IMMUNE-MEDIATED DISEASES OF THE GI TRACT – TREAT TO TARGET APPROACH

March 21-22, 2025

Symposium 239
SYDNEY, AUSTRALIA

12 CME CREDITS

12 CME CREDITS

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

PREFACE

It is with great excitement and anticipation that we welcome you cordially to the 2025 Sydney Falk Foundation Symposium on “Immune-Mediated Diseases of the GI Tract - Treat to Target Approach.” As we gather in the vibrant city of Sydney, we reflect on the remarkable strides made in understanding and treating immune-mediated diseases of the gastrointestinal tract, including eosinophilic esophagitis, inflammatory bowel disease, and microscopic colitis.

This year’s symposium promises to be a landmark event, building on the foundational discussions and insights from previous gatherings. The landscape of gastrointestinal immunopathology continues to evolve, with new discoveries and therapeutic advancements shaping our clinical practice. We bring forth the well-established paradigm of treat-to-target in inflammatory bowel diseases and extend this to other immune mediated conditions. Our program has been meticulously crafted to provide a comprehensive exploration of new ideas, featuring a blend of expert lectures and interactive sessions.

We will delve into cutting-edge research on pathophysiological mechanisms, the role of diet, the impact of environmental factors and new medical treatments. Current management strategies and innovative treatment options will be thoroughly examined, with an emphasis on their practical implications for patient care. Additionally, we will address treating beyond symptoms and appropriateness of this approach in GI immune diseases beyond inflammatory bowel disease.

Sydney, with its dynamic atmosphere and rich cultural heritage, offers an ideal backdrop for this symposium. We eagerly anticipate engaging discussions, collaborative learning, and the opportunity to reconnect with colleagues and friends from across the globe.

We look forward to welcoming you to Sydney for what promises to be an educational and inspiring event.

Warm regards, Professor Rupert Leong on behalf of the organising committee

Jane Andrews Axel Dignass Richard Gearry
Rupert Leong Gerhard Rogler

IMMUNE-MEDIATED DISEASES OF THE GI TRACT – TREAT TO TARGET APPROACH

March 21-22, 2025

Scientific Organization:
Prof. Jane Andrews, Adelaide
Prof. Dr. Axel Dignass, Frankfurt
Prof. Richard Gearry, Christchurch
Prof. Rupert Leong, Sydney
Prof. Dr. Gerhard Rogler, Zurich

Congress Venue:
Hyatt Regency Hotel Sydney
161 Sussex Street
Sydney NSW 2000
Australia

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

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

Start of Registration:
Thursday, March 20, 2025
16:00 - 20:00 h
at the congress office

Friday, March 21, 2025

9:00	Welcome and opening remarks *Rupert Leong, Sydney*

SESSION I

Is treat to target the ideal strategy in immune-mediated GI diseases (IBD, EoE, microscopic colitis)? Debate

Chairs: *Gerald Holtmann, Brisbane; Michael A. Kamm, Melbourne*

9:10	Pro *Axel Dignass, Frankfurt*
9:30	Con *Peter Gibson, Melbourne*
9:50	**Panel discussion** Moderation: Lena Thin, Murdoch Discussants: Axel Dignass, Peter Gibson, Gregory Moore, Dan Turner
10:10	Adjudication: Audience voting via smartphone
10:15	Summary *Richard Gearry, Christchurch*
10:30	**Coffee break with ePoster session**

SESSION II

IBD strategies & assessments

Chairs: *Richard Gearry, Christchurch; Ailsa Hart, London*

11:00	Holistic patient assessment: Depression, fatigue, pain, social integration - how does it impact therapy? *Akhilesh Swaminathan, Christchurch*
11:15	Trajectory modeling in IBD patients: A help to stratify treatment decisions? *Stefan Schreiber, Kiel*

11:30	IUS and calprotectin: Greater cost effectiveness over MRE and colonoscopy? *Yoon-Kyo An, Brisbane*
11:45	Early surgery in Crohn's disease: Underused? *Willem A. Bemelman, Amsterdam*
12:00	**Panel discussion** Discussants: Yoon-Kyo An, Willem A. Bemelman, Richard Gearry, Ailsa Hart, Stefan Schreiber, Akhilesh Swaminathan
12:30	**Lunch break with ePoster session**

SESSION III

Old and new drugs in IBD

Chairs: *Maria T. Abreu, Los Angeles; Aviv Pudipeddi, Concord*

13:30	5-ASA optimization & combination oral/topical *Britta Siegmund, Berlin*
13:50	The case for thiopurines (including thioguanine) *Mark Ward, Melbourne*
14:10	JAKi and S1P: Efficacy and safety *David T. Rubin, Chicago*
14:30	Anti-IL23 (including reimbursement barriers in Australia) *Taku Kobayashi, Tokyo*
14:50	Microbial therapies: Prebiotic, postbiotic and FMT *Damjana Bogatic, Adelaide*
15:10	**Coffee break with ePoster session**

Friday, March 21, 2025

SESSION IV	
IBD into the future	
Chairs: *Jane Andrews, Adelaide; Charles N. Bernstein, Manitoba*	
15:40	Epidemiology: Compounding prevalence *Charles N. Bernstein, Manitoba*
16:00	How to deal with the tsunami of drug use/resource utilization *Johan Burisch, Hvidovre*
16:20	Models of care, innovations (remote) in IBD and non-IBD *Sang H. Park, Seoul*
16:40	Treatment options, treatment changes: What, when, why *Remo Panaccione, Calgary*
17:00	Closing remarks

Saturday, March 22, 2025

SESSION V

Eosinophilic esophagitis

Chairs: *Yoon-Kyo An, Brisbane; Susan Connor, Sydney*

8:30	The role of AI in endoscopy of EoE *Helmut Messmann, Augsburg*
8:50	EoE symptoms: Inflammation versus fibrostenosis *Santosh Sanagapalli, Sydney*
9:10	EoE therapy: Steroids versus biological agents *Luc Biedermann, Zurich*
9:30	EoE diet: Role of diet in EoE therapy? *Emma Halmos, Melbourne*
9:50	Maintenance therapy: Treat to target? *Hamish Philpott, Adelaide*
10:10	**Case discussion (local faculty)**
10:30	**Coffee break with ePoster session**

SESSION VI

Diarrhea & microscopic colitis: Treat to target paradigm

Chairs: *Jane Andrews, Adelaide; David T. Rubin, Chicago*

11:00	The diarrheal world: Causes & diagnostic approach *Gillian Watermeyer, Cape Town*
11:20	Microscopic colitis: Epidemiology, risk factors, treatment *Peter Katelaris, Sydney*
11:40	Eosinophilic enteritis & colitis *Rebecca Burgell, Melbourne*

Saturday, March 22, 2025

12:00	**Presentation of Poster Awards**
12:20	Checkpoint inhibitor colitis *Andrew D. Buckle, Launceston*
12:40	**Cases & discussion presented by registrars**
13:00	**Lunch break with ePoster session**

SESSION VII

Strictures in Crohn's disease

Chairs: *Raja Affendi Raja Ali, Selangor; Gerhard Rogler, Zurich*

14:00	Clinical, imaging and biomarker assessment of Crohn's disease strictures and applying a treat to target approach *Rupert Leong, Sydney*
14:20	Treatment of strictures in Crohn's disease 2025 - From anti fibrotics to diet *Iris Dotan, Petah Tikva*
14:40	Endoscopic approaches to strictures and combination with advanced therapy *John Chetwood, Melbourne*
15:00	Strictureplasty versus resection – Which one and how? *Willem A. Bemelman, Amsterdam*
15:20	**Coffee break with ePoster session**

SESSION VIII

Other opportunities to optimize; Comorbidities in IBD

Chairs: *Rupert Leong, Sydney; Kate D. Lynch, Adelaide*

15:50	Obesity (including management) *Patricia Kaazan, Adelaide*
16:10	Cardiovascular comorbidities and how to assess them *Ashwin N. Ananthakrishnan, Boston*
16:30	Mortality in IBD: Cancer, clots, infections *Kate D. Lynch, Adelaide*
16:50	Primary sclerosing cholangitis *Vineet Ahuja, New Delhi*
17:10	Anemia *Jane Andrews, Adelaide*
17:30	Fatigue *Fernando Magro Dias, Porto*
17:50	Closing remarks

LIST OF SPEAKERS, MODERATORS AND SCIENTIFIC ORGANIZERS

Maria T. Abreu, MD
Professor of Medicine
F. Widjaja Inflammatory Bowel Disease Institute
Cedars-Sinai Medical Center
8700 Beverly Blvd. Suite 4063
Los Angeles, CA 90048
maria.abreu@cshs.org

Dr. Vineet Ahuja
Departement of Gastroenterology
All India Institute of Medical Sciences
Ansari Nagar
New Delhi - 110029
India
vineet.aiims@gmail.com

Dr. Yoon-Kyo An
Departement of Gastroenterology
Mater Medical Centre
293 Vulture Street
Brisbane QLD 4101
Australia
yoon.an@mater.uq.edu.au

Ashwin N. Ananthakrishnan, MD, MPH
Crohn's and Colitis Center
Mass General Hospital
165 Cambridge Street, 9th Floor
Boston
Massachusetts 02421
United States
aananthakrishnan@mgh.harvard.edu

Prof. Jane Andrews
Adelaide Medical School
Faculty of Health and Medical Sciences
University of Adelaide
Adelaide SA 5005
Australia
jane.andrews@sa.gov.au

Prof. Dr. Willem A. Bemelman
Department of Surgery
Amsterdam UMC
University of Amsterdam
Location VUMC | 7F |Postbus 7057
1007 MB Amsterdam
The Netherlands
w.a.bemelman@amsterdamumc.nl

Prof. Charles N. Bernstein
Max Rady College of Medicine
University of Manitoba
715 McDermot Avenue
Winnipeg
Manitoba R3E 0W2
Canada
charles.bernstein@umanitoba.ca

Dr. Luc Biedermann, PD
Klinik für Gastroenterologie und Hepatologie
UniversitätsSpital Zürich
Rämistr. 100
8091 Zürich
Switzerland
luc.biedermann@usz.ch

Dr. Damjana Bogatic
Inflammatory Bowel Disease Fellow
The Queen Elizabeth Hospital
28 Woodville Road
Woodville SA 5011
Adelaide
Australia
damjana.bogatic@sa.gov.au

Dr. Andrew D. Buckle
Gastroenterology
Launceston General Hospital
274-280 Charles Street
Launceston TAS 7250
Australia
andrew.buckle@ths.tas.gov.au

A/Prof. Rebecca Burgell
Department of Gastroenterology
Alfred Hospital
55 Commercial Road
Melbourne, VIC 3004
Australia
r.burgell@alfred.org.au

Prof. Johan Burisch
Gastrounit, medicial division &
Copenhagen IBD Center
Copenhagen University Hospital -
Amager and Hvidovre
Kettegårds alle 30
2650 Hvidovre
Denmark
johan.burisch@regionh.dk

Dr. John Chetwood
St Vincent's Hospital
University of Melbourne
Victoria Parade
Fitzroy VIC 3065
Melbourne
Australia
johnchetwood@doctors.org.uk

Prof. Susan Connor
Departement of Gastroenterology and Liver
Liverpool Hospital
Corner of Elizabeth and Goulburn Streets
Liverpool NSW 2170
Australia
susan.connor1@health.nsw.gov.au

Prof. Dr. Axel Dignass
Medizinische Klinik I
Agaplesion Markus Krankenhaus
Wilhelm-Epstein-Str. 4
60431 Frankfurt
Germany
axel.dignass@agaplesion.de

Prof. Dr. Iris Dotan
Division of Gastroenterology
Rabin Medical Center
Beilinson Hospital
39 Jabotinski St
49100 Petah Tikva
Israel
irisdo@clalit.org.il

Prof. Richard Gearry
Department of Medicine
University of Otago
Christchurch
New Zealand
richard.gearry@cdhb.health.nz

Prof. Peter Gibson
Departement of Gastroenterology
School of Translational Medicine
Monash University and Alfred Health
Level 6/99 Commercial Rd
Melbourne VIC 3004
Australia
peter.gibson@monash.edu

Dr. Emma Halmos
Departement of Gastroenterology
School of Translational Medicine
Monash University and Alfred Health
99 Commercial Rd
Melbourne VIC 3004
Australia
emma.halmos@monash.edu

Prof. Dr. Ailsa Hart
Inflammatory Bowel Disease Unit
St. Mark's Hospital
Watford Road
Harrow HA1 3UJ
Great Britain
ailsa.hart@nhs.net

Prof. Dr. Gerald Holtmann
Princess Alexandra Hospital
Ipswich Road, Woollongabba
Brisbane QLD 4102
Australia
gerald.holtmann@health.qld.gov.au

Dr. Patricia Kaazan
Calvary Adelaide Hospital
120 Angas Street
Adelaide SA 5000
Australia
patricia.kaazan@sa.gov.au

Prof. Michael A. Kamm
St Vincent's Hospital
University of Melbourne
Victoria Parade
Fitzroy VIC 3065
Melbourne
Australia
mkamm@unimelb.edu.au

Prof. Peter Katelaris
Concord Hospital, University of Sydney
Gastroenterology Department
Hospital Rd, Concord West
Sydney NSW 2137
Australia
peter.katelaris@sydney.edu.au

Taku Kobayashi, MD, PhD
Center for Advanced IBD Research and Treatment
Kitasato University Kitasato Institute Hospital
Department of Gastroenterology
Kitasato University School of Medicine
5-9-1 Shirokane, Minato-ku
Tokyo 108-8642
Japan
drkobataku@gmail.com

Prof. Rupert Leong, MD
Professor of Medicine
University of Sydney
Concord Clinical School Department of Gastroenterology
Level 1 West. 1A Hospital Road
Concord NSW 2139
Australia
rupertleong@hotmail.com

Dr. Kate D. Lynch
Inflammatory Bowel Disease Service
Royal Adelaide Hospital
1 Port Road
Adelaide SA 5000
Australia
kate.lynch@sa.gov.au

Prof. Fernando Magro Dias
Hospital de S. Joao
Servico de Gastroenterologia
Av. Prof. Hernani Monteiro
4200-319 Porto
Portugal
fm@med.up.pt

Prof. Dr. Helmut Messmann
III. Medizinische Klinik
Universitätsklinikum Augsburg
Stenglinstr. 2
86156 Augsburg
Germany
helmut.messmann@uk-augsburg.de

Prof. Gregory Moore
Gastroenterology & Hepatology Unit
Monash Health
School of Clinical Sciences
Monash University
Monash Medical Centre
246 Clayton Road,
Clayton VIC 3168
Australia
gregory.moore@monash.edu

Prof. Dr. Remo Panaccione
Inflammatory Bowel Disease Clinic
Cumming School of Medicine
University of Calgary
Teaching Research and Wellness Building
Room 6D30, 3280 Hospital Drive NW
Calgary, Alberta, T2N 4Z6
Canada
rpanacci@ucalgary.ca

Sang H. Park, MD, PhD.
Department of Gastroenterology and
Inflammatory Bowel Disease Center,
University of Ulsan College of Medicine
Asan Medical Center,
88, Olympic-ro 43-gil
Seoul, 05505
Republic of Korea
umdalpin@hanmail.net

A/Prof. Hamish Philpott
MBBS MRCP FRACP PhD AGAF
Gastroenterologist
NALHN
University of Adelaide
Faculty of Health and Medical Sciences
Corner of North Terrace & George Street
Adelaide SA 5000
Australia
hamish.philpott@sa.gov.au

Dr. Aviv Pudipeddi
Level 1 West ACE Unit
Concord Hospital
Hospital Road
Concord NSW 2139
Australia
aviv.pudipeddi@health.nsw.gov.au

Prof. Dr. Raja Affendi Raja Ali
School of Medical and Life Sciences
Sunway University
No. 5, Jalan Universiti, Bandar Sunway
47500 Selangor
Malaysia
affendi@sunway.edu.my

Prof. Dr. Dr. Gerhard Rogler
Klinik für Gastroenterologie
und Hepatologie
UniversitätsSpital Zürich
Rämistr. 100
8091 Zürich
Switzerland
gerhard.rogler@usz.ch

David T. Rubin, MD
Professor of Medicine
Section of Gastroenterology, Hepatology
and Nutrition
The University of Chicago
5801 South Ellis Ave.
Chicago, IL 60637
United States
drubin@bsd.uchicago.edu

Prof. Santosh Sanagapalli
Departement of Gastroenterology
St. Vincent's Hospital Sydney
390 Victoria Street
Darlinghurst NSW 2010
Australia
santosh153@gmail.com

Prof. Dr. Stefan Schreiber
Innere Medizin I
Universitätsklinikum
Schleswig-Holstein, Campus Kiel
Arnold-Heller-Straße 3 (Haus 6)
24105 Kiel
Germany
s.schreiber@mucosa.de

Prof. Dr. Britta Siegmund
Medizinische Klinik für Gastroenterologie
Infektiologie und Rheumatologie
Charité Universitätsmedizin
Campus Benjamin Franklin
Hindenburgdamm 30
12200 Berlin
Germany
britta.siegmund@charite.de

Akhilesh Swaminathan, PhD
University of Otago
2 Riccarton Avenue
Christchurch 8011
New Zealand
akhilesh.swaminathan@otago.ac.nz

Dr. Lena Thin
Fiona Stanley Hospital
102-108 Murdoch Drive
Murdoch WA 6150
Australia
lena.thin@health.wa.gov.au

Prof. Dan Turner
Paediatric Gastrenterology and Nutrition
Shaare Zedek Medical Center
Hebrew University of Jerusalem
12 Bayit Street
Jerusalem 91031
Israel
turnerd@szmc.org.il

Prof. Mark Ward
Department of Gastroenterology
Alfred Hospital
55 Commerical Road
Melbourne, VIC 3004
Australia
m.ward@alfred.org.au

Prof. Gillian Watermeyer
Division of Gastroenterology
Department of Medicine
University of Cape Town
Cape Town
South Africa
gillian.watermeyer@uct.ac.za

REGISTRATION

You can register for the event via our homepage:
www.falkfoundation.org
Registration is only possible online.

CONGRESS FEES

Scientific Program of Symposium 239	EUR 300
Students (copy of student ID required)	EUR 150

The congress fees include:
- Pre-Opening and Welcome on Thursday, March 20, 2025, 19 h
- Refreshments during coffee breaks
- Lunch on Friday, March 21 and on Saturday, March 22, 2025
– A copy of the final program

CONGRESS OFFICE AND REGISTRATION

Opening Hours:

Thursday, March 20, 2025	16:00 - 20:00 h
Friday, March 21, 2025	08:00 - 17:30 h
Saturday, March 22, 2025	08:00 - 18:15 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

Hyatt Regency Hotel Sydney
161 Sussex Street
Sydney NSW 2000
Australia

By plane
The Hyatt Regency Sydney is located adjacent to Darling Harbour, about 8 km from Sydney Airport (SYD). You can either take a taxi (20-25 minutes in light traffic (45-55 AUD) or take public transportation (train) to Town Hall Station in 16-25 minutes, with one change at Central Station.

By train
Town Hall Train Station is just 650 m away (10 min walking distance).

By car
For self-drive visitors, there is a Wilson Parking directly opposite the hotel (please note there are two entrances at 383 Kent Street or 168 Sussex Street). Please park your car and keep hold of your ticket. Then, contact the concierge desk to obtain a 24-hour, multi-entry, self-parking ticket for the car park at Hyatt Regency Sydney rates.

