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GASTROINTESTINAL INFLAMMATION AND NEOPLASIA

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Refining Diagnosis, Monitoring, and Treatment Across Immune-Mediated GI Disease

by eickhoff kommunikation GmbH

Leading European and international experts gathered in Warsaw for the international Falk Foundation Symposium, *Gastrointestinal Inflammation and Neoplasia*, to examine the epidemiology, pathophysiology, diagnosis and management of a broad spectrum of immune-mediated gastrointestinal diseases, including eosinophilic esophagitis (EoE), inflammatory bowel disease (IBD), microscopic colitis, celiac disease, as well as selected gastrointestinal neoplasias. A consistent theme throughout the two-day meeting was the move toward more structured, personalized care, with objective endpoints replacing symptom-based decisions and emerging tools offering better discrimination of disease activity and risk of progression.

Monitoring EoE: Looking beyond symptoms

Prof. Ulrike von Arnim (Magdeburg, Germany) reminded delegates that EoE is a clinico-pathological entity in which clinical and histological findings must be interpreted together. Diagnosis requires symptoms of esophageal dysfunction, eosinophilic infiltration of the epithelium (≥ 15 eosinophils per high-power field, or ≥ 60 eos/mm2), and exclusion of other local or systemic causes [1]. Symptoms shift with age, from feeding intolerance and failure to thrive in infants to dysphagia and food impaction in adults. Allergic comorbidities are present in 60–80% of patients [2], so a thorough dysphagia history and review of atopic comorbidities are essential at presentation. Prof. von Arnim recommended consistent use of the EREFS scoring system at endoscopy (range 0–9; score > 2 indicates active disease) [3].

Its implementation has reduced reports of “normal-appearing” esophagus in active EoE from 21% to 7% of cases, although 11% of active cases still present with macroscopically normal endoscopy, particularly younger patients with atypical symptoms, in whom biopsies should be taken regardless of endoscopic appearance [4]. Routine use of the EoE Histology Scoring System (EoE HSS) was also advocated as it captures basal zone hyperplasia, dilated intercellular spaces and lamina propria fibrosis alongside eosinophil count [5]. Symptoms correlate only moderately with histological activity: More than one-third of patients in clinical remission are not in endoscopic or histological remission [6]. Esophagogastroduodenoscopy (EGD) with biopsy therefore remains the gold standard, as no minimally invasive biomarker is currently suitable for routine monitoring. The proposed

algorithm calls for endoscopic and histological reassessment 8–12 weeks after induction or major treatment change; stable, stricture-free patients can be followed every 12–24 months. A Swiss cohort of around 160 adults on topical corticosteroid maintenance showed significantly fewer strictures in those followed within 18 months [7], reinforcing the case for structured monitoring.

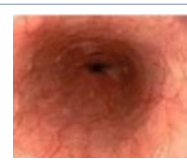
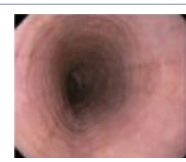
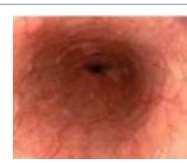
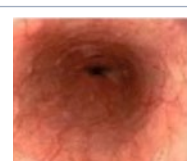
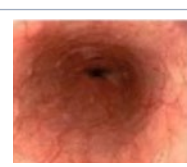
	Grade 0	Grade 1	Grade 2	Grade 3
Edema (loss of vascular markings)	Distinct vascularity	Decreased or absent		
Rings (trachealization)	 None	 Mild (ridges)	Moderate (distinct rings)	Severe (not pass scope)
Exudate (white plaques)	 None	Mild (≤ 10 % surface area)	Severe (> 10 % surface area)	
Furrows (vertical lines)	 None	Mild	Severe (depth)	
Stricture	 Absent	Present		

Figure 1: EREFS endoscopic reference score for EoE.
Standardized assessment of edema, rings, exudates, furrows and strictures (range 0–9); a score above 2 indicates active disease. Use of EREFS has reduced reports of "normal-appearing" esophagus in active EoE from 21% to 7%. Modified from [3].

