Clinical Conversations

LIVE WEBINAR

Bridging Conventional & Integrative Care in Crohn's & Colitis

Practical clinical insights with Chloe Turner

1

Speakers

Chloe Turner
Naturopath

Gabriella Sassi
Naturopath & Nutritionist

2

Disclaimer

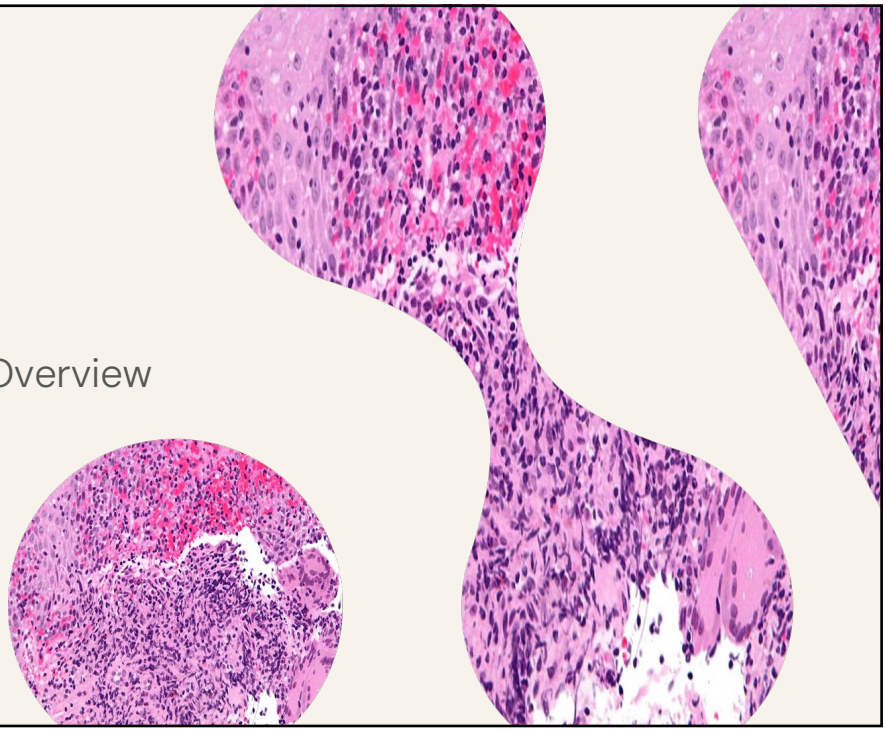
- The information provided in this webinar is for the use of qualified healthcare professionals.
- The information contained in this webinar is in no way to be taken as prescriptive or to replace a healthcare professional's duty of care and personalised care practices.
- The clinical opinions and patient case studies shared by presenters are solely those of the individual presenters and do not necessarily represent the view of Microba.

Learning Objectives

- Recognise clinical patterns suggestive of inflammatory bowel disease (including red flags) & interpret key gastrointestinal & inflammatory markers.
- Understand the role the microbiome plays in Crohn's & Colitis, specifically the role of pathogens in an acute flare.
- Know when to refer, when to treat & how to provide support alongside medical interventions.
- Identify key environmental, dietary, & psychosocial triggers that can initiate and / or exacerbate Crohn's & Colitis flares.
- Develop an evidence-based IBD plan addressing the microbiome, reducing systemic inflammation & providing complementary support alongside medications.

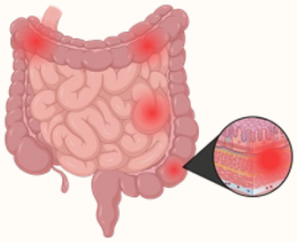
Clinical Conversations

Crohn's & Colitis Overview



5

Crohn's & Colitis



Crohn's Disease

Can occur anywhere along GI tract (mouth to anus), most commonly the terminal ileum. Transmural inflammation, with characteristic "skip lesions".[1-3]

VS

Ulcerative Colitis

Affects the colon and rectum, with inflammation limited to the mucosa and submucosa. Ulceration is continuous in nature, beginning in the rectum.[1-3]

Crohn's & Colitis make up the majority of IBD cases.

Symptoms include:

- Abdominal Pain
- Diarrhoea
- Rectal Bleeding
- Fatigue
- Weight Loss
- Constipation

6

Extra-intestinal Symptoms of IBD[2,4]

Musculoskeletal
- Peripheral arthritis (type 1 & 2)
- Axial Spondyloarthritis
- Arthralgia

Ocular
- Uveitis
- Episcleritis

Dermatologic
- Erythema nodosum
- Pyoderma gangrenosum

Complications of IBD[2,4]

- Anaemia (iron deficiency, Vitamin B12 deficiency, anaemia of chronic disease)
- Osteoporosis
- Strictures + Fistulas
- Venous thromboembolism
- Colorectal cancer

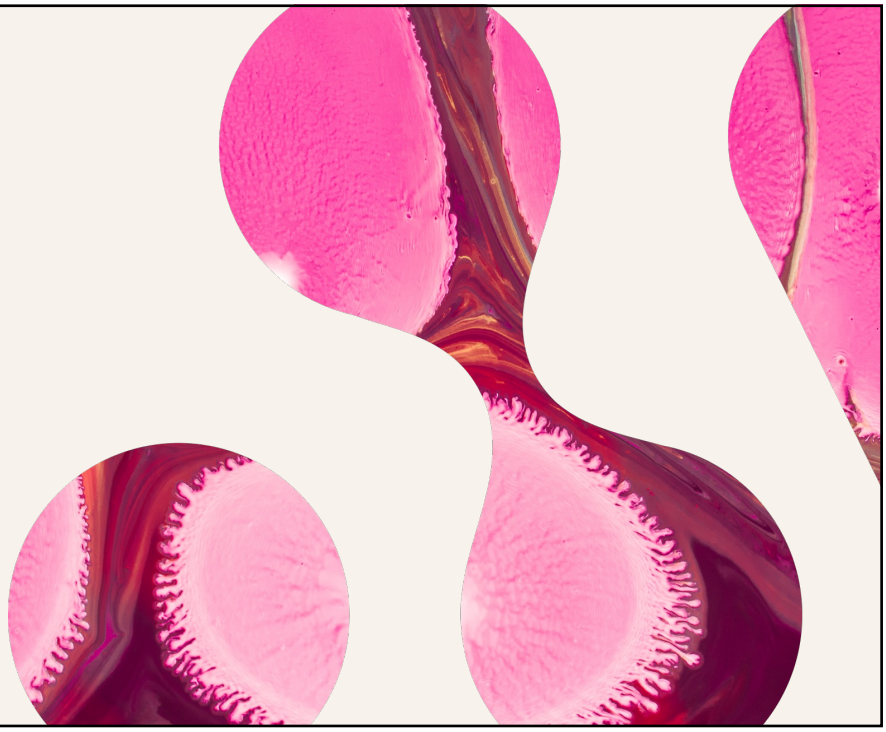
How Does IBD Develop?[2,5]

Complex mix of both genetic & environmental factors

- Medications
- Environmental pollutants
- Gut microbiome
- Stress
- Infections
- Diet

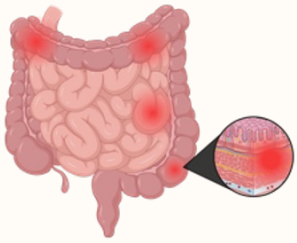
Clinical Conversations

Microbiome Patterns in IBD



9

Microbiome Patterns & IBD



Crohn's Disease[7,9,10]

- Decreased Microbial Diversity
- Decreased Butyrate producers
- Increased Hexa-LPS producers
- Increased Oral species
- ↑ *Escherichia coli*
- ↓ *Faecalibacterium* spp.
- ↓ *Akkermansia* spp.

Ulcerative Colitis[7,9,10]

- Decreased Microbial Diversity
- Increased Oral species
- ↓ *Faecalibacterium* spp.
- ↑ *Escherichia* / Proteobacteria signals

10

The Role of the Microbiome in Active Disease

- **Infections more commonly detected during flares**
  - *C. difficile* has been identified in up to 26% of patients with active disease.
  - Non–*C. difficile* enteric infection identified in ~16–18% of symptomatic IBD tests.[6]
  - Microba in-house data showed increased pathogen levels in active compared to inactive IBD.
- ***E. coli* pathotypes, and *Campylobacter spp.* matter**
  - UC cases had a **higher prevalence of *E. coli* species**, including pathogenic strains, **EAEC & EPEC**, compared with non-IBD controls.[6]
  - Both CD and UC had a **higher prevalence of *Campylobacter spp.*** compared to non-IBD controls.[6]
- **Inflammation can drive *E. coli* expansion**
  - Inflammatory response generates respiratory electron acceptors that selectively support growth of nitrate-respiring **Enterobacteriaceae** helping explain "blooms" during flares.[3,8]
- ***E. coli*-dominant states may reduce medication responsiveness.**
  - Enrichment of Proteobacteria is **associated with anti-TNF non-response** in Crohn's disease.[34]

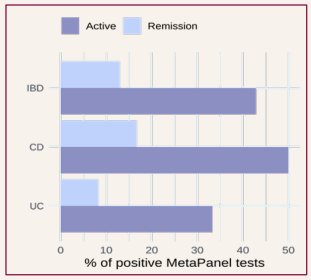


Figure 1: Microba's in-house data on the prevalence of pathogens in IBD patients with active disease vs remission.

