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WEBINAR

Managing Gut Autoimmunity:

Advanced microbiome strategies for IBD

Speaker

Hannah Braye
Nutrition Practitioner
Clinical Director – Microba UK

Housekeeping

All participants have been muted

Questions will be answered at the end of the presentation

Add your questions in the Q&A tab to have them answered

Microba's Pioneering Journey

From foundational metagenomics research to clinical-grade microbiome testing

30+

peer-reviewed papers on gut microbiome metagenomics

2004

Tyson, G. W., Et F. (2004). Seminal Nature paper on microbial genome reconstruction from the environment.

2012

Professors Gene Tyson and Philip Hugenholtz begin core metagenomics and bioinformatics research at The **University of Queensland.**

2017

Microba founded to turn advanced metagenomic science into clinical and research tools.

2018

Microba launch **high-resolution shotgun metagenomic testing**.

2021

Microba Scientific Review Process developed to help practitioners evaluate gut microbiome evidence.

2022

Microba Microbiome Explorer released as **Australia's first clinical-grade microbiome test**.

2023

Microba acquisition of **Invivo Healthcare** in the UK

2024

Microbiome Explorer upgraded with **faster reporting and new evidence summaries** for clinical decision-making.

2025

Microba launches clinical-grade microbiome testing in the **UK**

Our multidisciplinary team spans microbiology, bioinformatics and clinical practice, to translate microbiome science into real-world impact

OBJECTIVES

Learning objectives

01 **Understand** the key mechanisms driving autoimmunity, including the role of intestinal dysbiosis in autoimmune pathophysiology

02 **Identify** common environmental, dietary, psychosocial and microbiome-related triggers that may initiate IBD onset or exacerbate IBD flares.

03 **Recognise** key microbial patterns associated with Crohn's Disease and Ulcerative Colitis

04 **Interpret** GI inflammatory markers (such as faecal calprotectin) to differentiate between functional and organic disease presentations.

05 **Select** evidence-based interventions to help support remission, address microbial drivers of intestinal inflammation and support wider gut and systemic health in IBD

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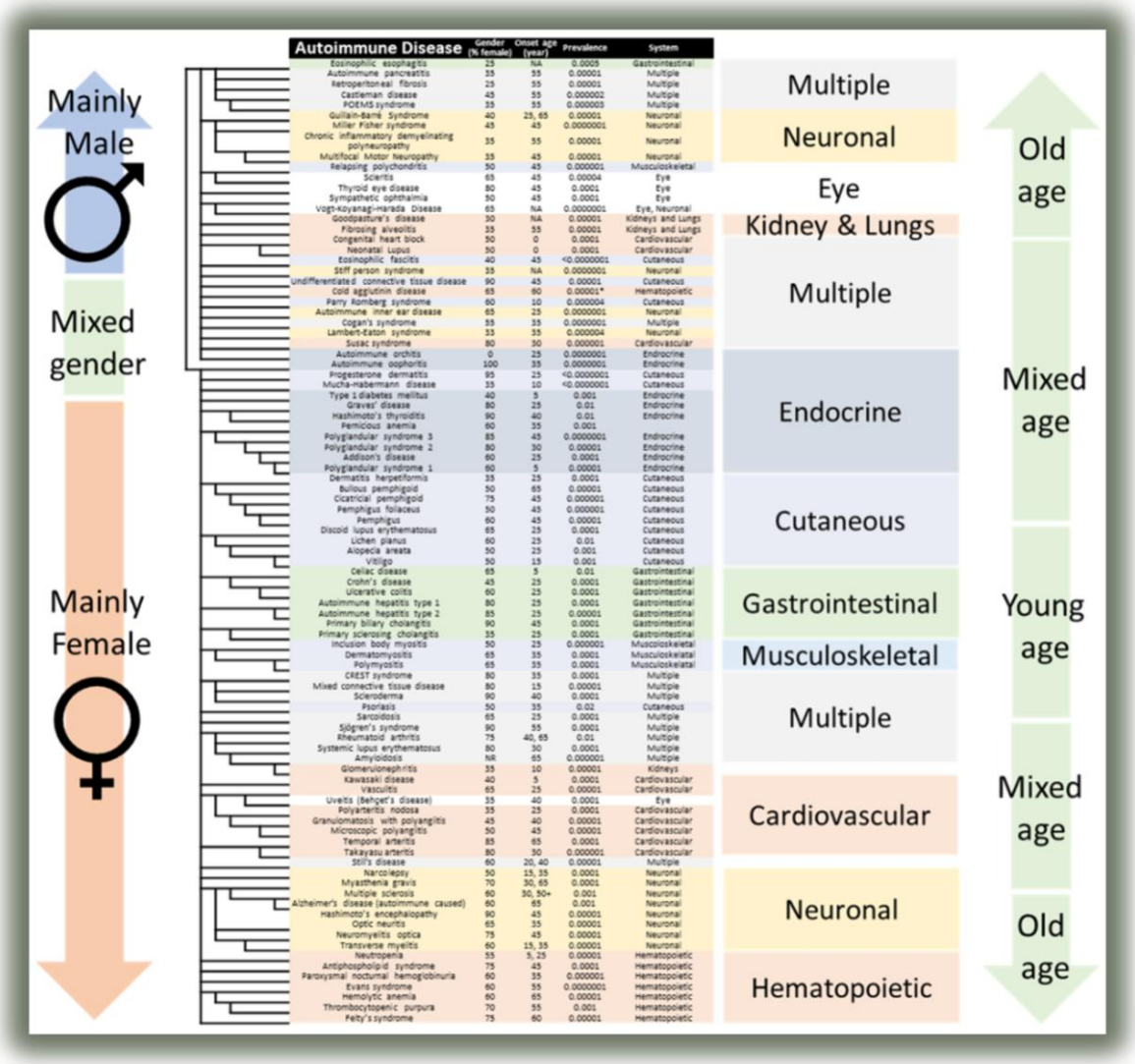
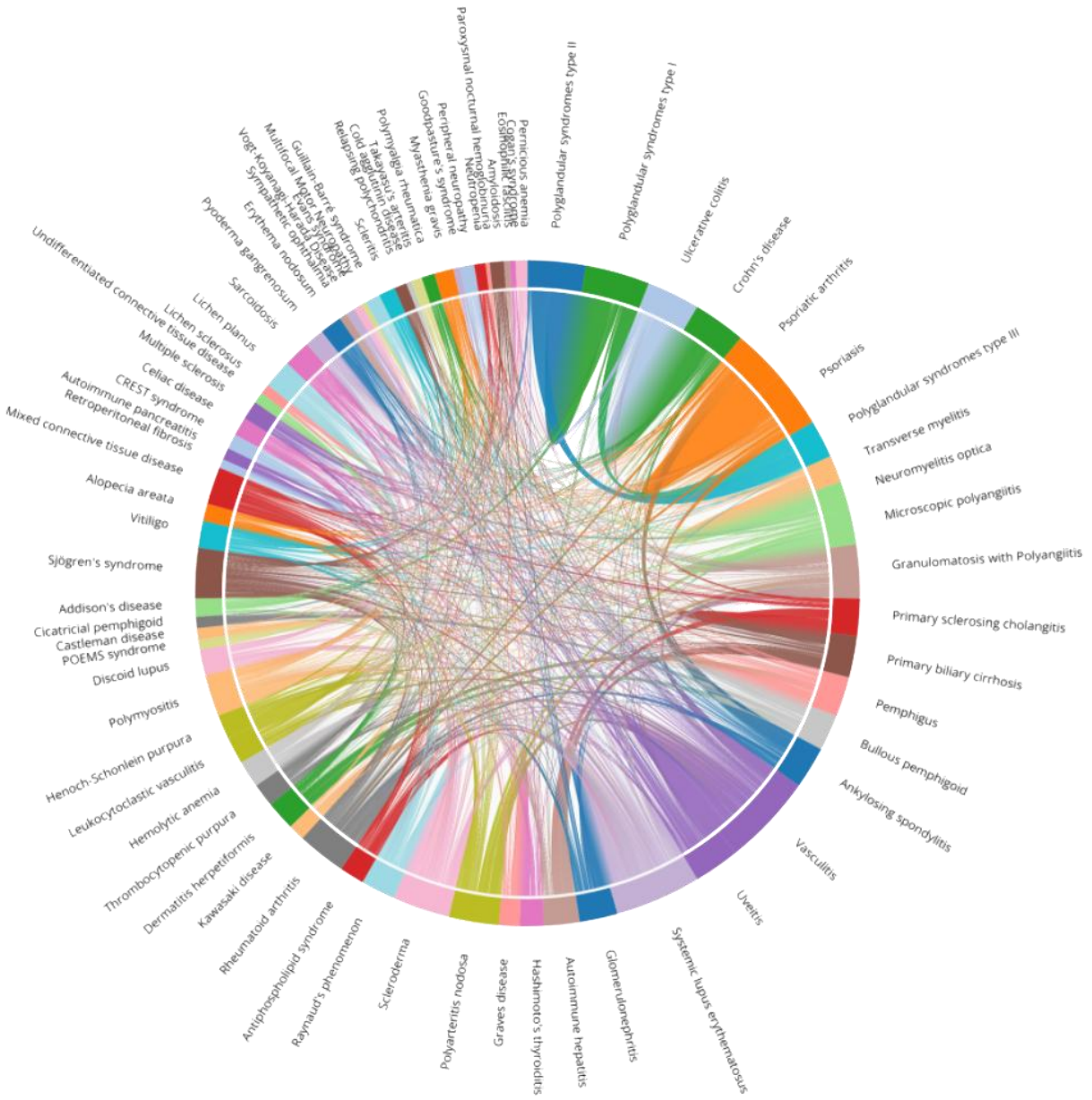
Managing Gut Autoimmunity

Autoimmune disease

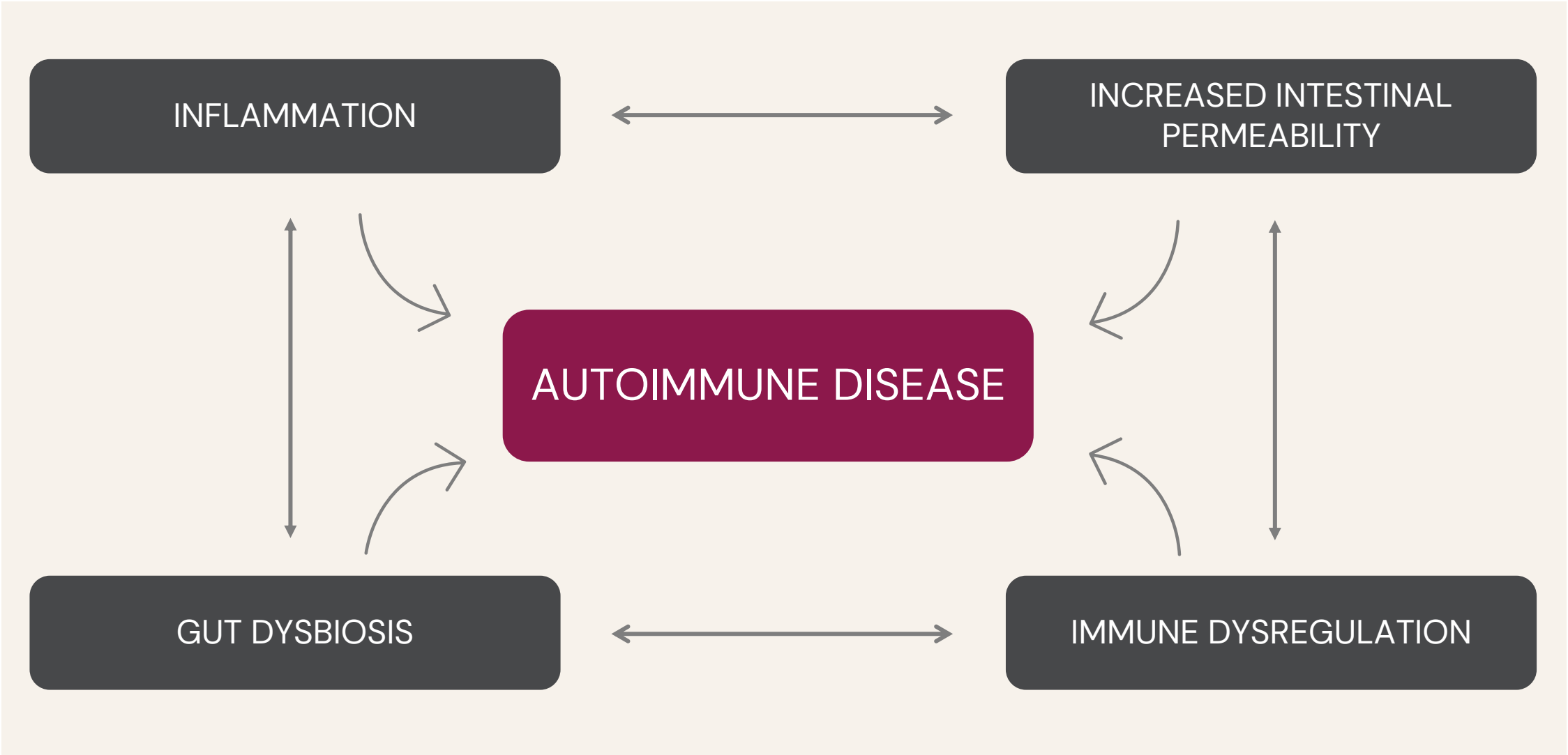
There are over 100 autoimmune diseases identified[1]





Autoimmune disease

"Once you develop one autoimmune disease, you're three times more likely to develop another autoimmune disease"[1]