IBD: Treatment goals in 2026

Prof. Alessandro Armuzzi (Milan, Italy) outlined how treatment targets in inflammatory bowel disease (IBD) have shifted from symptom control alone to composite, objective endpoints—including biomarker targets, endoscopic, histological, and transmural healing, with deep remission now the most desired outcome. No single biomarker reliably predicts disease progression or response to therapy, and no fixed algorithm guides treatment adjustment when targets are not met.

Current practice is based on the STRIDE-II consensus framework, which stratifies patient risk and defines short-, intermediate- and long-term targets [8]. Symptoms remain the most patient-relevant metric but correlate only moderately with objective disease measures, particularly in Crohn's disease (CD). Fecal calprotectin and CRP remain the most-used biomarkers; calprotectin correlates reasonably well with endoscopic activity, more reliably in colonic than small-bowel disease, and a treat-to-biomarker strategy outperformed a symptom-based strategy at one year in CD (CALM) [9]. Mucosal healing is associated with steroid-free remission, lower rates of surgery and hospitalization, and improved quality of life in both ulcerative colitis (UC) and CD [10,11].

Even with complete mucosal healing, around one in three patients still reports moderate disability, indicating that mucosal healing is the best available target, but not a complete

answer. Histological and transmural healing are emerging as adjunctive targets. The ongoing VERDICT trial in UC compares treat-to-symptoms, treat-to-endoscopy and treat-to-histology strategies [12], while VECTORS is evaluating intestinal ultrasound-guided transmural healing as a basis for therapy adjustment in CD [13]. Prof. Armuzzi expects both endpoints to be incorporated in the next STRIDE update. In advanced CD, only around 30% of patients achieve mucosal healing over a period of 8–10 years, supporting a proposed "minimal disease activity" concept for patients unlikely to reach complete remission. Surgery should be regarded as a legitimate therapeutic option rather than a failure [14]. The overall principle was summarized as "feel well, heal well, avoid damage."

Celiac disease: Modern strategies to improve and monitor adherence to lifelong diet

Dr. Luca Elli (Milan, Italy) discussed how current monitoring tools for celiac disease—serology, histology, and patient questionnaires—are all inadequate for the long-term monitoring phase, which can span decades. The goals of a gluten-free diet are symptom remission, antibody normalization and complete mucosal healing, alongside prevention of complications such as enteropathy-associated T-cell lymphoma [15]. Strict adherence is genuinely difficult: Gluten is widespread, up to one-third of foods labeled gluten-free still contain traces, and social situations make compliance especially hard.

Monitoring must distinguish two separate questions: Whether the patient is adhering to a gluten-free diet, and whether the celiac disease is active. Importantly, the two do not always correlate. Some patients adhere strictly yet show active disease, while others ingest gluten without symptoms and show normal duodenal histology with serology indistinguishable from fully adherent patients [16].

Dr. Elli highlighted gluten immunogenic peptides (GIP) measured in urine or stool as the only objective tool that correlates with mucosal atrophy [17,18]. Urine detects exposure within hours; stool over several days. Gluten has been detected in stool in over 40% of adult patients reporting a gluten-free diet [20], and three positive urine tests are associated with a high rate of duodenal atrophy [21]. Around 25% of patients with a positive GIP test are asymptomatic. Anti-tissue transglutaminase antibodies, while excellent for diagnosis, lose much of their value once a patient is on a gluten-free diet: A negative result does not confirm adherence, normalization is slow, and the timing of repeat testing is poorly defined. Routine duodenal biopsy is similarly problematic, as mucosal recovery can take years, and adherent symptomatic patients can still show atrophic lesions [19]. Personalized monitoring, with biomarkers chosen according to time since diagnosis, is now essential.

