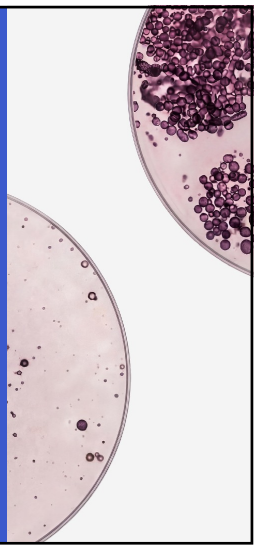
microba events

The starved microbiome

Gabriella Sassi



CLINICAL CONVERSATIONS
004

EXPECT BETTER
MICROBA.COM

1

Learning objectives

By the end of this session, you will be able to:

01 RECOGNISE

What a starved microbiome looks like

- Define the starved microbiome and how it differs from other forms of microbial imbalance
- Identify early signs of diversity decline — before symptoms force investigation

02 INTERPRET

Read markers across time, not snapshots

- Recognise the key microbiome marker clusters and how they progress across multiple reports
- Use Species Explorer in Microba Microbiome Explore to read trajectory, not just current status

03 MANAGE

Rebuild a depleted gut in the right order

- Support the gut after pathogen exposure, antimicrobial use or dietary disruption — and know why sequence matters
- Apply a staged rehabilitation framework mapped to the patient's specific marker deficits

04 COMMUNICATE

Translate findings into patient action

- Distinguish symptom resolution from genuine microbiome recovery
- Communicate longitudinal findings in a way that supports compliance and long-term follow-through

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.

3

Speakers

Dr Brad Leech

Nutritionist
Senior Clinical Educator

Gabriella Sassi

Clinical Nutritionist | Naturopath
Clinica Naturopathy

All participants have been muted

Questions will be answered at the end of the presentation

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

4

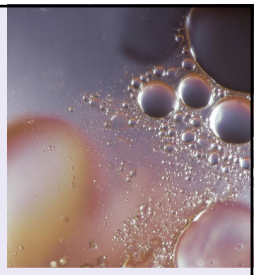
Why it matters

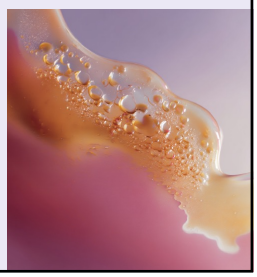
Key themes

- Silent depletion beneath a clinically improving presentation
- Loss of microbial resilience through cumulative ecological insults
- Post-infectious dysbiosis and incomplete recovery
- The clinical value of trajectory interpretation over time

Key message

- The greatest clinical value of microbiome testing if my experience comes from reading it longitudinally not as an isolated snapshot, but as a trajectory across time.





5

microba events

What is a starved microbiome?

001

CLINICAL CONVERSATIONS
004

EXPECT BETTER
MICROBA.COM

6

Defining the starved microbiome

A gut ecosystem chronically underfed not just from infection or dysbiosis, but from the sustained absence of the substrates beneficial microbes need to thrive. Remove fermentable fibre and keystone species decline, SCFA production falls, and the gut loses its capacity to sustain and protect itself.

KEY FEATURES

1. **Reduced microbial richness**
   Lower species diversity across the gut ecosystem.
2. **Depleted SCFA capacity**
   Butyrate, propionate and IPA pathways weakened.
3. **Gram-negative dominance**
   Elevated Hexa-acylated LPS producers.
4. **Persistent immune activation**
   Mucosal sIgA elevated.
5. **Mucin layer degradation**
   Progressive thinning of the protective layer.
6. **Protein fermentation shift**
   Elevated TMA and urease-producing microbes.

7

How this differs from pathogen-driven dysbiosis

STARVED MICROBIOME

Driven by ABSENCE

Insufficient substrate. Beneficial microbes lose ecological footing — no single culprit.

ONSET	Silent, gradual
SYMPTOMS	Non-specific, often misattributed
TESTING	No dominant pathogen
TREATMENT	Ecological rebuilding

PATHOGEN-DRIVEN DYSBIOSIS

Driven by PRESENCE

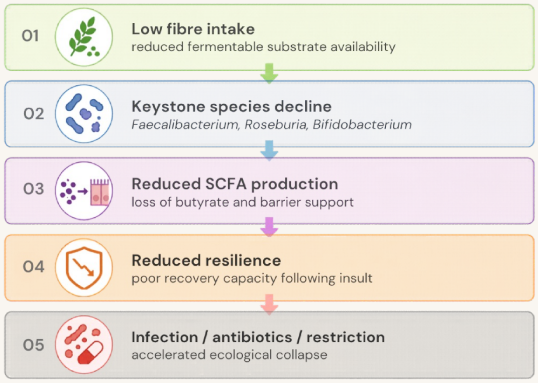
A pathogen or overgrowth disrupts the community — identifiable trigger.

ONSET	Acute, with clear trigger
SYMPTOMS	Patient symptomatic and aware
TESTING	Detectable organism out of range
TREATMENT	Pathogen management

Same patient presentation; opposite clinical logic.

8

How does a starved microbiome develop?



9

Symptoms of a starved microbiome

The combination — not any single symptom — defines the picture.