POSTER ABSTRACTS

1. A necroptotic-to-apoptotic signaling axis underlies inflammatory bowel disease
   A. Al-Ani, J. Pang, K. Patel, S. Young, I. Kong, J. Chen, M. Barrios, J. Rickard, S. Chen, S. Foroughi, W. Cawthorne, A. Jacobsen, A. Jois, A. Weir, L. Whitehead, P. Rajasekhar, C. Horne, I. Azeez, T. Tan, W. Liang, S. Sivanesan, A. Metz, A. Patwardhan, N. Shea, G. Iyngkaran, D. Scheider, A. Elford, W. Beattie, F. Macrae, G. Liccardi, H. Walczak, Y. Zhang, O. Sieber, T. Spelman, L. Giulino-Roth, E. Hawkins, K. Rogers, R. Bowden, S. Nicholson, K. Lawlor, A. Samson, J. Vince, J. Murphy, B. Christensen (Melbourne, Parkville, AU; New York, US; Guangzhou, Zhengzhou, CN; Cologne, DE)
2. Altered CD4+ T cell reactivity against yeasts in ASCA-positive patients with Crohn's disease
   P. Bacher, G. Rios Martini, B. Hube, I. Iliev, A. Scheffold, S. Schreiber (Kiel, Jena, DE; New York, US)
3. Characterization of drug-specific CD4+ T cells in patients with inflammatory bowel diseases
   P. Bacher, A. Weichberger, G. Rios Martini, S. Schreiber (Kiel, DE)
4. The role of bound anti-drug antibodies to infliximab in predicting future immunogenic failure when de-escalating from combination therapy with an immunomodulator to anti-TNF monotherapy – (RAPID-IM) study
   K. Cameron, M. Sam, N. Thennakoon, B. Gu, T. Skinner, A. Arzivian, E. Shelton, C. Haifer, S. Connor, C. Toong, P. Gibson, D. Gibson, A. Boussioutas, M. Sparrow, M. Ward (Melbourne, Sydney, AU)
5. Combined immunosuppressive therapies in inflammatory bowel disease, real-world registry data from the Persistence Australian National IBD Cohort (PANIC6) study
   J. Chetwood, S. Paramsothy, R. Leong (Melbourne, Sydney, AU)
6. Effect of IL-17A inhibitors on inflammatory bowel disease progress, real-world registry data from the Persistence Australian National IBD Cohort (PANIC6) study
   J. Chetwood, S. Paramsothy, R. Leong (Melbourne, Sydney, AU)
7. Predictors of long-term anti-TNF failure: A validation analysis via the prospective Persistence Australian National IBD Cohort (PANIC) registry
   J. Chetwood, S. Paramsothy, R. Leong (Melbourne, Sydney, AU)
8. Study on Crohn's disease obstructions and persistence with endoscopic dilatations (SCOPED): A dual-center retrospective cohort study
   J. Chetwood, S. Selvaratnam, A. Arzivian, P. Dhanji, N. Hung, F. Pan, S. Paramsothy, R. Leong (Melbourne, Sydney, AU)
9. Infliximab and adalimumab in the treatment of fistulizing Crohn's disease: A propensity score-matched analysis from the prospective Persistence Australian National IBD Cohort (PANIC4) study
   J. Chetwood, S. Paramsothy, R. Leong (Sydney, AU)
10. Immune checkpoint inhibitor esophagitis with excellent response to oral viscous budesonide
    M. Clark-Dickson, A. Khani, T. Hughes, G. Rich, K. Nahar (Wahroonga, Sydney, AU)
11. Clinical manifestations and implications of DAO deficiency in histamine intolerance
    E. Toader, A. Iacob, M. Filioreanu, A. Marcu, M. Piscuc (Iasi, RO)

12. Real-world effectiveness of risankizumab in Crohn's disease: The UK experience
A. Elford, N. Plevris, N. Constantine-Cooke, C. Lees; Pan UK Risankizumab study working group (Edinburgh, GB)

13. Increased advanced therapy prescribing correlates with decreased colectomy rates in ulcerative colitis: A twenty-year population-based cohort study in Lothian, Scotland
A. Elford, N. Constantine-Cooke, P. Jenkinson, B. Gros, N. Plevris, M. Lyons, S. Ong, N. Greenlees, C. Ohare, N. Ventham, D. Wilson, P. Henderson, S. Din, C. Noble, G. Jones, I. Arnott, C. Lees (Hobart, AU; Edinburgh, GB)

14. Topical upadacitinib is effective in distal ulcerative colitis – A case study and pre-clinical proof of concept
R. Giri, A. Amiss, M. Carpinelli de Jesus, B. Riches, J. Begun (Woolloongabba, Brisbane, AU)

15. Malnutrition in Crohn's disease: Prevalence, risk factors, and key predictors
H. Hassine, N. Krifa, H. Dabbebi, I. Kaffela, H. Yaacoub, D. Cherif, H. Kchir, N. Maamouri (Tunis, TN)

16. Acute abdomen in Crohn's disease: A comparative analysis of surgery – First diagnosis versus known cases
H. Hassine, N. Krifa, H. Dabbebi, R. Chaouachi, D. Cherif, H. Yaacoub, H. Kchir, N. Maamouri (Tunis, TN)

17. Can the occurrence of perianal lesions in Crohn's disease be predicted?
H. Hassine, N. Krifa, H. Dabbebi, R. Chaouachi, H. Yaacoub, D. Cherif, H. Kchir, N. Maamouri (Tunis, TN)

18. Clinical features of elderly-onset ulcerative colitis
H. Hassine, N. Krifa, H. Dabbebi, R. Chaouachi, H. Yaacoub, D. Cherif, H. Kchir, N. Maamouri (Tunis, TN)

19. Clinical, therapeutic, and evolutionary characteristics of severe acute colitis in Crohn's disease
H. Hassine, N. Krifa, H. Dabbebi, R. Chaouechi, D. Cherif, H. Yaacoub, H. Kchir, N. Maamouri (Tunis, TN)

20. Extent of disease as a predictor of bone loss in inflammatory bowel disease: A retrospective study
H. Hassine, N. Krifa, H. Dabbebi, I. Kaffela, H. Yaacoub, D. Cherif, H. Kchir, N. Maamouri (Tunis, TN)

21. Is proctitis in Crohn's disease a factor in the persistence of perianal fistulas?
H. Hassine, N. Krifa, H. Dabbebi, R. Chaouachi, D. Cherif, H. Yaacoub, H. Kchir, N. Maamouri (Tunis, TN)

22. Predictive factors for surgical intervention in Crohn's disease
H. Hassine, N. Krifa, H. Dabbebi, I. Kaffela, H. Yaacoub, D. Cherif, H. Kchir, N. Maamouri (Tunis, TN)

23. Infra-red microspectroscopy provides quantitative assessment of fibro-inflammation in Crohn's disease strictures and predicts postoperative recurrence
C. Keung, R. Pawlowski, A. Ryan, E. Kwan, A. Longano, R. Lim, W. Sievert, G. Moore, B. Wood (Melbourne, AU)

24. Infra-red spectroscopy predicts response to anti-tumour necrosis factor-α therapy in fibrostenotic Crohn's disease
C. Keung, J. Correia, A. Longano, J. Schulberg, E. Wright, A. Hamilton, M. Kamm, R. Lim, W. Sievert, B. Wood, G. Moore (Melbourne, AU)

25. Comparative accuracy and reliability of handheld versus cart-based ultrasound in assessing inflammatory bowel disease activity
M. Khaing, R. Fernandes, Y. An, B. Baraty, R. Bryant, S. Ghaly, R. Smith, A. Srinivasan, K. Hay, J. Begun (Brisbane, Sydney, Melbourne, AU)

26. Tofacitinib demonstrates preliminary efficacy in induction of remission in chronic pouchitis
E. Khoo, A. Amiss, J. Ding, S. Connor, L. White, R. Leong, W. Mohsen, R. Bryant, Z. Ardalan, P. De Cruz, A. Croft, K. Lynch, Y. An, G. Holtmann, J. Begun (Brisbane, Woolloongabba, Melbourne, Sydney, Sunshine Coast, Southport, Adelaide, Herston, AU)

27. Combined oral mesalamine and budesonide suppositories yield faster and better symptom resolution in active ulcerative colitis than oral mesalamine monotherapy: A randomized, double-blind, placebo-controlled add-on study
W. Kruis, A. Wrozek, L. Wolanski, O. Oliinyk, P. Andreev, A. Mueller, R. Mohrbacher, R. Mueller, B. Siegmund (Cologne, Freiburg, Berlin, DE; Staszów, Pulawy, PL; Zaporizhzhia, UA; Samara, RU)

28. Early faecal calprotectin – A novel predictor of clinical outcomes in acute severe ulcerative colitis: Results from PREDICT-UC
C. Li Wai Suen, M. Choy, D. Con, K. Driver, D. Bruen, J. Glason, F. Bonelli, I. Bittar, K. Boyd, R. Pena, K. Burrell, O. Rosella, D. Proud, R. Brouwer, A. Gorelik, D. Liew, W. Connell, E. Wright, K. Taylor, A. Pudipeddi, M. Sawers, B. Christensen, W. Ng, J. Begun, G. Radford-Smith, M. Garg, N. Martin, D. Van Langenberg, N. Ding, L. Beswick, R. Leong, M. Sparrow, P. De Cruz (Melbourne, Sydney, Adelaide, Geelong, Brisbane, AU; Saluggia, IT)

29. The role of early serum infliximab levels in predicting outcomes in acute severe ulcerative colitis: Results from PREDICT-UC
C. Li Wai Suen, M. Choy, D. Con, K. Cheng, J. Nigro, K. Breheney, K. Boyd, R. Pena, K. Burrell, O. Rosella, D. Proud, R. Brouwer, A. Gorelik, D. Liew, W. Connell, E. Wright, K. Taylor, A. Pudipeddi, M. Sawers, B. Christensen, W. Ng, J. Begun, G. Radford-Smith, M. Garg, N. Martin, D. Van Langenberg, N. Ding, L. Beswick, R. Leong, M. Sparrow, C. Grosserichter-Wagener, H. Te Velthuis, K. Visvanathan, P. De Cruz (Melbourne, Adelaide, Sydney, Geelong, Brisbane, AU; Amsterdam, NL)

30. Gut microbiota changes are associated with type of advanced therapy in patients with IBD: Baseline data from the Australian IBD Microbiome study
R. Little, T. Jayawardana, F. Zhang, S. Koentgen, W. Nan, P. Tavakoli, A. Chung, A. Ho, S. Read, A. Boussioutas, M. Ward, M. Sparrow, S. Ghaly, C. Corte, N. Talley, S. Leach, D. Lemberg, S. Dutt, S. Paramsothy, R. Leong, S. Connor, G. Hold (Melbourne, Sydney, Newcastle, AU)

31. Impaired health-related quality of life is associated with alterations in gut microbiota in patients with inflammatory bowel disease: Baseline sequencing from the Australian IBD Microbiome study
R. Little, T. Jayawardana, F. Zhang, S. Koentgen, N. Wu, P. Tavakoli, A. Chung, A. Cho, A. Boussioutas, M. Ward, M. Sparrow, P. Gibson, S. Ghaly, C. Corte, N. Talley, S. Leach, D. Lemberg, S. Dutt, S. Paramsothy, R. Leong, S. Connor, G. Hold (Melbourne, Sydney, Newcastle, AU)

32. Use of a convolutional neural network to quantify and predict ulcerative colitis disease endoscopic activity and histological outcomes
S. Morrison, C. Chiu, R. Cao, P. Lim, J. Shin, C. Mackenzie, J. Kench, C. Corte, A. Tamilarasan (Sydney, AU)

33. Incidence of acute severe ulcerative colitis in a changing landscape of increased disease prevalence and more effective therapies
S. Nario, M. Wu, C. Tung, C. Kiely (Sydney, AU)

34. Predicting early recognition of acute severe ulcerative colitis based on presence of Truelove and Witts' systemic features at time of presentation
S. Nario, M. Wu, C. Tung, C. Kiely (Sydney, AU)

35. Comparison of screening strategies for latent tuberculosis infection in patients with inflammatory bowel disease before anti-tumor necrosis factor-α agents therapy: TST-alone versus a combination of TST and IGRA
S. Park, Y. Kim, J. Kim, J. Lee, T. Shim, K. Jo (Seoul, KR)

36. Defining an eosinophilic leukocyte count per mm2 threshold for the diagnosis of eosinophilic esophagitis using a digital pathology platform
A. Patai, C. Molnar, M. Tamas (Budapest, Székesfehérvár, HU)

37. Multi-strain probiotics supplementation with standard care enhances quality of life in ulcerative colitis patients: A preliminary data
M. Rosali, R. Raja Ali, K. Muhammad Nawawi, D. Chew, H. Ahmad, N. Mohd Mokhtar (Cheras, Bandar Sunway, Gambang, MY)

38. Comprehensive risk assessment and prognostic factors in celiac disease: Impact of persistent villous atrophy on disease complications and survival outcomes
R. Saidani, D. Cherif, H. Hassine, H. Yacoub, H. Debbabi, H. Kchir (Bizerte, Tunis, TN)

39. The introduction of an intestinal ultrasound service significantly reduces diagnostic endoscopy usage in an inflammatory bowel disease service – The SCOPELESS study
M. Sparrow (Melbourne, AU)

40. Endoscopic severity at presentation and C-reactive protein predict immediate and twelve-month outcomes of second-line medical therapy in acute severe ulcerative colitis
D. Subhaharan, P. Kakkadasam Ramaswamy, J. Edwards, L. White, D. Patrick, A. Shahzad, L. Willmann, H. Moattar, D. Shukla, N. Ishaq, M. Bhullar, A. Dorrington, J. Satsangi, W. Mohsen (Gold Coast, Sunshine Coast, Logan, AU; Oxford, GB)

41. A preliminary report on the effects of microplastics on the gut microbiota and metabolites in patients with inflammatory bowel disease
D. V Thanga Velu, D. V Thanga Velu, R. Raja Ali, Y. Ibrahim, K. Muhammad Nawawi, D. Hsin Chew, H. Ahmad, M. Latif, N. Mohd Mokhtar (Cheras, Subang Jaya, Kuala Nerus, Gambang, Bangi, MY)

42. Assessing patient quality of care in acute severe ulcerative colitis using a composite textbook outcome approach
M. Wu, S. Nario, C. Tung, C. Kiely (Sydney, AU)

43. Number of Truelove and Witts' criteria met at time of presentation impacts on severity of systemic toxicity and investigative outcomes
M. Wu, S. Nario, C. Tung, C. Kiely (Sydney, AU)

FULL CONTENT OF POSTER ABSTRACTS

Poster Numbers 1 – 43

1. A necroptotic-to-apoptotic signaling axis underlies inflammatory bowel disease

Aysha Husam Al-Ani (Melbourne, AU), Jiyi Pang (Parkville, AU), Komal Patel (Parkville, AU), Samuel Young (Parkville, AU), Isabella Kong (New York, US), Jin-Jin Chen (Parkville, AU), Marilou Barrios (Parkville, AU), James Rickard (Parkville, AU), Siqi Chen (Parkville, AU), Siavash Foroughi (Parkville, AU), Wayne Cawthorne (Parkville, AU), Annette Jacobsen (Parkville, AU), Asha Jois (Parkville, AU), Ashley Weir (Parkville, AU), Lachlan Whitehead (Parkville, AU), Pradeep Rajasekhar (Parkville, AU), Christopher Horne (Parkville, AU), Imadh Azeez (Parkwville, AU), Tao Tan (Parkville, AU), Weiwei Liang (Guangzhou, CN), Suresh Sivanesan (Parkville, AU), Andrew Metz (Parkville, AU), Ash Patwardhan (Parkville, AU), Natalie Shea (Parkville, AU), Guru Iyngkaran (Parkville, AU), Daniel Scheider (Parkville, AU), Alexander Elford (Parkville, AU), William Beattie (Parkville, AU), Finlay Macrae (Parkville, AU), Gianmaria Liccardi (Cologne, DE), Henning Walczak (Cologne, DE), Yuxia Zhang (Zhengzhou, CN), Oliver Sieber (Parkville, AU), Tim Spelman (Parkville, AU), Lisa Giulino-Roth (New York, US), Edwin Hawkins (Parkville, AU), Kelly Rogers (Parkville, AU), Rory Bowden (Parkville, AU), Sandra Nicholson (Parkville, AU), Kate Lawlor (Parkville, AU), Andre Samson (Parkville, AU), James Vince (Parkville, AU), James Murphy (Parkville, AU), Britt Christensen (Parkville, AU)

Introduction: Dysregulated programmed cell death plays a key role in IBD pathogenesis, with clinical trials exploring anti-necroptotic therapies. While apoptosis and necroptosis are well-studied in murine colitis models, their prevalence, mechanisms, and therapeutic relevance in human IBD remain unclear. We examined cell death pathways in patients on advanced therapies, uncovering unique signaling mechanisms that could inform future treatments.

Methods: Over 900 paired intestinal biopsies were analysed from 52 patients with IBD (25 with Crohn's disease, 27 with ulcerative colitis) and 28 non-IBD controls. Biopsies were taken from inflamed, marginal, and non-inflamed areas. Cell death processes, including apoptosis (e.g. cleaved caspase-3) and necroptosis (e.g. RIPK3, MLKL), were assessed using histology, immunoblotting and RNA sequencing. The effects of IBD-associated inflammatory cytokines (TNF, IFNγ) on epithelial death were tested on human-derived intestinal organoids (1). Data was correlated with clinical parameters like disease indices and treatments, with tissue quality validated using S100A8/S100A9 expression and blinded histology scoring.

Results: Increased necroptotic and apoptotic signaling was prominent in IBD, correlating with intestinal inflammation, regardless of treatment. Necroptotic signaling was detected in non-inflamed IBD tissue, suggesting early activation in disease progression, while apoptotic signaling was primarily in inflamed tissue. Gene set enrichment analysis identified dysregulated TNF and IFNγ-related pathways linked to cell death signaling, exacerbated by inflammation. Bulk RNA sequencing demonstrated that inflammation-induced transcriptional reprogramming of epithelial cells triggered a macrophage-like phenotype, promoting RIPK1-independent necroptotic signaling (1). In vitro, exposure to TNF and IFNγ in intestinal organoids synergistically induced cell death, primarily through mitochondrial apoptosis. IFNγ enhanced epithelial cell reprogramming, amplifying cell death signaling, while both cytokines together induced mitochondrial apoptosis. In intestinal stem cells, apoptosis was driven by increased PUMA, while upregulated iNOS expression mediated epithelial cell death.

Discussion/Conclusion: Early IBD inflammation reprograms epithelial cells into a macrophage-like state, promoting RIPK1-independent necroptotic signaling. IFNγ and TNF synergise to induce iNOS- and PUMA-mediated mitochondrial apoptosis in epithelial and stem cells. This aberrant epithelial cell death signaling persists despite histologic remission or treatment. Necroptotic signaling occurs upstream of apoptotic cell death, suggesting that targeting the necroptotic-to-apoptotic axis could facilitate molecular healing and sustained deep remission in IBD.

Reference: (1) Jiyi Pang, Aysha H. Al-Ani, Komal M. Patel, Samuel N. Young, Isabella Kong, Jinjin Chen, Marilou Barrios, James A. Rickard, Siqi Chen, Siavash Foroughi, Wayne Cawthorne, Annette V. Jacobsen, Asha Jois, Ashley L. Weir, Lachlan W. Whitehead, Pradeep Rajasekhar, Christopher R. Horne, Imadh Azeez, Tao Tan, Weiwei Liang, Suresh Sivanesan, Andrew Metz, Ash Patwardhan, Natalie Shea, Guru Iyngkaran, Daniel Schneider, Alexander T. Elford, William Beattie, Finlay Macrae, Gianmaria Liccardi, Henning Walczak, Yuxia Zhang, Oliver M. Sieber, Tim Spelman, Lisa Giulino-Roth, Edwin D. Hawkins, Kelly L. Rogers, Rory Bowden, Sandra E. Nicholson, Kate E. Lawlor, Britt Christensen, Andre L. Samson, James E. Vince, James M. Murphy A necroptotic-to-apoptotic signaling axis underlies inflammatory bowel disease. bioRxiv 2024.11.13.623307; doi: https://doi.org/10.1101/2024.11.13.623307

2. Altered CD4+ T cell reactivity against yeasts in ASCA-positive patients with Crohn's disease

Petra Bacher (Kiel, DE), Gabriela Rios Martini (Kiel, DE), Berndhard Hube (Jena, DE), Iliyan Iliev (New York, US), Alexander Scheffold (Kiel, DE), Stefan Schreiber (Kiel, DE)

Introduction: Aberrant CD4+ T cell reactivity against intestinal microbes is considered to drive mucosal inflammation in inflammatory bowel diseases (IBD). The disease-relevant microbial species and the corresponding microbe-specific pathogenic T cell phenotypes remain largely unknown.