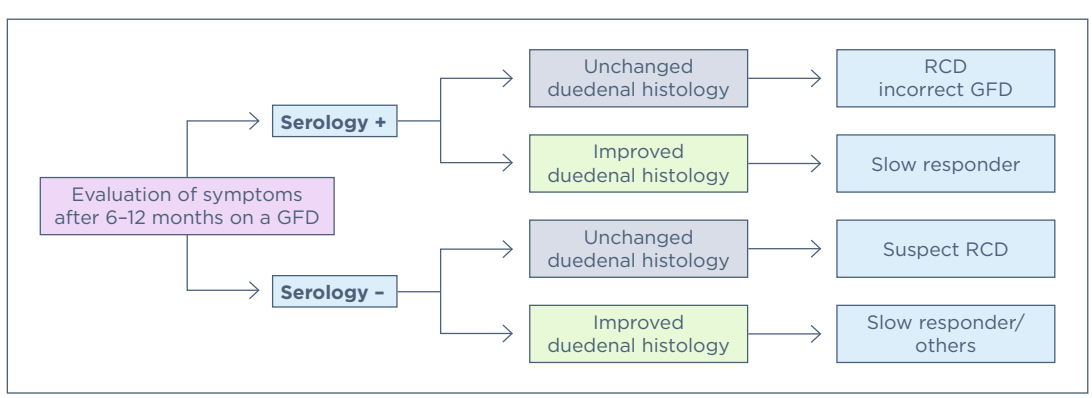


Figure 2: How we currently monitor and categorize response to treatment. Modified from [22, 23].

Immune-mediated gastrointestinal disorders during pregnancy and lactation

Prof. Uma Mahadevan (San Francisco, USA) presented the 2025 Global Consensus Consortium recommendations on pregnancy and lactation in IBD [24]. Active disease, not therapy, is the principal threat to the pregnancy, and withdrawing treatment out of caution is usually suboptimal. New IBD diagnoses during pregnancy are almost always ulcerative colitis. Monoclonal antibodies are too large to cross the placenta passively; instead, they are transferred actively via the Fc receptor, which develops around gestational weeks 12–14, after the bulk of organogenesis has occurred. This timing explains why biologics have not been associated with an increased risk of congenital anomalies. Small molecules cross the placenta during organogenesis and therefore warrant greater caution.

Practical recommendations from the 2025 Global Consensus include continuing all biologics (anti-TNFs, anti-integrins, anti-IL-12/23, anti-IL-23) and thiopurines throughout pregnancy

and lactation; avoiding small molecules unless no other option, with washout periods before conception of 3 months for ozanimod, 1–2 weeks for etrasimod and at least 4 weeks for tofacitinib and upadacitinib; and discontinuing methotrexate at least one month before conception as it is teratogenic.
Preliminary human data on JAK inhibitors in 55 exposed pregnancies showed no birth defects, although numbers remain insufficient for safety conclusions [25]. All IBD pregnancies should be followed as high-risk, with low-dose acetylsalicylic acid (150 mg) initiated at weeks 12–16 to reduce preterm preeclampsia [26]. Monoclonal antibody transfer to breast milk results in negligible infant exposure (~0.005% of maternal serum concentration), so breastfeeding is safe on 5-ASAs, sulfasalazine, thiopurines, anti-TNFs, anti-integrins and anti-IL-23s; small molecules should be avoided. All inactivated infant vaccines should be given on schedule, as should live vaccines, with the single exception of Bacillus Calmette-Guérin (BCG), which should be deferred to 6 months (or until biologic levels are undetectable). Rotavirus is now considered safe regardless of biologic exposure [27].

Colorectal cancer screening and surveillance by FIT

Prof. Antoni Castells (Barcelona, Spain) presented the 10-year results of the COLONPREV trial, comparing single-examination colonoscopy with biennial fecal immunochemical testing (FIT) for population-based colorectal cancer (CRC) screening [28]. The pragmatic non-inferiority trial randomized men and women aged 50–69 years across 15 Spanish tertiary hospitals, with crossover permitted to capture real-world participation. In the intention-to-screen analysis, FIT was non-inferior to colonoscopy for CRC mortality and incidence at 10 years, confirming for the first time in a head-to-head randomized setting that the two strategies are clinically equivalent at the population level. Importantly, participation was significantly higher in the FIT arm (~40%) than in the colonoscopy arm (~34%), an important practical advantage.
The findings sit alongside the Nordic NordICC trial, which showed that screening colonoscopy reduces CRC incidence but did not significantly reduce CRC-related mortality in intention-to-screen analysis [29]. For post-polypectomy surveillance, FIT may safely reduce the number of follow-up colonoscopies in low-risk individuals. A UK study of around 60,000 patients due for 3-year surveillance found that FIT (cut-off 40 µg/g) at years 1–3 could reduce colonoscopy demand by 70% but missed 40% of cancers and 70% of advanced adenomas [30].
The Dutch DEMOCRACY study compared FIT, stool DNA and a combined DNA+FIT test i n around 3,400 post-polypectomy patients. Using the ASCAP simulation model, it found no difference in CRC incidence or mortality versus colonoscopy, with 40–50% fewer colonoscopies required in the FIT arm. The updated ESGE guidelines now permit return of low-risk individuals (up to four small/non-advanced adenomas, or non-advanced serrated lesions) to FIT-based screening rather than ongoing surveillance colonoscopy [31]. However, the most pressing programmatic challenge remains low participation in CRC screening programs across Europe.