INFLAMMATION
INCREASED INTESTINAL PERMEABILITY
AUTOIMMUNE DISEASE
GUT DYSBIOSIS
IMMUNE DYSREGULATION

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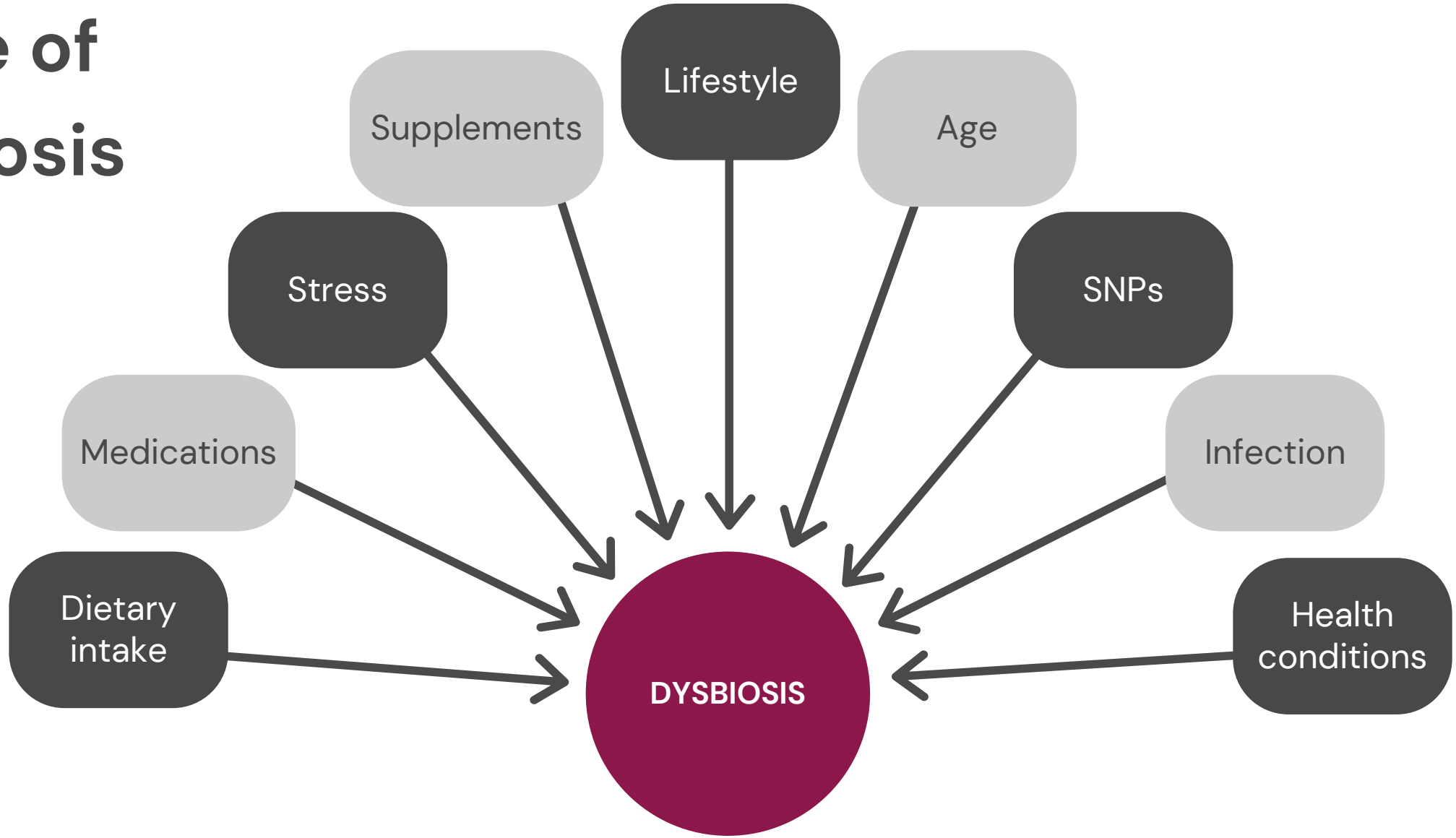
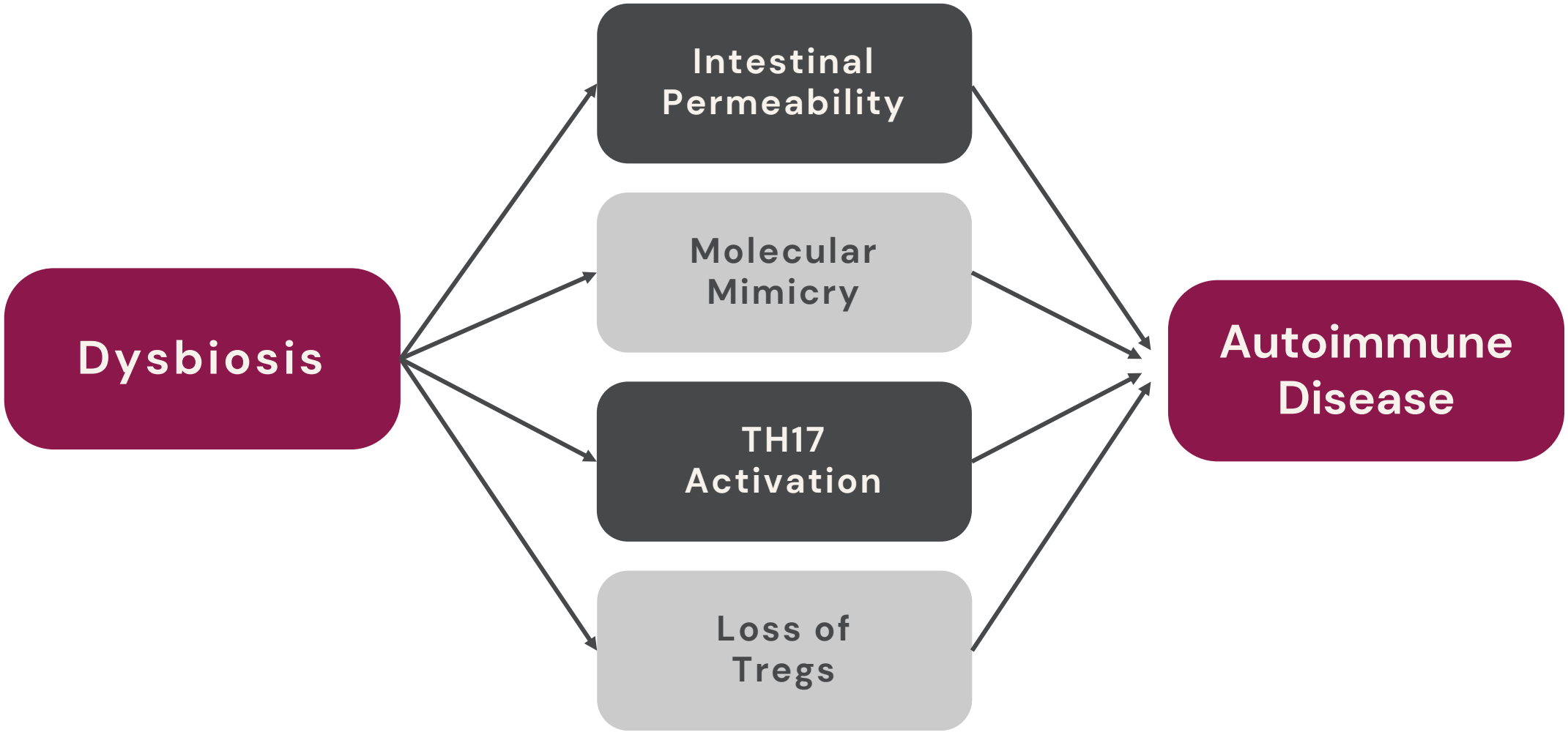
Managing Gut Autoimmunity

Intestinal dysbiosis

Cause of dysbiosis



Link between dysbiosis & autoimmunity



Molecular mimicry & autoimmunity

The mechanism

- Microbial proteins resemble self-antigens, triggering an immune attack on host tissues.

Clinical considerations

- Identify mimicry risk in autoimmune patients with chronic infections or dysbiosis.
- Reduce cross-reactive immune activation with microbiome modification.
- **AVOID AGGRESSIVE ANTI-MICROBIAL ERADICATION STRATEGIES**

Key microbial evidence

- *Prevotella copri* — elevated in new-onset RA; mimics joint proteins.[3]
- *Klebsiella pneumoniae* — linked to ankylosing spondylitis; resembles HLA-B27 & collagen.[4]
- *Clostridium perfringens* — mimics aquaporin-4 the autoantibody target in neuromyelitis optica (NMO), implicating gut bacteria in CNS autoimmunity.[5]
- Oral pathogens like *Porphyromonas gingivalis* can contribute to molecular mimicry & citrullinated autoantigens

TH17 activation & autoimmunity

The mechanism

- Th17 cells are essential for immune defence, but when dysregulated they drive chronic inflammation causing tissue damage in autoimmune disease (MS, psoriasis, Crohn's).[6]

Key evidence

- Adherent-Invasive *E. coli* (AIEC) — hyper-activates Th17 immunity in IBD via IL-23 & CD4 T cells.[7]
- *Eggerthella lenta* — produces cardiac glycoside reductase (Cgr2), which directly activates Th17 cells & interacts with host immunity.[8]
- Multiple Sclerosis microbiome activates Th17 cells & worsens symptoms in germ-free mice — implicating gut bacteria in neuroinflammation.[9]

Clinical considerations

- Assess the microbiome for Th17-promoting bacteria in Th17-driven autoimmunity.
- Diets rich in omega-3s, polyphenols, vitamin D & prebiotics may help modulate Th17 responses.
- Probiotics like *Lactobacillus reuteri* may suppress Th17 activation & reduce inflammation.[10]
- Modulating the gut microbiome is being explored as a potential strategy to help manage Th-17 driven autoimmune conditions.

Loss of Tregs & autoimmunity

The mechanism

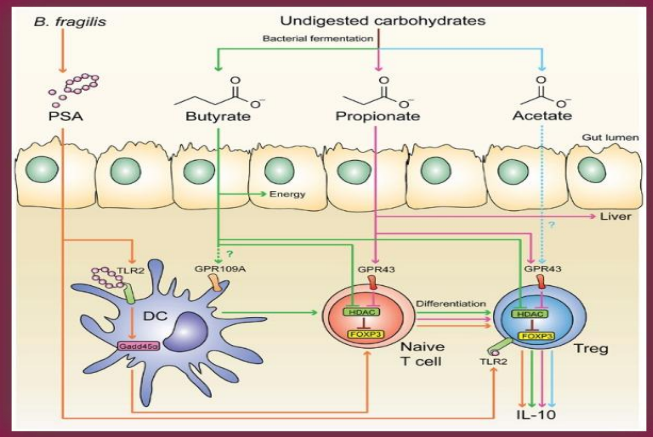
- Tregs maintain immune tolerance by suppressing excessive inflammation to prevent tissue damage.

Key evidence

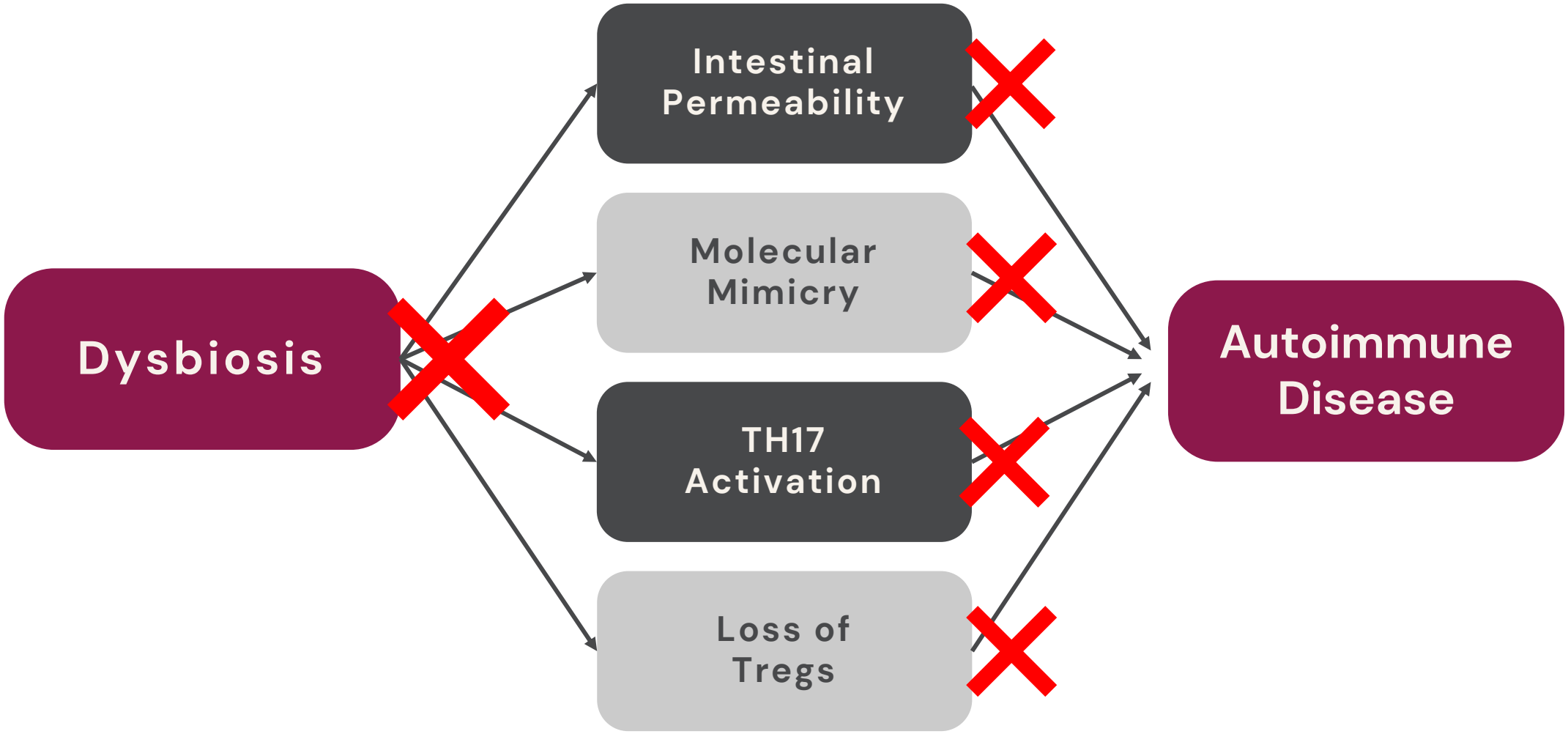
- SCFA-producing bacteria (*Clostridia*, *Bifidobacteria*) induce Treg development — butyrate enhances Treg polarisation but not Th17.[12]
- Autoimmune patients show lower levels of these bacteria, correlating with diminished Treg populations.[13]

Clinical considerations

- Identify dysbiosis-related Treg deficiency in autoimmunity by evaluating SCFA-producing bacteria via stool testing.
- Support Tregs with SCFA-inducing diet (fibre, resistant starch) & probiotics like *Bifidobacterium longum*.



