated with micro-encapsulated and free PTSO (1 mg/kg) for 2 weeks and then, CAC was induced by administration of azoxymethane followed by three cycles of dextran sulfate in drinking water (2%). PTSO treatment was maintained during all assay and a group treated with 5-fluorouracil (15 mg/kg) was used as control. Tumoral process was assessed using the disease activity index (DAI) and by colonoscopy. Moreover, inflammatory markers and immune populations in colon and mesenteric lymph nodes (MLNs) samples were analyzed by flow cytometry. Additionally, the immunomodulatory and antiproliferative/antitumoral properties of PTSO have been analyzed in vitro by using human colon cells lines (HCT-116, CACO-2, NCM-356) and co-culture models combining these cells lines with human immunity-cells (HMC-1.2).

Results: PTSO pretreatment reduced the macroscopic colonic inflammation, thus resulting in an amelioration of tumor development in CAC model. This was associated with a reduction of myeloid immune cell infiltration in the colonic mucosa, including macrophages (CD45+CD11b+Ly6G-MHCII-Ly6C-) and neutrophils (CD45+CD11b+Ly6G+). Moreover, PTSO increased lymphoid cells (CD3+), specifically Th cell (CD3+CD4+) population in MLNs. This effect was associated to an antiproliferative effect evidenced by a reduction of pSTAT3/STAT3 ratio in colon. In vitro studies revealed that PTSO supplementation reduced tumor cell proliferation and migration.

32. Parabacteroides goldsteinii mediates the antitumor effects of tigecycline in colorectal cancer under an obesity-associated microenvironment

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Introduction: Obesity and colorectal cancer (CRC) share alterations in cellular pathways and gut microbiota dysbiosis, which promote tumorigenesis via metabolite-mediated mechanisms. Preliminary results showed that tigecycline ameliorates CRC in mice, partly through enrichment of *Parabacteroides*, suggesting a protective role for this bacterium. This study aimed to evaluate the impact of

tigecycline and *Parabacteroides goldsteinii* on obesity- and CRC-associated dysbiosis and tumor development using humanized mouse models.

Methods: Tigecycline was first tested in a colitis-associated CRC model in HFD-fed obese C57BL/6 mice. Colitis-associated CRC (CAC) was induced with azoxymethane/DSS, and tigecycline (25 mg/kg/day) was administered for 7 weeks. Stool samples were analyzed by Illumina HiSeq to identify bacteria with therapeutic potential. Subsequently, humanized C57BL/6 mice were generated via fecal microbiota transplantation (FMT) from lean, obese, or CRC patients (n = 3–4) and CAC was induced. Mice received either *P. goldsteinii* (1×10^8 CFU/day) or vehicle for 7 weeks. Disease severity index (DAI) was assessed regularly, and colonoscopy and tumor burden, at sacrifice. In parallel, fecal metabolomes from lean, obese and CRC patients were tested in vitro for their effects on tumor (HCT116) and non-tumor (NCM356) cell proliferation.

Results: The tigecycline study confirmed an amelioration of tumor progression in HFD-fed mice, correlating with reduced inflammation (IL17, IL23, COX2) and proliferation (pSTAT3/STAT3, pAKT/AKT, and pCTNNB1/CTNNB1), accompanied by *P. goldsteinii* enrichment. In humanized models, CAC induction was aggravated in obese and CRC FMT mice, showing higher DAI and tumor burden than lean FMT mice. Administration of *P. goldsteinii* significantly reduced DAI and tumor burden in FMT obese mice, whereas no improvement was observed in CRC FMT mice. In vitro, fecal metabolomes from CRC patients increased proliferation of HCT116 cells, while metabolomes from lean individuals enhanced proliferation of non-tumor NCM356 cells.

Discussion/Conclusion: Obesity-associated dysbiosis plays a key role in CRC development, and *P. goldsteinii* supplementation hinders tumor progression, likely underlying the anti-tumor effects previously observed with tigecycline.

33. Development and validation of a novel patient-reported outcome for microscopic colitis – Microscopic Colitis Score (MCS)

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Introduction: Despite debilitating symptoms, no standardized disease severity index exists for microscopic colitis (MC). This gap hinders alignment with US Food and Drug Administration (FDA) and European Medicines Agency (EMA) standards, which emphasize the importance of patient-reported outcome measures (PROMs) in new therapy approval. This study aimed to validate the Microscopic Colitis Symptom Questionnaire (MCSQ) and develop the Microscopic Colitis Score (MCS), a novel disease severity index.