Early-onset colorectal cancer

Prof. Willem Bemelman (Amsterdam, The Netherlands) addressed the rising worldwide incidence of early-onset colorectal cancer (CRC), defined as diagnosis before the age 50 [32]. Most early-onset CRCs occur on the left side of the colon, are diagnosed at an advanced stage due to patient and clinician delays and exhibit more aggressive histology with a higher proportion of mucinous and signet-ring cell carcinomas. Despite this, overall survival is similar to late-onset disease because younger patients tolerate more intensive treatment. Etiology mirrors population-level trends, including rising rates of obesity and type 2 diabetes, sedentary behavior, high sugar intake, smoking, alcohol consumption, and processed food consumption.
Adipose tissue is increasingly viewed as an active organ driving chronic inflammation and microbiome alteration, both plausibly contributing to carcinogenesis [33]. Non-modifiable risk factors include genetic syndromes (Lynch syndrome, familial adenomatous polyposis) and IBD with extensive colonic involvement or primary sclerosing cholangitis. Around 16% of early-onset cases are linked to a hereditary syndrome and approximately 25% have a positive family history [34].
Surgical management follows the principles of late-onset CRC but emphasizes organ and stoma preservation given patients' age and life expectancy. Anastomotic leakage after total mesorectal excision has historically resulted in a permanent stoma in around 20% of cases, but Prof. Bemelman's team now successfully manages over 90% of acute leaks endoscopically using vacuum therapy, substantially reducing permanent stoma rates [35].

For early rectal cancer, options range from endoscopic submucosal dissection or transanal local excision up to total mesorectal excision, while locally advanced tumors are managed with neoadjuvant chemoradiotherapy. Patients with complete pathological response can be considered for watch-and-wait in selected cases [36]. The practical message is to lower the threshold for colonoscopy in any symptomatic patient under 50 and discuss every early-onset case in a multidisciplinary team. In the United States, the recommended screening age has already been lowered to 45 years on this basis [37].

Impaired coagulation: What gastroenterologists need to know

Prof. Jeanin van Hooft (Leiden, The Netherlands) presented a practical framework for managing antiplatelets and anticoagulants around endoscopic procedures, based on the 2021 joint European and British guidelines [38]. Each management decision balances procedure-specific bleeding risk against patient-specific thrombotic risk; stopping anticoagulation is not automatically a safe option. Procedures carrying a low bleeding risk (<1% bleeding risk) include diagnostic endoscopy, endoscopic retrograde cholangiopancreatography with biliary stenting, and enteral stenting. High-risk procedures include polypectomy, endoscopic submucosal dissection, and stricture dilation.

Patient-specific thrombotic risk varies substantially. Discontinuing antiplatelets for one week carries a substantial risk of stent thrombosis in patients with a recent drug-eluting coronary stent and warrants consultation with cardiology. Stopping anticoagulation for a week in a patient with a mechanical heart valve confers up to 4% thrombotic risk, with similar concern for recent venous thromboembolism. In contrast, anticoagulation for primary prevention can usually be paused with very low thrombotic risk. Acetylsalicylic acid can generally be continued across procedures. For clopidogrel, prasugrel and ticagrelor, the decision to discontinue depends on the patient's thrombotic risk category and the procedure's bleeding risk.