Methods: We used the highly sensitive antigen-reactive T cell enrichment approach in combination with single cell sequencing technologies for direct analyses of CD4+ T cell reactivity against microbial species in human blood and tissue samples.

Results: We identified commensal and food-derived yeasts as direct activators of altered CD4+ T cell reactions in patients with Crohn's disease (CD). Those altered anti-yeast T cell responses are characteristic for CD patients who are positive for anti-Saccharomyces cerevisiae antibodies (ASCA), indicating different host-fungal interactions in this subgroup of patients. Yeast-responsive CD4+ T cells in CD display a cytotoxic Th1 phenotype and show selective expansion of T cell clones that are highly cross-reactive to several fungal species. This indicates cross-reactive T cell selection by repeated encounter with conserved fungal antigens in the context of chronic intestinal disease.

Discussion/Conclusion: Our findings highlight the role of commensal and food-derived yeasts as triggers of aberrant CD4+ T cell reactivity in ASCA-positive patients with CD, paving the way for new therapeutic approaches. The discovery that a specific subgroup of CD patients develops altered CD4+ T cell responses and ASCAs against yeasts, while other patients with IBD do not, emphasizes the existence of fundamentally different host-microbe interactions within this subgroup of patients.

3. Characterization of drug-specific CD4+ T cells in patients with inflammatory bowel diseases

Petra Bacher (Kiel, DE), Amos Weichberger (Kiel, DE), Gabriela Rios Martini (Kiel, DE), Stefan Schreiber (Kiel, DE)

Introduction: Biologicals are commonly used to treat chronic inflammatory diseases. Treatment failure is a major limitation for the efficacy of biologicals, which can be caused by the development of anti-drug antibodies. The formation of high-affinity anti-drug antibodies suggests the involvement of specific CD4+ T cell responses. However, the CD4+ T cell reaction against biologicals remains poorly characterized. Therefore, in this study we analyzed CD4+ T cells specific to various biologicals and investigated potential differences of drug-specific CD4+ T cells in patients with continued remission versus those with secondary nonresponses or allergic reactions.

Methods: Peripheral blood samples were collected at baseline, and at week 6, week 14, week 22, week 30 after biological treatment. Drug-specific CD4+ T cells were analyzed using Antigen-Reactive T cell Enrichment (ARTE) directly ex vivo from human peripheral blood. To detect immunogenic regions, drug-specific CD4+ T cells were expanded and restimulated with individual peptides covering the whole variable chain of the therapeutic antibodies. Additionally, single-cell RNA sequencing was performed to investigate the gene expression profiles of drug-specific CD4+ T cells.

Results: Of the analyzed biological agents, the chimeric antibody Infliximab showed significantly higher activation of reactive CD4+ T cells compared to other humanized or fully human biological agents. Notably, this response was characterized by a highly proliferative Th1 phenotype with evidence of long-term memory. In patients with secondary nonresponses or allergic reactions IFX-specific showed elevated production of TNF-α, IFN-γ and IL-21. Restimulation experiments indicated that drug-specific CD4+ T cell responses are driven by small immunodominant peptide regions.

Discussion/Conclusion: Infliximab exhibits a higher activation of specific CD4+ T cell reactions compared to other biologicals, characterized by a proliferative Th1 response with long-term memory capabilities. Infliximab is a chimeric antibody consisting of murine variable regions, which likely contributes to its immunogenicity. The increased production of TNF- α, IFN-γ and IL-21 in patients experiencing secondary nonresponses or allergic reactions highlights the potential role of drug-specific T cells in treatment outcomes and formation of anti-drug antibodies. Overall, our data identify differences in anti-drug CD4+ T cell reactions for different biologics and as well as different treatment outcomes which may contribute to enhance drug efficacy and improve long-term success with immunogenic biologics.

4. The role of bound anti-drug antibodies to infliximab in predicting future immunogenic failure when de-escalating from combination therapy with an immunomodulator to anti-TNF monotherapy – (RAPID-IM) study

Karla Cameron (Melbourne, AU), Melissa Sam (Sydney, AU), Nithya Thennakoon (Melbourne, AU), Bonita Gu (Sydney, AU), Thomas Skinner (Sydney, AU), Arteen Arzivian (Sydney, AU), Edward Shelton (Melbourne, AU), Craig Haifer (Sydney, AU), Susan Connor (Sydney, AU), Catherine Toong (Sydney, AU), Peter R. Gibson (Melbourne, AU), David Gibson (Melbourne, AU), Alex Boussioutas (Melbourne, AU), Miles Sparrow (Melbourne, AU), Mark G. Ward (Melbourne, AU)

Introduction: Withdrawing immunomodulators (IM) from combination therapy with infliximab (IFX) may result in antibodies to IFX (ATI) and immunogenic failure. Conventional drug-sensitive ELISAs detect ATI when IFX drug levels are undetectable. We investigated whether bound-ATIs detected using a novel drug-tolerant ELISA at time of de-escalation, predicted subsequent loss of response and unfavourable IFX pharmacokinetics.

Methods: Retrospective, multicentre case-control study of IBD patients treated with combination intravenous IFX and IM. Patients had > 6 months steroid-free clinical (Harvey Bradshaw Index ≤ 4 / Partial Mayo Score ≤ 2) and biochemical remission (C-reactive protein (CRP) < 5 mg/l or faecal calprotectin (FCP) < 150 µg/g). Cases (withdrew IM and continued IFX monotherapy) were compared to controls (continued combination therapy). Primary endpoint was relapse (clinically active disease with elevated FCP/CRP) over 24 months follow-up; secondary endpoints included changes in therapy and IFX drug levels.

Results: Baseline characteristics were well matched between groups. Bound-ATI were detected in 16% cases and 14% of controls, none of whom tested positive for ATI using drug-sensitive ELISA. No patients with bound-ATI at baseline met the primary endpoint of disease relapse. Higher rates of treatment escalation (p = 0.015) in the withdrawal group were noted, but ATI status was not associated with this outcome. While there was no significant difference in change of median IFX levels over subsequent 12 months between cases (-0.99 mg/l) and controls (-0.84 mg/l; p = 0.20), all IM withdrawal patients with ATI had reduced IFX levels, with larger decreases in IFX drug levels than those without ATI (p = 0.05), an effect not observed in those continuing combination therapy.

Discussion/Conclusion: Drug-tolerant ELISA detects bound-ATI not measurable using drug-sensitive ELISA. While presence of bound-ATI did not predict poorer clinical/biochemical outcomes, it was associated with greater risk of reduction in IFX levels in those in whom immunomodulator was withdrawn. Whether this is a precursor to future immunogenic failure warrants further investigation.

5. Combined immunosuppressive therapies in inflammatory bowel disease, real-world registry data from the Persistence Australian National IBD Cohort (PANIC6) study

John Chetwood (Melbourne, AU), Sudarshan Paramsothy (Sydney, AU), Rupert Leong (Sydney, AU)

Introduction: Combination use of immunosuppressive medications are employed for refractory disease, multi-organ manifestations and multiple concurrent autoimmune diseases. However, there are extremely limited data on prescribing data and outcomes.

Methods: We interrogated the Persistence Australian National IBD Cohort (PANIC) registry for all patients with inflammatory bowel disease (IBD) on an advanced therapy (AT) receiving another immunosuppressive AT via the pharmaceutical benefits scheme (PBS) up until December 2021, with an overlap ≥ 1 month, lines were censored if overlap medications were in the same medication class. The PBS prospectively collects AT population-based prescribing data. Non-persistence was defined as failure to dispense AT prescriptions for > 6 months. Kaplan-Meier survival curves were generated for AT persistence and compared using the log-rank test.

Results: 65 combinations in 64 patients were identified. Via combination, 50/65 (76.9%) contained a tumour necrosis alpha inhibitor (TNFi), 29/65 (44.6%) contained vedolizumab (VED), 17/65 (26.1%) contained ustekinumab (UST), and 9/65 (13.9%) contained a Janus kinase inhibitor (JAK). Via class, the commonest combinations were: vedolizumab/TNFi

(16/65, 24.6%), ustekinumab/TNFi (15/65, 23.0%) and vedolizumab/dupilumab (5/65 (7.7%). Commonest IBD indications were luminal Crohn's disease (CD) (34/65, 52.3%) and ulcerative colitis (UC) (20/65, 30.8%). Commonest co-indications were rheumatoid arthritis and ankylosing spondylitis (both 16/65, 24.6%), followed by psoriatic arthritis (12/65, 18.5%) and chronic plaque psoriasis (13/65, 20.0%). Median medication overlap was 6.1 months (interquartile range [IQR], 1.0–11.2 months). There was no difference between luminal CD vs. UC for medication overlap duration (p = 0.93), nor between non-IBD indications (all p > 0.05). For IBD indications UST and VED had the longest persistence, and JAK had the poorest (overall p = 0.019). For non-IBD indications, p19 inhibitors were associated with the longest overlap duration (16.0 months, IQR, 0.98–21.3), whereas dupilumab was associated with the shortest (3.0 months, IQR, 1.0–7.1). When using TNFi for an IBD indication, ustekinumab co-therapy was associated with the greatest persistence and tocilizumab with the poorest (p = 0.12). For vedolizumab, there was no persistence difference according to co-therapy agent (all p > 0.05).

Discussion/Conclusion: Combination advanced therapy use dispensed via the PBS is uncommon. We demonstrate signals for efficacy with ustekinumab and vedolizumab when used in combination with other potent immunosuppressives. Furthermore, combinations using p19 inhibitors as co-therapy, and ustekinumab with TNFi appear to be associated with improved persistence.

6. Effect of IL-17A inhibitors on inflammatory bowel disease progress, real-world registry data from the Persistence Australian National IBD Cohort (PANIC6) study

John Chetwood (Melbourne, AU), Sudarshan Paramsothy (Sydney, AU), Rupert Leong (Sydney, AU)

Introduction: Few real-world studies have demonstrated a clear association between interleukin (IL)-17 inhibitors and inflammatory bowel disease (IBD) onset and severity.

Methods: We interrogated the Persistence Australian National IBD Cohort (PANIC) registry for all patients with inflammatory bowel disease (IBD) on an advanced therapy (AT) up until Dec 2021. The PBS prospectively collects AT population-based prescribing data. Patients who had received an IL-17 inhibitor prior to IBD AT were identified. Non-persistence was defined as failure to dispense AT prescriptions for > 6 months. Corticosteroid-free persistence was defined as non-persistence or corticosteroid use after 3 months of initiation, whichever came first. Kaplan-Meier survival curves were generated for AT persistence and compared using the log-rank test.

Results: There were 106 cases of IL-17 inhibitor use (87 with secukinumab, 19 with ixekizumab), for the indications of ankylosing spondylitis (24/106, 22.64%), chronic plaque psoriasis (35/106, 33.0%) and psoriatic arthritis (47/106, 44.3%). IL-17 use was associated with a shorter time to start an AT agent for an IBD indication vs. etantercept (p < 0.0001). Prior IL-17 use was also associated with a shorter persistence of the 1st IBD related biological agent (p < 0.0001), including with tumour necrosis alpha inhibitors (TNFi) & ustekinumab (both p < 0.0001), and vedolizumab (p = 0.030) and tofacitinib (p = 0.0018) vs. no prior use. In secondary analysis, IL-17 was associated with poorer corticosteroid-free persistence in a 1st line IBD biological agent (p < 0.0001), including with TNFi (p < 0.0001), ustekinumab (both p = 0.0083), tofacitinib (p = 0.010) though not vedolizumab (p = 0.080) vs. no prior use. Similarly for all IBD lines of therapy, prior IL-17 exposure was associated with poorer overall and corticosteroid-free persistence (both p < 0.0001), and with individual ATs (all p < 0.05) except overall persistence with vedolizumab (p = 0.16), though corticosteroid-free persistence

was poorer (p = 0.023). Overall persistence after IL-17 inhibitor was poorer versus non-us, in both ulcerative colitis and Crohn's disease (p = 0.0055 & p = 0.0088 respectively) with a non-significant trend in fistulising CD (p = 0.053). Stratified by indication, prior IL-17 use was associated with poorer IBD AT overall and corticosteroid-free persistence in patients with prior psoriatic arthritis indication for AT (p = 0.033 & p = 0.040 respectively), but not with a prior chronic plaque psoriasis nor ankylosing spondylitis indication (both p > 0.05).

Discussion/Conclusion: IL-17 inhibitor use is associated with poorer subsequent IBD persistence outcomes. Candidates for IL-17 inhibitor use should be assessed for future IBD risk.

7. Predictors of long-term anti-TNF failure: A validation analysis via the prospective Persistence Australian National IBD Cohort (PANIC) registry

John Chetwood (Melbourne, AU), Sudarshan Paramsothy (Sydney, AU), Rupert Leong (Sydney, AU)

Introduction: There are limited data on factors that predict long-term anti-TNF treatment failure. A recent UK study (PANTS-E) of 1164 patients recently suggested female gender, thiopurine commencement before biological agent, and thiopurine dose quartile predicted failure with infliximab but not adalimumab in bio-naïve Crohn's disease (BN CD). We sought to validate these findings in the Australian population

Methods: We interrogated the Persistence Australian National IBD Cohort (PANIC) registry for all patients on their 1st-line advanced therapy (AT) for IBD until Dec 2021, with a luminal CD indication. The PBS prospectively collects AT population-based prescribing data. Non-persistence was defined as failure to dispense AT prescriptions for > 6 months. Corticosteroid-free persistence was defined as non-persistence or corticosteroid use after 3 months of initiation, whichever came first. Kaplan-Meier survival curves were generated for AT persistence and compared using the log-rank test.

Results: The study population comprised 15,672 patients with a median age of 36.9 years (IQR, 26.5–50.1), and 8475 (54.1%) females, with 50,897 patient-years of follow-up. Female gender was adversely associated with overall persistence (OP) and corticosteroid-free persistence (CFP) persistence with both infliximab and adalimumab (all p < 0.0001) in follow-up > 10 years. Thiopurine co-use was protective for OP and CFP with adalimumab (p < 0.0001 & p = 0.0017), and OP but not CFP with infliximab (p = 0.018 & p = 0.26 respectively). Higher thiopurine dispensing quartile improved adalimumab persistence (p < 0.0001 & p = 0.0035 for OP and CFP respectively), and thiopurine commencement prior to biological agent induction compared to co-induction improved OP for adalimumab (p = 0.0017) though not cfp (p = 0.20). Conversely in the PANIC registry, neither affected infliximab outcomes (thiopurine dispensing quartile: p = 0.93 and p = 0.82 for OP and CFP respectively, thiopurine initiation timing: p = 0.14 & p = 0.96 for OP and CFP respectively). Independent predictors of both adalimumab and infliximab persistence were: age, gender, use of corticosteroids and immunomodulator co-therapy at induction (all p < 0.05).

Discussion/Conclusion: These findings reinforce the findings of PANTS-E which guide clinicians in how to optimise long-term outcomes with anti-TNF therapies in Crohn's disease. We confirm female gender and non-use of thiopurine at induction are associated with poorer long-term anti-TNF outcomes beyond 10 years, and there are also signals for biological-agent benefit with thiopurine dosing and timing of thiopurine commencement. However, the subtle differences in outcomes with adalimumab and infliximab between PANTS-E and PANIC merit further investigation.

8. Study on Crohn's disease obstructions and persistence with endoscopic dilatations (SCOPED): A dual-center retrospective cohort study

John Chetwood (Melbourne, AU), Sri Selvaratnam (Sydney, AU), Arteen Arzivian (Sydney, AU), Pranita Dhanji (Sydney, AU), Natalie Hung (Sydney, AU), Fei Pan (Sydney, AU), Sudarshan Paramsothy (Sydney, AU), Rupert Leong (Sydney, AU)

Introduction: Symptomatic stricturing Crohn's disease (CD) represents a challenging treatment cohort with a high proportion requiring surgery. Endoscopic balloon dilation (EBD) has shown promise in surgery-free management. There are limited data evaluating the performance of different medications combined with EBD, particularly for newer agents such as ustekinumab.

Methods: We performed a dual-center retrospective cohort study of all adult patients who underwent CD stricture dilation 2015–2023. Strictures were subclassified according to concomitant use of biological agent. Primary outcome was clinical success (1-year of surgery-free medication persistence without requirement for repeat EBD). Secondary outcomes included short-term success (1-year of surgery-free medication persistence after index EBD), surgery-free medication persistence, and adverse events. Inverse probability of treatment weighting was used to address baseline cohort imbalance.

Results: A total of 525 dilations were performed on 199 strictures in 94 patients over 120 lines of therapy. Per intention-to-treat per line of therapy, medical therapies were: immunomodulator without biologic in 42/120 (35.0%), ustekinumab in 31/120 (25.8%), vedolizumab in 13/120 (10.8%) and tumor necrosis factor inhibitors (TNFi) in 34/28.3 (28.3%). Ustekinumab was associated with the greatest surgery-free medication persistence ($p = 0.038$). At 1-year post index dilatation, surgery-free persistence was highest with vedolizumab and ustekinumab, followed by TNFi then immunomodulator ($p = 0.046$). The only independent predictor of medication failure was use of a non-ustekinumab agent (aHR = 2.80 (95% CI: 1.11–7.07, $p = 0.030$). Using inverse probability weighting adjustment, ustekinumab showed superior long-term clinical surgery-free persistence to TNFi and immunomodulator monotherapy ($p = 0.048$ and $p = 0.007$ respectively).

Discussion/Conclusion: This dual-center observational study confirms the safety and efficacy of EBD with immunomodulation for the management of stricturing CD. We demonstrate the efficacy and safety of using non TNFi agents with EBD, and that ustekinumab was associated with the highest surgery-free persistence rates.

9. Infliximab and adalimumab in the treatment of fistulizing Crohn's disease: A propensity score-matched analysis from the prospective Persistence Australian National IBD Cohort (PANIC4) study

John Chetwood (Sydney, AU), Sudarshan Paramsothy (Sydney, AU), Rupert Leong (Sydney, AU)

Introduction: There are spare data on comparative medication efficacy in fistulizing Crohn's disease (FCD), particularly with immunomodulator co-therapy. Persistence is a unique way to assess real-world outcomes.

Methods: The persistence of all dispensed biological agents were analysed from the Australian Pharmaceutical Benefits Scheme (PBS) registry data 2005–2021 for FCD. Propensity score matching was performed to account for baseline cohort imbalance.