Link between dysbiosis & autoimmunity



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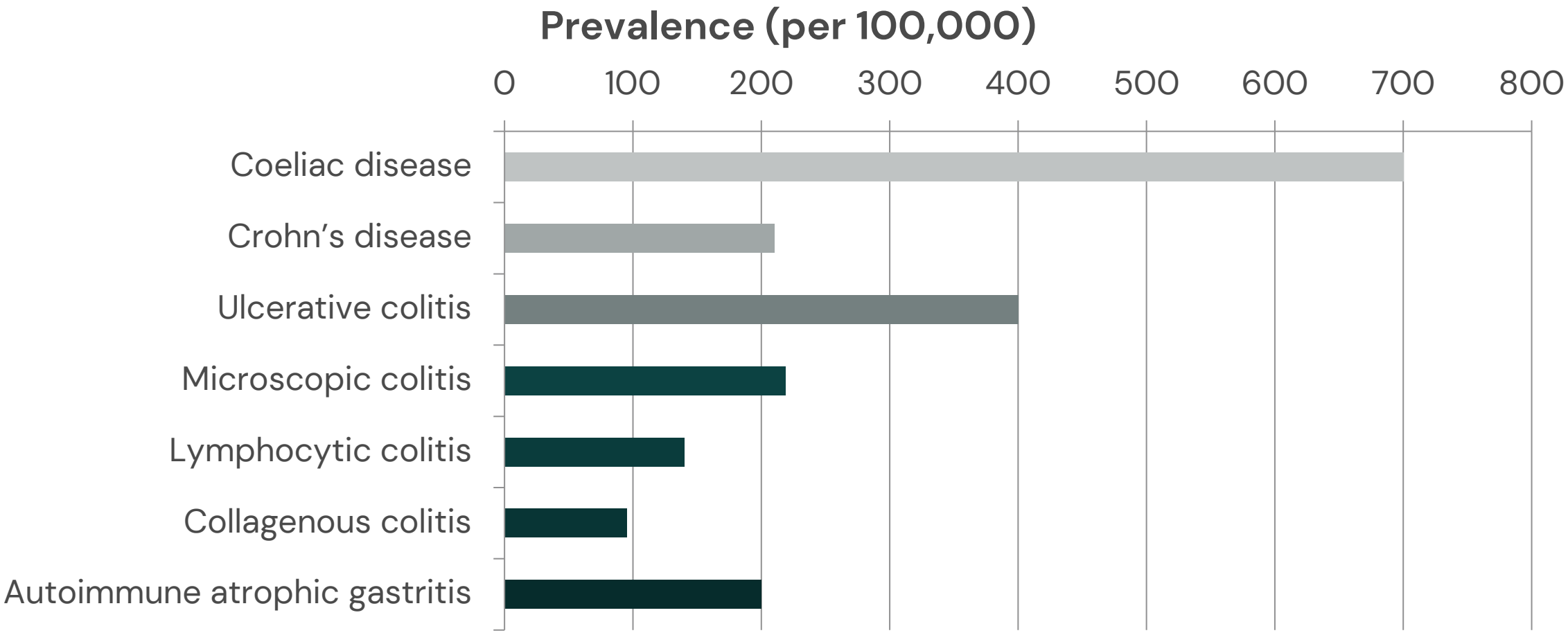
Managing Gut Autoimmunity

Gut autoimmune conditions



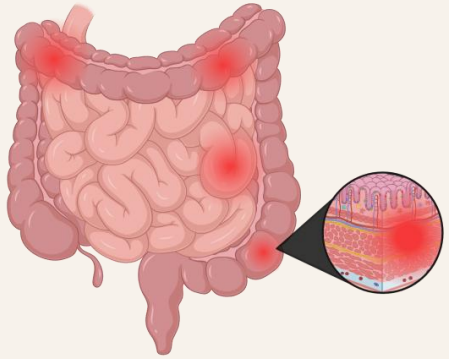
Inflammatory bowel disease (IBD)

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

What is IBD?

Abnormal immune response in the digestive tract, where the immune system mistakenly attacks healthy tissue leading to **chronic inflammation & sores**.

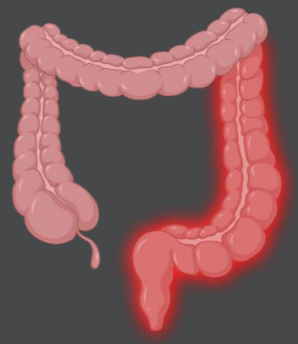
Crohn's Disease



Inflammation can occur in both the small & large intestine, often involving the deep tissue layers

VS

Ulcerative Colitis



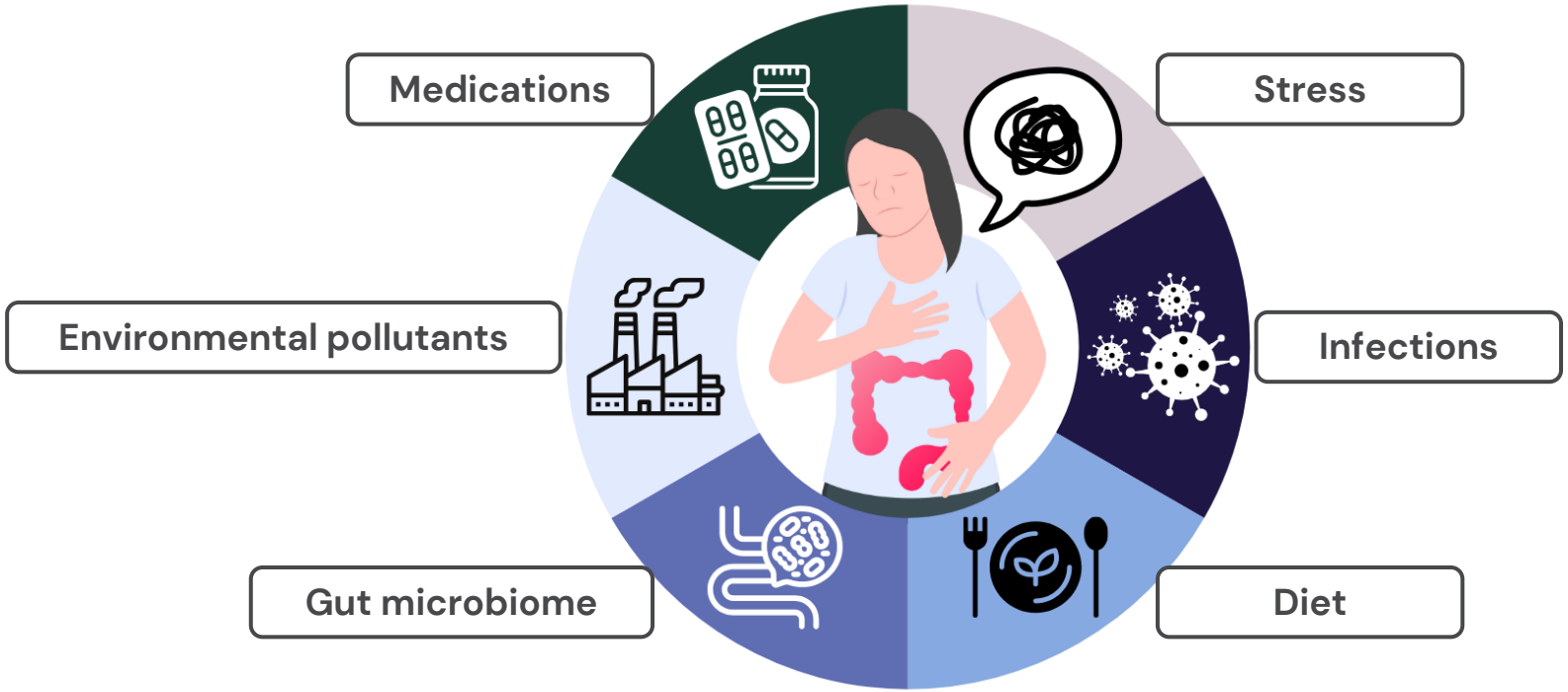
Inflammation can occur in the colon & rectum, usually involving only surface tissue

Symptoms

- Abdominal pain
- Diarrhoea
- Rectal bleeding
- Fatigue
- Weight loss

How does IBD develop: Overview

Complex mix of both **genetic** & **environmental** factors[14-16]



How does IBD develop: Dietary intake

Dietary factor	Effect	Mechanism of action	Ref.
Ultra-processed foods	↑	Barrier disruption, dysbiosis, immune activation	[25-27]
Emulsifiers (CMC, P80)	↑	Mucus thinning, bacterial encroachment	[28-29]
High n-6 PUFAs	↑	Pro-inflammatory eicosanoids	[30-31]
Saturated fat	↑	Bile acid shift → H_2S-producing bacteria	[32]
Simple sugars	↑	SCFA loss, barrier damage	[33-34]
High salt	↑	Th17 activation	[35]
Sulfur-rich/processed meats	↑	H_2S-mediated mucosal toxicity	[36]
Titanium dioxide	↑	Oxidative stress, inflammasome activation	[37]
High fiber	↓	SCFA production, Treg support	[38]
Mediterranean diet	↓	Anti-inflammatory, microbiome modulation	[39-40]
Omega-3 fatty acids	↓	Resolvins/protectins, NF-κB inhibition	[31,41]
Minimally processed foods	↓	Stable microbiota, fewer additives	[25,39]
Polyphenol-rich foods	↓	Antioxidant, prebiotic, immune regulation	[40,42]

Meta-analysis[88] has found a **Western dietary pattern** is linked to an **increased risk of** developing IBD.

How does IBD develop?: Infection

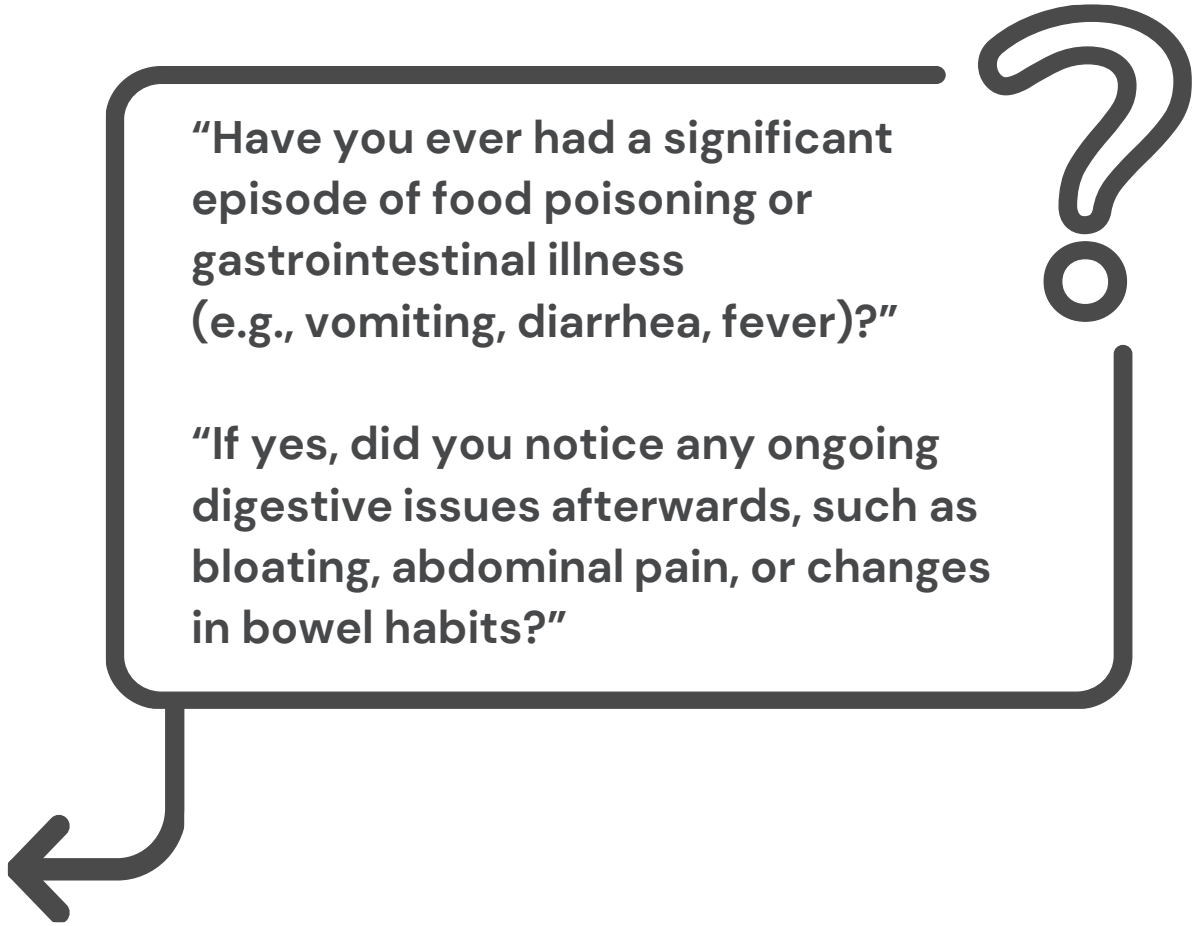
Food poisoning: Clinical question

Clinical Interpretation

- History of acute gastroenteritis is a well-documented risk factor for post-infectious IBS & can act as a triggering event in autoimmune pathophysiology via mechanisms like molecular mimicry, microbiome disruption, & increased intestinal permeability.