Methods: This prospective, multicenter study included 131 patients with biopsy-confirmed MC (67 remission, 64 active disease). Patients completed MCSQ and

HRQoL assessments (IBDQ-32, SHS) at baseline and follow-up. Clustering analysis systematically identified distinct disease severity groups. MCS was developed as a composite score derived from MCSQ.

Results: Factor analysis revealed a three-factor MCSQ model with good internal consistency (Cronbach's alpha = 0.88). Test-retest reliability (ICC = 0.88) and responsiveness to treatment ($p < 0.01$) of all MCSQ items were high. MCS, ranging from 0 (asymptomatic) to 15 (maximum symptoms), correlated strongly with HRQoL measures such as IBDQ-32 total score ($r_p = 0.78$), IBDQ-32 bowel symptoms ($r_p = -0.80$) and SHS bowel symptoms ($r_s = 0.69$). Receiver-operating characteristic curves indicated that MCS could accurately identify patients in remission (as per Hjortswang criteria; AUC = 0.85), as well as mild (AUC = 0.97), moderate (AUC = 0.93), or severe disease (AUC = 0.96).

Discussion/Conclusion: MCSQ and MCS are valid, reliable, and responsive tools that meet FDA and EMA standards. Both accurately reflect the diverse symptoms of MC. Compared to the binary Hjortswang criteria, MCS provides a nuanced evaluation of disease activity and holds promise for assessing therapeutic efficacy in future trials.

34. The modern method of differential diagnosis for patients with suspected colorectal cancer and inflammation colon

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Introduction: One of the main tasks of the doctor during the examination of a patient with suspected cancer of the colon, rectum and anal canal is to take the biopsy material as correctly as possible.

Despite the fact that this method is the most reliable and widespread throughout the world, it takes a long time to perform and obtain a result - up to 6 days, so an alternative or additional method has been developed and proposed that allows you to determine and differentiate the nature of the lesion during biopsy material collection, as the duration of the new study is 60 seconds.

Methods: This method is based on the difference in the concentration of intracellular trace elements in tumor, inflammatory and normal tissues of the subject being studied.

Results: The authors use the well-known phenomenon of the photo effect. Irradiation of a tissue sample with a mini- γ -quantum causes a loss of negative charge by cells, thus creating a photoelectric effect (Stoletov effect), which will be strictly individual for neoplastic, inflammatory and normal tissues. To differentiate various pathological formations, the authors measure the intensity of absorption of γ -quantum by cells, as well as the characteristic emission of trace elements present in the tissue.

Discussion/Conclusion: Thus, the key point of the presented method is to distinguish the concentration of trace elements in malignant, non-malignant, inflammatory and normal tissues. This circumstance can be explained by the complex and related function of the sodium pump in carcinogenesis.

We recommend the use of the express method of the mini- γ -quantum examination when taking biopsy material during the examination of patients with suspected colon, rectal or anal cancer for the differentiation of pathologically changed tissues.

35. Assuming overlapping genetic background in eosinophilic esophagitis and inflammatory bowel disease

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Introduction: The inflammatory bowel disease (IBD) and eosinophilic esophagitis (EoE) are increasingly recognized as linked, immune-mediated conditions, with studies showing significantly higher rates of EoE in IBD patients and vice versa, suggesting shared mechanisms like epithelial barrier dysfunction, disrupted gut microbiome, and exaggerated T-cell responses (especially Th2/IL-13), though research continues to clarify the exact bidirectional relationship and underlying causes. The thymic stromal lymphopoietin receptor (TSLPR) attracts major attention as there is evidence that the TSLP signalling pathway may contribute to the formation of perverted immune response in EoE, while exact polymorphisms and their roles have to be determined. In terms of IBD, the Th1 immune response is considered to greatly contribute to the pathogenesis of CD, whereas the Th2 immune mechanisms are considered to participate to UC. Moreover, some recent studies showed decreased mRNA expression levels of TSLP and a negative correlation between expression of TSLP and severity of UC disease. Therefore, we aimed on studying TSLPR genetic single nucleotide polymorphisms (SNP) in both EoE and IBD patients in attempt to clarify its possible role in their pathogenesis.

Methods: The study consists of two parts. As for the EoE part, we observed 75 school children with symptoms of food allergy (FA) based on clinical symptoms and prick skin testing as major criteria for FA. Based on questioning, clinical and laboratory observations, individuals were divided into following groups: 1st group included 68 FA patients with FA without EoE; 2nd group (7 patients, all males) – FA+EoE. EoE diagnosed based on symptoms and oesophageal endoscopy with biopsies (not less than 15 eosinophils per $\times 400$ hpf in at least one sample). In addition, 36 IBD patients (17 – CD, 19 – UC) participated in the study and formed study groups 3 and 4, respectively. IBDQ and CDAI were used to evaluate disease severity, IBD diagnosis according to ECCO Guidelines. Twenty-three practically healthy individuals formed the control group. TSLPR gene's single nucleotide polymorphism (rs36133495) was selected following literature search and studied in lymphocytes by PCR.