Direct oral anticoagulants (DOACs) have largely replaced warfarin and require planning that accounts for renal function: Apixaban can be stopped 2 days before a high-risk procedure, while dabigatran may require a 4-day hold in the setting of impaired renal function. For low-risk procedures, stopping one day before is generally sufficient. In acute GI bleeding, platelet transfusion is the primary intervention to consider for patients on antiplatelets, while vitamin K antagonists may be reversed with prothrombin complex concentrate, fresh frozen plasma or vitamin K. With DOACs, given their short half-lives proceeding without specific reversal agents is often reasonable in this setting. Common practice is to restart DOACs after polypectomy at 24 hours, extending to 48 hours for higher-risk procedures [38].

State of the art: Management of antibiotic-associated colitis

Prof. Christoph Högenauer (Graz, Austria) provided an update on antibiotic-associated colitis. Antibiotic-associated diarrhea occurs during or within two months of initiating antibiotic therapy and is usually mild and self-limiting. Antibiotic-associated colitis accounts for 10–20% of cases and is most commonly caused by *Clostridioides difficile*, a gram-positive, spore-forming, toxin-producing organism [39]. Highest-risk antibiotics include broad-spectrum cephalosporins, carbapenems, fluoroquinolones, clindamycin, and broad-spectrum penicillins. Risk increases with treatment duration and combination antibiotic regimens. Asymptomatic *C. difficile* carriage is common, so testing strategy is important.

Prof. Högenauer endorsed the two-step approach: a high-sensitivity test (glutamate dehydrogenase antigen) followed, if positive, by a high-specificity toxin enzyme immunoassay. Polymerase chain reaction (PCR) is a third option when suspicion remains high [40].

Three agents are available: oral vancomycin (125 mg 4 times daily for 10 days; preferred in pregnancy), oral fidaxomicin (200 mg twice daily), and metronidazole (500 mg 3 times daily). However, several guidelines no longer recommend metronidazole except in mild disease [41]. Severe and fulminant *C. difficile* infection (CDI) warrants high-dose oral vancomycin (500 mg 4 times daily) plus IV metronidazole, with vancomycin enemas when ileus prevents oral delivery.

Recurrence is the main challenge, occurring in approximately 25% of patients after a first episode and 50% after a second. First recurrence is best managed with extended pulse-and-taper vancomycin or extended fidaxomicin, with fidaxomicin preferred when recurrence risk is high [42]. For multiply-recurrent CDI, fecal microbiota transplantation has transformed practice, with cure rates of approximately 90% [43]. Two FDA-approved microbiota-based therapeutics are now available in the US: oral SER-109 [44] and rectal RBX2660 [45]. Antibiotic stewardship remains the strongest approach to prevention: A Scottish antimicrobial stewardship program targeting the '4C' antibiotics reduced CDI incidence by approximately 50% [46].

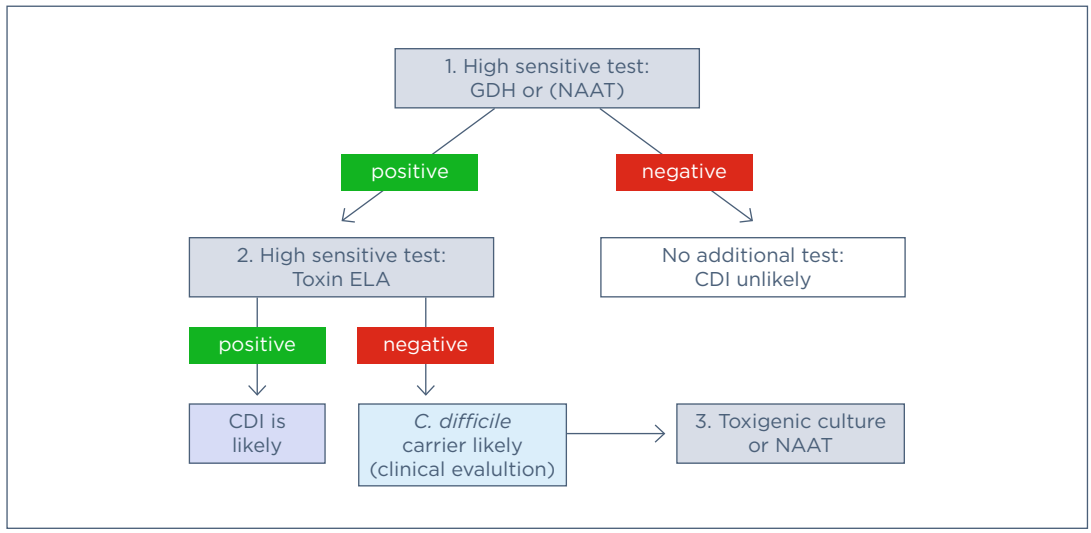


Figure 3: CDI: Two-test strategy. Modified from [40].