Clinical Management

- Measure microbiome
- Check if any remaining pathogens, treat if identified.
- Support gut barrier repair
- Address post-infectious dysbiosis



Infection as a risk factor for IBD

Pathogen	Linked to	Evidence type / strength	Refs
POST-INFECTION RISK			
***Salmonella* spp. or *Campylobacter* spp.**	IBD (CD & UC)	**1.2%** exposed vs **0.5%** unexposed (≤15 y); adjusted HR **2.9** overall, HR **1.9** excl. first year	[43]
Acute gastroenteritis (any pathogen)	IBD (signal for CD stronger in year 1)	**0.068%/yr** after GE vs **0.030%/yr** in controls (68.4 vs 29.7 per 100k PY); HR **2.4** overall; HR **4.1** in year 1	[44]
EMERGING ASSOCIATIONS			
Nontyphoidal *Salmonella*	IBD	Population-cohort **association**; absolute % not reported	[45]
Campylobacter concisus	Microscopic colitis	Markedly increased risk of **microscopic colitis**; IBD signal but no reliable post infection incidence %	[47-48]
Adherent-invasive *E. coli* (AIEC)	CD	Pathobiont **association**; not a discrete post-infection risk	[49]
***Mycobacterium avium* ssp. paratuberculosis (MAP)**	CD	Anti-MAP **treatment signal**; not post-infection incidence	[50]
Yersinia enterocolitica	Terminal ileitis	Can mimic/trigger Crohn-like disease (**controversial**); no quantified post-infection % for IBD	[46, 51]

Infection as a contributor to IBD flares

- **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.[89]
  - 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.[89]
  - Both CD and UC had a **higher prevalence of *Campylobacter spp***. compared to non-IBD controls. [89]

- **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.[90,91]

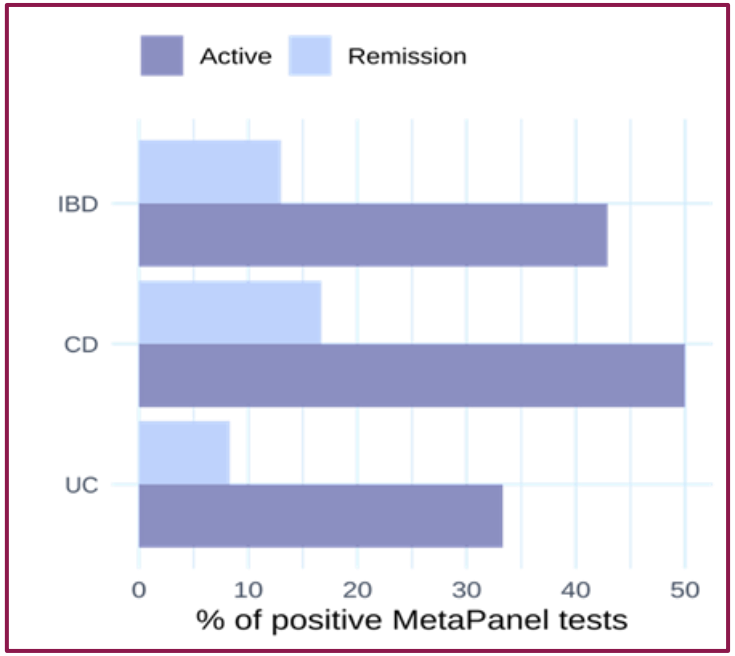


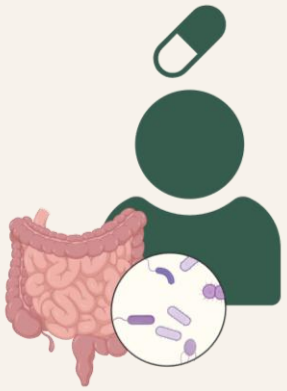
Figure 1: Microba’s in-house data on the prevalence of pathogens in IBD patients with active disease vs remission.

How does IBD develop: Microbiome

While a causal role for the gut microbiome in IBD has not been definitively proven, mounting evidence points to a significant contribution.

Antibiotic therapy

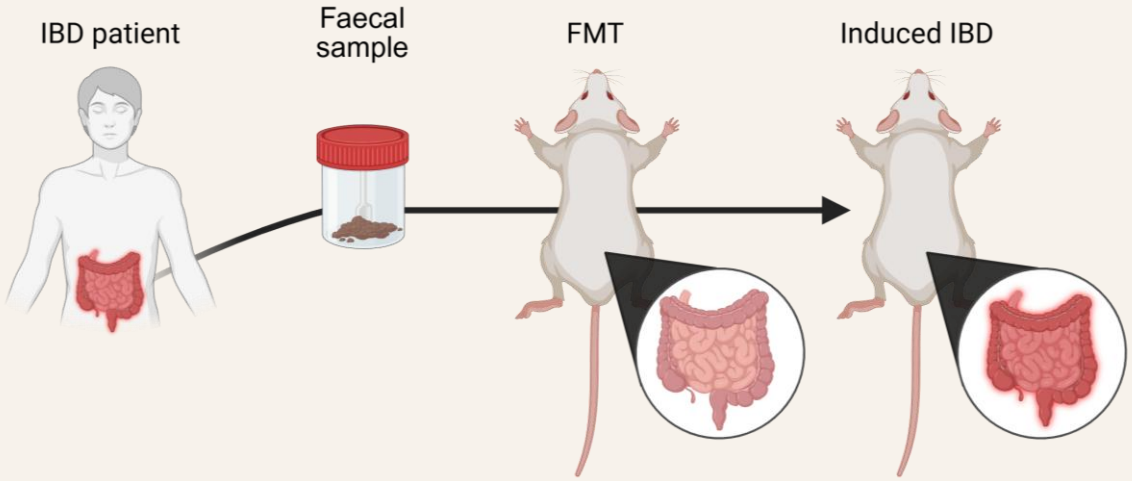
Meta-analyses[52-53] indicate that antibiotic therapy in IBD is associated with **remission induction** and **lower relapse rates**, highlighting a potential role of microbiome modulation in disease regulation.



Faecal microbiota transplantation (FMT)

FMT from donors with IBD can **induce or exacerbate** IBD in animal models[54].

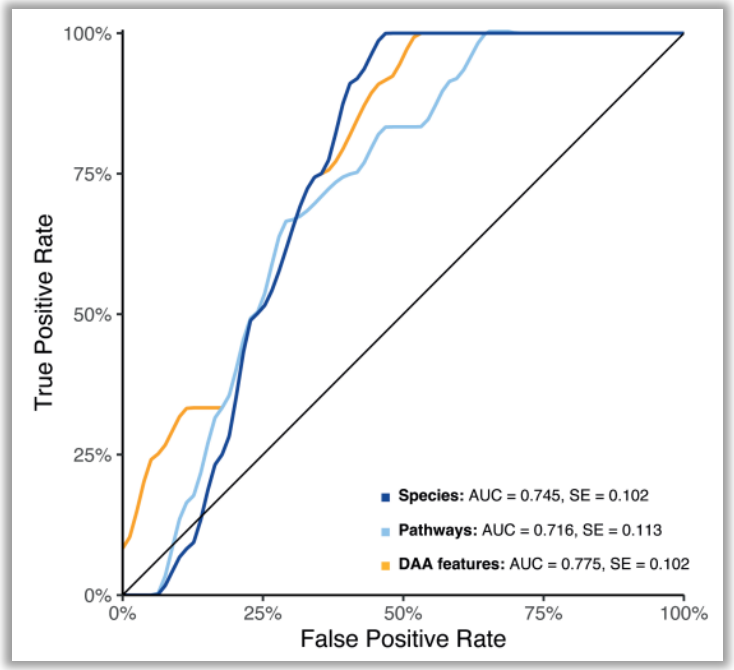
FMT from healthy donors may **induce remission** in patients with IBD[55].



The microbiome may predict IBD activity

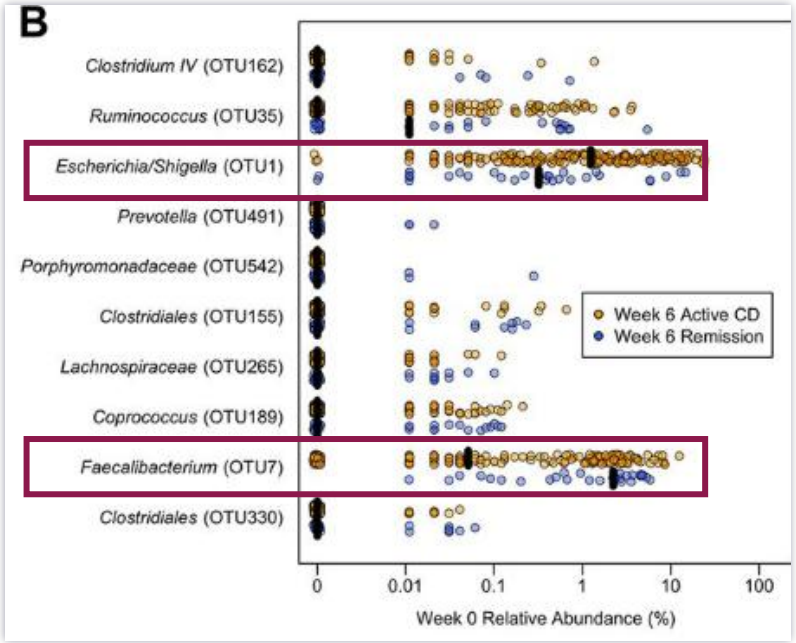
Microbial prediction

Baseline gut microbiome features were able to predict **future therapy intensification** with an AUC > 0.7[56].



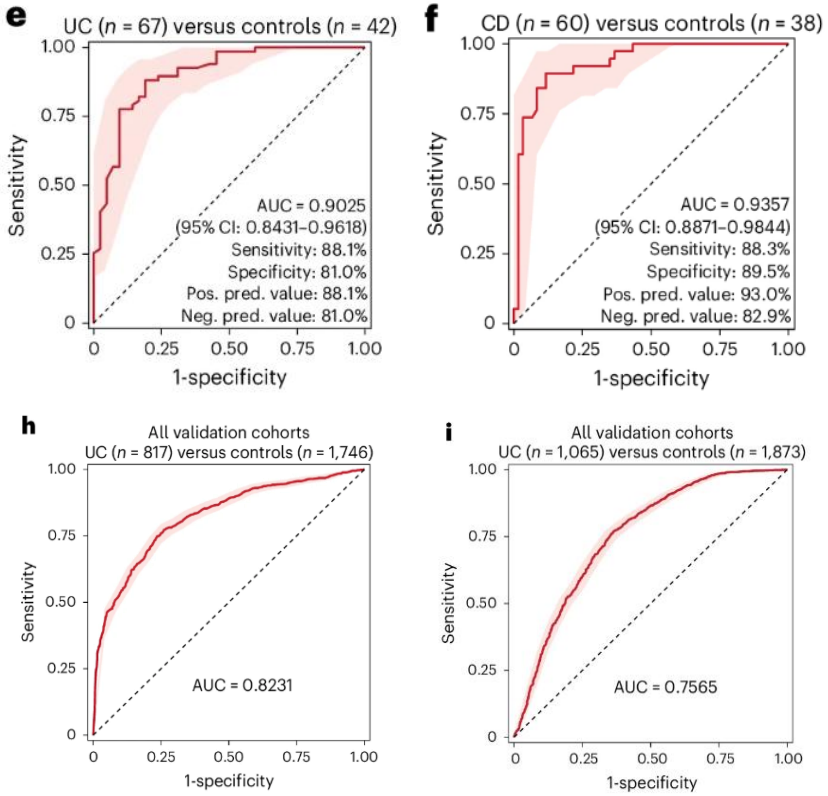
Microbial species

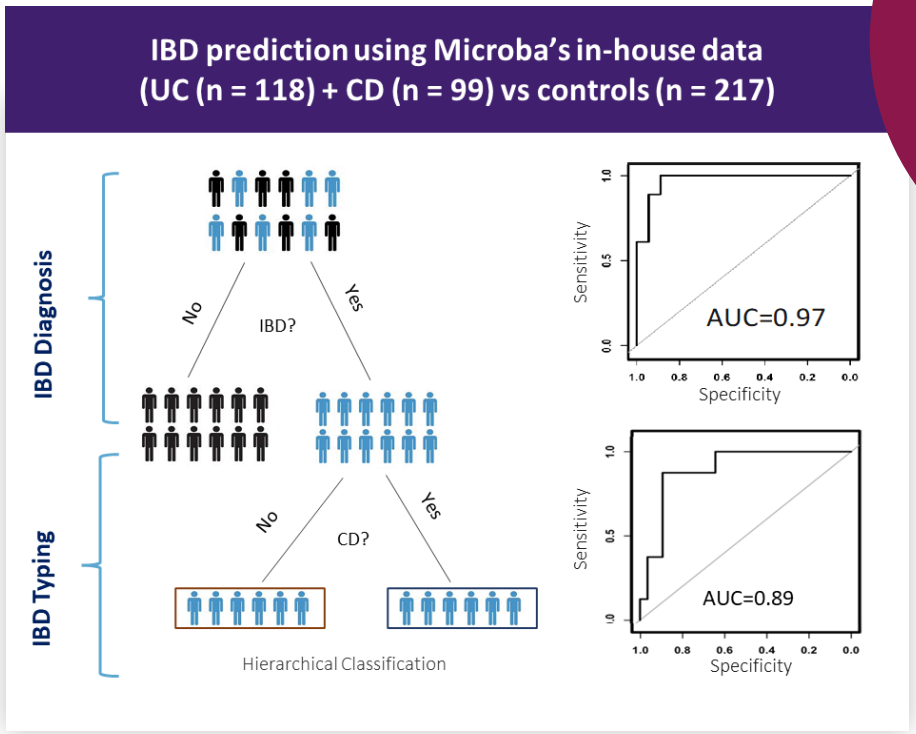
Baseline levels of ***Faecalibacterium spp*** were higher and ***Escherichia spp*** lower in CD patients in remission after six weeks[57].