Results: The A-allele of the TSLPR gene was detected in 88.89% of group 1, 14.29% of group 2, 88.2% of group 3, 42.1% of group 4 and 91.30% of controls. Minor G-allele was found in 11.11% of group 1, 85.71% of group 2, 11.8% of group 3, 57.9% of group 4, and 8.70% of controls, respectively. No GG-genotype carriers observed in

control. The likelihood of EoE increases in G-allele carriers of the TSLPR gene (OR = 2.46; 95% CI: 0.43–6.16; $p < 0.001$). However, emphasizing IBD, TSLPR gene's polymorphism doesn't show remarkable association (OR close to 1.0 even in UC group), which may be explained by the fact that besides pro-inflammatory activity, TSLP may as well play an anti-inflammatory and tolerogenic roles. Moreover, there were statistically valid differences in genotypes and alleles between 3rd (CD) and 4th (UC) groups. The distribution pattern of SNP in studied population meets the Hardy-Weinberg equilibrium ($p > 0.05$) with a slight excess of heterozygosity, which generally does not disturb the population balance.

Discussion/Conclusion: TSLP, is mainly expressed by epithelial cells, especially those located in the gut. Some of the studies have tied the genetic variations of TSLP/TSLPR with a wide range of Th2 determined diseases, including EoE and IBD. This study shows that the particular rs36133495 (A/G) TSLPR gene polymorphism may be involved in the development of EoE and partially IBD, and requires further investigation emphasizing possibilities for associated treatment approaches. A/G SNP (rs36133495) in the TSLPR gene suggests a potential mechanism for the difference between CD and UC.

36. The crosstalk between eotaxin, eosinophil activation, and TGF- β signalling in chronic gut inflammation

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Introduction: Variety of gastrointestinal disorders associate with type 2 immune response and eosinophilic involvement include allergic vasculitis, EoE, eosinophilic gastroenteritis, carcinomatosis, drug-related conditions, inflammatory bowel disease IBD and others. The increased number of eosinophils is described in IBD, in both active disease, and in remission. In addition, a set of molecules participate in pathogenesis among them different chemokines, which form the largest subgroup of cytokines, consisting of about 45 currently identified in humans. Their main role is the recruitment and activation of leukocytes during the inflammatory process, additionally, they modulate various biological effects, such as T-lymphocyte activation, angiogenesis, and hematopoiesis via interaction with different receptors expressed on different types of leukocytes. Recent studies showed eosinophilic activation and accumulation in IBD potentially involving certain chemokines in the pathophysiology of IBD. Eotaxin, has no activity on neutrophils and monocytes but it does show a certain degree of chemotactic activity toward Th2 cells and basophils. The role of eosinophils in IBD was demonstrated in recent studies, alongside with the eotaxin's significance in selective recruitment of eosinophils into. Furthermore, dysregulation of TGF- β signalling is observed in IBD patients, too. TGF- β promotes intestinal fibrosis and tissue remodelling in IBD, making eosinophils key contributors to the chronic inflammation and scarring seen in IBD, though their exact function is complex, with evidence suggesting

roles in both pathology and resolution. Targeting eotaxin or eosinophils (like with anti-eotaxin-1 or anti-IL-5R drugs) shows promise for perspective treatment. Whereas there are multiple studies on the issue, present data is still confusing. Therefore, the aim of this study was to investigate the interplay of eotaxin, eosinophils and TGF- β in IBD.

Methods: Thirty-six IBD patients (17 – UC, 19 – CD) participated in the study. IBDQ and CDAI were used to evaluate disease severity. 24 and 12 were in remission or have active disease, respectively, 13 – receive corticosteroids. 30 practically healthy individuals formed control group. Cells subpopulations were determined in fresh blood samples in an automated analyser, serum quantitative assay for eotaxin by solid phase sandwich ELISA. Colonic biopsies (screening colonoscopies in practically healthy adults as controls) were used to immunohistochemically (double immunostaining) identify and quantify eosinophils, and TGF- β 1+ cells via immunofluorescence method in 10–20 high power fields of view in each sample using automated computer-assisted system.