Results: There were 5739 lines of therapy in 4466 patients over the 16-year period with 17,144 patient-years of follow-up; via therapy 2605/5739 (45.4%) used adalimumab and 3134/5739 (54.6%) used infliximab; 1697/5739 (29.6%) used thiopurine co-therapy at induction, whilst 242/5739 (4.2%) used methotrexate. As a first-line biologic (biologic-naïve), infliximab showed superior overall- and corticosteroid-free persistence to adalimumab (p = 0.0002 and p = 0.0021 respectively). used after first-line (biologic-exposed), there was no difference between agents for overall persistence (p = 0.064) though infliximab showed greater corticosteroid-free persistence (p = 0.030). Co-induction with thiopurine was associated with improved overall- and corticosteroid-free persistence (p = 0.0002 and p = 0.045 respectively). After propensity score matching, infliximab showed superior overall and corticosteroid-free persistence compared to adalimumab in bio-naïve (p < 0.0001 and p = 0.0016 respectively), not in bio-exposed patients (p = 0.12 and p = 0.074 respectively). Thiopurine was associated with superior overall- and corticosteroid-free persistence use, though no difference was seen with methotrexate.

Discussion/Conclusion: The PANIC cohort with real-world data of non-hierarchical prescribing of biological agents supports the superiority of infliximab over adalimumab in bio-naïve FCD patients, but did not show a therapeutic difference in bio-exposed FCD. Thiopurine co-therapy was independently associated with improved biological agent persistence in FCD.

10. Immune checkpoint inhibitor esophagitis with excellent response to oral viscous budesonide

Mccawley Clark-Dickson (Wahroonga, AU), Alireza Khani (Sydney, AU), T. Michael Hughes (Sydney, AU), Graeme Rich (Sydney, AU), Kazi Nahar (Sydney, AU)

Introduction: Immune checkpoint inhibitors (ICIs) are increasingly utilised in the treatment of advanced malignancy. Targeting immune checkpoints can result in significant anti-tumour effects but is also important in the regulation of autoimmunity. ICIs can result in a wide array of immune-related adverse events (irAEs) of which gastrointestinal toxicities are common. The management of upper GI irAEs (including oesophagitis) is not well defined.

Methods: A 52-year-old woman presented with a 3-month history of progressive heartburn, dysphagia and odynophagia whilst receiving ICI therapy (pembrolizumab) for early breast cancer. The symptoms were non-response to PPI and topical antacids. To investigate a gastroscopy was performed which identified oesophageal oedema and linear ulceration (figure 1). Biopsies were obtained and demonstrated an active chronic oesophagitis consistent with an ICI-related oesophagitis (see figure 2). The patient was started on oral viscous budesonide (OVB). OVB consisted of budesonide solution (1 mg in 2 ml budesonide respule) mixed with 1 teaspoon of honey and ingested BD. There was an immediate response to OVB with complete resolution of her symptoms. A follow-up gastroscopy 4 weeks later demonstrated normal oesophageal mucosa (see figure 3). The patient was treated with OVB for a further 2.5 weeks before stopping. The patient remains in clinical remission. The pembrolizumab was ceased after discussion between the patient, oncologist and gastroenterologist.

Discussion/Conclusion: This case demonstrates the successful treatment of severe ICI-related oesophagitis with OVB avoiding the need for systemic immunosuppression. The optimal treatment for ICI-related oesophagitis is not well established. Treatment for mild cases typically includes PPI, H2 receptor blockers and sucralfate but more severe cases usually require immunosuppression. The use of systemic corticosteroids, infliximab, vedolizumab and tocilizumab have been reported. Systemic immunosuppression is usually effective but is not without risks including infection and reduced cancer-related survival. In our case, it was decided

to use OVB instead of systemic corticosteroids or other immunomodulators due to its local effects and limited systemic immunosuppression. We describe the first case in the literature of ICI-related oesophagitis treated with OVB. This prevented the need for high dose systemic steroids which is associated with worse cancer-related outcomes.

11. Clinical manifestations and implications of DAO deficiency in histamine intolerance

Elena Toader (Iasi, RO), Andreea Iacob (Iasi, RO), Magda Maria Filioreanu (Iasi, RO), Alexandra Marcu (Iasi, RO), Mirela Piscuc (Iasi, RO)

Introduction: Histamine intolerance (HIT), a condition associated with an imbalance between the intake and degradation of histamine, affects approximately 1% of the general population. One of the main causes is the deficiency of diamine oxidase (DAO), the enzyme responsible for the degradation of histamine.

Methods: We present three cases of HIT due to DAO deficiency, characterized by the predominance of digestive symptoms, sometimes associated with dermatological manifestations.

Results: Case 1: Woman, 58-year-old, presented with persistent digestive symptoms, including abdominal pain and postprandial bloating, ongoing for 10 years, at times leading to episodes of food refusal. Case 2: Young man, 30-year-old, reported experiencing recurrent episodes of abdominal pain for approximately 5 years, followed by skin rashes, with no identified cause. Case 3: 54-year-old woman presents with chronic abdominal pain lasting for approximately 8 years, with variations in intensity and frequency, associated with iron deficiency anemia, vitamin deficiency and osteoporosis. A distinctive feature was identified in Case 1, where the consumption of histamine-rich foods was correlated with the exacerbation of symptoms. All patients had in common negative test results for organic digestive and extra-digestive pathologies, a protracted clinical course, refractory to symptomatic treatments, marked by restrictive diets, weight fluctuations, anxiety and depression. In all cases, DAO activity testing confirmed the enzymatic deficiency, at low values (7–10 IU/ml) associated with clinical manifestations, due to histamine overload in the body.

Discussion/Conclusion: HIT is an underdiagnosed condition, but with a significant impact on quality of life. DAO deficiency represents an essential factor in its pathogenesis. The diagnosis involves correlating symptoms with low enzymatic activity while excluding other organic causes. Management consists of early recognition, dietary adjustment, and where necessary, supplementation with DAO preparations.

12. Real-world effectiveness of risankizumab in Crohn's disease: The UK experience

Alexander Elford (Edinburgh, GB), Nikolas Plevris (Edinburgh, GB), Nathan Constantine-Cooke (Edinburgh, GB), Charlie Lees (Edinburgh, GB); Pan UK Risankizumab study working group

Introduction: Risankizumab is an IL-23 inhibitor which received approval for Crohn's disease (CD) by the NICE committee in May 2023. Our aim was to evaluate the real-world outcomes of risankizumab in the United Kingdom (UK).

Methods: We conducted a retrospective, multicentre, cohort study across 24 health boards in the UK, of patients with CD treated with risankizumab between the 1st of January, 2021 and the 1st of November 2024. Our primary outcome was treatment persistence at 6 months.

Our secondary endpoints were treatment persistence, steroid-free clinical remission (Harvey-Bradshaw index < 5), biochemical remission (CRP ≤ 5 mg) and faecal biomarker remission (FCAL < 250 µg/g), at 3, 6 and 12 months. We explored whether risankizumab effectiveness was different between ustekinumab naive and exposed patients. Patients who discontinued therapy for any reason were considered treatment-failure for all indices after the time point they ceased therapy. We recorded all adverse events, including hospitalisation, bowel resection, infection, and death.

Results: We identified 737 patients who commenced risankizumab, of which 689 patients had 3 month outcome data available with a median follow-up time of 25 weeks (IQR, 17–37 weeks). The median number of advanced therapy exposures were 3 (2–4), with 92% (635/689) having failed anti-TNF therapy, and 71% (492/689) having failed ustekinumab. Treatment persistence was 97.4%, 94.6% and 87.7% at 3, 6 and 12 months. Unadjusted persistence rates for ustekinumab naïve vs. ustekinumab exposed patients were 92.7% vs. 95.3% and 89.0 % vs. 74.2% at 6 and 12 months respectively (p = 0.2). Rates of clinical remission were 57% (262/462), 48% (93/195), and 45% (17/38) at 3, 6 and 12 months. Rates of clinical remission for ustekinumab naive vs. exposed were 61% (79/129) vs. 55% (184/333) and 55% (24/44) vs. 46% (69/151) at 3 and 6 months respectively. Biochemical remission rates were 57% (359/628), 53% (142/269), and 54% (34/63) at 3, 6 and 12 months. Faecal biomarker remission rates were 54% (163/300), 44% (60/138), and 43% (17/40) at 3, 6 and 12 months. There was a significant reduction in median HBI, CRP and FCAL during follow-up. Of the 67 patients with active perianal disease at baseline, 19% (n = 13) had a perianal complication. We observed that 3% (n = 24) underwent CD related resectional surgery, and 9% (n = 65) of patients had a hospitalisation due to symptomatic CD (n = 58) or an implicated adverse event (n = 7). Adverse events occurred in 17% (n = 120) of the cohort, of which 48% were due to symptomatic CD disease hospitalisations. Serious adverse events occurred in 12% (n = 82), of which 70 were hospitalisations.

Discussion/Conclusion: Risankizumab was effective in a large, real-world, medically refractory CD cohort with excellent short-term persistence and good clinical and biochemical remission rates. Persistence rates were similar between ustekinumab naive and exposed patients, however clinical remission rates were higher in the naive group.

13. Increased advanced therapy prescribing correlates with decreased colectomy rates in ulcerative colitis: A twenty-year population-based cohort study in Lothian, Scotland

Alexander Elford (Hobart, AU), Nathan Constantine-Cooke (Edinburgh, GB), Philip Jenkinson (Edinburgh, GB), Beatriz Gros (Edinburgh, GB), Nikolas Plevris (Edinburgh, GB), Mathew Lyons (Edinburgh, GB), Solomon Ong (Edinburgh, GB), Neil Greenlees (Edinburgh, GB), Claire Ohare (Edinburgh, GB), Nicholas Ventham (Edinburgh, GB), David Wilson (Edinburgh, GB), Paul Henderson (Edinburgh, GB), Shahida Din (Edinburgh, GB), Colin Noble (Edinburgh, GB), Gareth-Rhys Jones (Edinburgh, GB), Ian Arnott (Edinburgh, GB), Charlie Lees (Edinburgh, GB)

Introduction: The evidence for the impact of increased advanced therapy prescribing on colectomy rates in ulcerative colitis (UC) are conflicting. Our aim was to describe prescribing trends of advanced therapies (biologics and small molecules) for patients with UC in Lothian, UK and colectomy rates (elective and emergency) between January 1st, 2004, to December 31st, 2023.

Methods: Incidence and prevalence data were obtained from the validated Lothian IBD Registry. We identified patients who commenced an advanced therapy using multiple ad-

ministrative databases, and identified patients who underwent colectomy using the NHS Lothian pathology database, TrakCare theatre lists, NHS Lothian multidisciplinary meetings database, and an inpatient coding database. We collected data by manual review of the electronic health records using the TrakCare (InterSystems Corporation, Cambridge, Massachusetts, USA) system. We used SPSS Version 25 [IBM Inc., Chicago, IL, USA], Prism Version 10.0 [Graphpad Software, San Diego, CA, USA], and R 4.4.0 [R Core Team, Vienna, Austria] for statistical analyses and generation of graphs. We report advanced therapy prescription and colectomy data as both raw numbers and annual incidence rates.

Results: The prevalence of UC increased from 216 to 441 patients per 100,000 population in the twenty years from 2004, culminating in a total of 4115 patients with UC in 2023 (total Lothian population 1 million). We identified 720 patients who had received an advanced therapy. Rates of first line advanced therapy increased from 0 per 100 UC patients in 2004 to 2.82 in 2023. At the conclusion of 2023, 41%, 18%, 8%, and 3% of the advanced therapy exposed patients had received second, third, fourth, and fifth-line advanced therapy, respectively.

We identified 563 patients of the prevalent UC population who had a colectomy, of which 68% were emergency colectomies. Absolute colectomy numbers decreased from 42 in 2004 to 7 in 2023, equating to 2.48 and 0.22 per 100 UC patients. We observed that 65% of colectomies occurred in patients with extensive disease (E3), with 68% performed for acute severe UC, of which 31% occurred within 90 days of diagnosis. A total of 4% of colectomies were for cancer (n = 15) or dysplasia/suspected cancer (n = 9). Colectomies for "chronic active UC refractory to medical treatment" have almost completely disappeared with only 5 colectomies performed for this indication in the last 2 years of follow-up.

Discussion/Conclusion: We found the incidence of colectomy in the UC population progressively decreased over time, and correlated with an increased use, and number, of available advanced therapies.

14. Topical upadacitinib is effective in distal ulcerative colitis – A case study and pre-clinical proof of concept

Rabina Giri (Woolloongabba, AU), Anna Amiss (Woolloongabba, AU), Matheus Carpinelli de Jesus (Brisbane, AU), Bruce Riches (Brisbane, AU), Jakob Begun (Woolloongabba, AU)

Introduction: Ulcerative colitis (UC) is a chronic and relapsing inflammatory disease of the colon and rectum. Despite the availability of an increasing number of therapies, achieving short-term and long-term disease control remains challenging. Urgency due to distal inflammation is one of the more disabling symptoms reported by patients. Janus kinase (Jak) pathways regulate immune signalling and are implicated in UC pathogenesis. Upadacitinib, an oral Jak-1 selective inhibitor, has demonstrated efficacy in UC, but there are concerns about its use in older patients due to safety. Upadacitinib 45 mg daily is used for induction with maintenance dosing decreasing to 30 or 15 mg. In this study, we report a case on the use of upadacitinib (10 mg) daily as a suppository in a patient with severe ulcerative proctitis that had previously failed multiple advanced therapies with pharmacokinetic measurements and discuss mechanistic outcomes in pre-clinical mouse models of colitis.

Methods: Clinical data was collected from the electronic medical record. Serial endoscopic biopsies (ranging from 10–50 cm) were collected to examine a gradient of inflammatory changes and quantification of upadacitinib concentration. Single cell transcriptomics were performed to assess changes in inflammatory signatures associated with upadacitinib exposure. Comparison between oral and rectal administration of upadacitinib was determined in two colitis models: a spontaneous colitis model, Winnie, and chemically induced colitis model (DSS). Histological inflammation was assessed and correlated with tissue concentrations.

Results: There was significant improvement in endoscopic disease activity and patient-reported outcome with gradient of inflammation diminishing in severity distally. Jak/STAT related gene expression and phosphorylation of STAT-3 was negatively co-related with Upadacitinib tissue concentrations. In the pre-clinical colitis model, rectal enema significantly improved disease activity index (comprising rectal bleeding, weight loss, diarrhea) compared to oral administration of Upadacitinib. This co-related with significant reduction in histological score of inflammation in distal colon. Furthermore, upadacitinib mediated epithelial specific transcriptomics changes based on scRNA seq, supported by pre-clinical in-vivo studies.

Discussion/Conclusion: We have reported a unique case of patient undertaking Upadacitinib suppository compared to regular oral formulation which demonstrated significant efficacy with significant lower dose of Upadacitinib and shown superior efficacy of rectal enema in pre-clinical colitis models.

15. Malnutrition in Crohn's disease: Prevalence, risk factors, and key predictors

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Imen Kaffela (Tunis, TN), Haithem Yaacoub (Tunis, TN), Dhouha Cherif (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Crohn's disease (CD) is a chronic inflammatory bowel disorder often associated with significant nutritional challenges. Malnutrition in CD arises from a combination of factors, including reduced food intake, increased energy expenditure, and exudative enteropathy. This state of malnutrition poses serious risks, including increased susceptibility to infectious complications and delayed mucosal healing, which can exacerbate disease progression in these vulnerable patients. Identifying the prevalence and predictive factors of malnutrition in CD is essential to improve patient care and outcomes. This study aims to evaluate the prevalence of malnutrition in patients with CD and to investigate its predictive factors, providing valuable insights for early identification and targeted interventions.

Methods: We conducted a retrospective comparative study that included all patients diagnosed with Crohn's disease (CD) and managed in the Gastroenterology Department between January 2018 and December 2023. Nutritional status was evaluated using body mass index (BMI), with malnutrition defined as a BMI of less than 18.5 kg/m2. Comprehensive data on epidemiological, clinical, therapeutic, and disease-specific characteristics were collected. Statistical analyses were performed using SPSS software to identify predictive factors for malnutrition in CD.

Results: A total of 142 patients with Crohn's disease (CD) were included in the study. The mean age was 34 years (range: 16–86 years), with a sex ratio of 0.9 (49 men and 53 women). The mean disease duration was 3.25 years (range: 3 months–28 years). The localization of CD was colonic in 14.3% of cases, ileocolonic in 33.3%, and ileal in 52.4%. Active disease, based on the Crohn's Disease Activity Index (CDAI), was observed in 76 patients (53.5%). Malnutrition was identified in 26% of the patients. Epidemiological factors, including age ($p = 0.305$), gender ($p = 0.219$), comorbidities ($p = 0.454$), smoking status ($p = 0.471$), disease duration ($p = 0.373$), and treatments with corticosteroids ($p = 0.1$) or immunosuppressants (thiopurines: $p = 0.87$; anti-TNF alpha: $p = 0.77$), were not significantly associated with malnutrition. In contrast, univariate analysis showed that active disease ($p = 0.04$), colonic localization of the disease ($p = 0.001$), hypoalbuminemia ($p = 0.049$), and anemia ($p = 0.001$) were significantly associated with malnutrition. Multivariate analysis confirmed hypoalbuminemia and anemia as the only independent predictive factors of malnutrition in CD patients.

Discussion/Conclusion: Our study revealed that malnutrition affected one in four patients with Crohn's disease. Hypoalbuminemia and anemia were identified as the two primary predictive factors for malnutrition.

16. Acute abdomen in Crohn's disease: A comparative analysis of surgery – First diagnosis versus known cases

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Rahma Chaouachi (Tunis, TN), Dhouha Cherif (Tunis, TN), Haithem Yaacoub (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Crohn's disease (CD) is a heterogeneous condition with unpredictable clinical behavior. Surgical bowel resection is a frequent outcome during the disease course. This study aimed to compare the clinical and surgical characteristics of patients whose CD was first diagnosed during surgery for acute abdomen and those with known CD who underwent surgery for acute abdomen.

Methods: We conducted a retrospective study from January 2012 to September 2024, including CD patients who underwent surgery for acute abdomen. Patients were categorized into two groups: Group A (GA) included those whose CD was first diagnosed during surgery, and Group B (GB) included patients with known CD who developed acute abdomen. Data on personal history, clinical features, and medical or surgical management during follow-up were collected from patient records.

Results: A total of 74 patients were included, comprising 55 males and 19 females, with a mean age of 37 years (range: 16–61). CD was first diagnosed during surgery in 28.4% of cases (GA), while 71.6% of patients had a prior diagnosis of CD (GB). Presentations included intestinal obstruction (60.8%), acute peritonitis (24.3%), and appendicitis (14.8%).

No significant differences were observed between the two groups regarding sex, extraintestinal manifestations, perineal involvement, or disease location. However, disease behavior was more commonly penetrating and/or stricturing in GA compared to GB (penetrating: GA 52.4% vs. GB 15.1%, $p = 0.0031$; stricturing: GA 71.4% vs. GB 26.4%, $p = 0.022$). In GB, 67.9% of patients were receiving corticosteroids at the time of acute abdomen onset.

Discussion/Conclusion: CD first diagnosed during surgery for acute abdomen was significantly associated with a family history of inflammatory bowel disease, active smoking, and penetrating or stricturing disease behavior. Conversely, acute abdomen in known CD patients was associated with corticosteroid use, underscoring the need for tailored management strategies in these distinct clinical scenarios.

17. Can the occurrence of perianal lesions in Crohn's disease be predicted?

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Rahma Chaouachi (Tunis, TN), Haithem Yaacoub (Tunis, TN), Dhouha Cherif (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Perianal lesions (PAL) are a frequent and debilitating complication of Crohn's disease (CD), which can occur at any stage of its evolution. Identifying predictive factors for their occurrence is essential for early intervention and management. This study aimed to determine the risk factors associated with the development of PL in CD.

Methods: We conducted an analytical retrospective study including patients diagnosed with CD and followed between January 2016 and December 2023. All patients underwent systematic proctological examination, and pelvic MRI was performed if a fistulous tract was

identified during clinical assessment. Clinical, biological, endoscopic, and radiological data were collected and analyzed.