IBD can be predicted using species profile

- Almost 6000 faecal samples from 13 cohorts globally, Zheng et al[59] correctly predicted UC with an AUC of 0.82, & CD with an AUC of 0.76.
- Using smaller cohorts from the same region, capability increased to an AUC of 0.90[59]

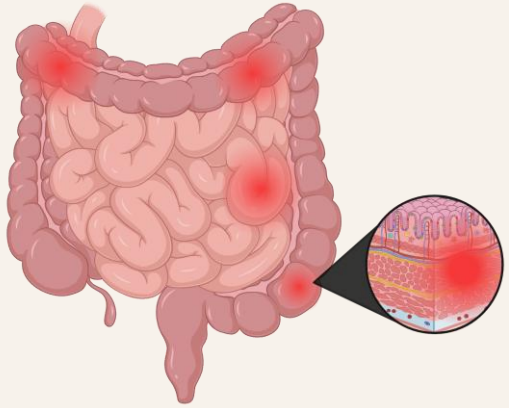




IBD has a strong, unique microbiome signature

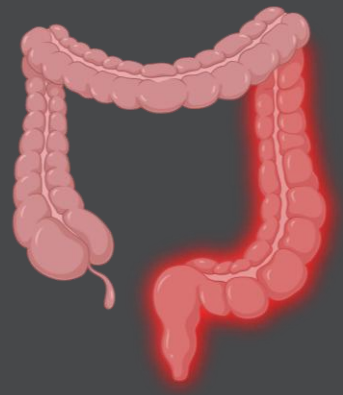
Associations between microbiome markers & IBD from existing literature

Crohn's Disease



- Decreased **Microbial Diversity**[60-63]
- Decreased **Butyrate** producers[60-63]
- Increased **Hexa-LPS** producers[61-63]
- Increased **Oral species**[60]

Ulcerative Colitis



- Decreased **Microbial Diversity**[1-3]
- Increased **Oral species**[1]

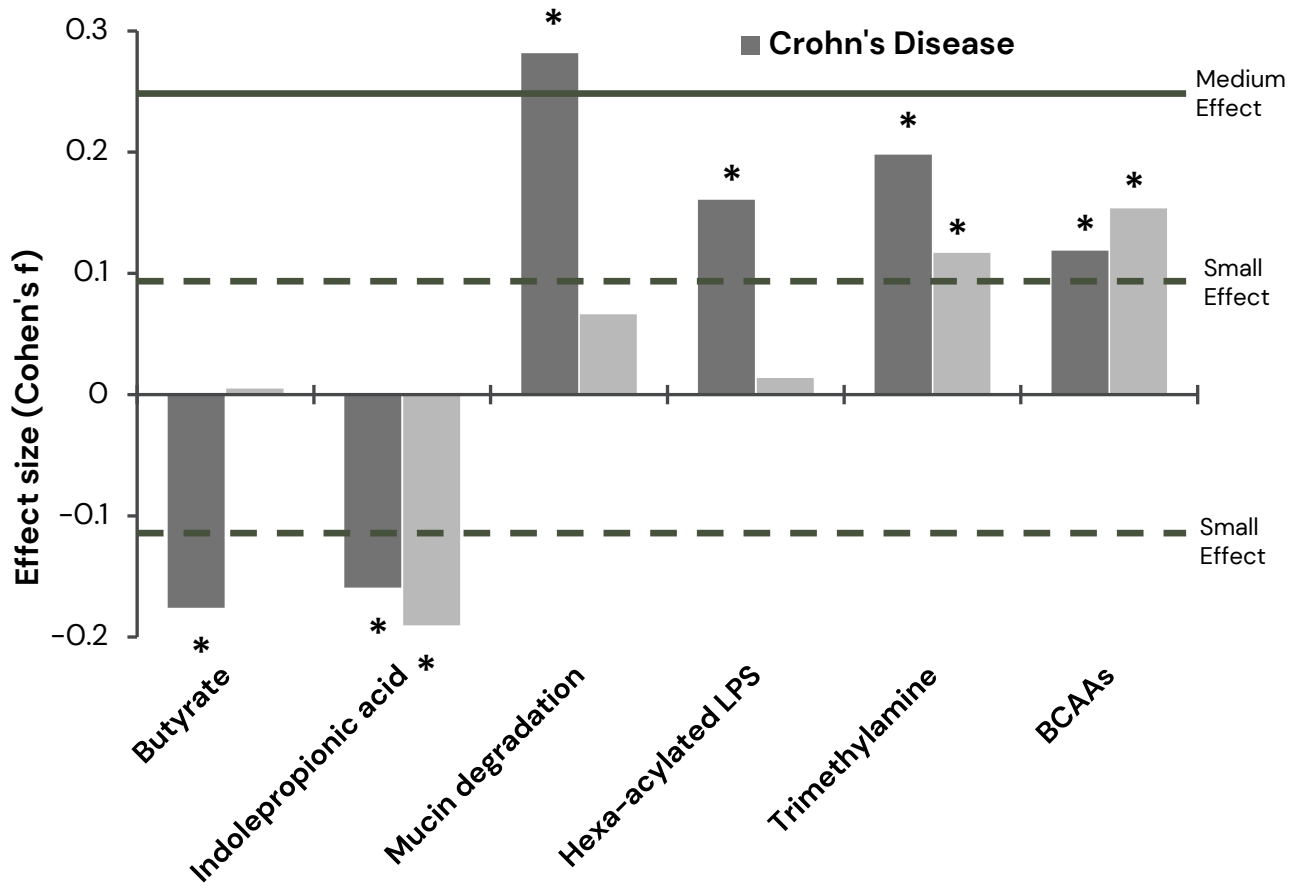
Associations of microbial markers with IBD from Microba's in-house dataset

Analysis

- Compared metagenomic microbiome profiles from IBD (CD = 45, UC = 88) vs healthy (n = 484)
- All analyses corrected for confounders: age, gender, BMI & DASS depression scores

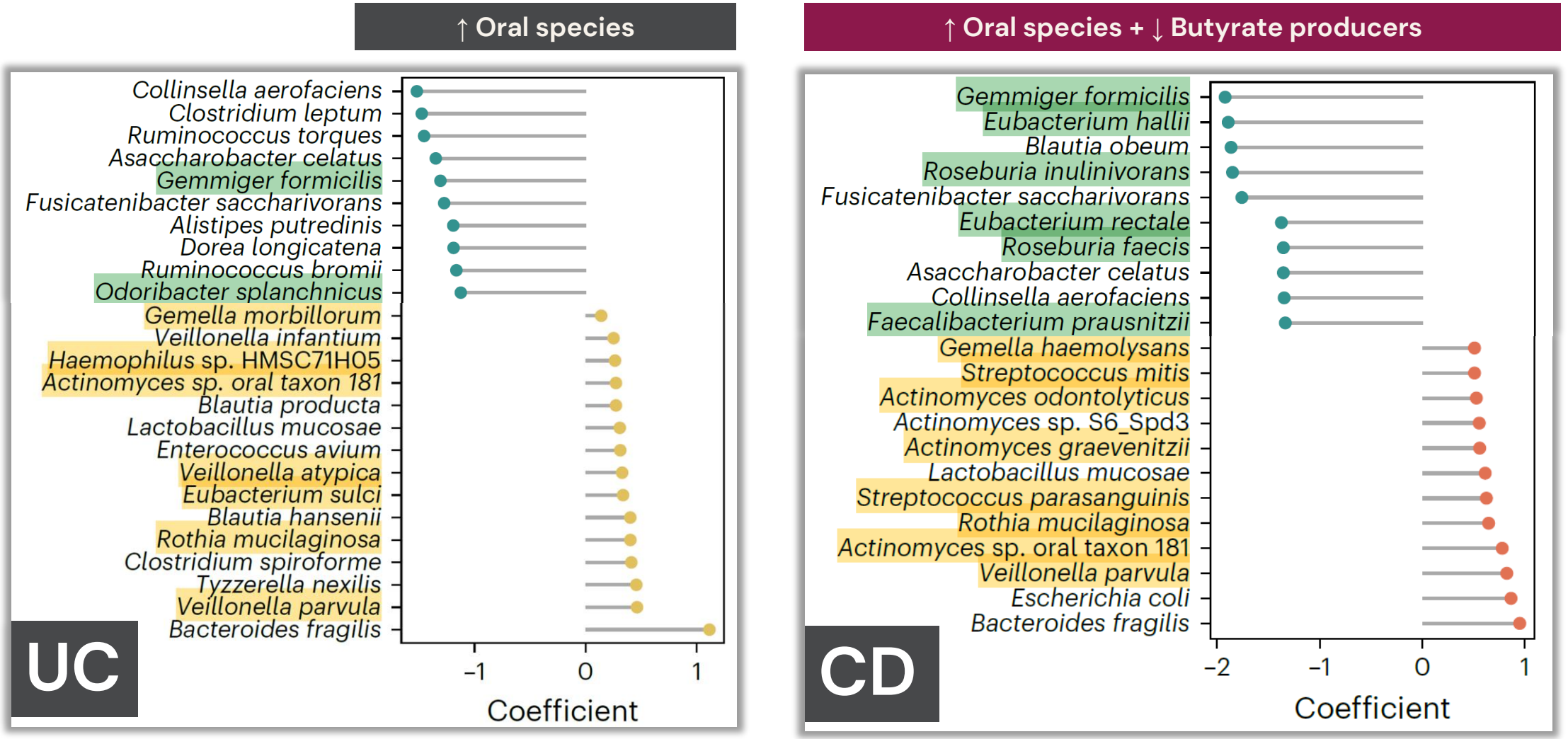
Results

- In CD, microbes involved in **butyrate, IPA, mucin degradation, hexa-LPS, TMA & BCAAs** production are altered
- **Stronger associations** with microbial markers in **CD compared to UC**
- Only microbes involved in production of **IPA, BCAAs & TMA** significantly altered in UC



* FDR < 0.05

Association of oral species with IBD[60]



Oral gut microbial overlap & signatures in IBD

Studies have found **greater similarity between oral and gut bacterial** in patients with ulcerative colitis (UC) and Crohn's disease (CD) compared to healthy controls (HC)[64-65].

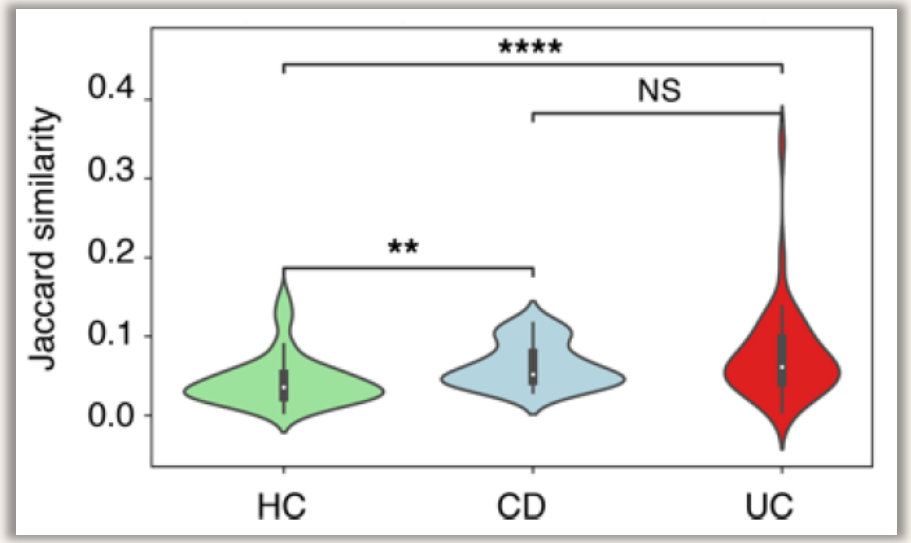
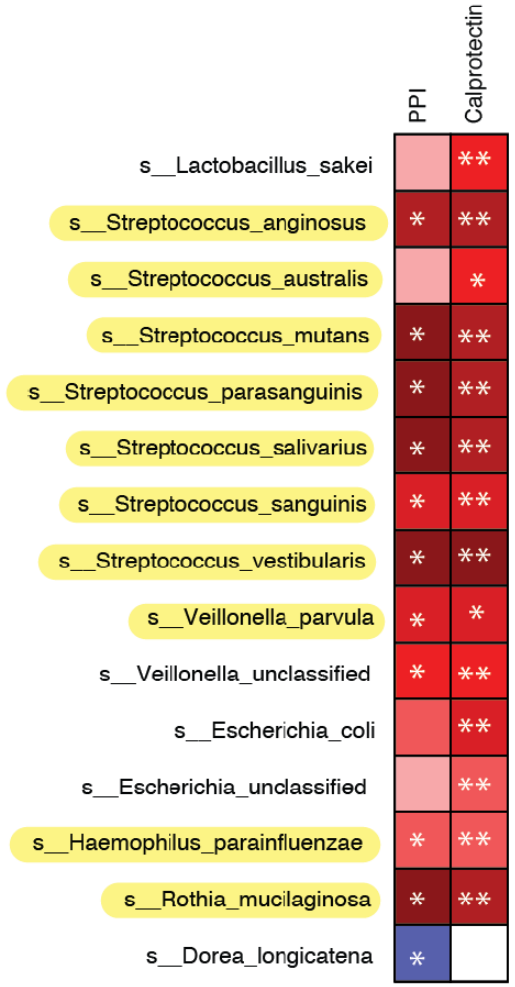
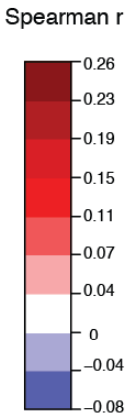


Image from: Imai et al 2021



Oral species were the dominant taxa associated with higher **faecal calprotectin** in a large cross-sectional study[66] (n > 1000)



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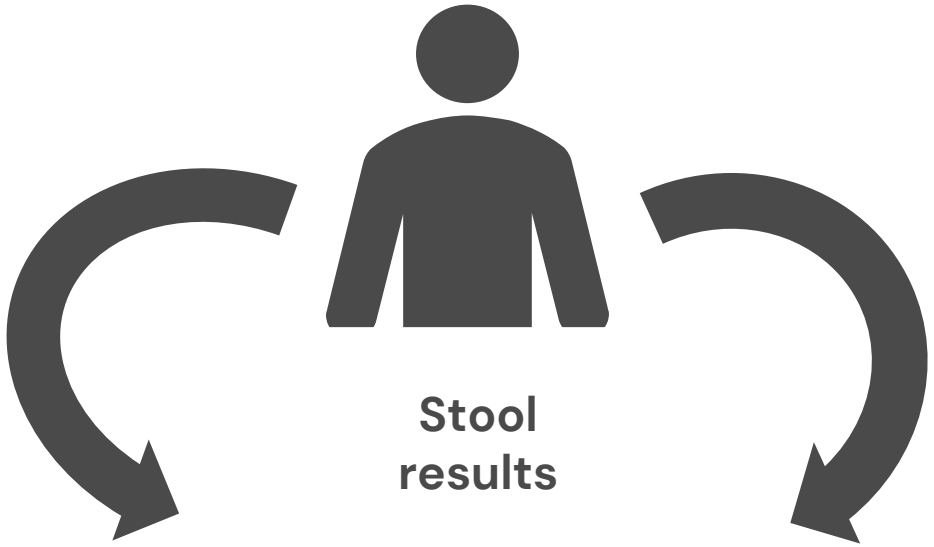
Managing Gut Autoimmunity