The Microbiome in Apparent Remission

- **Remission ≠ symptom-free:**[9,10,13]
  - IBS-like symptoms can persist despite mucosal healing (motility changes, visceral hypersensitivity, SIBO etc.)
- **Microbiome still matters in remission:**[9, 10]
  - IBD remission commonly remains **microbiome-impaired** vs healthy controls (lower "keystone" SCFA producers; higher inflammatory taxa)
- ***E. coli* as a relapse-prone signal:**[35]
  - Across IBD cohorts, remission is often characterised by **lower *Escherichia*** and higher beneficial anaerobes (e.g., *Faecalibacterium spp.*)
- **Clinical Significance:**[34]
  - Baseline microbiome features can help predict **future therapy intensification** (i.e., who may flare or preventative interventions)

Signs to Look For & When to Refer

If symptoms change, assess for inflammation & exclude infection

- **Red flags for active inflammatory disease**: rectal bleeding, diarrhoea, fever, weight loss, persistent pain, anaemia/iron deficiency, elevated CRP[1,2,]
  - ***Faecal Calprotectin[14,15]**
    <100 µg/g: inflammation less likely (consider IBS/dysbiosis)
    100–200 µg/g: repeat/trend + clinical context
    >200 µg/g: active inflammation likely → escalate assessment/referral
  - ***Faecal Lactoferrin[16,17]**
    <7.2 µg/g: inflammation less likely (consider IBS/dysbiosis)
    >7.2 µg/g: active inflammation likely → escalate assessment/referral

    *Cut-offs vary by assay and clinical context

Rule-out infection first when symptoms "flare": stool tests are more likely to present with infection in symptomatic IBD patients.[8]

Investigations for Diagnosis & Monitoring

Blood Tests[1-3]
- **FBC + ferritin:** check for anaemia/iron deficiency
- **CRP/ESR:** supportive markers of systemic inflammation

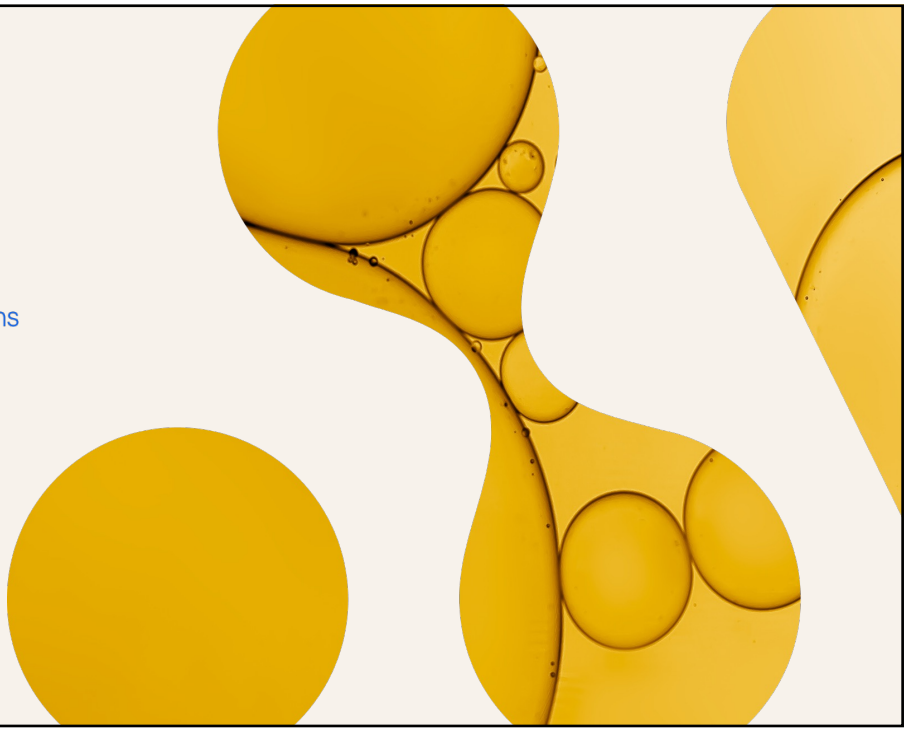
Intestinal Ultrasound (IUS)[18]
- Non-invasive way to assess bowel inflammation (e.g., bowel wall thickness, vascularity) & can be used for **monitoring response to treatment**, especially in Crohn's

Faecal Calprotectin + Lactoferrin[14-17]
- Stool marker of gut inflammation; helpful to **distinguish inflammatory activity vs functional symptoms**
- Calprotectin can rise with infection or NSAID use

Colonoscopy[2,3]
- Gold standard to diagnose IBD & to obtain biopsies
- Used to confirm remission

Clinical Conversations

Microbiome Testing in IBD



15

When You Might Consider Microbiome Testing

Baseline Assessment
- Establishes the patient's microbial ecosystem prior to initiating therapy

Monitoring Therapy
- Evaluate microbial shifts in response to therapeutic interventions
- Facilitates early detection of microbial regression before symptomatic relapse

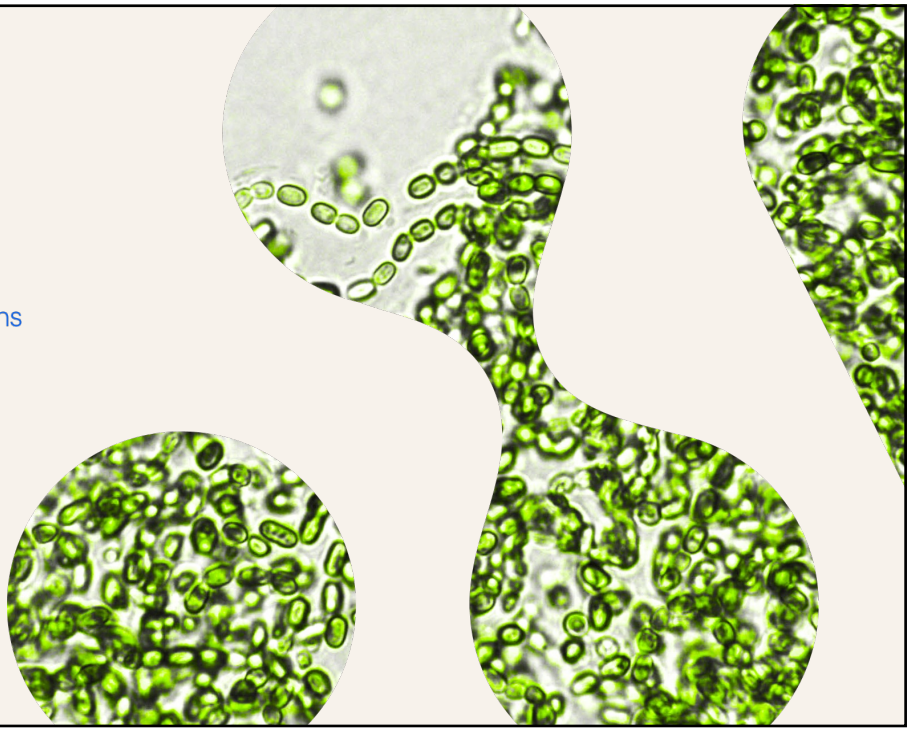
Regular Monitoring (Every 12 Months)
- Annual assessment to track long-term microbial stability & resilience

During A Suspected Flare
- Garner Insight into microbial & functional changes

16

Clinical Conversations

Interventions for IBD



17

Mediterranean Diet in IBD Management

- A recent consensus from the American Gastroenterological Association (AGA) highlights the Mediterranean diet can be beneficial in the management of IBD
- A diet rich in a variety of fresh fruits & vegetables, monounsaturated fats, complex carbohydrates, & lean proteins & low in ultra-processed foods, added sugar, & salt[23,24]
- No diet has consistently been found to decrease the rate of flares in adults with IBD

Conversely, a meta-analysis has found a Western dietary pattern is linked to an increased risk of developing IBD[20,21]

18

Crohn's Disease Exclusion Diet[24, 26]

A structured whole-food diet designed to reduce gut inflammation in Crohn's disease
- promote microbial restoration
- reduce dietary exposures that impair barrier function/microbiome
- Supports mucosal healing

CDED + PEN (Partial Enteral Nutrition) has better compliance than EEN (Exclusive Enteral Nutrition) due to inclusion of **whole foods, with similar success rates!**

Microbial changes have been observed in patients utilising CDED + PEN
- Increased *Faecalibacterium prausnitzii*
- Increased *Bifidobactrium spp.*
- Reduced *E. coli* & *Fusobacterium*

19

Crohn's Disease Exclusion Diet[24, 26]