Poster competition

All three winning posters addressed eosinophilic esophagitis:

- **1st prize:** Dr. Rohith Kumar (Stevenage, UK). **Can capsule sponge sampling replace gastroscopy for monitoring eosinophilic oesophagitis? A single-centre prospective study**
- **2nd prize:** Dr. Elena Grueso-Navarro (Tomelloso, Spain). **Evolution of histologic and endoscopic assessment of eosinophilic esophagitis in Europe and its effects on diagnostic delay: Data from the EUREOS EoE CONNECT registry**
- **3rd prize:** Dr. William Fong (Nottingham, UK), **Real-world effectiveness and safety of budesonide in adult eosinophilic oesophagitis: A UK tertiary-centre cohort**

The winning study by Dr. Kumar and colleagues prospectively assessed whether minimally invasive capsule sponge (CS) cytology could replace repeat gastroscopy for monitoring disease activity in non-stenotic adults with EoE, recruiting patients at a general hospital between July 2024 and December 2025. Among 112 paired same-day CS and gastroscopy episodes (71.4% male, median age 48 years), CS distinguished active disease (≥ 15 eosinophils per high-power field (eos/HPF) from remission with a sensitivity of 82.5%, specificity of 88.9%, PPV 80.5%, NPV 90.1% and overall accuracy of 86.6%, with substantial agreement against histology (Cohen's $\kappa = 0.71$; $p < 0.01$). The authors concluded that CS sampling may reduce the need for routine gastroscopy in selected patients and provides a platform for future biomarker development beyond eosinophil counts alone.

Interview with Professor Axel Dignass

Bridging Chronic Gastrointestinal Inflammation and Cancer

Professor Axel Dignass, Director of the Department of Medicine I at Agaplesion Markus Hospital, Goethe University, Frankfurt (Germany), co-organized the symposium "Gastrointestinal Inflammation and Neoplasia", which deliberately bridged two fields that usually keep to themselves: chronic inflammation and cancer. We spoke shortly after the meeting about the thinking behind that concept, where monitoring and treatment targets stand in 2026, and the questions he believes the field most urgently needs to answer.

What was the guiding idea behind the 243rd Falk Symposium program?

We deliberately combined inflammation and neoplasia. The clinical rationale is clear: Chronic inflammation drives dysplasia and, ultimately, cancer—yet the two specialties rarely meet. By interlinking sessions, we wanted oncologists and inflammation specialists to step out of their "silos" to learn from one another. Strong attendance and positive feedback over two days validated the approach.

How important is cross-regional learning in GI practice?

Very important. Differences in access, reimbursement, and regulation exist not only between the United States and Europe but also within Europe. Colorectal cancer surveillance is a good example: Established in Germany roughly 25 years ago, it is now being adopted more widely. Instead of transplanting one country's model, the goal is to exchange data and adapt to local systems. That approach raises standards of care while respecting context.

Where do you see endoscopy and biomarkers in diagnosis and monitoring?

For immune-mediated gastrointestinal disorders, endoscopy remains the gold standard. Biomarker availability, however, varies significantly. In inflammatory bowel disease (IBD), fecal calprotectin and C-reactive protein are well established for monitoring inflammation. In celiac disease, tissue transglutaminase is a highly sensitive and specific serologic marker for diagnosis and follow-up. In microscopic colitis, we lack reliable biomarkers and endoscopic findings are often normal, so colonoscopic biopsy remains essential. Strong markers in some diseases should spur discovery in others; the long-term goal is noninvasive monitoring to reduce patient burden and health-economic pressure.

Symptoms often don't match disease activity. How should clinicians respond?