Pathology assessment for IBD

Organic diseases vs DGBI

Calprotectin
< 100 µg/g
and/or
Lactoferrin
< 7.2 µg/g



Stool results

Calprotectin
> 100 µg/g
and/or
Lactoferrin
> 7.2 µg/g

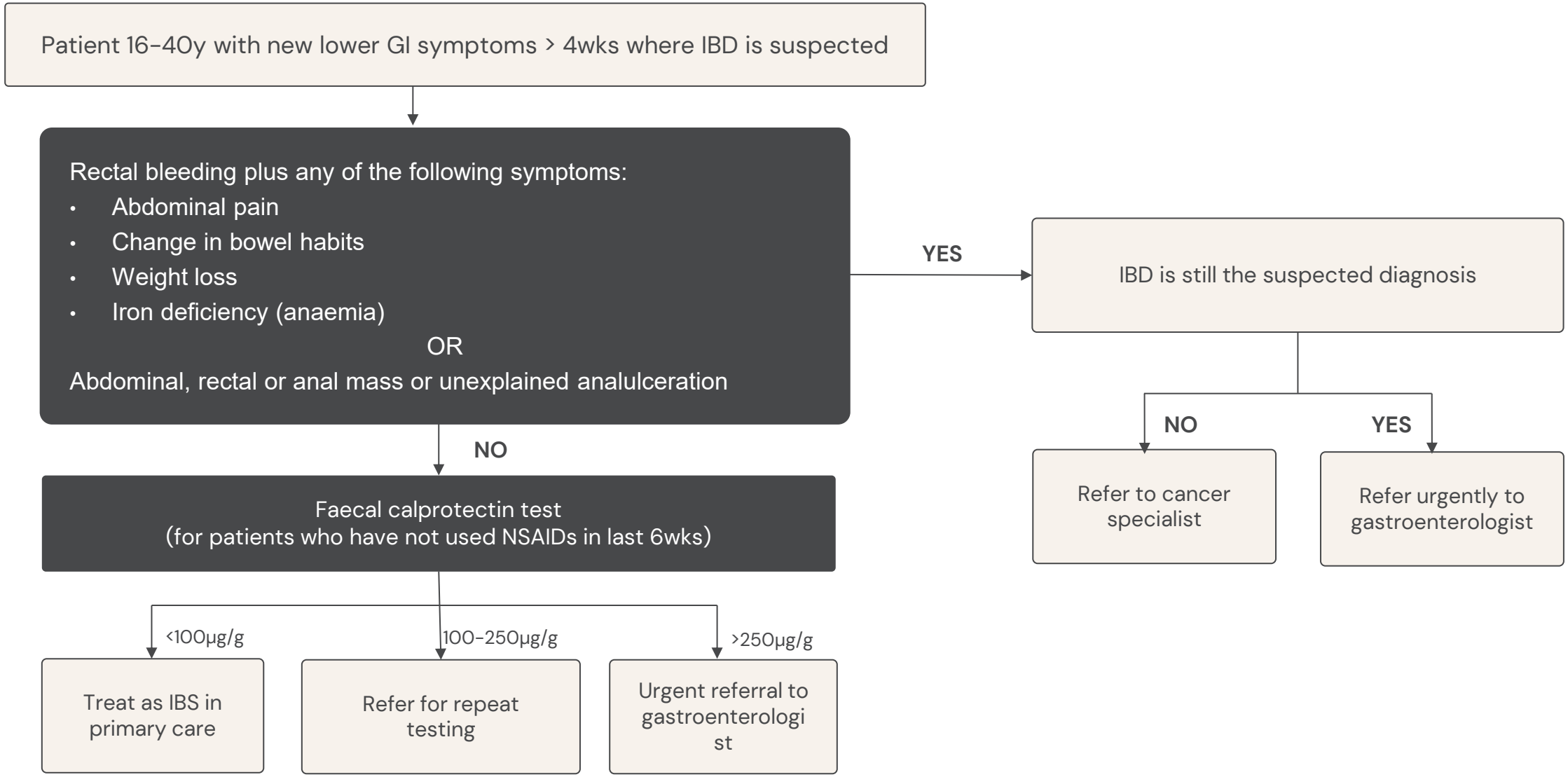
Disorders of gut-brain interaction (DGBI)

Irritable Bowel Syndrome (IBS), Functional Dyspepsia, Functional Abdominal Pain Syndrome (FAPS), Functional Constipation, Functional Diarrhea, Functional Bloating, Functional Nausea & Vomiting

Organic disease

Inflammatory Bowel Disease (Crohn's Disease, Ulcerative Colitis), Infectious Colitis (Bacterial/Viral), Diverticulitis, Gastroenteritis, Coeliac Disease, Colorectal Cancer, Microscopic Colitis, Radiation Colitis, Behçet's Disease, Ischemic Colitis

IBD suspected: Assessment[67]



Autoantibody testing

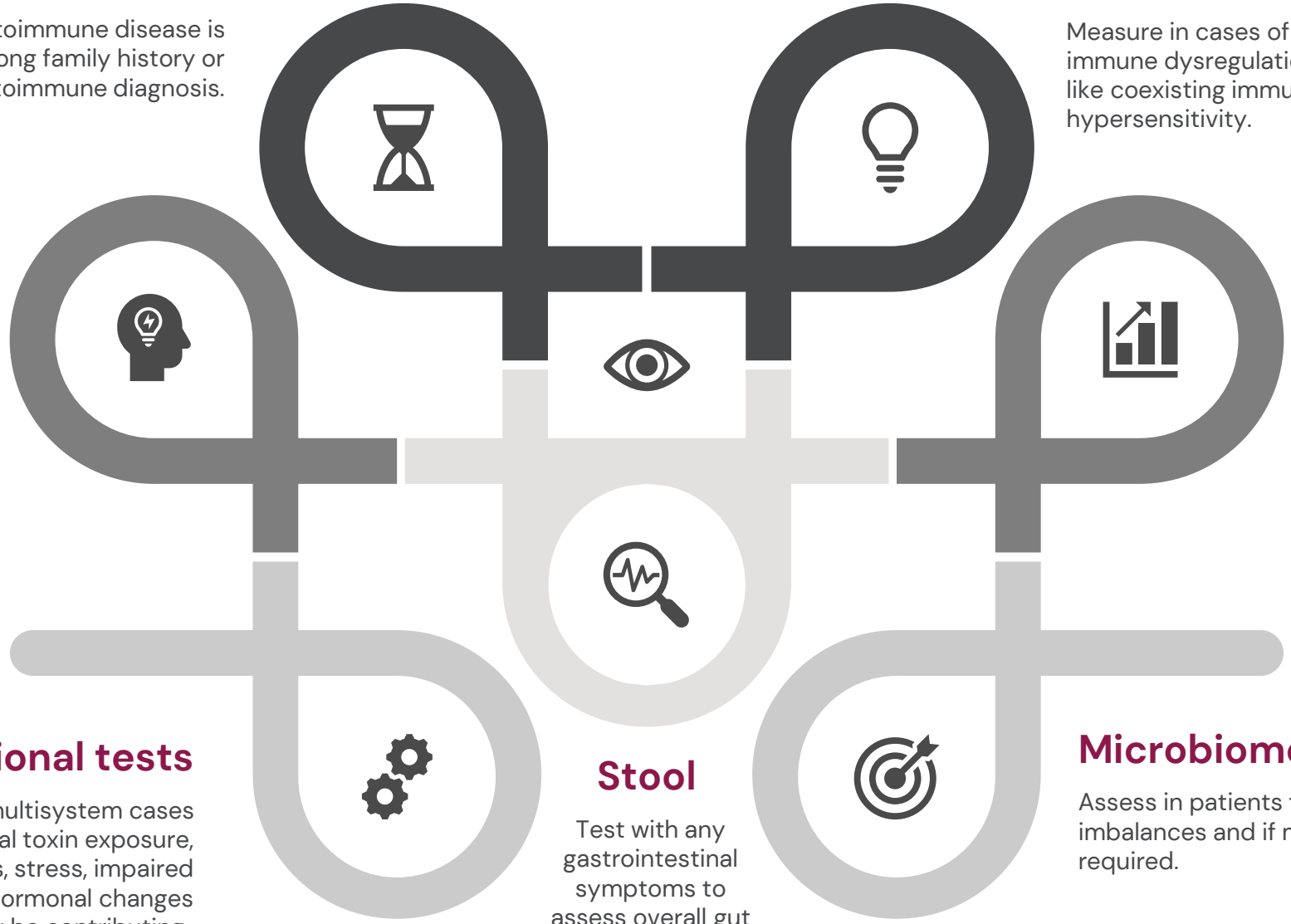
Consider testing when autoimmune disease is suspected, in anyone with a strong family history or existing autoimmune diagnosis.

Immune cell activity

Measure in cases of chronic infections, unresolved immune dysregulation, or paradoxical presentations like coexisting immune suppression and hypersensitivity.

Inflammation

Measure in autoimmune presentations to assess disease activity and guide intervention.



Nutrient status

Test patients to identify hidden deficiencies (iron, vitamin D, B12, folate, zinc, calcium, magnesium). Especially important with chronic inflammation, fatigue, digestive issues, or restrictive diets.

Functional tests

Use in complex, multisystem cases where environmental toxin exposure, genetics, stress, impaired detoxification or hormonal changes may be contributing..

Stool

Test with any gastrointestinal symptoms to assess overall gut function/health

Microbiome composition

Assess in patients to identify driving imbalances and if microbiome modulation is required.

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Managing Gut Autoimmunity

Stool testing in IBD

When to measure IBD clients

Baseline Assessment

- Establishes the patient's microbial ecosystem prior to initiating therapy.
- Identifies key dysbiotic signatures, loss of keystone taxa, & functional deficits guiding individualised intervention.
- Provides a reference point for future comparative analysis.

Monitoring Therapy (after 4-6 months)

- Evaluate microbial shifts in response to therapeutic interventions (e.g., dietary, probiotic, pharmacological, biologic).
- Helps distinguish therapy-induced improvements from spontaneous disease fluctuation.
- Facilitates early detection of microbial regression before symptomatic relapse.

Regular Monitoring (Every 12 Months)

- Annual assessment to track long-term microbial stability & resilience.
- Supports proactive management & treatment optimization, even in remission phases.
- Demonstrates objective progress for both clinician & patient engagement.

During Frequent Loose Stools

- Insight into microbial instability & functional changes associated with ongoing inflammation or malabsorption.
- Assists in differentiating inflammatory activity from microbial-driven dysfunction.

When NOT to measure IBD patients

During an acute flare or if visible blood is present in stool

- Blood introduces a high proportion of **human DNA**, which can overwhelm microbial DNA reads & lead to **sequencing inaccuracies** or reduced taxonomic resolution.
- High host DNA interferes with the detection of low-abundance microbial species & functional genes.

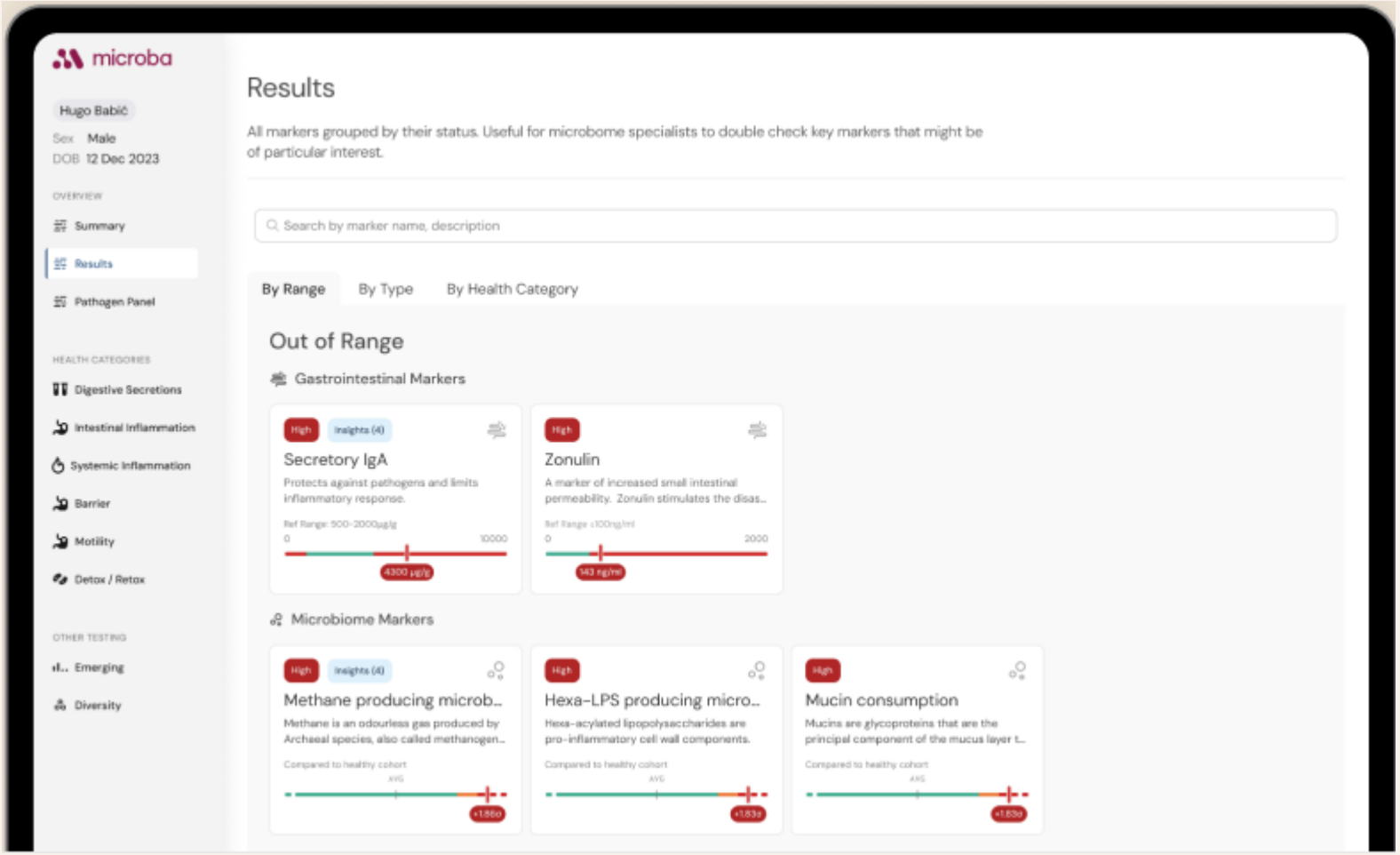
Immediately following antibiotic use & colonoscopy prep

- These conditions temporarily distort the microbiome & may not represent true baseline composition.