Results: Seventy-four patients were included, with a mean age of 33 years (range: 14–60) and an equal sex ratio (1:1). Active smoking was reported in 31% of patients. Disease localization was ileal in 28%, colonic in 32%, and ileocecal in 40%. Rectal involvement was observed in 60% of cases. The disease exhibited a stenosing phenotype in 28% and a fistulizing phenotype (excluding PAL) in 29%. PAL were identified in 33% of patients (n = 25). PAL were classified as follows: 60% (n = 15) perianal complex fistulas, 12% (n = 3) simple perianal fistulas, 12% (n = 3) anovulvar fistulas, 8% (n = 2) rectovaginal fistulas, one case of anal margin abscess, and one case of anal fissure. Univariate analysis identified several factors associated with the occurrence of PL: younger age at CD onset (p = 0.02), male sex (p = 0.03), rectal involvement (p = 0.001), and active smoking (p = 0.002). Multivariate analysis confirmed that active smoking, rectal involvement, and male sex were independent predictive factors for the development of PL.

Discussion/Conclusion: Our study demonstrated that male sex, rectal involvement, and active smoking are the primary predictive factors for the occurrence of PL in CD. Efforts to reduce smoking should be prioritized to decrease the frequency and severity of PL in these patients.

18. Clinical features of elderly-onset ulcerative colitis

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Rahma Chaouachi (Tunis, TN), Haithem Yaacoub (Tunis, TN), Dhouha Cherif (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Ulcerative colitis (UC) exhibits a bimodal age distribution and is increasingly recognized as a disease affecting the elderly. It remains unclear whether elderly-onset UC represents a distinct phenotypic subgroup requiring tailored treatment strategies. Information on the disease course and treatment response in elderly-onset UC is limited and often conflicting. This study aimed to compare the disease course and treatment response between adult-onset (AO) and elderly-onset (EO) UC in our population.

Methods: We conducted a retrospective study between January 2012 and September 2014, including patients hospitalized for the treatment of their first UC flare. Patients were categorized into two groups: adult-onset UC (AO), defined as diagnosis before 50 years of age, and elderly-onset UC (EO), defined as diagnosis at or after 50 years of age. Epidemiological, clinical, and therapeutic characteristics were extracted from medical records for analysis.

Results: A total of 92 patients were included (52 males, 40 females), with a mean age of 39 years (range: 18–69). Among these, 21 patients (22.8%) were identified as EO and 61 (66.2%) as AO. In the EO group, males represented 61.9% of cases. EO patients were more likely to present with left-sided disease (57.1% vs. 44.2%) and less likely to have rectal disease (23.8% vs. 36%) at diagnosis. There were no significant differences between the two groups regarding the risk of multiple hospitalizations, colectomy, or disease extent progression. EO patients, however, were less likely to receive immunosuppressive or anti-TNFα therapy compared to AO patients.

Discussion/Conclusion: Elderly-onset UC exhibits distinct clinical behavior compared to adult-onset UC, characterized by reduced use of immunosuppressive and anti-TNFα therapies without an associated increase in hospitalization rates or colectomy risk. These findings suggest that treatment strategies for elderly-onset UC may differ from those for adult-onset UC, warranting further investigation.

19. Clinical, therapeutic, and evolutionary characteristics of severe acute colitis in Crohn's disease

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Rahma Chaouechi (Tunis, TN), Dhouha Cherif (Tunis, TN), Haithem Yaacoub (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Isolated colonic or rectal involvement in Crohn's disease (CD), known as Crohn's colitis, occurs in approximately 25% of patients. Limited data are available on the clinical course of severe colonic Crohn's disease. This study aimed to evaluate the clinical, therapeutic, and evolutionary characteristics of severe acute colitis (SAC) in patients with CD.

Methods: We conducted a retrospective comparative study over eight years (January 2016–December 2023), including all patients hospitalized for SAC. The diagnosis of SAC was based on True-Love and Witts criteria. Clinical data, type of inflammatory bowel disease (IBD), disease duration, therapeutic management, response to medical treatment, and colectomy rates were collected. These parameters were compared between two groups: Group 1 (G1) with SAC in CD and Group 2 (G2) with SAC in ulcerative colitis (UC).

Results: Sixteen patients were included during the study period, of whom ten (63%) had SAC complicating CD, and six (37%) had SAC complicating UC. The mean age was significantly lower in G1 (28 years, range: 18–34) compared to G2 (42 years, range: 22–72) ($p = 0.01$). The sex ratio (male/female) was 1:9 in G1 and 1:2 in G2, with no significant difference ($p > 0.05$). Steroid response rates were notably higher in G1 (80%) than in G2 (16.6%) ($p = 0.002$). Second-line treatment with cyclosporine was used in 20% of G1 cases and 33.3% of G2 cases ($p > 0.05$). Subtotal colectomy was not performed in G1 but was required in 50% of G2 patients ($p = 0.03$). These findings highlight distinct clinical and therapeutic profiles between the groups, with G2 demonstrating older age, lower steroid response rates, and a higher need for surgical intervention.

Discussion/Conclusion: In our study, SAC complicating CD primarily affected younger patients and responded favorably to first-line treatment, with no cases requiring colectomy. These results warrant confirmation through larger-scale studies to better characterize these observations.

20. Extent of disease as a predictor of bone loss in inflammatory bowel disease: A retrospective study

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Imen Kaffela (Tunis, TN), Haithem Yaacoub (Tunis, TN), Dhouha Cherif (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Bone mineralization disorders are well-recognized complications of inflammatory bowel disease (IBD), encompassing both Crohn's disease (CD) and ulcerative colitis (UC). These disorders, including osteopenia and osteoporosis, significantly contribute to the morbidity associated with IBD by increasing the risk of fractures and impairing quality of life. The pathophysiology of bone loss in IBD is multifactorial, involving chronic inflammation, corticosteroid use, malnutrition, and altered calcium and vitamin D metabolism. While numerous studies have investigated the prevalence and risk factors for bone loss in IBD, the specific influence of disease extent on bone mineral density (BMD) remains insufficiently addressed. This study aimed to evaluate the correlation between disease extent and bone loss in patients with IBD.

Methods: This was a retrospective study conducted between January 2016 and June 2024, including all patients with IBD who underwent dual-energy X-ray absorptiometry (DXA) for BMD evaluation. BMD was measured as bone mass in g/cm2, and the T-score was expressed in standard deviations (SD). Patients with CD were classified according to the Montreal classification into four categories based on disease location: L1 (ileal), L2 (colonic), L3 (ileocolonic), and L4 (upper gastrointestinal involvement). Similarly, patients with ulcerative colitis (UC) were categorized into three classes based on disease extent: E1 (proctitis), E2 (left-sided colitis), and E3 (pancolitis). Patients who had undergone surgical interventions for IBD were excluded from the study.

Results: A total of 182 patients with IBD were included in the study, comprising 132 patients with CD (72.5%) and 50 patients with UC (27.4%). The mean age at diagnosis was 33.2 years (range: 15–78 years). According to the Montreal classification, among UC patients, 42% (21/50) were classified as E1, 36% (18/50) as E2, and 22% (11/50) as E3. Among CD patients, 38.6% (51/132) were classified as L1, 31% (41/132) as L2, 28.8% (38/132) as L3, and 1.3% (2/132) as L4. A total of 39 patients (21.4%) had extensive digestive involvement. BMD was normal in 76 patients (41.7%), while 106 patients (58.2%) exhibited mineralization disorders, including 71 cases of osteopenia (39%) and 35 cases of osteoporosis (19.2%). In the CD group, 60.6% (80/132) had low BMD, with 53 cases of osteopenia and 27 cases of osteoporosis. In the UC group, 53.6% (26/50) had low BMD, with 20 cases of osteopenia and 6 cases of osteoporosis. A significant positive correlation was observed between the extent of digestive involvement and reduced BMD in both univariate ($p = 0.001$) and multivariate analyses ($p = 0.006$).

Discussion/Conclusion: Bone loss is a prevalent complication in IBD. Our study highlights a significant correlation between extensive disease involvement and the risk of bone demineralization, underscoring the critical importance of prioritizing skeletal health in the management of IBD.

21. Is proctitis in Crohn's disease a factor in the persistence of perianal fistulas?

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Rahma Chaouachi (Tunis, TN), Dhouha Cherif (Tunis, TN), Haithem Yaacoub (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Rectal involvement in Crohn's disease (CD) is recognized as a factor contributing to poor therapeutic outcomes for perianal fistulas (PAF). This study aimed to evaluate the impact of rectal mucosal resolution on PAF healing in CD patients treated with anti-TNFα therapy.

Methods: This was a single-center retrospective study, including records of patients with CD and PAFs treated with anti-TNFα therapy over a 10-year period (2010–2020). Clinical, biological, endoscopic, and radiological data were collected, along with therapeutic outcomes under anti-TNFα therapy. Therapeutic response was assessed both clinically and radiologically using pelvic MRI. Rectal mucosal healing was evaluated and compared to PAF recovery.

Results: A total of 38 patients were included, with a mean age of 42 years (range: 26–73) and a male-to-female ratio of 0.4. Perianal involvement was isolated in 5.2% of cases, while 94.7% had associated luminal disease. Rectal involvement was present in 60.5% of patients. PAFs were complex in 92.1% of cases, accompanied by perianal abscesses in 44.7% and anorectal strictures in 7.9%.

All patients received anti-TNFα therapy (infliximab: 92.1%; adalimumab: 7.9%) after resolution

of collections, combined with thiopurines in 97.4% of cases. After a mean follow-up of 40 months, rectal mucosal healing was associated with PAF healing in 10 patients (43.5%). PAF healing with persistent rectal inflammation was observed in 6 patients (26.1%), while rectal healing with persistent PAFs occurred in only 1 patient (4.3%). Neither rectal nor PAF healing was observed in 6 patients (26.1%). Univariate analysis using the Chi-square test found no statistically significant correlation between rectal mucosal healing and PAF healing after anti-TNFα therapy (p = 0.069, 95% confidence interval).

Discussion/Conclusion: Our findings suggest that rectal mucosal healing does not significantly influence the natural history of suppurative PAF in CD. Larger-scale studies are needed to confirm these results.

22. Predictive factors for surgical intervention in Crohn's disease

Hajer Hassine (Tunis, TN), Nesrine Krifa (Tunis, TN), Habiba Dabbebi (Tunis, TN), Imen Kaffela (Tunis, TN), Haithem Yaacoub (Tunis, TN), Dhouha Cherif (Tunis, TN), Hela Kchir (Tunis, TN), Nadia Maamouri (Tunis, TN)

Introduction: Surgery remains a crucial component in the management of Crohn's disease (CD), despite the increasing and earlier use of immunosuppressive therapies. The persistent need for surgical intervention underscores the importance of identifying factors that predict surgical outcomes. This study aimed to determine the key predictive factors associated with the need for surgery in patients with CD, providing insights into risk stratification and optimizing patient outcomes.

Methods: We conducted an analytical retrospective study by reviewing the medical records of patients diagnosed with CD and followed between January 2016 and December 2023. Data collected included patient demographics, disease characteristics, complications, and treatment modalities, with a particular emphasis on cases requiring surgical management. This approach provided a comprehensive understanding of the characteristics and severity of surgical forms of CD. Statistical analyses were performed to assess correlations and determine independent predictors of surgical outcomes.

Results: A total of 74 patients were included in the study, with a mean age of 33 years (range, 14–60) and an equal sex distribution (sex ratio 1:1). Nearly half of the patients (48%) were under 30 years of age, and 31% were active smokers. The most frequent disease localization was ileocecal (40%), followed by colonic (32%) and ileal (28%) involvement. The disease exhibited a stenosing phenotype in 28% of cases and a fistulizing phenotype (excluding perianal involvement) in 29%. Perianal disease was observed in 33% of patients (n = 25). Among the study cohort, 29 patients underwent surgical intervention. In 20 cases, surgery was performed on an emergency basis, while 9 cases were managed electively. The most frequently performed surgical procedures included ileocecal resection (23 patients), subtotal colectomy (3 patients), and small bowel resection (2 patients). Univariate analysis identified several factors significantly associated with the need for surgery: age under 30 years (p = 0.013), fistulizing phenotype (p = 0.02), active smoking (p = 0.03), corticosteroid resistance (p = 0.025), and the presence of severe endoscopic findings (p = 0.015). Multivariate analysis further confirmed age under 30 years, active smoking, fistulizing phenotype, and corticosteroid resistance as independent predictors of surgical intervention.

Discussion/Conclusion: Our study demonstrated that the primary predictive factors for surgical intervention in Crohn's disease are age under 30 years, active smoking, a fistulizing disease phenotype, and corticosteroid resistance. These findings underline the importance of early identification and targeted management of high-risk patients to potentially reduce the need for surgical treatment.

23. Infra-red microspectroscopy provides quantitative assessment of fibro-inflammation in Crohn's disease strictures and predicts postoperative recurrence

Charlotte Keung (Melbourne, AU), Rhonda Pawlowski (Melbourne, AU), Andrew Ryan (Melbourne, AU), Edward Kwan (Melbourne, AU), Anthony Longano (Melbourne, AU), Rebecca Lim (Melbourne, AU), William Sievert (Melbourne, AU), Gregory Moore (Melbourne, AU), Bayden Wood (Melbourne, AU)

Introduction: Quantitative assessment of fibro-inflammation in Crohn's disease strictures remains a challenge despite its crucial role in clinical decision making using medical therapies or endoscopic/surgical interventions. Fourier-transform infra-red (FTIR) spectroscopy is a reproducible, label-free and non-destructive approach for analysing biochemical tissue composition. We have previously showed accurate assessment of inflammation using this technique in colitis models. The aim of this study was to characterise Crohn's disease strictures using FTIR spectroscopy and to assess utility for predicting postoperative Crohn's disease recurrence.

Methods: Full-thickness Crohn's stricture resections (n = 72) were obtained in paraffin and sectioned for hyperspectral imaging using the Agilent Cary 670 FPA-IR in transflection mode in the wavenumber region 1800–800 cm-1. Spectra was transformed to the second derivative and normalised prior to analysis using partial least squares discriminant analysis (PLSDA) with internal cross-validation. Adjacent tissue sections were stained with H&E, Masson's trichrome and α-smooth muscle actin with histology scored by three independent clinical histopathologists, with their final scores averaged for inflammation, fibrosis and muscular changes.

Results: Tissue infra-red spectra demonstrated excellent performance defined by area under the receiver operating characteristic curve (AUC) of > 0.9 using PLSDA to classify severe from mild inflammation and good (AUC, > 0.8) to excellent (AUC, > 0.9) performance to classify severe from mild fibrosis and muscular hyperplasia/hypertrophy. In those with complete macroscopic Crohn's disease resection (n = 51), spectra from index stricture resections demonstrated good performance (AUC, > 0.8) in discriminating between future significant endoscopic recurrence defined by Rutgeert's score ≥ i2 and excellent performance for surgical recurrence (AUC, > 0.9) defined as future surgical resection of Crohn's recurrence.

Discussion/Conclusion: FTIR microspectroscopy can detect differences in biochemistry underlying inflammation, fibrosis and muscular changes in Crohn's strictures and provide simultaneous quantitative assessment. The biochemical tissue fingerprints demonstrate good to excellent discrimination for predicting endoscopic and surgical Crohn's recurrence, respectively.

24. Infra-red spectroscopy predicts response to anti-tumour necrosis factor-α therapy in fibrostenotic Crohn's disease

Charlotte Keung (Melbourne, AU), Jeanne Correia (Melbourne, AU), Anthony Longano (Melbourne, AU), Julien Schulberg (Melbourne, AU), Emily Wright (Melbourne, AU), Amy Hamilton (Melbourne, AU), Michael Kamm (Melbourne, AU), Rebecca Lim (Melbourne, AU), William Sievert (Melbourne, AU), Bayden Wood (Melbourne, AU), Gregory Moore (Melbourne, AU)

Introduction: Crohn's fibrostenotic strictures lack validated biomarkers that predict treatment response. Fourier-transform infra-red (FTIR) spectroscopy provides reproducible biochemical information using a rapid, label-free, non-destructive technique via molecular vibrations. The

aim was to investigate if FTIR spectroscopy could predict patient response to anti-tumour necrosis-factor-α therapy and identify spectral biomarkers for treatment response.

Methods: Hyperspectral images were acquired using the Agilent Cary 670 FPA-IR in transflection mode in the wavenumber region 1800–800 cm-1 from endoscopic biopsies from 62 patients with Crohn's disease and 12 healthy controls including a subset (n = 24) from STRIDENT (NCT03220841), a randomised study assessing adalimumab therapy for intestinal Crohn's strictures. Spectra were transformed to the second derivative and normalised prior to analysis using partial least squares discriminant analysis (PLSDA) with internal cross-validation. Spectral biomarkers were identified by the most contributive infra-red bands from the latent variables and variable importance in projection scores of > 1. Adjacent histological sections were stained with H&E, Masson's trichrome and α-smooth muscle actin with inflammation and fibrosis scored by a clinical pathologist.

Results: Spectra from baseline biopsies from 24 patients in the STRIDENT study demonstrated excellent performance using PLSDA in discriminating between responders and non-responders in relation to the 12-month clinical outcomes, defined by area under the receiver operating characteristic curve (AUC) > 0.9 for MRI and IUS responses, normalisation of faecal calprotectin and CRP, CDAI and pain responses. Spectral biomarkers for stricture response to adalimumab include the amide I and II protein bands (peaks at 1651 and 1543 cm-1, respectively), the carboxylate group of proteins (1454 cm-1), lipid bands (1750 and 1395 cm-1), collagen band (1236 cm-1), glycoprotein bands (1081 and 1030 cm-1), and nucleic acid bands (1236, 1081 and 966 cm-1).

Discussion/Conclusion: FTIR spectroscopy detects tissue biochemical "fingerprints" in fibrostenotic Crohn's disease that demonstrate excellent discrimination for predicting clinical response to adalimumab therapy.

25. Comparative accuracy and reliability of handheld versus cart-based ultrasound in assessing inflammatory bowel disease activity

Myat Myat Khaing (Brisbane, AU), Richard Fernandes (Brisbane, AU), Yoon-Kyo An (Brisbane, AU), Brandon Baraty (Brisbane, AU), Robert Bryant (Brisbane, AU), Simon Ghaly (Sydney, AU), Rebecca Smith (Melbourne, AU), Ashish Srinivasan (Melbourne, AU), Karen Hay (Brisbane, AU), Jakob Begun (Brisbane, AU)

Introduction: Bowel wall thickness (BWT) and vascularity are key parameters for assessing inflammatory bowel disease (IBD) activity. Handheld ultrasound devices (HHUS) provide a portable and affordable alternative for incorporating intestinal ultrasound (IUS) into IBD management. However, their diagnostic accuracy and reliability compared to cart-based ultrasound (CBUS) remain uncertain. This study assesses the diagnostic accuracy, reproducibility, and practitioner confidence with HHUS in comparison to CBUS for assessing BWT and vascularity.

Methods: In this prospective, comparative study, 12 patients with IBD, with body mass index (BMI) < 23 kg/m2, were assessed. Each patient underwent ultrasound scanning of 2–3 pre-specified bowel segments using five devices: one CBUS device (Samsung™) and four HHUS devices (Clarius™ L15 HD3, GE™ Vscan Air CL, Mindray™ TE Air, and Philips™ Lumify L12-4). Linear probes were used for all devices except Mindray™, which employs a phased array probe optimised for cardiac imaging. Scans were conducted by two to three gastroenterologists accredited by the Gastroenterology Network of Intestinal Ultrasound (GENIUS), who were blinded to each other's findings to reduce bias. BWT and Doppler vascularity, as well as other standard IUS parameters, were assessed for each segment. Diagnostic accuracy and inter-/intra-expert agreement were analysed using statistical models.