Results: Significantly increased eotaxin levels ($p < 0.01-0.05$) were observed in both CD (301.2 ± 135.0 pg/ml) and UC patients (218.4 ± 142.5 pg/ml) when compared to controls (145.2 ± 112.7 pg/ml). Active IBD demonstrated higher eotaxin levels than patients with quiescent disease – 395.1 ± 172.3 pg/ml vs. 118.5 ± 63.7 pg/ml in CD and 289.5 ± 152.8 versus 143.6 ± 95.7 pg/ml in UC, respectively ($p < 0.05$). A significant dominance of eosinophils was observed in the colonic biopates in active CD patients (537.5 ± 913 cells per sq. mm) and UC (299.4 ± 45.1 cells per sq. mm) compared to controls (52.6 ± 7.4 cells per sq. mm), $p < 0.05$. However, eosinophils' number significantly ($p < 0.001-0.01$) dropped in remission (221.6 ± 31.1 and 72.1 ± 8.1 cells per sq. mm in CD and UC, respectively), still remaining slightly higher than in control. Ratio of eosinophils expressing TGF- β 1 dropped significantly in all IBD patients with lowest figures observed in active IBD (12%). In remission, number of TGF- β 1+ cells (48%) raised significantly closer to control (58%).

Discussion/Conclusion: This study shows that eotaxin is significantly increased in serum of patients with active IBD, suggesting that this cytokine may be responsible for the IBD activity. Previous studies have shown that TGF- β level is elevated in active IBD patients, but activation of TGF- β signalling is insufficient to suppress active IBD. Therefore, obtained data shows that eosinophils may play significant role in downregulating TGF- β release in IBD contributing into its pathogenesis via both pathways. These mechanisms may play the interconnecting role with other immune dependant GI conditions and beyond.

37. Intricate amalgamation of genetics, pro- and anti-inflammatory cytokines, and connexins in chronic intestinal inflammation

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Introduction: IBD is characterized by the functional impairment of intestinal epithelial cells concomitant with the infiltration of the lamina propria by inflammatory cells. Gap junction proteins (connexins) are high-resistance pathways that mediate intercellular coupling, allowing passive spread of ions and large molecules in the GI tract. Gap junction intercellular mechanism plays an important role in multiple normal and pathophysiological processes such as acute pancreatitis, cholestasis, diabetes, and nephritis. However, little is known about its role in the intestinal inflammation and some reports have suggested that several pro-inflammatory mediators are involved in its regulation, too. The close proximity of immunocompetent cells to the epithelium advocate a cross-talk between these cell types, which may execute directly via gap junctional channels or via mediators and exosomes. Taking into account known significance of several cytokines in IBD pathogenesis and their possible proximity to gap junction mechanism, we selected several target genes and hypothesized that C-590T polymorphism of IL-4 gene and 35delG polymorphism of Gap junction β -2 protein/connexin (GJB2) gene may have pathogenetic significance in chronic intestinal inflammation.

Methods: One hundred and two IBD patients participated in the study. Diagnosis and management provided according to ECCO Guidelines. Female - 31 (30.4%), male - 71 (69.6%), control group was formed by 40 practically healthy individuals (female - 17 (42.5%), $\chi^2 = 1.88$, $p > 0.05$, male - 23 (57.5%), $\chi^2 = 1.38$, $p > 0.05$). Levels of pro- and anti-inflammatory cytokines' production (by ELISA) statistically calculated compared to control group quartiles. 'Low' (LQ) was L-1 β < 23 pg/mL (lower quartile of control), TNF- α \leq 15 pg/mL, IL-4 \leq 4.95 pg/mL, IL-10 & IL-13 \leq 15 pg/ml & \leq 28 pg/ml, respectively. 'High' (HQ) was TNF- α > 32 pg/mL (upper quartile), IL-1 β \geq 60 pg/mL, IL-4 \geq 45 pg/mL, IL-10 & IL-13 \geq 25.96 pg/mL & \geq 38 pg/mL, respectively. Allelic and genotypes distributions of GJB2 (rs80338939) and IL-4 (rs2243250) mutations analysed in PCR.

Results: All observed polymorphisms fully adhered to the Hardy-Weinberg equilibrium in both the IBD and control groups. Homozygous GJB2 gene mutation (35delG) in control has frequency of 5.0%, whereas among IBD patients in every second person, by 20.58% more often in male, $\chi^2 = 38.32$, $p < 0.001$. The distribution of IL-4 (C-590T) genotypes between groups including gender stratification was similar. The presence of GJB2 mutation in haplotype, regardless of IL-4 (C-590T) genotypes, increases the likelihood of IBD (UC, CD) 7.5- and 15.0-fold (OR = 9.67, 95% CI: 2.13-43.9, $p < 0.001$ and OR = 19.67, 95% CI: 2.53-102.9, $p < 0.001$, respectively). Number of patients with LQ of TNF- α and IL-4 gene's CC/CT genotypes dominate over TT-genotype: 22.06%/26.47% vs. 4.41% ($\chi^2 = 34.0$, $p < 0.001$). The same trend registered for IL-1 β . Lower IL-1 β production found in 35delG genotype of CJB2 gene, compared to non-del-carriers by 30.35%: 63.16% vs. 32.81% $\chi^2 = 8.91$, $p = 0.003$).