Symptoms correlate poorly with inflammatory activity in IBD and eosinophilic esophagitis (EoE). Patients may report few or no symptoms while inflammation progresses, leading to fibrosis, strictures, and ultimately dysplasia and cancer. Symptom-based management alone risks missing the intervention window. Follow-up should include objective assessment—endoscopy, histology, and biomarkers—not symptom assessment alone. This is consistent with treat-to-target principles increasingly used in IBD care.

What are the main barriers to implementing this objective, treat-to-target strategy?

The gap is multifactorial: financial constraints; limited endoscopy capacity and trained workforce; uneven awareness of less common entities (some primary care physicians are unfamiliar with microscopic colitis); and patient reluctance to undergo invasive procedures. Screening participation shows the challenge: Despite invitations, only a minority of eligible individuals take part. Improving uptake requires public education, primary care engagement, and streamlined pathways that minimize logistical friction.

How do you view histologic remission as a treatment target in IBD?

Histologic remission should not be a universal goal. With STRIDE-II under revision to STRIDE-III, histology is expected to remain an adjunctive, not definitive, target. Where achievable without excessive risk or burden, pursue it. But if a patient is already in steroid-free clinical and endoscopic remission on a third-line agent, chasing histology may jeopardize gains. These trade-offs demand experienced clinical judgment—something artificial intelligence cannot yet replace in nuanced, patient-specific decisions.

How do you approach shared decision-making and steroid use?

I use shared decision making to balance efficacy, safety, and quality of life. When a patient is not happy with the ideal plan, we agree on realistic second or third best targets that still move outcomes forward. A consistent priority is steroid avoidance and withdrawal. Glucocorticoids bring rapid symptom control but carry significant long-term harms: osteoporosis, fragility fractures, cataracts, glaucoma, and dermatologic adverse effects, and many more. Transparent conversations about these risks are essential to minimize cumulative exposure.

What is your stance on CRC prevention and screening?

Strong endorsement. Diet matters—Mediterranean rather than Westernized patterns likely reduce risk. Population-based colorectal cancer screening is the most actionable lever today. Where implemented, screening reduces both total and advanced cancers. Even imperfect fecal occult blood testing confers benefit; the fecal immunochemical test improves detection further. Combining stool testing with endoscopy enhances sensitivity and, ulitmately leads to polypectomy, the only screening intervention that prevents cancer outright by removing preneoplastic lesions. Participation remains the obstacle: In Germany, roughly 15% of men and 22% of women participate in colorectal cancer screening, compared with about 60–70% for breast cancer screening. Closing that gap would yield substantial population level benefit.

Early-onset disease featured prominently. What were the key takeaways?

Talks by Michał Kamiński (surveillance strategies), Antoni Castells (FIT versus other approaches), and Nastazja Pilonis (endoscopic resection of early cancers) converged on a simple message: combine methods and act early. Integration, not substitution, should guide practice: align noninvasive tests, high-quality endoscopy, and timely intervention to optimize outcomes across risk groups and health systems.

What should the field prioritize next to advance personalized care?

Predictive markers for disease course and treatment response. After three decades of work, immune-mediated inflammatory diseases still lack the genetic or molecular predictors that already guide therapy selection in oncology. Clinicians therefore navigate an expanding therapeutic armamentarium largely by trial and error. Identifying robust predictors is the key to genuinely personalized medicine: matching the right patient to the right therapy at the right time, shortening time to control, reducing steroid exposure, and preserving organ function long term.

Any reflections on the symposium's organization and format?

The scientific organization granted complete freedom in selecting topics and speakers, which ensured that clinically important questions were addressed by the best available faculty. Bringing disciplines together, focusing on objective targets, and elevating standards through international exchange were, in my view, the symposium's most durable contributions.