Results: Among the 12 patients (42% male; median age: 26 years, IQR: 23–36) with CD (n = 6) and UC (n = 6), a total of 29 bowel segments were assessed with all devices (Table 1). BWT measurements showed high concordance across devices for active segments (mean BWT: 4.3–4.6 mm, p = 0.73). However, significant discrepancies were noted in inactive segments ($p < 0.001$), particularly with the Mindray™ device showing larger deviations from the CBUS measurements. Intraclass Correlation Coefficients (ICC) for intra-expert BWT measurements were good to excellent across devices (ICC 0.79–0.97), while inter-expert agreement was good for all devices (ICC 0.70–0.77) except for Mindray™ (ICC 0.36) (Figure 1B). Significant confidence was noted for BWT, vascularity, stratification, mesenteric fat, and inflamed bowel length across devices, highlighting consistent measurement reliability for these parameters (Table 1).

Discussion/Conclusion: The majority of HHUS devices used in this study demonstrate high diagnostic accuracy and reproducibility in assessing IBD activity and are comparable to CBUS for identifying and assessing active disease. This study supports the integration of HHUS into IBD management as a portable and accessible tool while highlighting the importance of recognising device-specific limitations when selecting devices for clinical practice.

26. Tofacitinib demonstrates preliminary efficacy in induction of remission in chronic pouchitis

Emi Khoo (Brisbane, AU), Anna Amiss (Woolloongabba, AU), John Nik Ding (Melbourne, AU), Susan Connor (Sydney, AU), Lauren White (Sunshine Coast, AU), Rupert Leong (Sydney, AU), Waled Mohsen (Southport, AU), Robert Bryant (Brisbane, AU), Zaid Ardalan (Melbourne, AU), Peter De Cruz (Melbourne, AU), Anthony Croft (Herston, AU), Kate Lynch (Adelaide, AU), Yoon-Kyo An (Brisbane, AU), Gerald Holtmann (Woolloongabba, AU), Jakob Begun (Brisbane, AU)

Introduction: Pouchitis, a common complication after total proctocolectomy for ulcerative colitis (UC), usually responds to antibiotics. However, up to 20% of patients develop antibiotic-dependent or antibiotic-refractory pouchitis, termed chronic pouchitis.

Methods: We report preliminary induction results from a multi-center open label induction with randomized withdrawal trial to assess the efficacy of tofacitinib in chronic pouchitis. Eligible participants received 10 mg tofacitinib twice daily for 8 weeks, with an additional 8 weeks for non-responders. Primary endpoint was clinical response (a reduction of clinical PDAI > 2) at week 8. Secondary endpoints included endoscopic response (a reduction of endoscopic PDAI > 2), mPDAI remission (modified pouchitis disease activity index (mPDAI) < 4 and a reduction of mPDAI > 2), mPDAI response (a reduction of mPDAI > 2) and improvement in health-related quality of life (HRQoL). Serum cytokine levels were analyzed using LEGENDplex human inflammation panel 1. Univariate analysis was performed to identify clinical and biochemical predictors of response to tofacitinib.

Results: A total of 47 patients with chronic pouchitis were included in the preliminary analysis. The clinical and endoscopic response rates at week 8 were 57% and 52% respectively, with a median improvement of the score of 2 ($p < 0.05$) and 2 ($p < 0.05$) respectively. The mPDAI remission rate was 34% at week 8 with an mPDAI response rate of 62%. There was significant improvement in HRQoL, with the median SIBDQ score increasing from 36 at baseline to 53 at week 8 ($p < 0.05$). Vedolizumab-exposed and naïve groups had similar clinical response rates at week 8 (50% vs. 59%, respectively). Twenty patients received a further 8 weeks of therapy with a clinical response rate of 30%, resulting in an overall response rate including extended induction of 72%. A significant reduction in average bowel movements per day, decreased urgency, improved confidence in passing wind, reduced nocturnal symp-

toms, decreased faecal calprotectin levels, and increased high-density lipoprotein (HDL) cholesterol levels from baseline to the end of the induction phase were documented ($p < 0.05$). However, there were no significant changes observed in hemoglobin, creatine kinase, total cholesterol, or low-density lipoprotein (LDL) cholesterol levels. Univariate analysis revealed that patients with nocturnal symptoms, endoscopic friability, and elevated baseline levels of IL-1β, IFN-α, and IL-33 were more likely to experience a treatment response at week 8 compared to those without these factors ($p < 0.05$).

Discussion/Conclusion: Tofacitinib demonstrated efficacy in inducing both clinical and endoscopic response, as well as improving quality of life, in patients with chronic pouchitis who have undergone IPAA for UC. Notably, tofacitinib was equally effective in patients who had previously been exposed to vedolizumab and those who were naïve to the treatment.

27. Combined oral mesalamine and budesonide suppositories yield faster and better symptom resolution in active ulcerative colitis than oral mesalamine monotherapy: A randomized, double-blind, placebo-controlled add-on study

Wolfgang Kruis (Cologne, DE), Adrian Wrozek (Staszów, PL), Lukasz Wolanski (Pulawy, PL), Oleksandr Oliinyk (Zaporizhzhia, UA), Pavel Andreev (Samara, RU), Anne Mueller (Freiburg, DE), Ralf Mohrbacher (Freiburg, DE), Ralph Mueller (Freiburg, DE), Britta Siegmund (Berlin, DE)

Introduction: Quick resolution of rectal symptoms affects the therapeutic success in active ulcerative colitis (UC) considerably. The combination of oral and rectal therapy leads to high drug concentrations in the rectum and thus, yields accelerated improvement of active UC.

Methods: This randomized, double-blind, multicentric, placebo-controlled, 8-weeks trial compared once daily (OD) oral mesalamine 3 g granules (morning) and OD rectal budesonide placebo suppository (bedtime) (MES; monotherapy) to a combination treatment with OD oral mesalamine 3 g granules (morning) and OD rectal budesonide 4 mg suppository (bedtime) (MES/BUS; combination therapy). Primary and secondary endpoints were time to resolution of clinical symptoms (defined as time from baseline visit to the first of 3 consecutive days with a score of 0 for both rectal bleeding and stool frequency or duration of observation for patients without resolution) and treatment effects at week 4 and 8. The war between Russia and Ukraine caused premature termination of the trial. Inclusion criteria: Confirmed diagnosis of UC, bloody stools occurring at least during 28 days prior to baseline visit; mildly to moderately active disease ($3 <$ modified UC-DAI < 11 with rectal bleeding subscore of ≥ 1, endoscopic subscore of ≥ 2).

Results: A total of 99 patients with histological confirmed diagnosis of UC were enrolled. 30 patients failed screening and 69 were randomized. Thereof 35 patients received MES and 34 patients MES/BUS. Patients were analyzed in a modified Intention-to-treat analysis set (mITT, analyzed as treated). The extent of UC ranged between 5 and 80 cm ab ano and was comparable between both groups. Mean (± SD) time to resolution of clinical symptoms was 37.0 ± 22.0 days for MES/BUS and 43.6 ± 15.5 days for MES ($p < 0.03$) counting for a shorter resolution time of 6.6 days for MES/BUS. Overall, there was a non-significant trend towards therapeutic superiority of MES/BUS combination therapy. Intragroup comparisons of individual UC-DAI subscores between baseline and week 4 as well as week 8/withdrawals showed significant improvements ($p < 0.001$) for both therapies. Treatment with BUS and MES were well tolerated and safe. No unexpected or serious events were reported during the treatment phase including the follow-up and no patients had to be withdrawn from the study prematurely. A decrease of serum-cortisol occurred within the normal range in 5.9% of

subjects in the MES/BUS group. At follow-up, the values increased again but didn't normalize completely since some patients were prescribed corticosteroids as follow-up treatment.

Discussion/Conclusion: Both, once daily oral mesalamine monotherapy (MES) and the combination of once daily oral mesalamine and rectal budesonide suppository (MES/BUS) are very effective treatments for active UC, but resolution of symptoms is accomplished more rapidly, and improvement of clinical symptoms is achieved by more subjects with combination therapy. This suggests combination therapy as the preferred treatment in UC.

28. Early faecal calprotectin – A novel predictor of clinical outcomes in acute severe ulcerative colitis: Results from PREDICT-UC

Christopher Li Wai Suen (Melbourne, AU), Matthew Choy (Melbourne, AU), Danny Con (Melbourne, AU), Kate Driver (Sydney, AU), Dylan Bruen (Sydney, AU), Joshua Glason (Sydney, AU), Fabrizio Bonelli (Saluggia, IT), Intissar Bittar (Melbourne, AU), Kristy Boyd (Melbourne, AU), Raquel Pena (Melbourne, AU), Kathryn Burrell (Melbourne, AU), Ourania Rosella (Melbourne, AU), David Proud (Melbourne, AU), Richard Brouwer (Melbourne, AU), Alexandra Gorelik (Melbourne, AU), Danny Liew (Adelaide, AU), William Connell (Melbourne, AU), Emily Wright (Melbourne, AU), Kirstin Taylor (Melbourne, AU), Aviv Pudipeddi (Sydney, AU), Michelle Sawers (Geelong, AU), Britt Christensen (Melbourne, AU), Watson Ng (Sydney, AU), Jakob Begun (Brisbane, AU), Graham Radford-Smith (Brisbane, AU), Mayur Garg (Melbourne, AU), Neal Martin (Brisbane, AU), Daniel Van Langenberg (Melbourne, AU), Nik Ding (Melbourne, AU), Lauren Beswick (Geelong, AU), Rupert Leong (Sydney, AU), Miles Sparrow (Melbourne, AU), Peter De Cruz (Melbourne, AU)

Introduction: The role of faecal calprotectin in the management of acute severe ulcerative colitis (ASUC) is unknown. We aimed to evaluate the relationship between FCP and outcomes in ASUC.

Methods: We included ASUC patients who were screened/randomised as part of PREDICT-UC (NCT02770040), a randomised controlled trial that evaluated escalated infliximab (IFX) dosing strategies in steroid-refractory ASUC. Stool was collected at screening, and in steroid-refractory patients at day 0 (pre-IFX), and days 1, 3, 5, 7, 14, 30, 42, and months 3, 6, 9, and 12 post-IFX. FCP levels were measured by Liaison® XL (Diasorin). Outcomes included initial IFX response by day 7 (Lichtiger score < 10), month 3 colectomy, and month 3 remission (partial Mayo ≤ 1 & Mayo endoscopic subscore ≤ 1).

Results: Among 185 ASUC patients, 136 were steroid-refractory and received IFX. Of these, 85 responded to IFX, and 17 required colectomy. Screening FCP was higher in steroid-refractory compared to steroid-responsive patients (median 3745 ug/g vs. 2305 ug/g, p = 0.020). In steroid-refractory patients, day 0 FCP correlated with CRP (rho = 0.251, p = 0.031) and the erosion/ulcer sub-score of UCEIS (rho = 0.298, p = 0.010). In linear mixed modelling, FCP dynamics in the first 3 days differed between IFX responders and non-responders (daily 22% decrease vs. 3% decrease, p = 0.018). A higher day 3:day 0 FCP ratio also predicted non-response (AUROC = 0.72, p = 0.006). FCP dynamics in the first two weeks after IFX predicted colectomy (p = 0.012). Month 3 colectomy was also predicted by a higher day 7 FCP (AUROC = 0.71, p = 0.044) and a higher day 3:day 0 FCP ratio (AUROC = 0.75, p = 0.018). A lower day 14 FCP was associated with month 3 remission (AUROC = 0.31, p = 0.004).

Discussion/Conclusion: FCP levels and dynamics are novel predictors of outcomes in ASUC. Decreases in FCP by day 3 after IFX predict response, while day 7 FCP and early dynamics predict colectomy. Day 14 FCP levels may help identify patients who may benefit from treatment optimisation.

29. The role of early serum infliximab levels in predicting outcomes in acute severe ulcerative colitis: Results from PREDICT-UC

Christopher Li Wai Suen (Melbourne, AU), Matthew Choy (Melbourne, AU), Danny Con (Melbourne, AU), Kaylene Cheng (Melbourne, AU), Julie Nigro (Melbourne, AU), Kerry Breheney (Melbourne, AU), Kristy Boyd (Melbourne, AU), Raquel Pena (Melbourne, AU), Kathryn Burrell (Melbourne, AU), Ourania Rosella (Melbourne, AU), David Proud (Melbourne, AU), Richard Brouwer (Melbourne, AU), Alexandra Gorelik (Melbourne, AU), Danny Liew (Adelaide, AU), William Connell (Melbourne, AU), Emily Wright (Melbourne, AU), Kirstin Taylor (Melbourne, AU), Aviv Pudipeddi (Sydney, AU), Michelle Sawers (Geelong, AU), Britt Christensen (Melbourne, AU), Watson Ng (Sydney, AU), Jakob Begun (Brisbane, AU), Graham Radford-Smith (Brisbane, AU), Mayur Garg (Melbourne, AU), Neal Martin (Brisbane, AU), Daniel Van Langenberg (Melbourne, AU), Nik Ding (Melbourne, AU), Lauren Beswick (Geelong, AU), Rupert Leong (Sydney, AU), Miles Sparrow (Melbourne, AU), Christina Grosserichter-Wagener (Amsterdam, NL), Henk Te Velthuis (Amsterdam, NL), Kumar Visvanathan (Melbourne, AU), Peter De Cruz (Melbourne, AU)

Introduction: The role of infliximab (IFX) therapeutic drug monitoring (TDM) in acute severe ulcerative colitis (ASUC) is unclear. We study aimed to assess whether IFX levels are linked to outcomes in ASUC.

Methods: PREDICT-UC (NCT02770040) was a randomised controlled trial involving 138 steroid-refractory ASUC patients. Serum and faecal IFX levels were measured using ELISA (MabTrack level infliximab, Essange Reagents, Netherlands), and correlated with outcomes including initial IFX response by day 7 (Lichtiger score [LS] < 10, with ≥ 3-point reduction and decrease in rectal bleeding/stool frequency), eventual response at day 14 (LS < 10), and colectomy by month 3. IFX clearance was estimated using a pharmacokinetic model.

Results: 681 serum IFX levels were analysed from 135 patients, with 91 receiving an initial 5 mg/kg IFX and 44 receiving 10 mg/kg. 85 patients responded by day 7, and 17 required colectomy by month 3. Serum IFX levels on days 1 and 3 were higher in the 10 mg/kg group than the 5mg/kg group ($p < 0.001$). A high day 3:day 1 serum IFX ratio predicted initial response ($p = 0.006$; AUC = 0.67), while lower day 3 serum IFX levels predicted colectomy ($p = 0.003$; AUC = 0.23). IFX clearance based on days 1–7 serum levels was higher in non-responders compared to responders ($p < 0.001$) and in those requiring colectomy ($p = 0.011$). Patients with high clearance (≥ 0.62 l/day) were more likely to respond to an initial 10 mg/kg compared to a 5 mg/kg IFX dose ($p = 0.046$), and were less likely to need colectomy if they received an initial 10 mg/kg dose ($p = 0.039$). Patients with high clearance who didn't initially respond had a better day 14 response with a second 10 mg/kg dose ($p = 0.041$). Faecal IFX levels correlated with clearance, CRP, and UCEIS.

Discussion/Conclusion: Early IFX levels predict outcomes in ASUC. Elevated day 3:day 1 serum IFX ratio predicted initial response while low day 3 serum IFX predicted colectomy. High IFX clearance may be overcome by higher IFX dosing.

30. Gut microbiota changes are associated with type of advanced therapy in patients with IBD: Baseline data from the Australian IBD Microbiome study

Robert Little (Melbourne, AU), Thisun Jayawardana (Sydney, AU), Fan Zhang (Sydney, AU), Sabrina Koentgen (Sydney, AU), Wu Nan (Sydney, AU), Paris Tavakoli (Sydney, AU), Andrew Chung (Sydney, AU), Anson Ho (Sydney, AU), Sophina Read (Sydney, AU), Alex Boussioutas (Melbourne, AU), Mark Ward (Melbourne, AU), Miles Sparrow (Melbourne, AU), Simon Ghaly

(Sydney, AU), Crispin Corte (Sydney, AU), Nicholas Talley (Newcastle, AU), Steven Leach (Sydney, AU), Daniel Lemberg (Sydney, AU), Shoma Dutt (Sydney, AU), Sudarshan Paramsothy (Sydney, AU), Rupert Leong (Sydney, AU), Susan Connor (Sydney, AU), Georgina Hold (Sydney, AU)

Introduction: Gut microbiota are associated with both disease course and treatment outcomes in inflammatory bowel disease (IBD). This study aimed to characterise differences in gut microbial signatures according to medication use in the Australian IBD Microbiome (AIM) study.

Methods: Faecal and oral samples alongside participant characteristics were collected at baseline from all patients with IBD enrolled in the AIM study from June 2019 to November 2023. Faecal and oral microbial samples were collected in DNA stabilizing buffer, aliquoted and stored at -80oC. Samples underwent 16S rRNA sequencing with annotation of DNA sequences to operational taxonomic units at the genus level. Differences in alpha diversity (Shannon index) and relative abundance of observed genera between medication groups were assessed using Mann-Whitney U and Kruskal-Wallis tests. Linear discriminant analysis was performed to identify significantly enriched bacterial taxa between groups. Beta diversity (Bray-Curtis dissimilarity) between bacterial communities were shown using the Principal Coordinate Analysis (PCoA), and significance of variance tested using ADONIS within R.

Results: 446 participants (232 CD, 214 UC) returned baseline faecal (n = 403) and/or oral samples (n = 436). Mean age was 43.7 years, mean BMI 25.9 kg/m2, and median faecal calprotectin (FCP) was 39 µg/mg. 46% of patients were on advanced therapy and 39% of patients were on an immunomodulator. No differences in FCP were observed between patients on advanced therapy vs. patients who were not, nor between each advanced therapy class. Patients on advanced therapy had lower alpha diversity ($p = 0.003$) and distinct beta diversity (R2 0.64, $p = 0.002$) than those not on advanced therapy. Patients on anti-TNF therapy had lower alpha ($p < 0.001$) and distinct beta diversity (R2 1.75, $p = 0.008$) compared to patients on no advanced therapy. Alpha diversity remained lower when comparing those on either infliximab ($p = 0.008$) or adalimumab ($p = 0.012$) to patients on no therapy. There were no differences in alpha or beta diversity between other advanced therapy classes, between types of immunomodulator and between patients on 5-ASA compared to patients who were not. Pairwise comparisons between patients on no therapy and each class of advanced therapy demonstrated significantly different relative abundances of multiple genera observed in faecal samples. No differences were observed in oral samples.

Discussion/Conclusion: Distinct differences in both community structure and the relative abundance of multiple genera were observed in IBD patients according to type of advanced therapy despite no significant difference in biochemical activity. No microbiota differences were observed according to conventional 5-ASA or immunomodulator use. Future analysis with greater sequencing depth and including longitudinal samples and treatment outcomes may allow identification of unique microbial signatures predictive of treatment response.

31. Impaired health-related quality of life is associated with alterations in gut microbiota in patients with inflammatory bowel disease: Baseline sequencing from the Australian IBD Microbiome study

Robert Little (Melbourne, AU), Thisun Jayawardana (Sydney, AU), Fan Zhang (Sydney, AU), Sabrina Koentgen (Sydney, AU), Nan Wu (Sydney, AU), Paris Tavakoli (Sydney, AU), Andrew Chung (Sydney, AU), Anson Cho (Sydney, AU), Alex Boussioutas (Melbourne, AU), Mark Ward (Melbourne, AU), Miles Sparrow (Melbourne, AU), Peter Gibson (Melbourne, AU), Simon Ghaly (Sydney, AU), Crispin Corte (Sydney, AU), Nicholas Talley (Newcastle, AU), Steven

Leach (Sydney, AU), Daniel Lemberg (Sydney, AU), Shoma Dutt (Sydney, AU), Sudarshan Paramsothy (Sydney, AU), Rupert Leong (Sydney, AU), Susan Connor (Sydney, AU), Georgina Hold (Sydney, AU)

Introduction: Inflammatory Bowel Disease (IBD) activity as well as disease monitoring and management have a substantial impact on the health-related quality of life (HRQoL) of affected patients. This study investigated the association between HRQoL and gut microbial composition in IBD patients using the 32-item Inflammatory Bowel Disease Questionnaire (IBDQ-32).