Summary of methods used to measure the **gut microbiome**

Method	Sensitivity	Bacteria coverage	All microbes coverage	Species-level ID	Functional pathways
Culture	✓			Limited	
qPCR	✓			Limited	
16S rRNA sequencing	✓	✓		Imprecise	
Long-read 16S/ITS/23S	✓	✓		✓	
Metagenomic sequencing	✓	✓	✓	✓	✓

Report Demo


microba
Hugo Babić
Sex Male
DOB 12 Dec 2023
OVERVIEW
Summary
Results
Pathogen Panel
HEALTH CATEGORIES
Digestive Secretions
Intestinal Inflammation
Systemic Inflammation
Barrier
Motility
Detox / Retox
OTHER TESTING
Emerging
Diversity
Results
All markers grouped by their status. Useful for microbome specialists to double check key markers that might be of particular interest.
Search by marker name, description
By Range
By Type
By Health Category
Out of Range
Gastrointestinal Markers
High
Insights (4)
Secretory IgA
Protects against pathogens and limits inflammatory response.
Zonulin
A marker of increased small intestinal permeability. Zonulin stimulates the disas...
Microbiome Markers
Methane producing microb...
Methane is an odourless gas produced by Archaeal species, also called methanogen...
Hexa-LPS producing micro...
Hexa-acylated lipopolysaccharides are pro-inflammatory cell wall components.
Mucin consumption
Mucins are glycoproteins that are the principal component of the mucus layer t...

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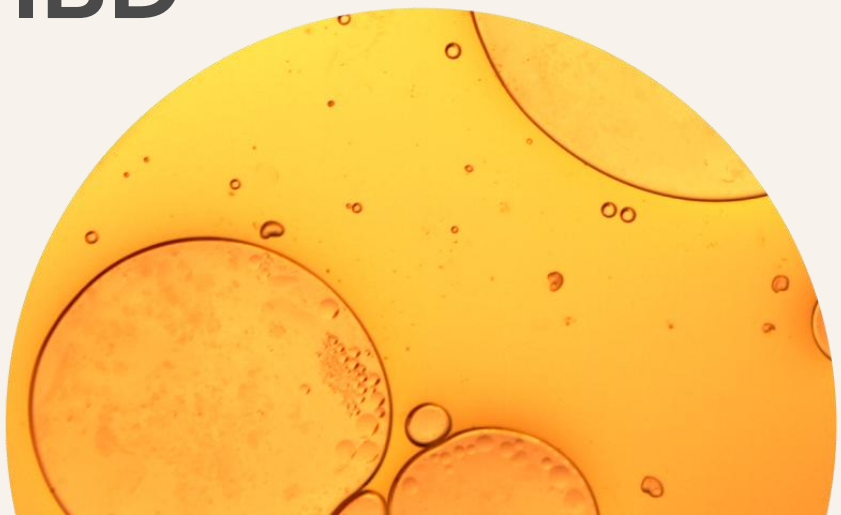
Managing Gut Autoimmunity

Interventions in IBD



NT Scope of practice in IBD care

PRE-DIAGNOSIS

Recognise and refer

- Refer red flag symptoms or markers promptly
- Hold off interventions until diagnosis is confirmed
- Avoid masking symptoms or delaying diagnosis

ACUTE FLARE

Medically led

- Defer to the gastroenterology team and work in collaboration
- May require medically prescribed nutrition (e.g. EEN or PEN in Crohn's).
- Do not initiate or alter medically prescribed diets

REMISSION

Supportive role

- Gradual reintroduction of dietary diversity
- Correcting nutritional deficiencies left by restriction or malabsorption
- Addressing food fears
- Managing IBS-type symptom overlap

MAINTENANCE

Long-term support

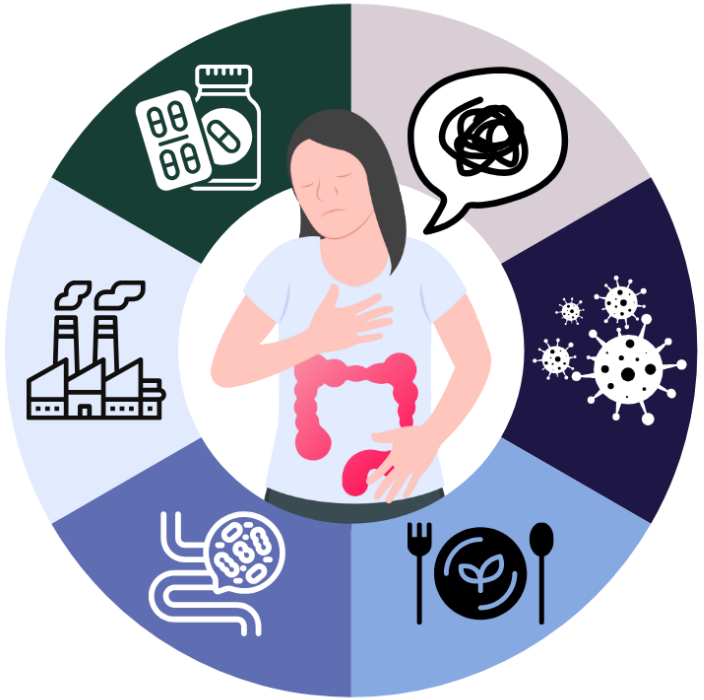
- Increasing microbial diversity and resilience
- Supporting overall gut health and function
- Whole-diet quality
- Monitoring symptoms and the microbiome for relapse risk

Remove aggravating factors

Medication review – Explore alternatives where medically appropriate

Reduce Environmental pollutant exposure

Reduce sedentary lifestyle as appropriate to energy levels/disease status



Stress Management, Talking & Bodywork Therapies

Screen for Gut Infections – Discuss management strategies with medical team

Remove UPFs & Alcohol

Assess and address dysbiosis

Crohn's Disease Exclusion Diet[92, 93]

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

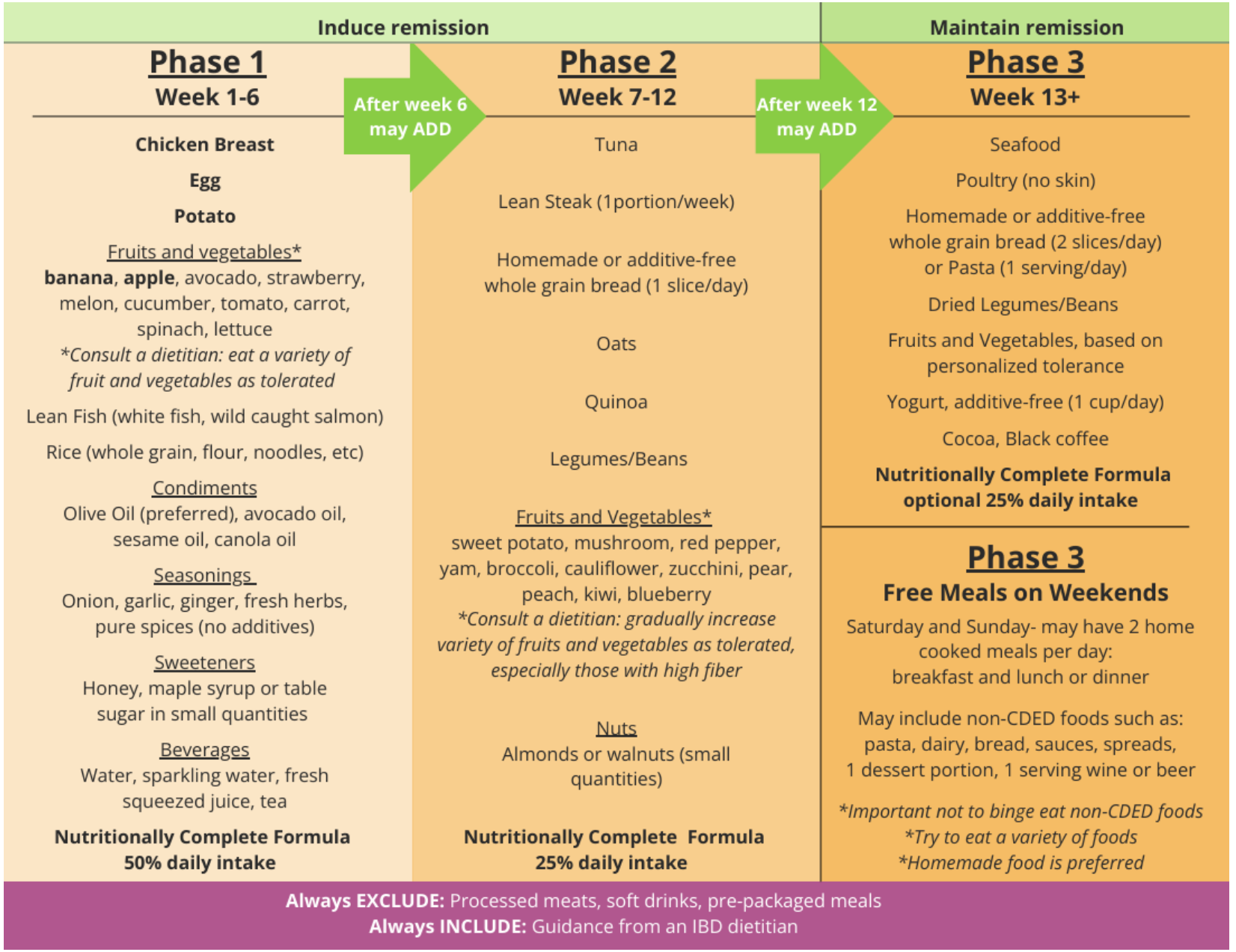
CDED + PEN (Partial Enteral Nutrition) has better compliance than EEN (Exclusive Enteral Nutrition) due to inclusion of **whole foods, with similar success rates**

Microbial changes have been observed in patients utilising CDED + PEN

- Increased *Faecalibacterium prausnitzii*
- Increased *Bifidobactrium spp.*
- Reduced *E. coli* & *Fusobacterium*

Crohn's Disease Exclusion Diet[24, 26]

- 3 phase diet
- Often combined with PEN to improve efficiency to induce remission
- PEN becomes optional in later phases
- Phase 3 = Maintenance (Mediterranean diet eventual goal)



CDED Guide for Crohn's Disease | GI Nutrition Foundation

Low residue dietary recommendations

Category	What to Include	What to Avoid	Goals / Purpose
Overall eating pattern	Soft, low-fibre, easy-to-digest foods; peeled/cooked vegetables; ripe/soft fruits; small frequent meals	Raw veg, fibrous peels, skins, seeds, tough stalks; large meals; fasting if fatiguing	Reduce irritation, improve stool consistency, maintain nutrition
Low-residue / Low-fibre foods	Soups, purées, mash, peeled potatoes; pumpkin, carrot, sweet potato; banana, stewed apple, pear	Whole grains; raw veg; cabbage family; legumes; popcorn; nuts/seeds	Minimise mechanical irritation & bloating
Protein choices	Poached/steamed chicken or turkey; soft-boiled eggs; tofu; mild white fish (trial)	Fried meats, red meat, charred foods, spicy marinades	Support healing without irritation
Fat management	Small amounts of olive/avocado oil; lightly cooked lean proteins	Fried foods; fatty meats; cream, butter-heavy dishes; oily meals	Prevent worsening diarrhoea & cramping
Dairy & alternatives	Lactose-free dairy; simple plant milks without gums; small amounts of fermented dairy if tolerated	High-fat dairy; dairy/plant milks with gums; large amounts of milk/cream	Reduce symptoms; allow tolerated options
Hydration	2–3 L/day; herbal teas (peppermint, chamomile, ginger); diluted electrolytes	Coffee, alcohol, carbonated drinks, strong black tea, energy drinks	Replace losses from diarrhoea; maintain electrolytes
Soothing / Repair-focused foods	Bone broth; rice congee; rice noodles in broth; pumpkin/carrot/sweet potato purées; peeled potato mash; plain crackers	Spicy foods; acidic/vinegar dressings; highly processed foods	Calm the gut and restore digestive balance

Mediterranean diet in IBD management

No diet has consistently been found to decrease the rate of flares in adults with IBD.

A recent consensus[77] 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.
- "A diet low in red & processed meat may reduce ulcerative colitis flares but has not been found to reduce relapse in Crohn's disease."

Conversely, a meta-analysis[88] has found a **Western dietary pattern** is linked to an **increased risk of** developing IBD.

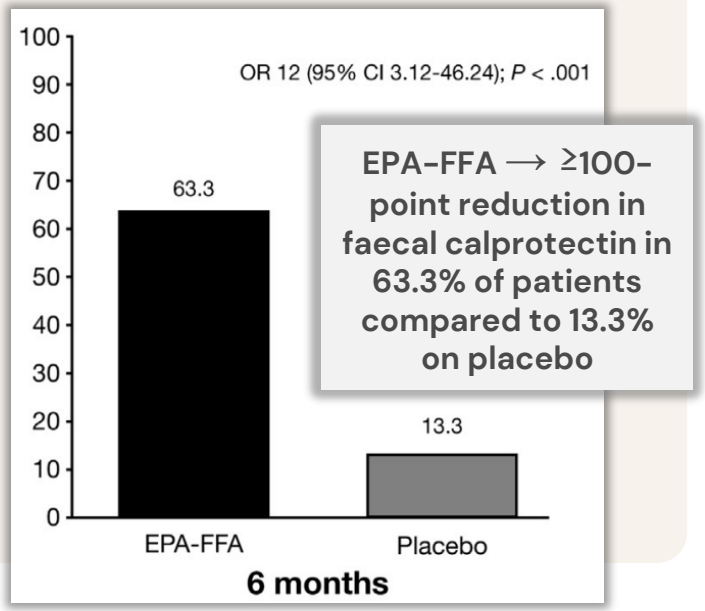
Interventions to reduce faecal calprotectin

Omega-3 fatty acids

NHMRC GRADE B

Ground flaxseeds (30g/day)[78]

EPA-FFA capsules (2g/day)[79-80]

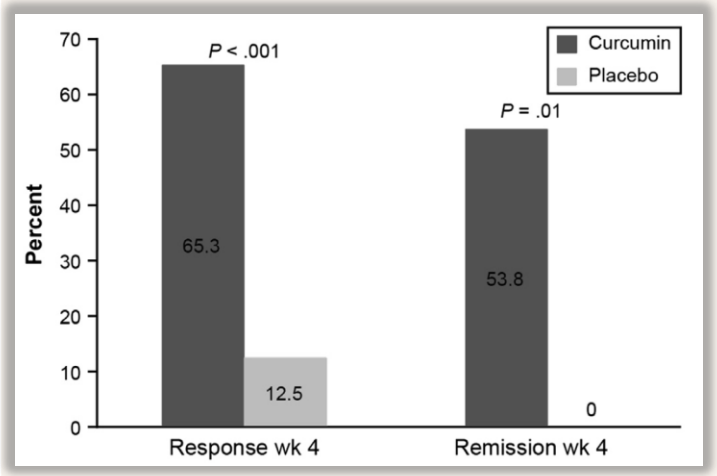


Curcumin

NHMRC GRADE B

Bio-enhanced (50mg twice daily)[81]