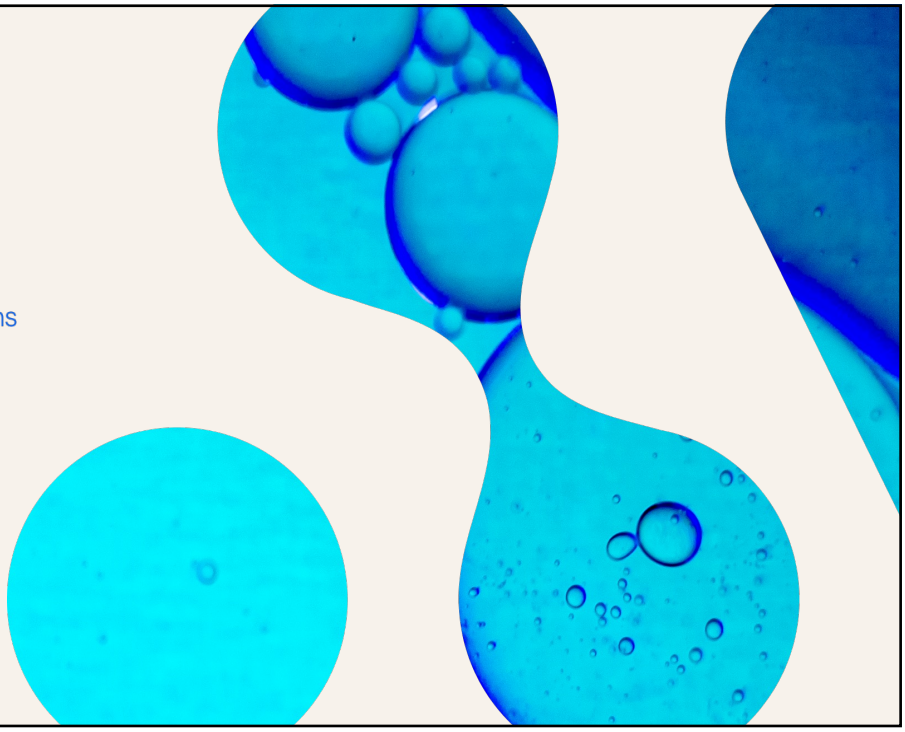
3 phase diet

- **Phase 1** (week 1-6) is often combined with PEN to improve efficacy to induce remission
  - Whole food focus, including:
    - Lean protein (chicken, eggs, white fish, tuna in water)
    - Some whole grains (white rice, potatoes, sweet potatoes, oats)
    - Cooked and peeled veg (carrots, zucchini, pumpkin, cucumber, beans)
  - Exclusion of processed foods, including:
    - Ultraprocessed foods
    - Emulsifiers (gums + carrageenan)
    - Animal fats (including dairy red meat and processed meats)
    - Alcohol, sweetened beverages
- **Phase 2** (week 7-12)
  - Increase variety of foods, including fruits and veg
  - Increase Omega 3 fatty foods, including oily fish
  - PEN optional
  - Maintain strict avoidance to processed foods, inc. emulsifiers
- **Phase 3** (week 13~)
  - Maintenance diet

20

Clinical Conversations

Case Study



21

Case Overview

- Patient: Female in early 40s
- Symptom onset: December 2024, presenting with a Crohn's flare
- Initial pattern: Diarrhoea, cramping, urgency
- Progression:
  - In and out of remission since 2019 (initial diagnosis)
  - Strict vegan diet for 2 years.
  - Presented with iron deficiency anaemia
  - Sex hormones were low across the board (oestrogen, progesterone, testosterone and DHEA-s)
  - Experiencing Increased stool frequency and urgency
- Contributing risk factors:
  - Stress from job
  - Managing a work-life balance with 3 kids at home
  - Travel overseas, triggered flare
  - Diet lacking in quality protein and fat

22

Microbiome Explorer Comprehensive Results, March 2025

High

Faecal pH

A measure of faecal acid-base balance.

0 — 2000

7.79

High

Lactoferrin

A marker of acute gut inflammation.

0 — 40

33.51

High

Methane producing microb...

Methane is an odourless gas produced by Archaeal species, also called methanogen...

AVG

+2.46

High

Calprotectin

A marker of acute gut inflammation.

0 — 1000

204.90

High

Secretory IgA

Protects against pathogens and limits inflammatory response.

0 — 10000

6180.64

Microbiome Explorer Comprehensive Results, March 2025

Borderline

Propionate producing micro...

When produced in balance with the other SCFAs propionate is associated with healt...

Avg

+0.79

Borderline

Trimethylamine producing...

Trimethylamine is produced by gut microbes from the breakdown of choline...

Avg

+0.81

	Species	Phylum	Prevalence	Relative Abundance %	Distance from Average
⊖	Escherichia coli (flexneri)	Proteobacteria	Less common	0.27%	+1.10

Microbiome Explorer Comprehensive Results, March 2025

Optimal

Microbial Diversity

A measure of the number of species detected and how evenly spread they are.

AVG

+0.63

Optimal

Microbial Richness

Microbial richness is s measure of the number of different species in samples, L...

AVG

+0.14

Microbiome Explorer Comprehensive Results, March 2025

Bacterial

Not detected

Aeromonas spp.

Bacteria

Not detected

Clostridium difficile toxin B

Bacteria

Not detected

Enteroaggregative *E. coli* (EAEC)

Bacteria

Not detected

Campylobacter spp.

Bacteria

Not detected

E. coli O157

Bacteria

Detected

Enteropathogenic *E. coli* (EPEC)

Bacteria

Clinical Interpretation: GI Markers

Strong biochemical evidence of active inflammatory bowel disease (IBD)

Faecal Calprotectin: elevated (204.9 mcg/g)
- Indicates neutrophil-mediated mucosal inflammation
- High levels correlated with disease activity
- May be elevated in presence of an infection

Faecal Lactoferrin: elevated (33.5 mcg/g)
- Reflects activated neutrophils in the intestinal lumen
- Confirms active inflammation even when symptoms temporarily improve
- Highly specific for IBD

Faecal Occult Blood: Not Detected

Clinical Objectives: Primary

Primary objective – integrative care + ensuring patient safety
- Collaborate with gastroenterology/GP (rule out complications, ensure appropriate infection management & escalation)
- Patient wants to avoid immune-suppressants & will need monitoring

---

Referral back to gastroenterologist with:
- Microbiome Explorer results
- Discuss interventions to treat EPEC

Ongoing monitoring:
- Regular reviews with intestinal ultrasounds
- Reassessment of faecal calprotectin & CRP
- Assess for malnutrition, dehydration & nutritional deficiencies

Gastroenterology Findings

Colonoscopy in 2019

- Mild inflammation in the duodenum
- Mild-moderate Colitis with granulomas
- Inflammation throughout colon, except rectum
- Terminal ileum looks normal
- Chronic constipation identified

Treatment

- Mesalazine 4g daily (inconsistent)
- Prescribed azathioprine, did not start
- Vegan diet
- *Dientamoeba* treated with doxycycline

Investigations in 2024 / 2025

- Colonoscopy identified inflammation throughout large bowel
- Follow up Colonoscopy showed continued inflammation with mid transverse colon reaching moderate levels of inflammation at 5mm bowel wall thickness

Treatment

- Mesalazine 4g daily
- Prescribed azathioprine, refused
- Mesalazine suppository, 1g daily

Clinical Objectives: Secondary

Secondary functional objectives (integrative care)

Reduce pro-inflammatory microbial load

- Identify & address pro-inflammatory microbes using evidence-informed antimicrobial strategies (dietary + herbal/nutraceutical as appropriate)

Downshift neutrophil-driven inflammation

- Use evidence-based nutritional + herbal interventions to **downregulate pro-inflammatory cytokines** & oxidative stress while supporting immune tolerance

Support mucosal healing + barrier integrity

- Support epithelial repair & mucus layer integrity; reduce permeability

Symptom control + relapse risk reduction

- Reduce stool urgency & fatigue while maintaining adequate intake
- Support stress–gut axis regulation (sleep, vagal tone, nervous system strategies) to reduce flare susceptibility

Replete nutrients

- Screen for common Crohn's-related nutritional deficiencies (iron, B12, folate, vit D, zinc, magnesium)

Naturopathic Supplement Plan: Week 1-2

Supplement	Dosage	Purpose
Gut powder, with glutamine, PHGG, slippery elm, L-theanine	Glutamine 10g twice daily between meals	Support mucosal gut healing, barrier integrity Prebiotic support
Saccharomyces boulardii	1000mg daily	Reduce calprotectin Support microbial resilience[31]
Herbal supplement containing: • *Punica granatum* ext. 283 mg • *Allium sativum* ext. 50 mg • *Carum carvi* oil 100 mg • *Nigella sativa* ext. 100 mg	1 cap twice daily	Address pathobionts Target-methane associated dysbiosis.
Fish oil, containing: • EPA 500mg • DHA 200mg	4 caps daily	Support inflammatory resolution[28-29]
Curcumin (C3 complex)	550mg x 3 daily	Anti-inflammatory & antioxidant support[27]

31

Naturopathic Supplement Plan: Week 3-6

Supplement	Dosage	Purpose
Gut powder, with glutamine, PHGG, slippery elm, L-theanine, **added GOS & lowered glutamine**	Glutamine 5g twice daily GOS 1.5g twice daily	Support mucosal gut healing, barrier integrity Prebiotic support
Lactobacillus gasseri KS-13, 0.75 bill. CFU *Bifidobacterium bifidum* G9-1, 0.125 bill. CFU *Bifidobacterium longum* MM2, 0.125 bill CFU	3 tablets daily (60 days in total)	Reduce *E. coli* overgrowth
Herbal supplement containing: • *Punica granatum* ext. 283 mg • *Allium sativum* ext. 50 mg • *Carum carvi* oil 100 mg • *Nigella sativa* ext. 100 mg	1 cap twice daily	Address pathobionts Target-methane associated dysbiosis
Saccharomyces boulardii	1000mg daily	Reduce calprotectin Support microbial resilience[31]
Fish oil, containing: • EPA 500mg • DHA 200mg	4 caps daily	Support inflammatory resolution[28-29]
Curcumin (C3 complex)	550mg x 3 daily	Anti-inflammatory & antioxidant support[27]

32

Medical Interventions

Medication	Dosage	Duration	Purpose
Azithromycin	500mg	2 days	Address EPEC infection
Mesalazine	1000mg QID	Ongoing	Targets topical GI inflammation
Mesalazine suppositories	1000mg	Ongoing	As above
Iron infusion	1000mg	Once only	Replenish iron stores, bypassing GI

Dietary Recommendations[23,26,29-30]

Diet objective:

- Maintain an anti-inflammatory, whole-food dietary pattern **low in saturated fat & ultra-processed foods**, prioritising **plant diversity** & **prebiotic fibres (including GOS where tolerated)** to help reduce pro-inflammatory dysbiosis & support commensal microbes

Foods to limit/avoid:

- **Ultra-processed foods**, especially additives such as **gums/ emulsifiers**
- **Red meat**
- **High-starch foods (including resistant starches);** aim to reduce **methane-associated fermentation**
- **Saturated & trans fats**, including high-fat dairy & animal fats, to reduce inflammatory load
- **Gluten-containing foods** (confirmed intolerance)
- **Coffee** + Alcohol

Foods to include:

- **Fish**, especially **small oily fish**
- **Lean protein** (lean poultry, eggs, tofu/tempeh)
- **A wide variety of fruits & vegetables**, prioritising **cooked** forms while inflammation is high
- **Plant proteins** such as **tofu & lentils** (as tolerated)
- **Ginger tea**, to support gastric motility / MMC support

Lifestyle Recommendations

Lifestyle objective:

Support flare recovery by **reducing physiological stress load. Encourage a gentle, consistent routines** that support digestion, immune regulation, & adherence

Daily habits:

- **Daily meditation / down-regulation (10–20 min/day)**
- **Daily gentle movement (20–40 min/day)**
- **Sleep as treatment (target 7.5–9 hours)**
- **Daily sunshine**

Nervous system support:

- **Reduce cognitive load during flare**
  Simplify schedule where possible; plan meals, rest, & appointments to reduce decision fatigue
- **Connection + co-regulation**
  Regular check-ins with trusted person; i.e., EMDR therapist, NET practitioner

Clinical Progress: 6 weeks

Objective findings

- Remission induced as per intestinal ultrasound
- Stool markers/symptom profile consistent with reduced inflammatory activity

Bowel symptoms

- Well-formed stools, once daily *(no blood or mucus)*
- Mild urgency Monday mornings, likely work-stress related
- No abdominal pain
- Bloating improved

Systemic health

- Energy excellent
- Hormone testing: improvements across sex hormone markers

Psychological / nervous system

- Anxiety reduced (not fully resolved)

Naturopathic Supplement Plan: Week 6-12

Supplement	Dosage	Purpose
Gut powder - glutamine, PHGG, slippery elm, L-theanine, zinc carnosine **Added GOS & lowered glutamine**	Glutamine 5g twice daily GOS 1.5g daily to begin	Support mucosal gut healing, barrier integrity Prebiotic support[30]
Saccharomyces boulardii	500mg daily	Reduce calprotectin Support microbial resilience[31]
Fish oil, containing: • EPA 500mg • DHA 200mg	2 caps daily	Support inflammatory resolution[28-29]
Curcumin (C3 complex)	550mg x 1 daily	Anti-inflammatory & antioxidant support[27]
Lactobacillus reuteri DSM 17938	100mg twice daily	Address methane-producing archaea[33]
Mushroom complex - turkey tail, cordyceps	1 tsp of powder daily	Immune system support

Follow Up: 6 months

Current presentation

- **Increased bowel motions** since returning to work
- Change appears **context-dependent, unclear if symptoms are IBD or IBS related**

Clinical interpretation

- Pattern suggests a **stress–gut axis contribution**
- Prior remission history supports reassessing **microbiome & gastrointestinal health markers**

Key priorities

- Screen for red flags: blood/mucus, escalating pain, fevers, weight loss
- **Re-check markers**
  - Stool: **Repeat Microbiome Explorer Comprehensive test**
  - Blood tests: **CRP, FBC, Iron studies, Vitamin B12, Vitamin D**
- **Reinforce nervous system supports during work weeks**

Microbiome Explorer Comprehensive Results, Jan 2026

Intestinal Inflammation

High | Insights (4)

Lactoferrin

Lactoferrin is an iron-binding glycoprotein released from neutrophils and is a marker...

Ref Range: < 7.20 µg/g

0.42 — 240.00

18.61 µg/g

Low | Insights (3)

Secretory IgA

Secretory IgA (sIgA) is a marker of intestinal inflammation and increased intestinal...

Ref Range: 500.00 µg/g - 2000.00 µg/g

149.56 — 7500.00

446.88 µg/g

Borderline High | Insights (8)

Calprotectin

Calprotectin is a protein released by neutrophils and is a non-specific marker...

Ref Range: < 50.00 µg/g

11.91 — 2100.00

61.93 µg/g

Microbiome Explorer Comprehensive Results, Jan 2026

Microbiome Markers

High | Insights (4)

Methane producing archaea

Methane is an odourless gas produced by Archaeal species which can also be called...

Compared to Healthy Cohort

Avg

+2.22

Borderline High | Insights (5)

Beta-glucuronidase producing...

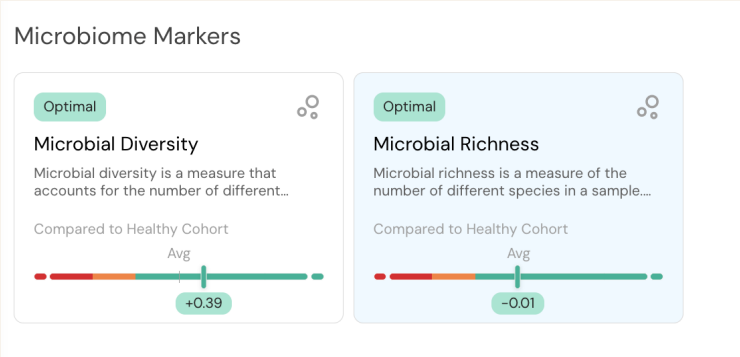
Inulin supplementation may reduce faecal beta-glucuronidase activity.

Compared to Healthy Cohort

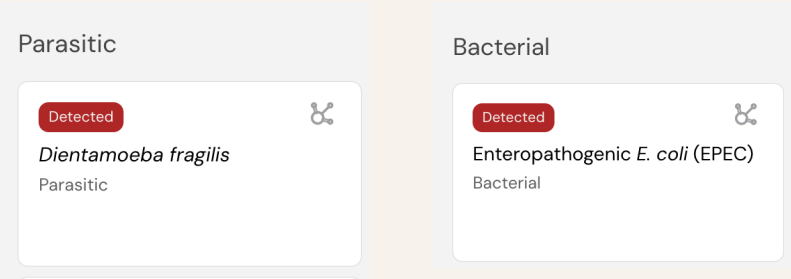
Avg

+1.10

Microbiome Explorer Comprehensive Results, Jan 2026



Microbiome Explorer Comprehensive Results, Jan 2026



E. coli (flexneri) no longer showing in the species explorer (less than 0.01% relative abundance)

Clinical Interpretation

Biochemical evidence of a mild flare of inflammatory bowel disease (IBD)

Faecal Calprotectin: Borderline elevated (62mcg/g)
- Significantly decreased from March 2025 (204.9mcg/g)

Faecal Lactoferrin: elevated (18.6mcg/g)
- Decreased from March 2025 (33.5mcg/g)

Faecal Occult Blood: Not Detected

Clinical Interpretation: Comparison

Improved Crohn's-relevant microbial risk axes:
- ***E. Coli*** **reduced in the Species explorer (0.27% relative abundance to <0.01%)**
  - **Hexa-LPS signal → not detected**
  - Lower Trimethylamine **producing microbes**
- Lower **histamine producing microbes**
- Lower **methane producing microbes**
- Faecal pH has normalised

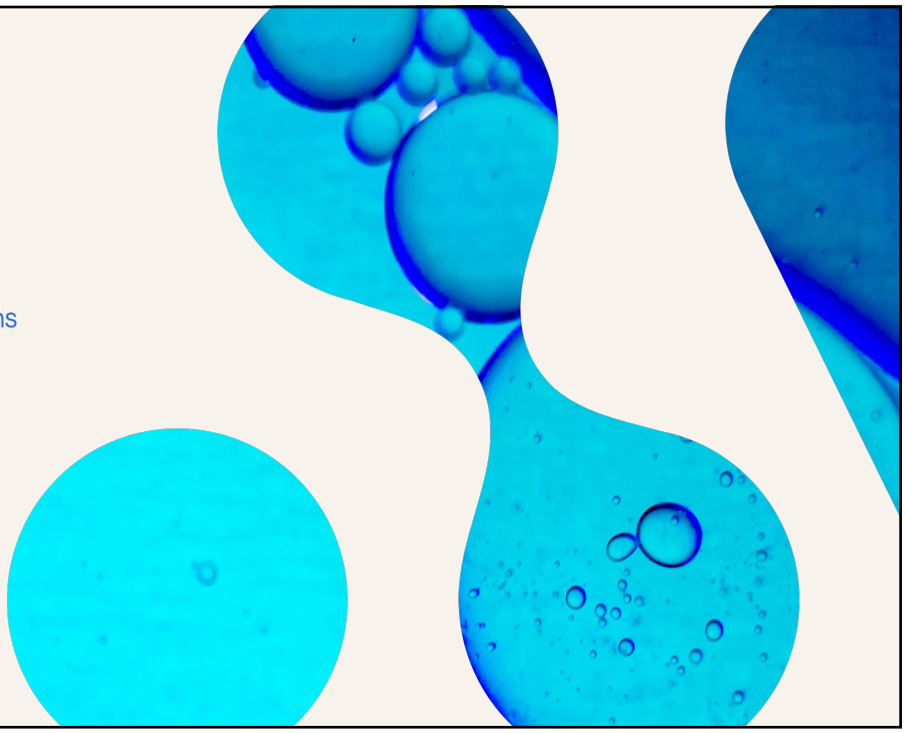
Potential new/ongoing microbial issues:
- **Beta-glucuronidase** rose sharply (now borderline high)
- ***Bifidobacterium*** **dropped to not detected**
- **EPEC persists**, & ***D. fragilis*** **now detected**

Clinical Reflections / Next Steps

Inflammatory burden improved
- Faecal calprotectin and lactoferrin both reduced vs March 2025, suggesting a shift toward better inflammatory control.
- Symptom picture significantly improved
- Borderline FCP + elevated lactoferrin in Jan 2026 suggests ongoing mucosal inflammation is still plausible.