Discussion/Conclusion: In this study the IL-4 gene's C-allele (CC/CT) associated with lower TNF- α ; high or normal IL-4, IL-10, IL-13 in 36delG-genotype of CJB2 gene. IL-4 hyperproduction in TT-genotype of IL-4 gene create predisposition for chronic intestinal inflammation. 35delG mutation of GJB2 gene is characterized by increased production of TNF- α , without significant growth of IL-1 β and hyper-

production of IL-4 backed by activity of IL-10, IL-13. The obtained results may be used for prognostic as well as treatment customization purposes, including anti-cytokine therapies. More data must be collected and further wider studies are needed for clinical implementations.

38. Inflammatory and pre-neoplastic lesions of the small bowel: Correlation between capsule endoscopy and double balloon enteroscopy

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Introduction: Inflammatory disorders of the small bowel comprise a heterogeneous group of diseases with variable endoscopic features in terms of location, extent, and morphology. This study aimed to assess the correlation between small-bowel capsule endoscopy (SBCE) and double-balloon enteroscopy (DBE) in the evaluation of inflammatory and pre-neoplastic lesions.

Methods: This retrospective, observational study included all SBCE and DBE procedures performed at an Italian tertiary referral center between 01/2018 and 06/2025, based on the availability of complete endoscopic data within the hospital PACS. Patients with at least one inflammatory SB mucosal lesion detected by SBCE or DBE were included. Lesions were defined according to the Delphi consensus for Crohn's disease capsule endoscopy and the I-CARE international consensus for atrophic patterns. SBCE was performed using PillCam SB3 or PillCam Crohn's, while Fujifilm DBE was carried out via anterograde or retrograde approach according to suspected lesion location. Diagnostic performance of SBCE was evaluated using DBE as the reference standard by calculating sensitivity, specificity, and Cohen's kappa.

Results: Overall, 295 procedures were analysed (195 SBCE and 100 DBE). Among these, 102 paired examinations performed within six months were available for comparison. SBCE showed high specificity (> 80%) for all inflammatory lesions. Sensitivity was high (> 80%) for aphthoid lesions and villous atrophy, moderate (~70%) for deep ulceration, stenosis, and scalloping, and lower for other lesions. Agreement was excellent ($\kappa \geq 0.6$) for stenosis, edema, and villous atrophy; good ($\kappa 0.4-0.6$) for deep ulceration, hyperemia, mucosal denudation, and mosaicism; and fair to weak for aphthous and superficial ulcerations. Low agreement for fold reduction and granular mucosa likely reflected the limited number of cases.

Discussion/Conclusion: SBCE showed good overall performance and agreement with DBE in detecting most inflammatory and pre-neoplastic SB lesions, including ulcerative-stenosing and atrophic patterns. Larger multicenter studies are needed to confirm these findings.

39. Shared genes, shared lesions: Mirror-image proximal esophageal inlet patches in monozygotic twins with familial colorectal and gastric cancer

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Introduction: Heterotopic gastric mucosa in the proximal esophagus (inlet patch, IP) is usually an incidental endoscopic finding, but familial clustering and its relationship with hereditary gastrointestinal malignancy remain poorly described.

Methods: We describe the clinical course and endoscopic findings in monozygotic twin sisters with a strong family history of gastrointestinal cancer, both previously operated for rectal carcinoma, who underwent screening upper gastrointestinal endoscopy.

Results: The patients are 59-year-old monozygotic twin sisters. The first twin underwent surgery with curative intent for rectal adenocarcinoma at the age of 51 years; the second twin had surgery for rectal adenocarcinoma at 58 years. Their mother died from rectal carcinoma at 46 years and their father from gastric carcinoma at 53 years. Following colorectal cancer treatment, both sisters were referred for upper gastrointestinal surveillance because of their family history. Esophagogastroduodenoscopy in each sister demonstrated a well-demarcated, salmon-coloured area of heterotopic mucosa in the proximal esophagus, consistent with IP, located at symmetrical positions (“mirror-image”) with respect to the esophageal circumference and distance from the incisors. There were no additional neoplastic lesions in the esophagus or stomach. The family history and endoscopic findings prompted consideration of an underlying hereditary cancer syndrome and referral for genetic counselling.

Discussion/Conclusion: This observation of mirror-image esophageal IPs in monozygotic twins with familial colorectal and gastric cancer supports the hypothesis that IP expression may be influenced by shared genetic and developmental factors. Recognition of such patterns may help to individualise endoscopic surveillance strategies in families with suspected hereditary gastrointestinal cancer.