Methods: Paired faecal and oral samples alongside participant demographics, disease characteristics and the IBDQ-32 survey were collected at baseline from patients with Crohn's disease (CD) or ulcerative colitis (UC) enrolled in the Australian IBD Microbiome (AIM) study from June 2019 to November 2023. IBDQ-32 scores range from 32 to 224, with a higher score indicating better HRQoL. Patients were divided into two groups: impaired HRQoL (cumulative score < 170) and preserved HRQoL (> 170). Faecal and oral samples were self-collected in DNA stabilising buffer, aliquoted and stored at -80°C until extraction. Samples underwent 16S rRNA sequencing with annotation of DNA sequences to operational taxonomic units at the genus level. Differences in alpha diversity (Chao1) between groups were assessed using Mann-Whitney U tests. Linear discriminant analysis was performed between groups to identify significantly enriched bacterial taxa. Beta diversity (weighted UniFrac dissimilarity) in bacterial communities were shown using Principal Coordinate Analysis (PCoA), and significance of variance was tested using ADONIS in R.

Results: 446 participants (232 CD, 214 UC) returned baseline faecal (n = 403) and/or oral samples (n = 436). Mean age was 43.7 years, mean BMI 25.9 kg/m2, and median faecal calprotectin (FCP) was 39 µg/mg. 46% of patients were on advanced therapy and 39% of patients were on an immunomodulator. Most CD and UC patients were in clinical remission at baseline (77% and 65% respectively). Despite this, impaired HRQoL was common in both CD (42%) and UC (41%). Patients with impaired HRQoL had lower faecal microbial alpha ($p = 0.003$) and beta diversity ($R2 = 0.005$, $p = 0.041$). There were no differences in alpha or beta diversity in oral samples. Linear discriminant analysis showed 45 genera with significantly altered relative abundance between patients with impaired and preserved HRQoL with greater abundance of beneficial, short-chain fatty acid-producing taxa in those with preserved HRQoL.

Discussion/Conclusion: Despite a high proportion of clinical and biochemical remission, impaired HRQoL was common amongst UC and CD patients within the AIM study. Lower faecal but not oral alpha diversity was associated with impaired HRQoL, indicating reduced microbial richness in the gut. A high number of bacterial taxa had differential enrichment according to HRQoL status. Identification of a distinct microbial signature associated with impaired HRQoL may help to identify patients that require both optimisation of disease control and greater psychosocial support.

32. Use of a convolutional neural network to quantify and predict ulcerative colitis disease endoscopic activity and histological outcomes

Savannah Morrison (Sydney, AU), Christopher Chiu (Sydney, AU), Rena Cao (Sydney, AU), Peter Lim (Sydney, AU), JooShik Shin (Sydney, AU), Catriona Mackenzie (Sydney, AU), James Kench (Sydney, AU), Crispin Corte (Sydney, AU), Aravind Gokul Tamilarasan (Sydney, AU)

Introduction: There is considerable inter-user variability in endoscopic and histologic scoring of the severity of ulcerative colitis (UC). The aim of this study was to create and train an artificial intelligence algorithm to predict the severity of UC using endoscopic videos.

Methods: This prospective cohort study recruited UC patients undergoing flexible sigmoidoscopy or colonoscopy between October 2023 and September 2024. The endoscopic examination was recorded from the splenic flexure to the anus. The videos were segmented into 3 second tubelets, and poor quality videos excluded. Objective disease activity was quantified by an experienced IBD gastroenterologist (by determining individual endoscopic Mayo Scores – eMS), and corresponding biopsies were scored according to the Nancy Histologic Index (NHI) by one of three gastrointestinal histopathologists. The dataset was divided into a training dataset (38 patients, 88% of video data) and a test dataset (6 patients, 12% of data), stratified by disease severity and equipment source. Two video vision transformer models were trained to recognise Mayo and NHI respectively, and their performance in differentiating disease activity from remission, and accuracy in determining endoscopic and histologic severity scores were evaluated. Standard diagnostic accuracy measures were used to calculate the models' ability to detect and quantitate UC disease activity and included area under the receiver operating characteristic curve (AUROC), binary and multi-class accuracy, sensitivity, specificity, and precision.

Results: 53 patients were recruited, and after exclusions a total of 44 were included in the final analysis. 30 patients had endoscopic activity and 30 patients had histological activity. The resultant eMS and NHI showed moderate correlation (R = 0.72). For eMS, the accuracy of the model was excellent and had an AUROC of 0.98 in identifying disease activity (eMS ≥ 1), with accuracy, sensitivity, specificity, and precision of 92%, 86%, 96%, 93% respectively. Multiclass accuracy was 87%, with a macro F1 score of 0.85. For NHI, the model was highly accurate in detecting histopathologic activity (NHI ≥ 1), with AUROC of 0.95, accuracy, sensitivity, specificity, precision at 89%, 92%, 86%, 85% respectively. However, it was less robust in predicting specific NHI scores, with multiclass accuracy at 69%, and a macro F1 score of 0.41.

Discussion/Conclusion: We developed an AI algorithm that is able to reliably predict endoscopic and histologic disease activity for patients with UC, and accurately differentiate those patients from those in remission. This may reduce clinician reliance on biopsies to determine disease activity, and have far-reaching implications in UC disease assessment.

33. Incidence of acute severe ulcerative colitis in a changing landscape of increased disease prevalence and more effective therapies

Steffanie Nario (Sydney, AU), Michael Yulong Wu (Sydney, AU), Carmen Tung (Sydney, AU), Christopher Kiely (Sydney, AU)

Introduction: Over the past 60 years, Ulcerative Colitis (UC) has seen increasing worldwide disease prevalence and incidence.1,2 With the rise in UC across the patient population, more effective therapeutic options to induce remission and maintenance have become available including biologic therapies and small molecule inhibitors. Approximately 15–25% of patients with UC will experience at least one episode of Acute Severe Ulcerative Colitis (ASUC). First diagnosis of UC accounts for 10–20% of ASUC presentations, and 34% of patients will experience ASUC within a year of UC diagnosis.3 We aimed to ascertain the effect of increased therapeutic options and increased UC prevalence on the incidence of ASUC hospital admissions. We hypothesised that with increased availability of more effective UC maintenance therapy through the Australian Pharmaceutical Benefits Scheme (PBS), there would be a reduction in ASUC presentations in patients with existing UC diagnosis, including within the first year of UC diagnosis. We further hypothesised that with the increase in UC incidence, there would be an increase in first diagnosis ASUC presentations.

Methods: Data was collected retrospectively on patients admitted with ASUC to a quaternary Sydney hospital between January 2014 and December 2023.1,2 A modified Truelove

and Witts criteria using the European Crohn's and Colitis Organisation guidelines was used to identify ASUC as ≥ 6 bloody bowel motions daily and presence of at least one systemic feature, including heart rate > 90 bpm, temperature > 37.8°C, haemoglobin < 105 g/l, and C-reactive protein (CRP) ≥ 30 mg/l. Patients admitted with ASUC were classified as "relapse" if they were on maintenance UC therapy at time of presentation, "refractory" if presented during treatment of a UC flare, and "new diagnosis" if first presentation and no previous history of UC.

Results: Over the 10-year period, there were a total of 167 admissions of ASUC among a total of 136 patients. On average, there were 13.6 ± 2.4 patients with 16.7 ± 3.4 presentations each year. The highest number of ASUC presentations were in 2022 and 2018. The average number of refractory and relapse presentations were not significantly different (6.8 vs. 7.1, p = 0.8). Over 38.4% of ASUC presentations were within the first year of diagnosis. Out of all ASUC presentations on average, 40.7% of presentations were relapsed ASUC, 42.5% were refractory ASUC and 14.5% were new diagnosis. The number of ASUC episodes and number of patients presenting with ASUC did not appear to increase over the past decade (Table 1, Figure 1).

Discussion/Conclusion: Despite an increasing worldwide incidence and prevalence of UC, we did not find an increasing incidence of ASUC admissions. The proportion of patients with relapsed and refractory UC presenting as ASUC were similar. The increased availability of more effective options for treatment of UC may explain the stable number of ASUC presentations, reflecting better disease control in the outpatient setting. Interestingly, despite increasing UC incidence, the incidence of ASUC presentation in first diagnosis UC did not appear to increase over time.

34. Predicting early recognition of acute severe ulcerative colitis based on presence of Truelove and Witts' systemic features at time of presentation

Steffanie Nario (Sydney, AU), Michael Yulong Wu (Sydney, AU), Carmen Tung (Sydney, AU), Christopher Kiely (Sydney, AU)

Introduction: Acute severe ulcerative colitis (ASUC) is a medical emergency. Early recognition and diagnosis are necessary to initiate timely and appropriate investigations and treatment. The Truelove and Witts criteria (TWC) is used to stratify severity of Ulcerative Colitis (UC) and thus identify those presenting with ASUC. However, TWC is underutilised in the emergency department which can lead to delayed diagnosis and management. We aimed to assess which TWC criteria are most commonly fulfilled at time of ASUC presentation. We further hypothesised that an increase in number of systemic features present may be associated with earlier diagnosis and treatment of ASUC.

Methods: A retrospective review was conducted on all patients presenting to a quaternary Sydney hospital over a 10-year period (January 2014 to May 2024) with "Ulcerative Colitis", as coded by ICD diagnosis. Each admission coded with UC was reviewed and those meeting criteria as ASUC on presentation were included for further analysis. Using a modified TWC based on the European Crohn's and Colitis Organisation guidelines, we defined ASUC as ≥ 6 bloody bowel motions daily and presence of at least one systemic feature, including heart rate (HR) > 90 bpm, temperature > 37.8°C, haemoglobin (Hb) < 105 g/l, and C-reactive protein ≥ 30. Authors classed recognition of ASUC when there were clear clinician documentation of ASUC as the diagnosis within 24 hours of presentation.

Results: During the study period, there were a total of 617 presentations to hospital with UC and 172 that met criteria for ASUC. Only 30.2% of all presentations were recognised as ASUC

by clinicians within 24 hours of presentation. In patients presenting with ASUC, mean HR was 101.7 bpm, temperature 37.2°C, Hb 126.0 g/l and CRP was 71.1 mg/l. We found no significant difference in early diagnosis of ASUC between patients who presented with one systemic feature compared to those who presented with more than one systemic feature (33.8% vs. 27.9%, p = 0.41). HR > 90 bpm and CRP ≥ 30 were the most common systemic features (Table 1, Figure 1). This was the only systemic feature present in 38 presentations, with 34.2% of these recognised and documented as ASUC within 24 hours. Time to commence steroids if ASUC was recognised within 24 hours was 0.16 (± 0.26) days, compared to 0.73 (± 2.5) days if ASUC was recognised > 24 hours (p < 0.05).

Discussion/Conclusion: Less than one third of patients that met ASUC criteria were diagnosed as such within 24 hours of presentation. Tachycardia and elevated CRP were the most frequent systemic features present met for diagnosis of ASUC at time of presentation. Clinicians should be more familiar with identifying systemic features of severity, as defined by TWC, in order to recognise patients with ASUC at time of presentation. Delays to diagnosis of ASUC may impact on patient treatment and outcomes.

35. Comparison of screening strategies for latent tuberculosis infection in patients with inflammatory bowel disease before anti-tumor necrosis factor-α agents therapy: TST-alone versus a combination of TST and IGRA

Sang Hyoung Park (Seoul, KR), Ye-Jee Kim (Seoul, KR), Jiyeon Kim (Seoul, KR), Jiwon Lee (Seoul, KR), Tae Sun Shim (Seoul, KR), Kyung Wook Jo (Seoul, KR)

Introduction: We aimed to evaluate if using the interferon-gamma release assay (IGRA) alone is effective for latent tuberculosis infection (LTBI) screening in preventing active tuberculosis in patients with inflammatory bowel disease (IBD) before initiating anti-tumor necrosis factor (TNF)-α therapy, compared to using both the tuberculin skin test (TST) and IGRA.

Methods: Using South Korea's Health Insurance Review and Assessment Service, we selected IBD patients treated with anti-TNF-α agents for ≥ 1 year who underwent LTBI screening between 2018 and 2021. We compared the 1-year incidence rate and standardized incidence ratio of active tuberculosis incidence after starting anti-TNF-α treatment to the general population based on the LTBI screening strategy.

Results: Of the 4215 enrolled patients, 3505 underwent IGRA alone for LTBI screening, while 710 received both TST and IGRA. Within 1 year of starting anti-TNF-α treatment, 15 patients (0.36%) developed active tuberculosis, with a mean follow-up period of 4200.6 person-years. The 1-year tuberculosis incidence rates were 372.3 (95% CI: 198.2–636.6) per 100,000 person-years for the IGRA alone group and 282.3 (95% CI: 34.2–1019.9) per 100,000 person-years for the combination group. The standardized incidence ratios were similar: 14.34 (95% CI: 7.63–24.52) for the IGRA alone group and 11.25 (95% CI: 1.26–40.61) for the combination group.

Discussion/Conclusion: Using IGRA alone may be an effective strategy for LTBI screening in IBD patients before starting anti-TNF-α therapy.

36. Defining an eosinophilic leukocyte count per mm2 threshold for the diagnosis of eosinophilic esophagitis using a digital pathology platform

Arpad Patai (Budapest, HU), Csilla Molnar (Székesfehérvár, HU), Micsik Tamas (Budapest, HU)

Introduction: Eosinophilic esophagitis (EoE) is a chronic, antigen mediated disease causing severe dysphagia. Diagnosis relies on clinical symptoms and eosinophilic leukocyte count (EoL) per High Power Field (HPF). However, histology samples frequently may not fill the whole objective and there is an almost 2.5-fold difference in HPF area of different microscopes. Thus, a standardized number of EoL/mm2 may decrease diagnostic discrepancies and may facilitate better diagnostic rate.

Methods: Our aim was to analyze EoL/mm2 and other histopathologic features in EoE using a digital pathology platform, and compare these to gastroesophageal reflux disease (GERD) to define a EoL/mm2 threshold for the diagnosis of EoE. 45 EoE and 45 matched GERD cases were selected retrospectively from the archives of the Pathology Departments of Semmelweis University, Budapest, Hungary. Clinicopathological data were collected, slides were digitalized using PannoramicTM P250 Flash digital slide scanner (3DHISTECH Ltd., Budapest, Hungary) and re-analyzed by 2 independent GI histopathologists. Epithelial and subepithelial compartments and EoLs were annotated digitally with CaseViewer.

Results: In our EoE cohort there was a male predominance (males, n = 30/45); mean age was 23.5 ± 19.7 years. Most common endoscopic manifestations were rings (36.8%) and longitudinal furrows (21.1%). Histopathologic EoE findings were: eosinophilic cell degranulation 86.6%, spongiosis 75.5%, eosinophilic aggregate formation 66.7%. Average sample areas were similar in both groups, whereas digital image analysis showed significantly higher EoL/mm2 count in both epithelial (171.2 ± 177.2 vs. 3.3 ± 11.6) and subepithelial (61.1 ± 67.11 vs. 4.5 ± 13.22) compartments of EoE as compared to GERD cases. ROC analysis found a reliable threshold for differentiating EoE from GERD at 33 EoL/mm2 (AUC = 0.993) in the epithelial and 16 EoL/mm2 in the subepithelial compartment (AUC = 0.882).

Discussion/Conclusion: In 2018 a consensus paper of international experts suggested an estimated 60 EoL/mm2 threshold for diagnosing EoE instead of 15 EoL/HPF. Our study with numerical counting of EoLs on a digital platform found that a lower, 33 EoL/mm2 threshold might reliably differentiate EoE from GERD. Further prospective studies are needed to validate these findings.

Reference: Dellon ES, Liacouras CA, Molina-Infante J, et al. Updated International Consensus Diagnostic Criteria for Eosinophilic Esophagitis: Proceedings of the AGREE Conference. Gastroenterology. 2018;155(4):1022 1033.

37. Multi-strain probiotics supplementation with standard care enhances quality of life in ulcerative colitis patients: A preliminary data

Muhammad Ikhmal Rosali (Cheras, MY), Raja Affendi Raja Ali (Bandar Sunway, MY), Khairul Najmi Muhammad Nawawi (Cheras, MY), Deborah Chia Hsin Chew (Cheras, MY), Hajar Fauzan Ahmad (Gambang, MY), Norfilza Mohd Mokhtar (Cheras, MY)

Introduction: Ulcerative colitis (UC) is a chronic inflammatory condition of the colon characterised by remitting and relapsing symptoms that can impair quality of life. Standardised treatments aim for clinical remission but often overlook the emotional and physical burdens associated with the disease. Probiotics, as an adjunctive therapy, may address these gaps by modulating dysregulated gut microbiota. Therefore, this study aims to evaluate the effects of probiotics combined with standard care on quality of life, gut microbiota, disease activity, and pro-inflammatory cytokines in UC patients.

Methods: Twenty-one UC patients were randomized to receive either probiotics or placebo, labelled as Groups G and J, for 12 weeks. Baseline and post-treatment assessments included the Partial Mayo Score (PMS), Short Inflammatory Bowel Disease Questionnaire (SIBDQ),

stool samples for 16S rRNA sequencing, and blood samples for pro-inflammatory cytokine analysis using qPCR.

Results: After 12 weeks of intervention, Group G showed significant improvements in quality of life parameters, including reduced abdominal pain ($p = 0.0357$), feelings of depression or discouragement ($p = 0.0183$), and urgency to use the bathroom ($p = 0.0074$) compared to Group J. Linear discriminant analysis effect size (LEfSe) revealed microbiota associations in both groups. Group G showed a positive correlation with Bifidobacterium adolescentis and Roseburia, whereas Group J was predominantly associated with Prevotella. Clinically, Group G exhibited a significant reduction in physician's global assessment ($p = 0.043$) and a significant decrease in IL-13 expression levels ($p = 0.037$). In contrast, Group J demonstrated a significant increase in the presence of blood in stool ($p = 0.006$).

Discussion/Conclusion: The enrichment of beneficial bacteria in Group G likely contributed to a significant improvement in quality of life and clinical symptoms, as well as a decrease in IL-13 gene expression, supporting gut barrier integrity and reducing inflammation. Conversely, Group J, which demonstrated a significant abundance of Prevotella, showed no improvement in quality of life and worsening symptoms due to the inflammatory response associated with pathogenic bacteria. This preliminary data suggests that the group showing significant improvements likely represents the probiotics group. Probiotics, in combination with standard care, significantly enhanced quality of life, modulated gut microbiota, and reduced inflammation-related symptoms in UC patients.

38. Comprehensive risk assessment and prognostic factors in celiac disease: Impact of persistent villous atrophy on disease complications and survival outcomes

Raouia Saidani (Bizerte, TN), Dhouha Cherif (Tunis, TN), Hajer Hassine (Tunis, TN), Haithem Yacoub (Tunis, TN), Habiba Debbabi (Tunis, TN), Hela Kchir (Tunis, TN)

Introduction: Celiac disease (CD) is an autoimmune disorder triggered by gluten ingestion, leading to intestinal villous atrophy. While a gluten-free diet (GFD) is the mainstay of treatment, some patients exhibit persistent villous atrophy (pVA) on follow-up biopsies. The long-term implications of pVA in CD patients adhering to GFD remain concerning, particularly regarding complications and mortality risk.