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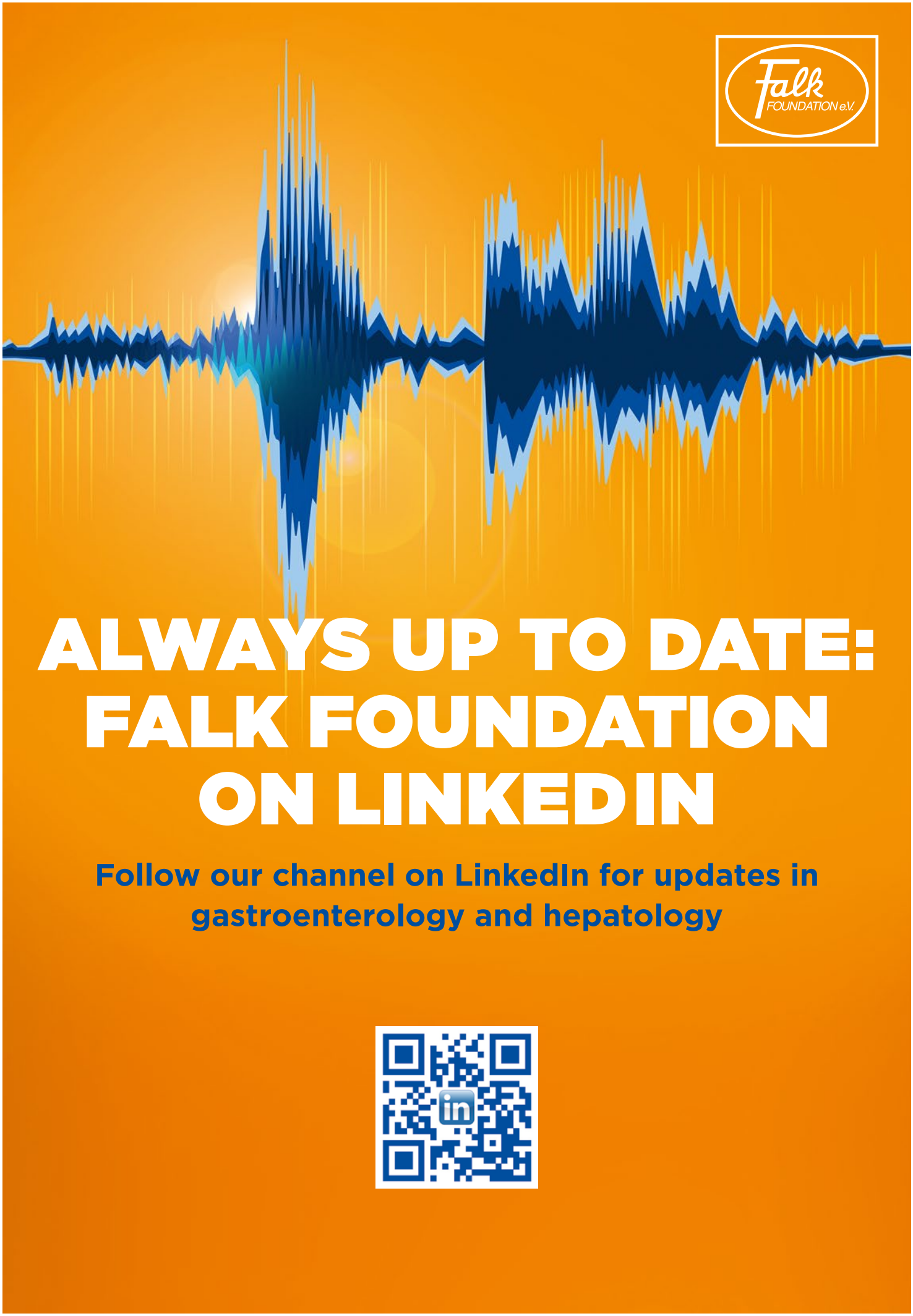
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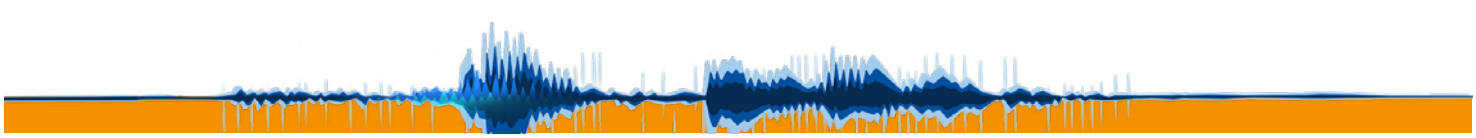

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