40. Vitamin D deficiency and oral manifestations in Helicobacter pylori infection – Preliminary results of a cross-sectional study

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Introduction: Helicobacter pylori infection has been associated not only with gastric pathology but also with a variety of extra-gastric manifestations. Increasing attention has been directed toward its potential role in oral health disorders and systemic biochemical alterations. The aim of this study was to assess the prevalence of selected oral complications and their biochemical characteristics in H. pylori positive patients.

Methods: This preliminary analysis is based on a cross-sectional observational study. A total of 36 H. pylori-positive patients were included. The primary outcomes were the incidence of oral complications, specifically periodontitis and extraoral halitosis. Biochemical parameters were also analyzed, with particular focus on serum vitamin D levels, alkaline phosphatase levels (ALP), and total cholesterol levels. Vitamin D status was categorized as severe deficiency (< 30 nmol/L), moderate deficiency (30–50 nmol/L), and mild insufficiency (50–75 nmol/L).

Results: Biochemical analysis revealed significantly reduced serum vitamin D levels, with (25/36 = 69%) patients classified as vitamin D deficiency, including (1/36 = 0.02%) with severe deficiency, (10/36 = 27%) with moderate deficiency, and (14/36 = 38%) with mild insufficiency. In addition, ALP levels were reduced in (11/36 = 30%), while total cholesterol levels were elevated in (28/36 = 77%) within the study population. Biochemical samples were collected evenly throughout the entire year, ensuring representation of all four seasons, and none of the patients were receiving vitamin D supplementation at the time of the study.

Discussion/Conclusion: The findings suggest a potential association between H. pylori infection, oral complications, and systemic biochemical alterations. Vitamin D deficiency, along with reduced ALP activity and increased total cholesterol levels, may contribute to inflammatory processes affecting periodontal tissues and oral-related halitosis. These observations support the concept of H. pylori infection as a systemic condition with extra-gastric implications. This cross-sectional study indicates a high burden of oral complications and notable biochemical abnormalities in H. pylori-positive patients. However, the absence of an H. pylori-negative control group represents a limitation. Further studies including a well-defined H. pylori-negative comparison group are necessary to confirm these associations and better elucidate potential causal relationships.

41.A2 beta casein milk is tolerated in the majority of patients with milk-induced eosinophilic esophagitis

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Introduction: Eosinophilic esophagitis (EoE) is a food allergen driven chronic T2 inflammatory disorder of the esophagus with milk being the most frequently identified food culprit. It remains elusive whether changes in dairy production are associated with the development of EoE. A dominant point mutation in beta casein occurred in ancestors to modern type cattle with a change from A2 milk (traditional non-mutated beta casein) to A1/A2 milk (at least one point mutation). Nothing is known about the allergenicity of standard vs. A2 milk and its implications in EoE pathogenesis.

Methods: This was an analysis of Swiss EoE cohort patients with proven milk-induced EoE who underwent a 3-month challenge with commercially available A2 milk (300 mL daily). EoE-activity was assessed before vs. after. Local responses to standard and A2 milk were analyzed using ex vivo allergen-stimulation models.

Results: Eleven patients with proven milk-induced EoE (6 males, median age 36.7 years) underwent A2 milk exposure (median time 13.9 weeks, IQR, 10.3–15.5). Clinical and histological remission was maintained in 7 patients (63.6%), while relapse occurred in 4 patients (36.4%). At 12 weeks of A2 exposure, median peak eosinophil counts did not increase (0 vs. 0 eos/hpf). Milk-induced IL-5 secretion was significantly attenuated by A2 milk in an ex vivo model using freshly collected esophageal biopsies, but not in peripheral blood mononuclear cells.

Discussion/Conclusion: Our study is the first to identify not only a food category but also a specific food protein as a potential allergen involved in the pathogenesis of EoE. Importantly, the finding that most patients with milk-induced EoE tolerate traditional A2 β -casein milk further emphasizes β -casein as a possible key driver of disease pathogenesis.

42. Voclosporin in experimental colitis: A novel therapeutic approach to modulating gastrointestinal inflammation

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Introduction: Chronic gastrointestinal inflammation is a critical driver in the development of colitis-associated neoplasia. Voclosporin, a novel calcineurin inhibitor with a structural similarity to cyclosporine A, has demonstrated improved efficacy and tolerability in conditions such as lupus nephritis. Our study evaluates the therapeutic potential of voclosporin in IBD by analyzing its ability to suppress pro-inflammatory signaling pathways that facilitate long-term malignant transformation.