Methods: Our primary objectives were to evaluate the association between pVA and long-term outcomes in CD patients and assess the performance of a novel scoring system identifying patients at high risk of pVA. We conducted a retrospective analysis including 151 patients diagnosed with CD between 2014 and 2020 who underwent follow-up duodenal biopsies. Patients with follow-up biopsies were older at diagnosis (mean 44 ± 16 years) and more frequently presented with classical CD patterns (50.6%). The median follow-up duration was 110 months (IQR, 74–161). Patients were categorized based on the Marsh classification (≥ 3a) for pVA assessment. CD complications included refractory CD, ulcerative jejuno-ileitis, abdominal lymphomas, and small bowel carcinomas. We implemented a 5-point scoring system incorporating age at diagnosis ≥ 45 years, classical CD pattern, lack of clinical response to GFD, and poor GFD adherence (weighted as two points).

Results: Approximately 24.5% of patients demonstrated pVA, with these individuals showing a significantly higher risk of complications (HR = 7.61, 95% CI: 3.18–17.64, $p < 0.001$). During follow-up, 9 patients (5.96%) developed complications, including three cases of refractory CD, one B-cell lymphoma, three cases of persistent malabsorption, and two cases of ulcerative jejuno-ileitis. Notably, 7 of these 9 patients had pVA. Patients who underwent follow-up duodenal biopsy showed improved overall survival compared to those who did not ($p <$

0.05). While most patients adhered to GFD, 35.1% maintained persistent VA at follow-up. Importantly, 29.72% of patients with pVA were asymptomatic. The median follow-up duration for patients with follow-up biopsies was significantly longer at 110 months (IQR, 74–161) compared to 60 months (IQR, 24–110) for those without follow-up biopsies ($p < 0.001$). Mortality causes in patients with CD complications were predominantly related to disease progression, with a higher frequency of CD-related deaths observed in the follow-up biopsy group ($p < 0.01$). Among patients presenting with classical CD patterns, 50.6% underwent follow-up biopsies, showing a higher tendency for careful monitoring. The scoring system effectively stratified patients into three risk categories: low risk (0–1 points) with 4.1% pVA prevalence (3 out of 73 patients), intermediate risk (2 points) with 20% pVA prevalence (9 out of 45 patients), and high risk (3–5 points) with 75.75% pVA prevalence (25 out of 33 patients). Multivariate analysis revealed that age at diagnosis ≥ 45 years and classical CD presentation were independent predictors of pVA development. Additionally, patients with pVA demonstrated higher rates of hospitalizations and nutritional deficiencies, requiring more intensive monitoring and dietary intervention.

Discussion/Conclusion: Our study demonstrates that pVA in CD patients following GFD significantly correlates with increased complication risks and mortality. The novel 5-point scoring system proves effective in stratifying patients by pVA risk, particularly in identifying high-risk individuals, including those who are asymptomatic. This scoring system enables personalized follow-up strategies and early intervention opportunities, potentially improving long-term outcomes in CD patients. Regular follow-up biopsies appear crucial for risk assessment and prognosis evaluation, especially in patients identified as high-risk by the scoring system.

39. The introduction of an intestinal ultrasound service significantly reduces diagnostic endoscopy usage in an inflammatory bowel disease service – The SCOPELESS study

Miles Sparrow (Melbourne, AU)

Introduction: Endoscopic assessment is the gold standard for monitoring of inflammatory bowel disease (IBD) activity. Increasingly, intestinal ultrasound (IUS) is utilised as a non-invasive alternative disease monitoring strategy. Our aim was to quantify and compare endoscopy usage for evaluation of IBD disease activity before and after the introduction of an IUS service.

Methods: A retrospective single-centre study was performed. Total numbers of lower GI endoscopies (ileocolonoscopy or flexible sigmoidoscopy) performed for luminal Crohn's disease (CD) or ulcerative colitis (UC) disease evaluation were collected across two 5-year time periods: the pre IUS era (2010–2014) and the IUS era (2015–2019). Endoscopies for dysplasia surveillance were excluded. The primary endpoint was a comparison of the cumulative number of endoscopies for IBD activity evaluation annually relative to the annual number of patients seen in clinic in the pre-IUS and IUS eras. Secondary endpoints included evaluating the number of endoscopies by individual year within each time period, endoscopies according to diagnosis (CD vs. UC), and the number of IUS performed within the IUS era. Categorical variables were compared using a Chi-squared test.

Results: The number of endoscopies performed for IBD disease evaluation decreased from 576 in the pre-IUS era to 474 in the IUS era despite an increase in cumulative annual patient reviews (1985 vs. 3337 patient reviews, respectively). The proportion of cumulative annual endoscopies relative to patients reviewed across the 5-years reduced from 29 per 100 patients in the pre-IUS era to 14 per 100 patients in the IUS era (OR = 2.47, 95% CI: 2.15–2.84; p <

0.001). There was a reduction in total endoscopies for CD evaluation from 325 to 264 and for UC evaluation from 251 to 210. The proportion of cumulative annual endoscopies relative to patients reviewed reduced from 30 to 14 per 100 patients in CD (OR = 2.60, 95% CI: 2.16–3.12; p < 0.001), and 37 to 17 per 100 patients in UC (OR = 2.90, 95% CI: 2.33–3.59, p < 0.001). In the IUS era, a total of 3319 IUS were performed (2673 CD, 646 UC). This included 1467 IUS for assessment of suspected activity (44 per 100 patients/year) and 1852 IUS for objective confirmation of clinical remission (55 per 100 patients/year).

Discussion/Conclusion: In the 5 years following introduction of an IUS service, the number of endoscopies performed for evaluation of IBD activity per patient review was halved. With IUS being performed for both assessment of disease activity and objective confirmation of clinical remission, the potential workflow and cost savings of reducing endoscopies for IBD disease activity evaluation are significant.

40. Endoscopic severity at presentation and C-reactive protein predict immediate and twelve-month outcomes of second-line medical therapy in acute severe ulcerative colitis

Delo Subhaharan (Gold Coast, AU), Pradeep Kakkadasam Ramaswamy (Gold Coast, AU), John Edwards (Gold Coast, AU), Lauren White (Sunshine Coast, AU), Desmond Patrick (Sunshine Coast, AU), Asif Shahzad (Logan, AU), Laura Willmann (Gold Coast, AU), Hadi Moattar (Gold Coast, AU), Dheeraj Shukla (Gold Coast, AU), Naveed Ishaq (Gold Coast, AU), Maneesha Bhullar (Gold Coast, AU), Alexander Dorrington (Gold Coast, AU), Jack Satsangi (Oxford, GB), Waled Mohsen (Gold Coast, AU)

Introduction: Medical rescue therapy (MRT) is effective in intravenous corticosteroid refractory acute severe ulcerative colitis (ASUC). Our primary objective was to identify parameters which identify initial and sustained response to MRT.

Methods: Two cohorts were studied retrospectively. Analysis of 66 adult ASUC admissions receiving MRT between 2015–19 at Gold Coast University Hospital and Logan Hospital was first performed. Clinical, endoscopic and laboratory data were collected. Response was defined as avoiding colectomy during the same admission. Univariable and multivariable logistic regression were employed to identify predictors of response. The predictors were validated in 99 patients admitted to Gold Coast University Hospital and Sunshine Coast University Hospital between 2020–23.

Results: In the development cohort, 55/66 patients (83.3%) responded to MRT. On multivariable analysis, UCEIS score at admission [Coef - 0.105 (-0.19 to -0.007), p = 0.03] and CRP on day 3 of post commencement of MRT (CRP on day R+3) [Coef -0.004 (-0.0008 to -0.0004), p = 0.03] identified response to MRT. All patients (n = 20) with a UCEIS score < 6 [sensitivity 41.7%, specificity 100%, PPV 100%, NPV 26.3%] and 97.1% (33/34) patients with a CRP on day R+3 < 22 mg/l responded to MRT [sensitivity 71.7%, specificity 90%, PPV 97.1%, NPV 40.9%]. In the validation cohort, 93/99 (94%) patients responded to MRT; 92.6% (25/27) with a UCEIS score < 6 [sensitivity 26.9%, specificity 66.7%, PPV 92.6%, NPV 5.6%] and 100% (72/72) patients with CRP on day R+3 < 22 mg/l responded to MRT [sensitivity 85.7%, specificity 100%, PPV 100%, NPV 33.3%]. In patients with both UCEIS < 6 and CRP on day R+3 < 22 mg/l, 15/15 (100%) and 18/18 (100%), in the development and validation cohorts respectively, responded to MRT.

At 12 months after hospitalisation, in the development cohort, 45 (68.1%) patients had avoided colectomy. Of the patients with a UCEIS score < 6, 19 (95%) patients and 28/34 (82.4%) patients with CRP on day R+3 < 22 mg/l avoided colectomy. In the validation cohort, at 12 months, 84 (84.9%) patients had avoided colectomy. Of the patients with a UCEIS score <

6, 24/27 (88.9%) and 66/72 (91.7%) patients with CRP on day R+3 < 22 mg/l had avoided a colectomy at 12 months. In patients with both UCEIS < 6 and CRP on day R+3 < 22 mg/l, 14/15 (93.3%) and 17/18 (94.4%), in the development and validation cohorts respectively, had avoided colectomy at 12 months.

Discussion/Conclusion: UCEIS score < 6 at admission and CRP on day R+3 < 22 mg/l predict response to second-line MRT and colectomy is extremely unlikely either on the index admission or within 12 months.

41. A preliminary report on the effects of microplastics on the gut microbiota and metabolites in patients with inflammatory bowel disease

Dinesh Prasad V Thanga Velu (Cheras, MY), Raja Affendi Raja Ali (Subang Jaya, MY), Yusof Shuaib Ibrahim (Kuala Nerus, MY), Khairul Najmi Muhammad Nawawi (Cheras, MY), Deborah Chia Hsin Chew (Cheras, MY), Hajar Fauzan Ahmad (Gambang, MY), Mohd Talib Latif (Bangi, MY), Norfilza Mohd Mokhtar (Cheras, MY)

Introduction: In Asia, environmental factors significantly influence the development of inflammatory bowel disease (IBD), including ulcerative colitis (UC) and Crohn's disease (CD). However, little is known about how microplastics (MPs) influence gut dysbiosis and affect the natural history of IBD. We postulated that MPs disrupt microbiota homeostasis, increase gut permeability, and facilitate toxin transport into circulation through their interactions with gut flora.

Methods: This study investigates the presence of MPs in inflamed colonic tissues and faeces of IBD patients and their impact on microbiota and metabolite profiles. Fecal and colonic samples were analyzed using filtration, microscopy, and advanced spectroscopic techniques (FT-IR and SEM/EDX) to determine polymer types and elemental composition. Genomic DNA from samples was extracted, and 16S rRNA sequencing, coupled with bioinformatic analysis, was conducted to evaluate gut microbiota.

Results: Twenty-six IBD patients (14 – UC, 12 – CD) were recruited. Deposition of MPs were detected in fecal and colonic samples of all patients, with UC showing the highest levels in colonic samples and CD in fecal samples. Notable correlations were observed between MPs levels and gut dysbiosis. Beneficial microbes, including Faecalibacterium prausnitzii, Bacteroides uniformis, and Prevotella copri, were more prevalent in CD, while opportunistic microbes, such as Escherichia and Collinsella, dominated in UC. Key SCFA producers, Faecalibacterium prausnitzii and Bifidobacterium longum, were inversely correlated with MPs levels. In CD, Faecalibacterium maintained SCFA production, whereas in UC, Escherichia compromised it. Higher MPs levels correlated with lower SCFA levels (acetate, propionate, and butyrate) in fecal and colonic samples of IBD patients.

Discussion/Conclusion: This study offers fresh proof that MPs are a novel environmental factor affecting the gut microbiota and metabolite profiles in IBD patients. Findings underscore the urgency of reducing MPs exposure and adopting comprehensive strategies for IBD management. Findings emphasise the necessity of focused approaches to reduce MPs exposure and provide all-encompassing IBD management.

42. Assessing patient quality of care in acute severe ulcerative colitis using a composite textbook outcome approach

Michael Yulong Wu (Sydney, AU), Steffanie Nario (Sydney, AU), Carmen Tung (Sydney, AU), Christopher Kiely (Sydney, AU)

Introduction: Textbook outcome (TBO) is a concept of quality performance measurement that has garnered traction in primarily the surgical field. While traditional methods evaluate individual outcome parameters, TBO is a composite measurement of multiple key performance indicators and is only achieved when all individual criteria are met. Studies suggest that a composite TBO provides a superior holistic assessment of patient outcomes, better captures the multidimensional aspects of patient care and sets a comparable benchmark for quality improvement across treatment centres. To date, TBO has not been defined or utilised in the setting of acute severe ulcerative colitis (ASUC). We aimed to define a TBO using guideline-based standards of care for ASUC presentations and assessed whether meeting TBO was associated with improved treatment outcomes.

Methods: All ASUC presentations to a Quaternary Sydney hospital over a 10-year period were included. A modified True-love Witts' Criteria was used to identify ASUC as ≥ 6 bloody bowel motions daily and presence of at least one systemic feature, including heart rate > 90 bpm, temperature > 37.8°C, haemoglobin < 105 g/l, and C-reactive protein (CRP) ≥ 30 mg/l. We defined achievement of TBO as IV steroids and venous thromboembolism prophylaxis commenced on admission, stool samples to exclude infective pathogens, abdominal imaging completed within 24 hours from presentation, and flexible sigmoidoscopy completed within 48 hours from presentation. Treatment outcomes measured were rates of rescue therapy, colectomy, length of stay, 30-day and 90-day readmission.

Results: During the study period, 172 presentations met criteria for ASUC and of these, 14.0% (n = 24) admissions achieved a TBO. The number of presentations meeting each TBO criteria is highlighted in Figure 1. Stool sample screening (91.3%, n = 157), IV steroids (79.1%, n = 136) and venous thromboembolism prophylaxis (73.3%, n = 126) on admission were the most frequently achieved parameters in a TBO. The least frequent achieved parameter were timely flexible sigmoidoscopy (36.0%, n = 62). A comparison of outcomes for patients achieving TBO compared to those without a TBO is shown in Table 1. Achieving a TBO was associated with significantly shorter length of stay.

Discussion/Conclusion: Composite outcome measures such as TBO have become an increasingly popular metric of comparing and benchmarking standards of care. ASUC patients who achieved a TBO, defined by meeting five different guideline-based criteria as part of their initial investigations and management, were found to have a significantly shorter length of stay. There were no differences in need for rescue therapy, colectomy and readmission rate. We found time to flexible sigmoidoscopy could be improved at our centre. More research is required to refine the consensus parameters that encompass a TBO in ASUC presentations.

43. Number of Truelove and Witts' criteria met at time of presentation impacts on severity of systemic toxicity and investigative outcomes

Michael Yulong Wu (Sydney, AU), Steffanie Nario (Sydney, AU), Carmen Tung (Sydney, AU), Christopher Kiely (Sydney, AU)

Introduction: Acute Severe Ulcerative Colitis (ASUC) is considered a medical emergency that occurs in 15–25% of UC patients during their disease course. The Truelove and Witts' criteria (TWC) is traditionally used to identify patients with ASUC at time of presentation. One study demonstrated that patients who met more systemic TWC on admission were more likely to undergo colectomy, however no other outcomes have been reported. We hypothesised that with an increasing number of systemic TWC met at time of presentation, this would correlate with increased systemic toxicity which may impact on patient outcomes. We evaluated whether ASUC patients who met a higher number of systemic TWC was associated with more timely investigations, use of empiric antibiotics, rescue therapy or need for colectomy.

Methods: A retrospective review was conducted on all ASUC presentations to a quaternary Sydney hospital over a 10-year period (January 2014 to May 2024). A modified TWC based on the European Crohn's and Colitis Organisation guidelines was used to identify ASUC as ≥ 6 bloody bowel motions daily and presence of at least one systemic feature, including heart rate (HR) > 90 bpm, temperature > 37.8°C, haemoglobin (Hb) < 105 g/l, and C-reactive protein (CRP) ≥ 30 mg/l. Patients were classified into four groups based on number of systemic TWC met at time of presentation. Data on antibiotic prescription, time taken for abdominal imaging (XR or CT), time to flexible sigmoidoscopy, time to rescue therapy, surgical review, colectomy, and length of stay were compared.

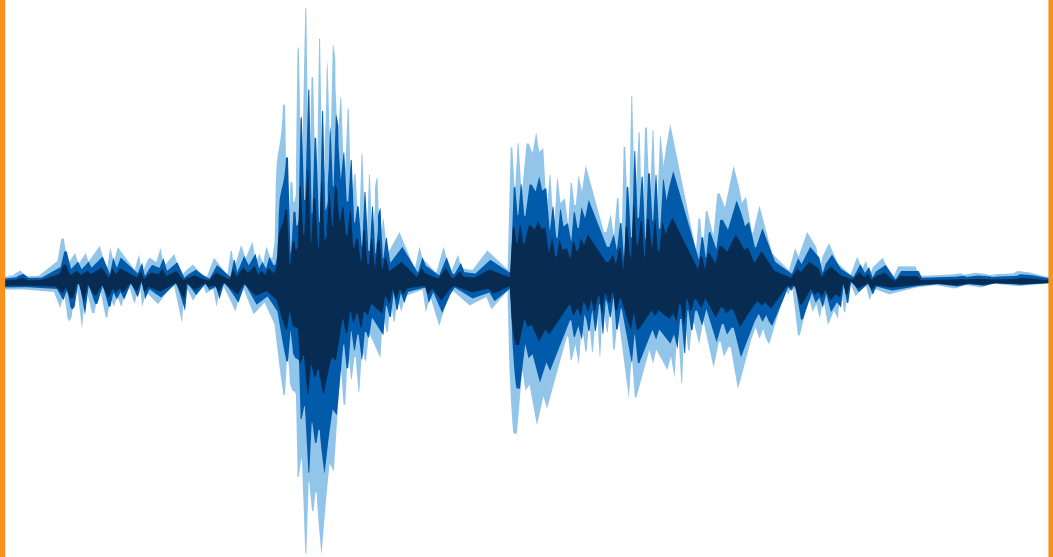
Results: During the study period, there were a total of 617 presentations to hospital with ulcerative colitis with 172 meeting criteria for ASUC. Most presentations had ≤ 2 systemic features present (73.8%, n = 127). Figure 1 demonstrates that systemic toxicity was significantly increased in patients who met more than one systemic TWC at time of presentation. These patients had higher average HR, higher temperature, higher CRP, and lower Hb ($p < 0.05$). Table 1 shows comparative results of investigation and treatment outcomes in those presenting with one, two, three or four systemic features. Patients with more systemic features were more likely to receive antibiotics and undergo flexible sigmoidoscopy.

Discussion/Conclusion: ASUC patients who met more systemic TWC on presentation had significantly increased features of systemic toxicity, with higher average HR, temperature, CRP and lower Hb. Patients presenting with a higher number of systemic TWC features are more likely to be recognised as ASUC and therefore, were more likely to receive empirical antibiotics and undergo flexible sigmoidoscopy. However, treatment outcomes, as defined by need for, and time to rescue therapy, colorectal surgery review, colectomy and length of stay, appeared similar regardless of number of systemic TWC met at presentation.

AUTHOR INDEX TO POSTER ABSTRACTS

(Name - Poster Number)

Registration via www.falkfoundation.org or simply scan and register.



Together we know more. Together we do more.
Falk Foundation e.V. | Leinenweberstr. 5 | 79108 Freiburg | Germany
T: +49 761 1514-400 | F: +49 761 1514-460 | E-Mail: meeting@falkfoundation.org
www.falkfoundation.org

P239E/2025/600Stü