Pure capsules (1-1.5g twice daily)[82-83]



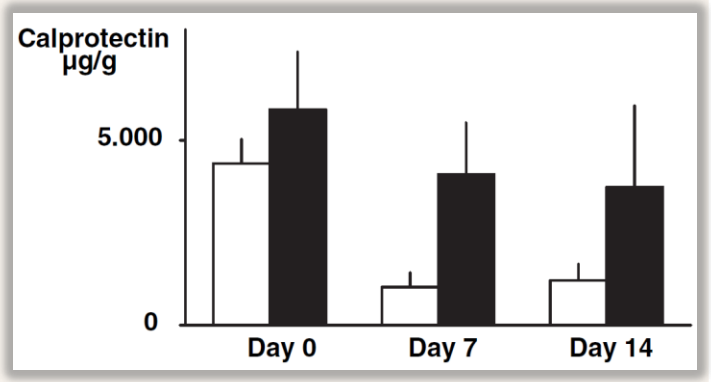
Image[82] from: Lang et al 2015

Inulin

NHMRC GRADE B

Oligofructose enriched (12g/day)[84]

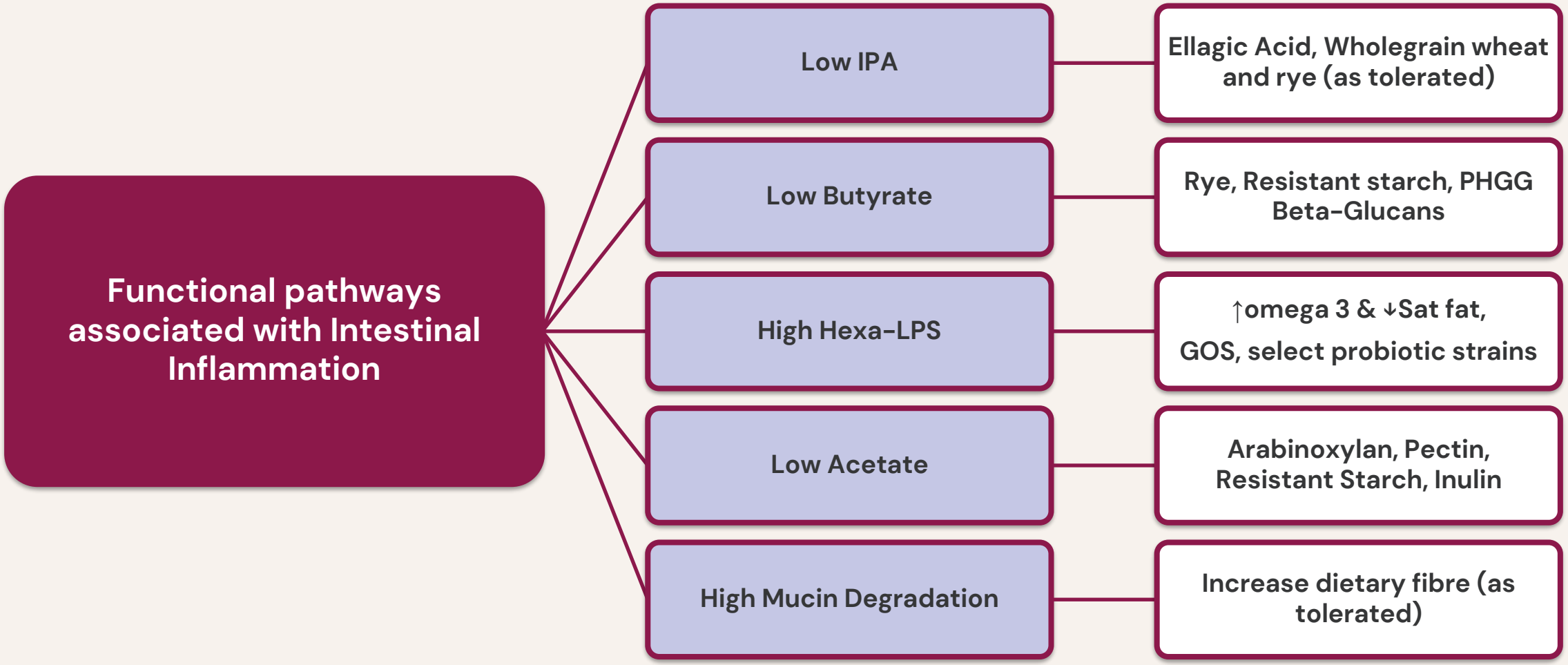
Native (15-16g/day)[85-86]



Image[84] from: Casellas et al 2007

NHMRC GRADES B Body of evidence can be trusted to guide practice in most situations

Target Microbial Drivers of Intestinal Inflammation



Other pillars of IBD management

1. Soothe the gut lining

Demulcent & mucosal-healing agents calm an irritated, inflamed lining.

e.g. slippery elm, marshmallow root, colostrum, glycyrrhizinated liquorice (DGL)

2. Gut barrier repair

Rebuild tight junctions & epithelial integrity to reduce permeability.

e.g. glutamine, zinc carnosine, vitamins A & D, Saccharomyces boulardii

3. Restore microbial diversity

Rebuild a resilient, diverse microbiome.

e.g. fermented foods, time in nature, High ALA (alpha-linolenic acid) oil, exercise

4. Modulate inflammation

Dampen pro-inflammatory signalling.

e.g. curcumin, omega 3, quercetin, ginger, green tea, Boswellia

5. Regulate the immune system

Rebalance over-active immunity toward tolerance.

e.g. vitamin D, specific probiotic strains, quercetin

6. Support the nervous system

Address gut–brain axis, stress & vagal tone to ease symptoms.

e.g. magnesium, ashwagandha, L-theanine, rhodiola, B-vits

7. Correct nutrient deficiencies

Assess and support iron, B12, folate, vit D, zinc, magnesium and others lost due to restrictive eating, malabsorption and inflammation

Key learnings

IBD is a complex mix of genetics and environment: Environmental triggers (food poisoning, ultra-processed foods, medications, stress) disrupt the microbiome and may trigger autoimmune inflammation in susceptible individuals.

Four microbiome-mediated mechanisms may contribute to autoimmunity: Intestinal permeability, Molecular mimicry, Th17 activation, Loss of Tregs

Stool & microbiome testing help differentiate disease: Calprotectin >100 µg/g increases suspicion for inflammatory bowel disease or other organic pathology. The microbiome in IBD has a strong and reproducible signature.

Dietary and microbiome-targeted interventions may complement conventional IBD management.

Best outcomes come from a multi-layered approach: inflammation, microbiome, nutrition, gut barrier function, and environmental factors.

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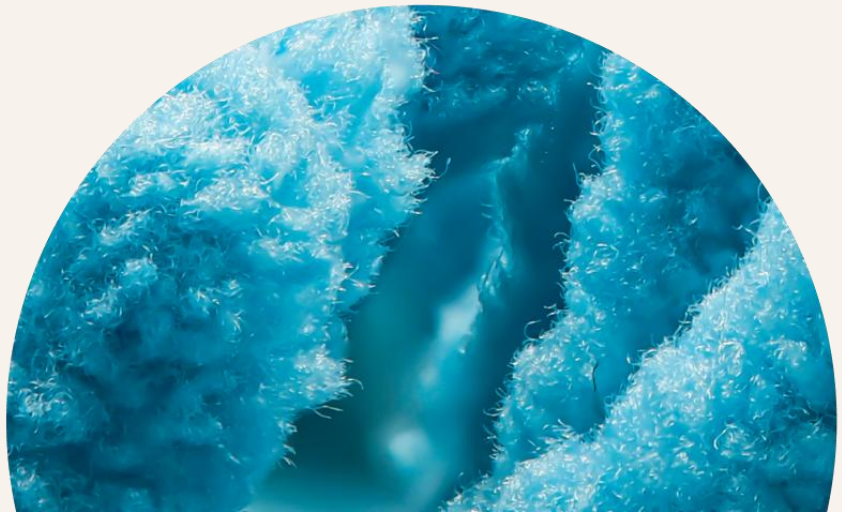
Managing Gut Autoimmunity with

Microbiome Explorer



Designed to scale to clinical need

The tiered approach allows you to choose the right depth of analysis for each patient.

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Metagenomics + GI Health Markers + Pathogen Screening

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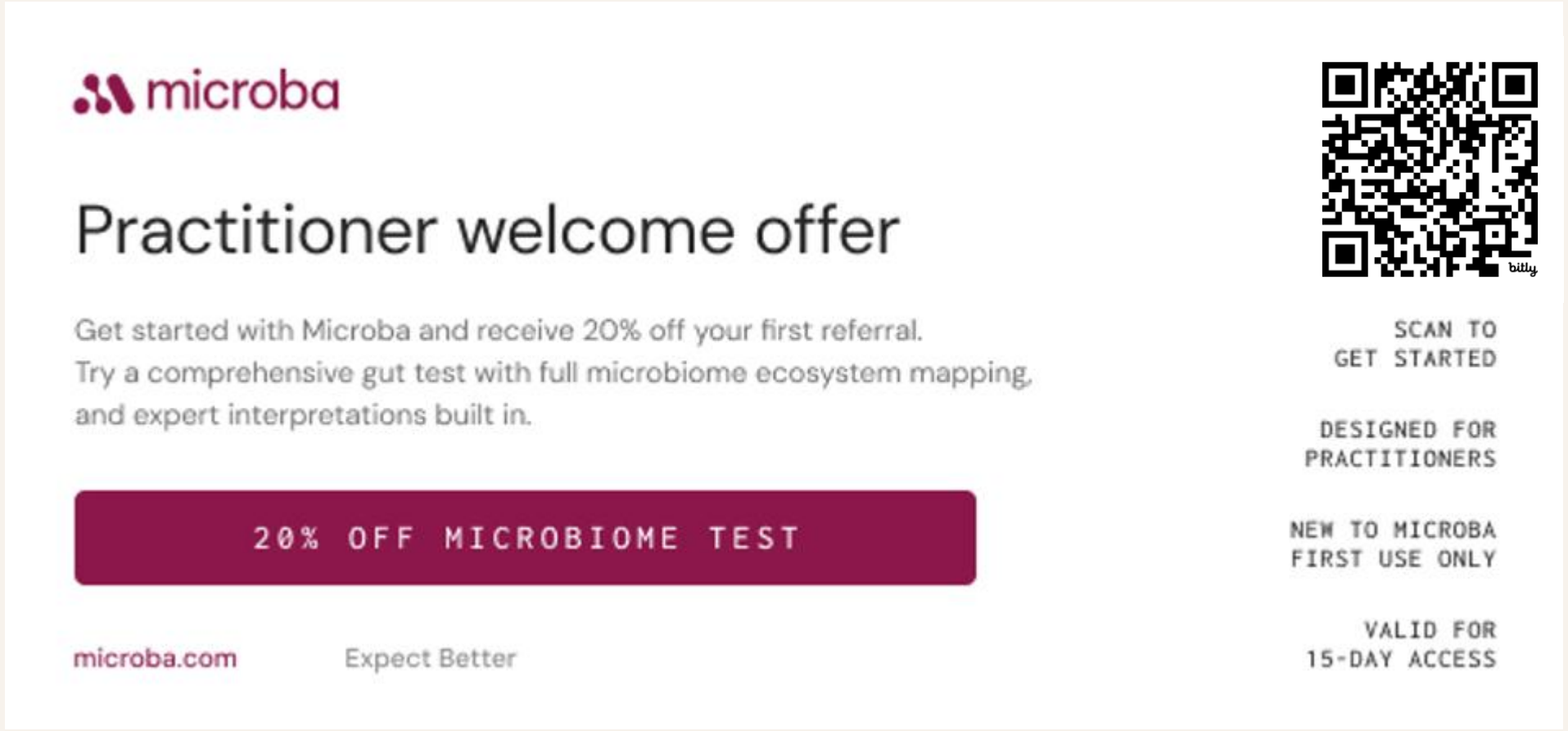

Clinical Growth Team

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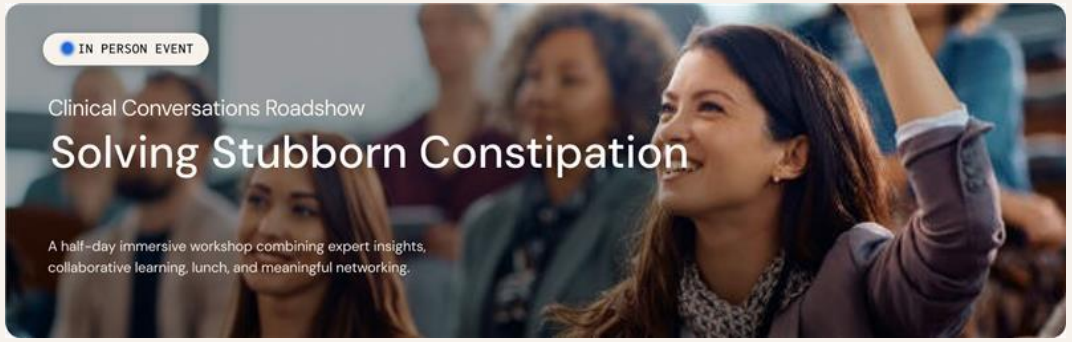
Contact us at: clinicalgrowth@microba.com
Book a Discovery call: https://microba.com/practitioners/support

Clinical Conversations Roadshow



- LONDON **Thursday 21 May 2026** – 10:00 am to 2:00 pm
- BRISTOL **Thursday 4 June 2026** – 10:00 am to 2:00 pm
- TUNBRIDGE WELLS **Thursday 25 June 2026** – 10:00 am to 2:00 pm
- YORK **Thursday 2 July 2026** – 10:00 am to 2:00 pm

WHY ATTEND

Explore stubborn constipation through two clinical lenses: colonic transit and obstructed defaecation patterns, and the role of the gut microbiome in ongoing symptoms.

Walk away with a practical management framework, clear decision points for investigation, and targeted, evidence-informed interventions you can use from day one.

WHAT'S INCLUDED

- Evidence-based presentation
- Interactive case-based workshop
- Nourishing lunch
- Practitioner networking
- 40% off a Microba test
- Stunning venues across four UK cities

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EARLY BIRD **£30**
FULL PRICE **£40**

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