Methods: Therapeutic effects were assessed in vivo using a hapten-induced oxazolone colitis model. Analyses included mini-endoscopy, histology, multiphoton endomicroscopy, flow cytometry, and immunofluorescence staining. Additionally, the in vitro molecular effects of voclosporin on ITK activation and T cell signaling LCK, ZAP70 were investigated in human PBMCs.

Results: Voclosporin treatment effectively suppressed established experimental colitis. It significantly reduced the activation of ITK, a key driver of intestinal inflammation. In human PBMCs, voclosporin decreased the protein expression of the kinases ITK, LCK, and ZAP70. Notably, voclosporin exhibited superior efficacy in reducing T cell activity compared to cyclosporine A, suggesting a more potent modulation of the inflammatory environment.

Discussion/Conclusion: Voclosporin represents a promising novel therapeutic approach for acute and chronic intestinal inflammation. By efficiently blocking

the ITK signaling pathway, voclosporin not only provides a potent tool for acute disease management but may also play a crucial role in the chemoprevention of inflammation-associated gastrointestinal neoplasia.

43. Endoscopic resection of upper gastrointestinal subepithelial tumours: Our clinical experience and results

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Introduction: Upper gastrointestinal subepithelial tumours (SETs) are generally asymptomatic and clinically insignificant and have malign, borderline and benign variants. In advanced endoscopic procedures, histopathological diagnosis and endoscopic resection are possible and feasible. In this study, we examined our approach to upper gastrointestinal subepithelial tumours and our clinical results.

Methods: Adult patients who applied to Surgical Endoscopy unit between January 2024 and January 2025 were included in the study. The patients' files and final histopathological diagnoses were recorded and analysed retrospectively for this single-center study. SET lesion lower than 30 mm and the lesion whose endoscopic submucosal dissection attempt was included in the study

Results: The total of 8 patients were four female (50%) and four male (50%), aged 31–66 years (median, 53 years). The tumoral lesions were located 4 (50%) patients in esophagus, 3 (37.5%) patients in stomach and one (12.5%) patient in duodenum and their diameter ranged from 5 to 30 mm (median, 14 mm). Post-interventional no complications or abdominal symptoms were encountered.

Discussion/Conclusion: in early follow-ups for six months, no recurrence was observed. Our experiences together with literature reported here, indicated endoscopic resection is a safe and effective method of treatment for most patients with upper gastrointestinal SETs.

44. Prevalence of perfusion disorders in the pulmonary circulation in patients with ulcerative colitis requiring hospitalization in the last 24 months

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Introduction: Inflammatory bowel disease (IBD) is associated with a 2–3-fold risk of venous thromboembolic complications. This risk is further increased by disease flare. However, there is a lack of studies systematically searching for these events and identifying other risk factors in IBD patients.

Methods: The aim of a prospective cohort study was to evaluate the occurrence of perfusion disorders in the pulmonary circulation in patients with acute severe ulcerative colitis (ASUC). A cohort of patients from a single tertiary IBD centre hospitalized with an episode of ASUC was recruited from January 2023 to April 2025. Ventilation-perfusion (V/P) SPECT was performed in all patients after hospital discharge. In case of detection of primary perfusion defect, anticoagulation treatment was initiated in patients not yet anticoagulated.

Results: Fifty-four patients with relapse of UC requiring hospital stay due to disease activity in the past 24 months in whom V/P SPECT was performed were identified. None of the patients had clinical symptoms of pulmonary embolism. Mean age of the cohort was 37.0 ± 13.4 years (min-max 19-76 years). Of them 53.7% (29/54) were males. Mean disease duration at the time of V/P SPECT was 7.1 ± 7.1 years (1-35 years). Disease extension included 25.9% patients with left-sided colitis and 74.1% with pancolitis. Active smoking was present in 13.0% of patients and history of using JAK inhibitors was positive in 63.0% of patients. Mean time elapsed between last hospital stay due to UC activity and V/P SPECT was 239.6 ± 216.4 days (12-745). Presence of a perfusion defect was found in 16.7% (9/54) of patients. There were no significant differences in baseline patient and disease characteristics including smoking and JAKi history between positive and negative group. None of the positive patients had previous history of known thrombotic event.

Discussion/Conclusion: We have identified 16.7% of patients with severe UC relapse in the past 2 years to have signs of perfusion defect on V/P SPECT warranting further research to identify potential risk factors and probability of developing perfusion disorders of pulmonary circulation in patients with ASUC.

45. Short-term efficacy of advanced therapy plus CurQD in patients with ulcerative colitis

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Introduction: Biologic and small-molecule therapies are established treatments for active ulcerative colitis (UC), yet many patients experience incomplete or lost response. Adjunctive approaches such as CurQD, a combination of curcumin and QingDai, may offer additional therapeutic benefit.