Microbiome overall improved, aligning with reduced inflammatory pressure
- Resolution of *E. coli* species in the species explorer, equates to reduced hexa-LPS producing species, & lower trimethylamine producing species.
- Reduced Methane producing archaea compared with March 2025

Not all microbial markers improved, indicating the need for ongoing support
- Rising beta-glucuronidase may indicate higher deconjugation activity—often a sign the microbiome still needs rebuilding, not just "calming".
- Persistent EPEC signal & detection of *D. fragilis* warrant further consideration.



Clinical Conversations

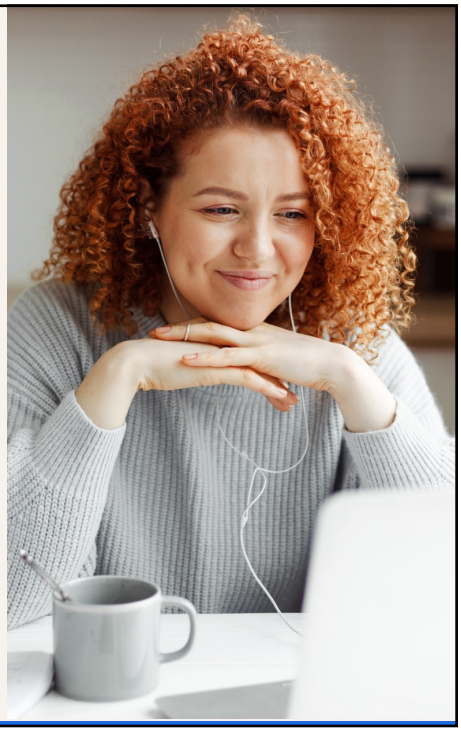
Case Study

Case Overview

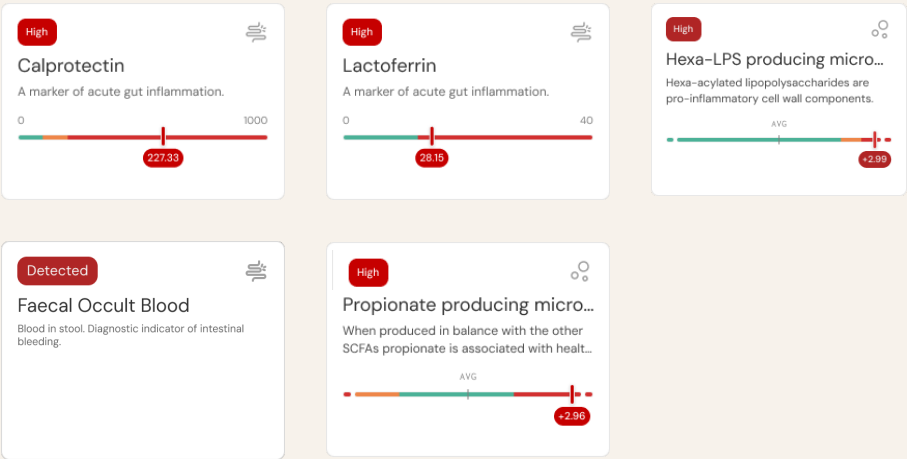
- Patient: Female in early 20s
- Symptom onset: February 2025, presenting with a UC flare
- Symptom pattern: Diarrhoea, cramping, urgency, blood in stool, weight loss, nausea + vomiting

- Progression:
  - In & out of remission since 2023 (initial diagnosis)
  - History of iron deficiency anaemia (infusions)
  - Remission not obtained since diagnosis

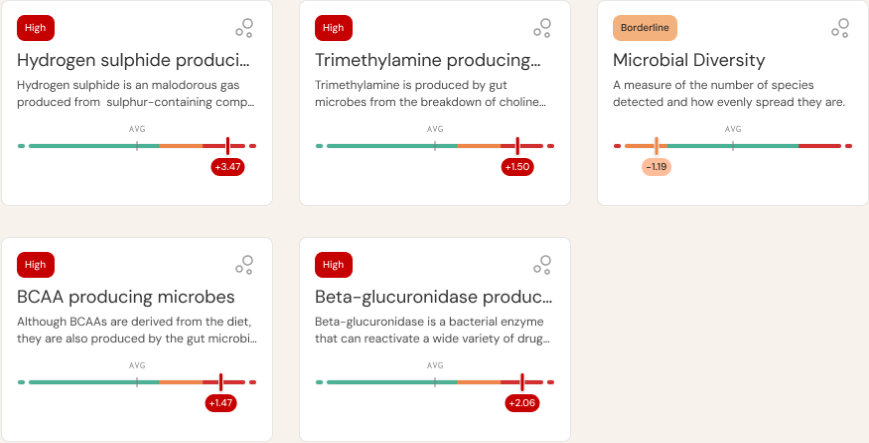
- Contributing risk factors:
  - Diagnosis & flares associated with study stress
  - Poor compliance to treatment due to severe nausea
  - Feels better on a white bread diet
  - Weight loss



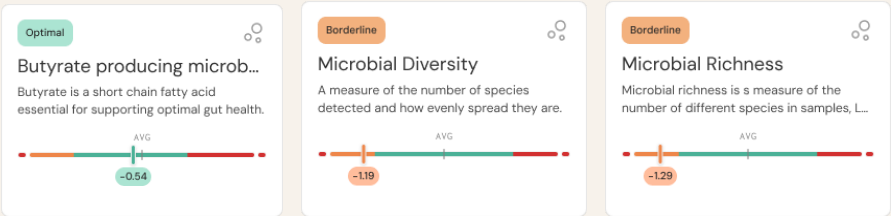
Microbiome Explorer Comprehensive Results, April 2025



Microbiome Explorer Comprehensive Results, April 2025



Microbiome Explorer Comprehensive Results, April 2025



Microbiome Explorer Comprehensive Results, April 2025

	Species	Phylum	Prevalence	Relative Abundance %	Distance from Average
⊖	Escherichia coli	Proteobacteria	Less common	19.62%	+4.18

No pathogenic strains of *E. coli* identified in PCR panel.

E. coli & Hexa-LPS[36]

- *E. coli* has the potential to produce Hexa-LPS (hexa-acylated lipopolysaccharide), a pro-inflammatory endo-toxin
- In the absence of pathogenic strains of *E. coli* (ie. EPEC/EAEC), *E. coli* detected in the species explorer is more indicative of LPS signalling potential of the microbiome
- Hexa-LPS signalling can **amplify inflammation** (NF-κB → cytokines like TNF-α, IL-6, IL-1β)
- Hexa LPS has the potential to amplify an inflammatory loop in the gut, worsening barrier disruption & permeability
- Signs & symptoms of elevated Hexa-LPS in a patient include malaise, diffuse aches & pain, GI cramping

Clinical Interpretation: GI Markers

Strong biochemical evidence of active inflammatory bowel disease (IBD)

Faecal Calprotectin: elevated (227.3mcg/g)

- Indicates neutrophil-mediated mucosal inflammation
- High levels correlate with disease activity, mucosal ulceration, & risk of flare

Faecal Lactoferrin: elevated (28.15mcg/g)

- Reflects activated neutrophils in the intestinal lumen
- Confirms active inflammation
- Highly specific for IBD

Faecal Occult Blood: DETECTED

The combination of these 3 clinical signs are highly correlated with active inflammatory bowel disease.

Clinical Objectives: Primary

Primary objective – Stabilise acute flare

- Patient is under the care of a GP & a gastroenterologist
- The patient is on multiple immune suppressant medications without remission
- Review by gastroenterologist is required.

Protect nutritional status

- Minimise loss of blood
- Stop diarrhoea
- Rebuild appetite, support rehydration.
- Increase calorie intake

Monitoring

- Colonoscopy required to investigate extent of inflammation
- Gastroscopy required to investigate nausea & vomiting
- Assess iron levels

Clinical Objectives: Secondary

Secondary functional objectives (integrative care)

Reduce pro-inflammatory microbial load
- Reduce *E. coli* to reduce Hexa-LPS production
- Use evidence-informed antimicrobial strategies (dietary + herbal/nutraceutical as appropriate)

Downshift neutrophil-driven inflammation
- Aim to **downregulate pro-inflammatory cytokines**

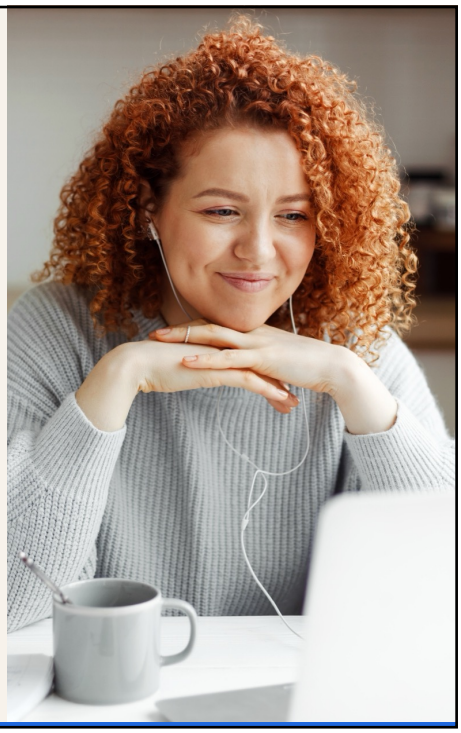
Support mucosal healing + barrier integrity
- Support epithelial repair & mucus layer integrity; reduce permeability

Support stress–gut axis regulation
- Identify strategies to support nervous system regulation to enable remission

Replete nutrients
- Screen/restore common IBD-related nutritional deficiencies (iron, B12, folate, vit D, zinc, magnesium)

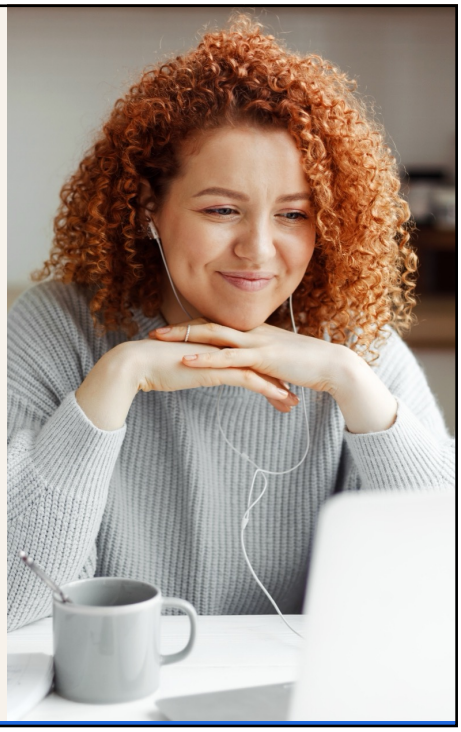
Naturopathic Supplement Plan

Supplement	Dosage	Purpose
Gut powder - glutamine, zinc carnosine, Vitamin A, Quercetin	7g twice daily	Support mucosal gut healing, reduce inflammation Replenish nutrients
Saccharomyces boulardii	1000mg daily	Reduce calprotectin Support microbial resilience[31]
Curcumin	550mg x 3 daily	Anti-inflammatory & antioxidant support[27]
Vitamin D	3000IU daily	Repletion Support healthy immune functioning[37]
Lactobacillus gasseri KS-13, 0.75 bill. CFU *Bifidobacterium bifidum* G9-1, 0.125 bill. CFU *Bifidobacterium longum* MM2, 0.125 bill CFU	3 tablets daily (60 days in total)	Reduce *E. coli* overgrowth[38]



Medical Interventions

Medication	Dosage	Duration	Purpose
Budesonide	9mg daily	30 days	Short-term corticosteroid to reduce GI inflammation
Mesalazine	4200mg daily	ongoing	Targets topical GI inflammation
Thioguanine	20mg daily	ongoing	Immune suppressant to reduce inflammation
Iron infusion	1000mg	Once only	Replenish iron stores, bypassing GI
Vedolizumab	Awaiting approval		Gut Selective Monoclonal Antibody to reduce white blood cell immune activation



Dietary Recommendations[23,26,29-30]

Diet objective

Increase calorie intake, reduce GI irritation, stabilise hydration/electrolytes, minimise further weight loss

Core principles

- **Small, frequent feeds**: 6–8 "mini-meals"/day (less nausea, easier calorie delivery)
- **Low residue diet** (temporarily): reduce bulky fibre, grains, skins, seeds, nuts, popcorn, raw salads
- **Keep meals bland,** Low fat, low spice
- Trial lactose-free dairy or avoid milk; yoghurt may be better tolerated

Preferred foods (gentle, calorie-efficient)

- **Carbs**: white rice, rice congee, potatoes (no skins), oats, spelt sourdough
- **Proteins**: eggs (if tolerated), chicken/turkey, fish, tofu, lactose-free yoghurt, smooth nut butters (small amounts)
- **Fruits/veg**: peeled cooked options (carrot, pumpkin, zucchini), mashed banana, stewed apple/pear
- **Fats (small additions for calories)**: olive oil, MCT oil, avocado only
- **Soups & stews** reduces pressure on the GI tract
- **Ginger tea**, to manage nausea

Foods to limit/avoid

- **Ultra-processed foods**, especially additives such as **gums/ emulsifiers**
- **High-starch foods (including resistant starches)** if they worsen gas/bloating
- **Saturated & trans fats**, including high-fat dairy & animal fats, to reduce inflammatory load
- **Gluten-containing foods** (confirmed intolerance)
- **Coffee**

Partial Enteral Nutrition[24,26]

Aim: deliver a meaningful portion of daily energy via formula while keeping some oral intake to improve nutrition uptake & calorie increase

PEN targets
- start: 50% of daily calories from formula for 3–5 days
- Practical adult range: ~500–1500 kcal/day from formula

Elemental Formula
Elemental, nutritionally complete hypoallergenic medical food (powder) providing 100% free-form amino acids, readily absorbed carbohydrates, & MCT as a gentle fat source.
- formulated to deliver the daily recommended allowances of essential nutrients, vitamins, & minerals in easily digestible forms
- **Low residue, easy to assimilate:** designed for situations with **impaired digestion/absorption** & to reduce symptom burden by limiting food-particle interaction further along the GI tract

Dosing
Divide the PEN portion into **~200–300 kcal servings** taken **every ~2–3 hours**

Duration
2-4 weeks to begin

Lifestyle Recommendations

Lifestyle objective
Support flare recovery by **reducing physiological stress load**, improving **sleep & autonomic balance**, & maintaining **gentle, consistent routines** that support digestion, immune regulation, & adherence

Daily habits
- **Daily prayer,** mindful practice (already in her practice)
- **Daily gentle movement (20–40 min/day)**
  Walking, mobility, Pilates/yoga (low intensity).
- **Sleep as treatment (target 7.5–9 hours)**
  Fixed wake time, morning light exposure, screens off 60 min pre-bed, & wind-down routine
- **Daily sunshine**
  Start with 5 mins of sunshine on the face in the morning (within 30 mins of waking)
  20-30 mins of sun on the skin for vitamin D support

Nervous system support
- **Prioritise rest**
  Simplify schedule where possible; plan meals, rest, & appointments to reduce decision fatigue
- **Connection + co-regulation**
  Social outings with family & friends

Clinical Progress: 16 weeks

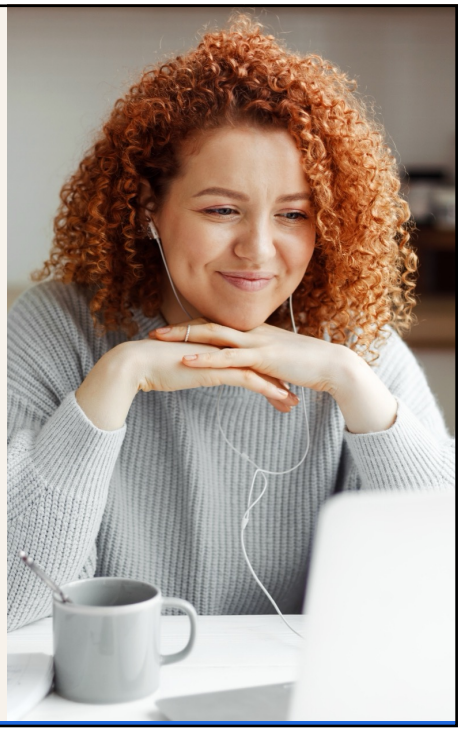
Short remission followed by relapse

Findings
- Treatment failure (including to biologic medication)
- Faecal calprotectin remained elevated

Bowel symptoms
- Frequent stools, watery diarrhoea
- Blood in stool
- Nausea persists

Systemic health
- Energy depleted

Psychological / nervous system
- Anxiety is high due to work stress



61

Treatment Progress

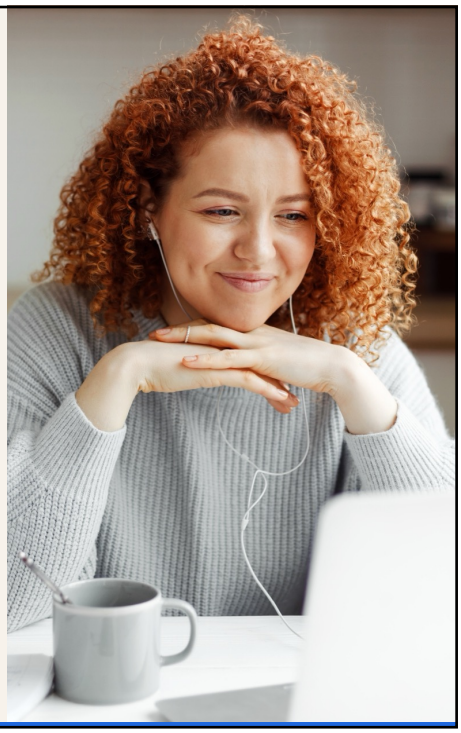
Medications
- Considered to have failed first biologic medication (vedolizumab)
- Started on prednisolone for 5 weeks

Supplements maintained
- *Saccharomyces boulardii*
- *Lactobacillus gasseri* KS-13, 0.75 bill. CFU, *Bifidobacterium bifidum* G9-1, 0.125 bill. CFU, *Bifidobacterium longum* MM2, 0.125 bill CFU

All other supplements were ceased due to nausea

Diet
- Gluten free
- Whole food primarily
- Did not try PEN

Aversion to powders



62

Microbiome Explorer Comprehensive Results, Jan 2026

Gastrointestinal Health Markers

High
Secretory IgA
Secretory IgA (sIgA) is a marker of intestinal inflammation and increased intestinal...
Ref Range: 500.00 µg/g - 2000.00 µg/g
149.56 — 7500.00
2715.33 µg/g

Borderline Low
Pancreatic Elastase
Pancreatic elastase is an enzyme produced by the pancreas that passes undigested...
Ref Range: ≥ 200.00 µg/mL
20.55 — 720.00
140.24 µg/mL

Optimal
Lactoferrin
Lactoferrin is an iron-binding glycoprotein released from neutrophils and is a marker...
Ref Range: < 7.20 µg/g
0.42 — 240.00
2.97 µg/g

Optimal
Calprotectin
Calprotectin is a protein released by neutrophils and is a non-specific marker...
Ref Range: < 50.00 µg/g
11.91 — 2100.00
< 11.91 µg/g

Optimal
Zonulin
Zonulin is a physiological regulator of intestinal permeability. Zonulin binds to a...
Ref Range: < 100.00 ng/mL
9.40 — 800.00
34.50 ng/mL

Optimal
Faecal pH
Faecal pH is a marker of gut transit time. Low faecal pH may be indicative of rapid...
Ref Range: 5.70 - 7.30
0.00 — 14.00
6.34

Not Detected
Faecal Occult Blood
Faecal occult blood is a marker of intestinal bleeding. Early diagnosis of faecal occult...