Methods: This prospective single-centre cohort study evaluated the week-12 efficacy of adding CurQD to advanced therapy in active UC compared with advanced therapy alone. Patients receiving vedolizumab (n = 17), infliximab (n = 1), ustekinumab (n = 1), mirikizumab (n = 8), or upadacitinib (n = 8) were enrolled between June 2023 and June 2025 and treated with CurQD using the “Orange protocol.” CurQD patients were matched 1:2 to a retrospective control group by

age, sex, treatment class, and baseline corticosteroid use. Disease activity was assessed using SCCAI, Mayo endoscopic subscore, and faecal calprotectin. Clinical response was defined as a ≥ 3 -point SCCAI decrease, clinical remission as SCCAI 0–2, endoscopic response as ≥ 1 -point Mayo improvement, endoscopic remission as Mayo 0, laboratory response as $\geq 50\%$ calprotectin reduction, and laboratory remission as $< 100 \mu\text{g/g}$.

Results: Thirty-five CurQD patients (mean age 36.9 ± 11.3 years; 65.7% female) were compared with 70 matched controls (37.0 ± 10.8 years; 65.7% female). Baseline disease duration was similar, and most patients in both groups had extensive colitis and Mayo 3 endoscopic severity. At week 12, CurQD produced significantly higher laboratory response (80.0% vs. 55.7%, $p = 0.02$) and numerically higher laboratory remission (65.7% vs. 48.6%, $p = 0.15$) than controls. Clinical response was identical between groups (68.6% in each group, $p = 1.00$), while clinical remission was numerically higher with CurQD (62.9% vs. 54.3%, $p = 0.53$). There was no difference in endoscopic response (65.7% vs. 65.7%, $p = 1.00$) and remission (31.4% vs. 37.1%, $p = 0.67$) between the CurQD cohort and controls. Corticosteroid use declined from 34.3% to 20.0% in the CurQD cohort and from 47.1% to 24.3% in controls.

Subgroup analyses showed the strongest effects with upadacitinib plus CurQD: clinical remission 87.5% (vs. 62.5% in controls), laboratory remission 75.0% (vs. 37.5%), and endoscopic remission 50.0% (vs. 25.0%). Safety of the CurQD cohort was favourable, with one case of reversible hair loss and one discontinuation due to pregnancy.

Discussion/Conclusion: CurQD added to advanced therapy was safe and associated with improved biochemical response, numerically higher clinical remission, and meaningful corticosteroid sparing at 12 weeks. Endoscopic outcomes were comparable. The greatest benefit appeared in combination with upadacitinib. More controlled trials are needed to confirm these findings.

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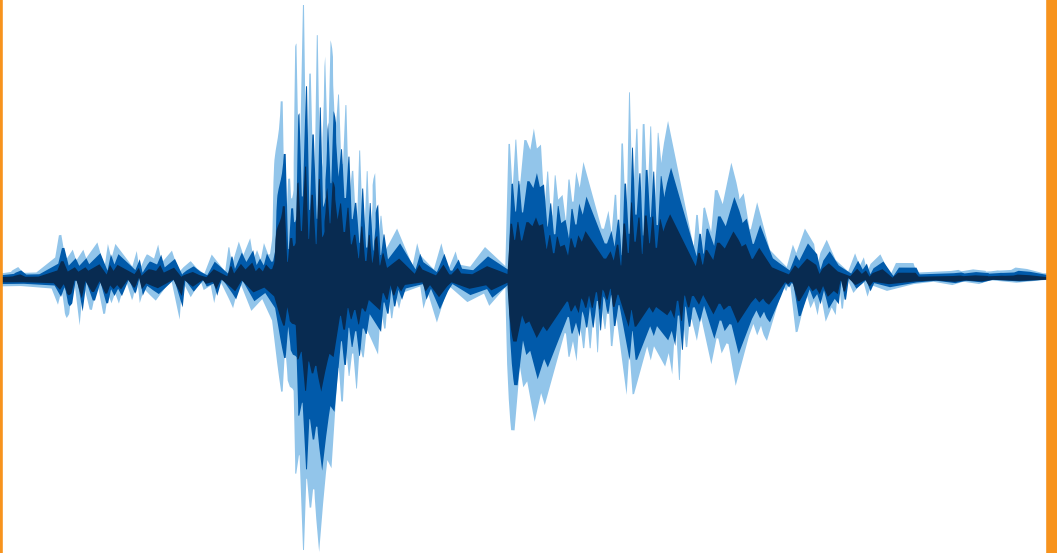
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