Microbiome Explorer Comprehensive Results, Jan 2026

Microbiome Markers

High
Oral Species
Oral species are species commonly found in the mouth. High levels may be...
Compared to Healthy Cohort
Avg
+1.43

Low Insights (7)
Microbial Diversity
Microbial diversity is a measure that accounts for the number of different...
Compared to Healthy Cohort
Avg
−2.71

Low Insights (8)
Butyrate producing microbes
Butyrate is a beneficial short-chain fatty acid that is important for gut health. Low...
Compared to Healthy Cohort
Avg
−3.32

Low
Microbial Richness
Microbial richness is a measure of the number of different species in a sample...
Compared to Healthy Cohort
Avg
−2.58

Not Detected Insights (3)
IPA producing microbes
3-indolepropionic acid (IPA) is a beneficial substance produced by some gut bacteri...

Borderline Low Insights (1)
Oxalate consuming microbes
Oxalate is a key component of calcium oxalate kidney stones. Low microbial...
Compared to Healthy Cohort
Avg
−1.04

Borderline Low Insights (5)
Propionate producing microbes
Propionate is a short-chain fatty acid produced by the gut microbiome. Optimal...
Compared to Healthy Cohort
Avg
−0.98

Optimal
Acetate producing microbes
Acetate is the most abundant short-chain fatty acid produced by our gut microbiom...
Compared to Healthy Cohort
Avg
−0.30

Optimal
Mucin Degradation
Mucin is a component of the mucus layer that lines and protects the epithelial gut...
Compared to Healthy Cohort
Avg
−0.55

Microbiome Explorer Comprehensive Results, Jan 2026

Optimal

Hexa-LPS producing microbes

Hexa-acylated lipopolysaccharides (hexa-LPS) are bacterial cell wall components of...

Compared to Healthy Cohort

Avg

-1.26

Optimal

Hydrogen sulphide producing...

The gas hydrogen sulphide is produced by gut microbes when they break down...

Compared to Healthy Cohort

Avg

-2.01

Bacterial

Detected

Enteropathogenic *E. coli* (EPEC)

Bacterial

Species	Phylum	Prevalence	Relative Abundance %	Distance from Average
Escherichia coli (flexneri)	Proteobacteria	Less common	0.02 %	-0.80

Oral Gut Microbial Overlap & Signatures in IBD[21,22]

Studies have found greater oral and gut bacterial similarity in patients with ulcerative colitis (UC) and Crohn's disease (CD) compared to healthy controls (HC)

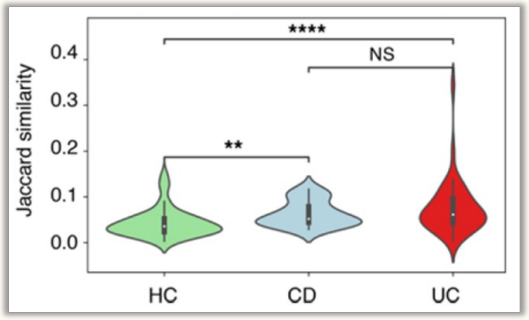
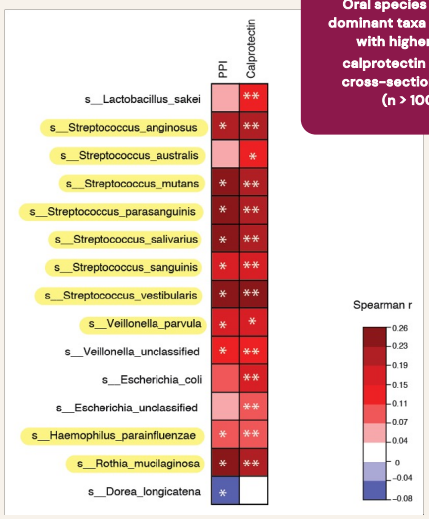


Image from: Imai et al 2021

Oral species were the dominant taxa associated with higher faecal calprotectin in a large cross-sectional study (n > 1000)



Clinical Interpretation

Remission achieved!

Faecal Calprotectin: In healthy range (11.9mcg/g)
- Significantly decreased from April 2025 (227.3mcg/g)

Faecal Lactoferrin: In healthy range (2.97mcg/g)
- Decreased from April 2025 (28.15mcg/g)

Faecal Occult Blood: Not Detected (was detected in April 2025)

Clinical Interpretation: Microbiome Comparison

Improved UC-related microbial risk axes:
***E. coli* has reduced from 19.62% relative abundance to <0.01%**
- Hexa-LPS producing microbes → significantly decreased
- Trimethylamine & H_2S producing microbes → significantly decreased

Potential new/ongoing microbial issues:
- Decreased diversity & richness
- Oral species have increased
- EPEC detected in pathogen panel, along with *E. coli* (flexneri) at 0.02% relative abundance, in the species explorer
- Decreased butyrate producing microbes

Clinical Context Between Reports

- Patient was **hospitalised with a severe UC flare** between reports
- **Vedolizumab was ineffective (treatment failure)**
- Patient achieved **clinical remission following a course of prednisolone**, & the repeat test was completed **4 weeks after stopping prednisolone** (i.e., *off-steroid at time of re-test*)
- Patient was **largely non-compliant with supplements** due to **severe nausea**

Clinical Reflections / Next Steps

- **Reduced *E. coli*** alongside normalised **calprotectin** & **lactoferrin**, is consistent with improved inflammation control. The drop in *E. coli* may be **secondary to reduced gut inflammation** (remission environment is less favourable for Proteobacteria expansion), rather than solely due to dietary/supplement changes

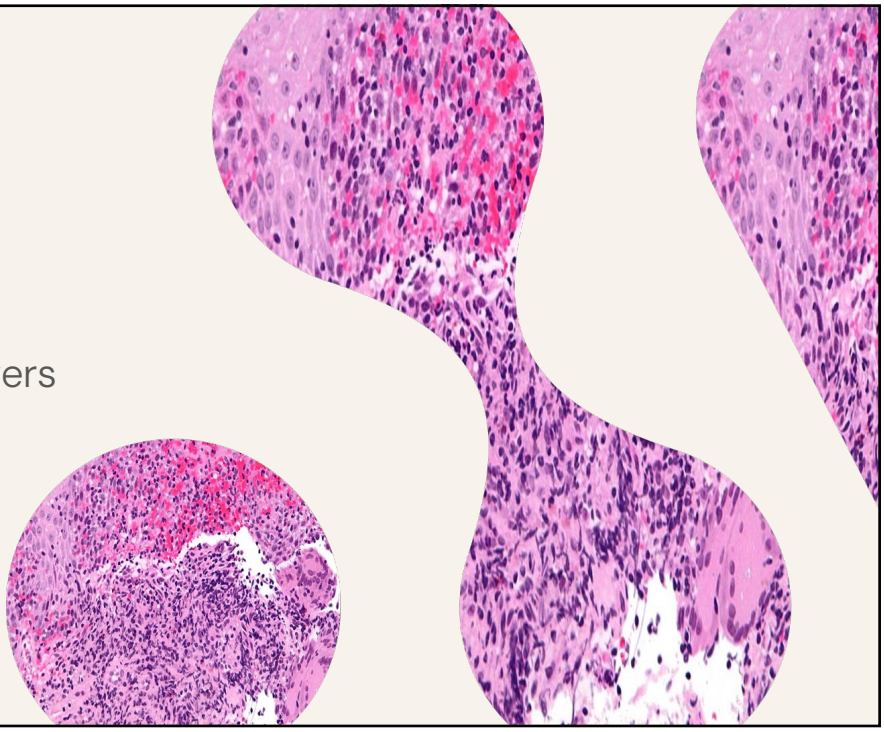
- **Keep the patient connected to care:** support ongoing treatment & follow-up **even if they can't make changes right now**, to ensure they receive the right medical management

- **Now focus on microbiome support:** with inflammation controlled medically, the next step is to **strengthen the microbiome** to help **maintain remission**

- **Microbiome & medication response:** early research suggests the microbiome may influence biologic response; the patient's earlier dysbiosis may have contributed to treatment failure.

Clinical Conversations

Questions & Answers



References

1. National Health Service (UK). Inflammatory bowel disease (IBD) [Internet]. NHS; [cited 2026 Feb 23].
2. Lamb CA, Kennedy NA, Raine T, et al. British Society of Gastroenterology consensus guidelines on the management of inflammatory bowel disease in adults. *Gut*. 2019;68(Suppl 3):s1–s106.
3. Kucharzik T, Ellul P, Greuter T, et al. ECCO-ESGAR-ESP-IBUS Guideline on diagnostics and monitoring of patients with inflammatory bowel disease: Part 1 (initial diagnosis, monitoring of known IBD, detection of complications). *J Crohns Colitis*. 2025;19(7):jjaf106. doi:10.1093/ecco-jcc/jjaf106
4. Kilic Y, Kamal S, Jaffar F, et al. Prevalence of extraintestinal manifestations in inflam9 matory bowel disease: a systematic review and meta-analysis. *Inflamm Bowel Dis*. 2024;30(2):230–239.
5. de Souza HSP, Fiocchi C, Iliopoulos D. The IBD interactome: an integrated view of aetiology, pathogenesis and therapy. *Nat Rev Gastroenterol Hepatol*. 2017;14(12):739–749. doi:10.1038/nrgastro.2017.110
6. Axelrad JE, Joelson A, Green PHR, Lawlor G, Lichtiger S, Cadwell K, Lebwohl B. Enteric infections are common in patients with flares of inflammatory bowel disease. *Am J Gastroenterol*. 2018;113(10):1530–1539.
7. Shin NR, Whon TW, Bae JW. Proteobacteria: microbial signature of dysbiosis in gut microbiota. *Trends Biotechnol*. 2015;33(9):496–503.
8. Winter SE, Winter MG, Xavier MN, et al. Host-derived nitrate boosts growth of *Escherichia coli* in the inflamed gut. *Science*. 2013;339(6120):708–711. doi:10.1126/science.1232467
9. Halfvarson J, Brislawn CJ, Lamendella R, et al. Dynamics of the human gut microbiome in inflammatory bowel disease. *Nat Microbiol*. 2017;2:17004. doi:10.1038/nmicrobiol.2017.4
10. Wills ES, Jonkers DMAE, Savelkoul PHM, Masclee AAM, Pierik MJ. Fecal microbial composition of ulcerative colitis and Crohn's disease patients in remission and subsequent exacerbation. *PLoS One*. 2014;9(3):e90981. doi:10.1371/journal.pone.0090981
11. Sanchis-Artero L, Martínez-Blanch JF, Manresa-Vera S, et al. Evaluation of changes in intestinal microbiota in Crohn's disease patients after anti-TNF-α treatment. *Sci Rep*. 2021;11:10016. doi:10.1038/s41598-021-88823-2
12. Ding NS, McDonald JAK, Perdones-Montero A, et al. Metabonomics and the gut microbiome associated with primary response to anti-TNF therapy in Crohn's disease. *J Crohns Colitis*. 2020;14(8):1090–1102. doi:10.1093/ecco-jcc/jjaa03
13. Al Radi ZMA, Prins FM, Collij V, et al. Exploring the predictive value of gut microbiome signatures for therapy intensification in patients with inflammatory bowel disease: a 10-year follow-up study. *Inflamm Bowel Dis*. 2024;30(10):1642–1653. doi:10.1093/ibd/izae064
14. York & Scarborough Teaching Hospitals NHS Foundation Trust. Faecal calprotectin care pathway [Internet]. [cited 2026 Feb 23].

References

15. National Institute for Health and Care Excellence (NICE). Faecal calprotectin diagnostic tests for inflammatory diseases of the bowel (DG11) [Internet]. 2013 [cited 2026 Feb 23].
16. Walsham NE, Sherwood RA. Measurement of faecal calprotectin and lactoferrin in inflammatory bowel disease. *Frontline Gastroenterol.* 2011;2(1):13–18.
17. Zhou XL, Liu W, Wu YJ, et al. Fecal lactoferrin in discriminating inflammatory bowel disease from irritable bowel syndrome: a diagnostic meta-analysis. *BMC Gastroenterol.* 2014;14:121. doi:10.1186/1471-230X-14-121
18. Chavannes M, Dolinger MT, Cohen-Mekelburg S, Abraham B. AGA Clinical Practice Update on the role of intestinal ultrasound in inflammatory bowel disease. *Clin Gastroenterol Hepatol.* 2024;22(9):1790–1795.e1. doi:10.1016/j.cgh.2024.04.039
19. Colombel JF, Shin A, Gibson PR. AGA Clinical Practice Update on functional gastrointestinal symptoms in patients with inflammatory bowel disease. *Clin Gastroenterol Hepatol.* 2019;17(3):380–390.e1. doi:10.1016/j.cgh.2018.08.001
20. Gracie DJ, Guthrie EA, Hamlin PJ, Ford AC. Prevalence of irritable bowel syndrome-type symptoms in inflammatory bowel disease in remission: systematic review and meta-analysis. *Lancet Gastroenterol Hepatol.* 2020;5(12):1053–1062.
21. Imai J, Kitamoto S, Sugihara K, et al. Oral–gut microbial overlap and oral taxa in the gut associated with inflammatory bowel disease. *JCI Insight.* 2021;6:e148543.
22. Salosensaari A, Laitinen V, Havulinna AS, et al. Faecal calprotectin and gut microbiome associations in a population cohort. *Sci Rep.* 2024;14:63893.
23. Narula N, Wong ECL, Dehghan M, et al. Association of ultra-processed food intake with risk of inflammatory bowel disease: prospective cohort study. *BMJ.* 2021;374:n1554. doi:10.1136/bmj.n1554
24. Levine A, Wine E, Assa A, et al. Crohn's Disease Exclusion Diet plus partial enteral nutrition induces sustained remission in a randomized controlled trial. *Gastroenterology.* 2019;157(2):440–450.e8. doi:10.1053/j.gastro.2019.04.021
25. Cox SR, Lindsay JO, Fromentin S, et al. Effects of Low-FODMAP diet on symptoms, fecal microbiome, and markers of inflammation in patients with quiescent inflammatory bowel disease in a randomized trial. *Gastroenterology.* 2020;158(1):176–188.e7. doi:10.1053/j.gastro.2019.09.024
26. ESPEN guideline on clinical nutrition in inflammatory bowel disease. *Clin Nutr.* 2023. (ESPEN Guideline document).
27. Lang A, Salomon N, Wu JCY, et al. Curcumin in combination with mesalamine induces remission in patients with mild-to-moderate ulcerative colitis in a randomized controlled trial. *Clin Gastroenterol Hepatol.* 2015;13(8):1444–1449.e1. doi:10.1016/j.cgh.2015.02.019

References

28. Prossomariti A, Scaioli E, Piazzi G, et al. Short-term treatment with eicosapentaenoic acid improves inflammation and affects colonic differentiation markers and microbiota in patients with ulcerative colitis. *Sci Rep.* 2017;7:7458. doi:10.1038/s41598-017-07992-1
29. Lyte JM, Gabler NK, Hollis JH. Postprandial serum endotoxin in healthy humans is modulated by dietary fat in a randomized, controlled, cross-over study. *Lipids Health Dis.* 2016;15:186. doi:10.1186/s12944-016-0357-6
30. Vulevic J, Drakoularakou A, Yaqoob P, Tzortzis G, Gibson GR. Modulation of the fecal microflora profile and immune function by a novel trans-galactooligosaccharide mixture (B-GOS) in healthy elderly volunteers. *Am J Clin Nutr.* 2008;88(5):1438–1446. doi:10.3945/ajcn.2008.26242
31. Zhou W. Effect of *Saccharomyces boulardii* powder on fecal calprotectin and inflammatory factors in active ulcerative colitis. *Shijie Huaren Xiaohua Zazhi* (World Chinese J Digestol). 2017;25(22):2065–2070. doi:10.11569/wcjd.v25.i22.2065
32. Spaiser SJ, Culpepper T, Nieves C Jr, Ukhanova M, Mai V, Percival SS, et al. *Lactobacillus gasseri* KS-13, *Bifidobacterium bifidum* G9-1, and *Bifidobacterium longum* MM-2 ingestion induces a less inflammatory cytokine profile and a potentially beneficial shift in gut microbiota in older adults: a randomized, double-blind, placebo-controlled, crossover study. *J Am Coll Nutr.* 2015;34(6):459–469. doi:10.1080/07315724.2014.983249
33. Ojetti V, Petruzziello C, Migneco A, et al. Effect of *Lactobacillus reuteri* (DSM 17938) on methane production in patients affected by functional constipation: a retrospective study. *Eur Rev Med Pharmacol Sci.* 2017;21:1702–1708.
34. Microbiome signatures and biologic response/nonresponse in inflammatory bowel disease (anti-TNF etc.). J Crohns Colitis. 2020;14(8):1090–1102. Available from: https://academic.oup.com/ecco-jcc/article/14/8/1090/5771407
35. Winter SE, Winter MG, Xavier MN, Thiennimitr P, Poon V, Keestra AM, et al. Host-derived nitrate boosts growth of *Escherichia coli* in the inflamed gut. Science. 2013;339(6120):708–711. doi:10.1126/science.1232467.
36. Raetz CRH, Whitfield C. Lipopolysaccharide endotoxins. *Annu Rev Biochem.* 2002;71:635–700. doi:10.1146/annurev.biochem.71.110601.135414.
37. Valvano M, Magistroni M, Cesaro N, et al. Effectiveness of vitamin D supplementation on disease course in inflammatory bowel disease patients: systematic review with meta-analysis. *Inflamm Bowel Dis.* 2024;30(2):281–291. doi:10.1093/ibd/izac253.
38. Spaiser SJ, Culpepper T, Nieves C Jr, Ukhanova M, Mai V, Percival SS, Christman MC, Langkamp-Henken B. *Lactobacillus gasseri* KS-13, *Bifidobacterium bifidum* G9-1, and *Bifidobacterium longum* MM-2 ingestion induces a less inflammatory cytokine profile and a potentially beneficial shift in gut microbiota in older adults: a randomized, double-blind, placebo-controlled, crossover study. *J Am Coll Nutr.* 2015;34(6):459–469. doi:10.1080/07315724.2014.983249.
39. Rohani P, Ansari A, Shaygantabar M, Hekmatdoost A, Sohouli MH. The effects of the Crohn's disease exclusion diet (CDED) alone versus CDED plus partial enteral nutrition (PEN) on gut microbiome composition in pediatric Crohn's disease patients. MicrobiologyOpen. 2025 Oct;14(5):e70099. doi:10.1002/mbo3.70099.