01 / GASTROINTESTINAL

What the gut shows

- Bloating and distension after meals
- Loose stools or IBS-type pattern
- Poor tolerance to fibre and fermentable foods
- Slow gut transit or constipation
- Nausea or early satiety

02 / SYSTEMIC

What the body shows

- Fatigue not explained by other causes
- Brain fog, poor cognitive clarity
- Frequent infections, slow immune recovery
- Skin changes — eczema, rashes, acne
- Progressive food intolerances

03 / CLINICAL PATTERN

The diagnostic signature

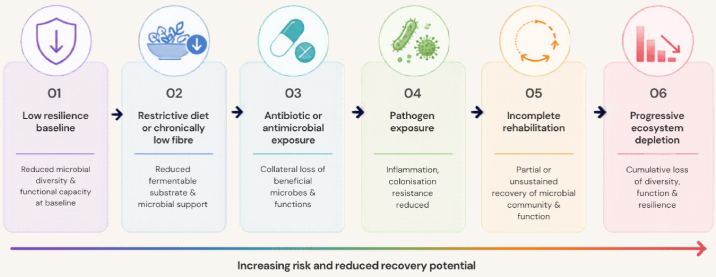
Partial treatment responses that never fully hold · gradual narrowing of food tolerance over months to years · fatigue that persists after GI symptoms improve.

Clinical trap: GI improvement with persistent fatigue is not recovery — it can signal a stable depleted state.

10

The cumulative insult

A depleted microbiome cannot buffer the same insult a diverse microbiome absorbs easily. The same pathogen, the same antibiotic, the same dietary disruption hits differently when the ecosystem is already starved.

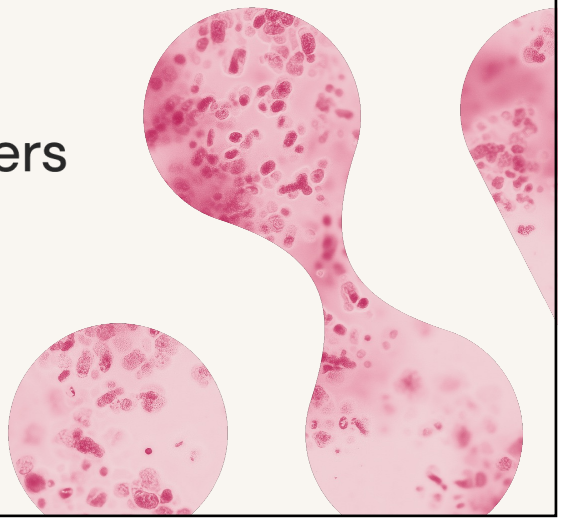


The insult is often unavoidable. The baseline is what we can influence.

11

microba events

Why this matters
002

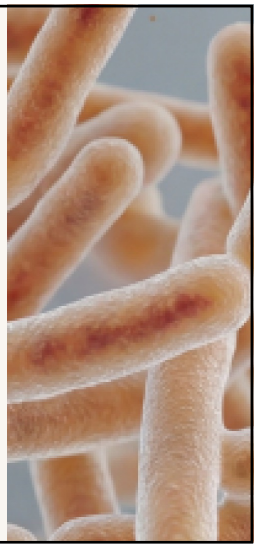


CLINICAL CONVERSATIONS
004

EXPECT BETTER
MICROBA.COM

12

Why microbial diversity matters clinically



What the microbiome regulates

- Immune regulation — Treg development, Th17-Treg balance, mucosal IgA
- Gut barrier integrity — butyrate as primary colonocyte fuel, tight junction maintenance
- Metabolic function — glucose metabolism, lipid signalling, bile acid recycling
- Neurological and mood — tryptophan metabolites, IPA, serotonin precursors
- Cardiovascular risk — TMA/TMAO pathway
- Pathogen defence — colonisation resistance

13

Conditions consistently associated with low microbial diversity

IBD (Crohns & UC)

Depleted butyrate producers, elevated Hexa-LPS, reduced *F. prausnitzii.*

IBS

Reduced Shannon diversity in IBS-D & IBS-M. Low SCFA production & altered fermentation.

Depression & Anxiety

Depleted Bifidobacterium, impaired IPA/tryptophan metabolism.

Metabolic Syndrome &T2D

Altered fermentation, impaired insulin signalling, elevated inflammatory load.

Cardiovascular disease

Elevated TMA/TMAO production, LPS-driven systemic inflammation.

Autoimmune conditions

Impaired Treg function, increased permeability, LPS-mediated immune activation.

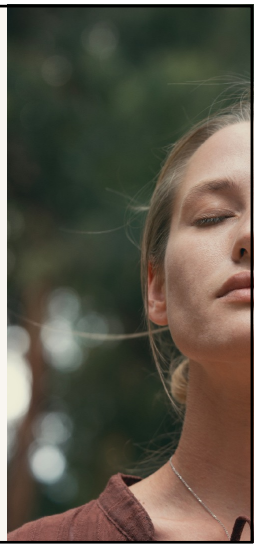
The starved microbiome is not a diagnosis. It is a low-resilience ecosystem state that increases vulnerability across all these systems.

14

Why clinicians miss the starved microbiome

- Symptom improvement is interpreted as recovery
- Normal calprotectin creates false reassurance — it measures active mucosal inflammation, not microbial recovery
- Pathogen-focused models overlook ecosystem function — a negative pathogen screen says nothing about richness or butyrate producers
- Single-report interpretation misses trajectory decline
- Patients may appear health-conscious despite low microbial resilience
- Functional symptoms fluctuate while microbial depletion progresses silently

Key insight: The microbiome can continue deteriorating beneath a clinically improved presentation.



15

microba events

Identifying the starved microbiome

003

CLINICAL CONVERSATIONS
004

EXPECT BETTER
MICROBA.COM

16

Clinical clues before testing

Red flags

- Restrictive dietary patterns — extended low-FODMAP, ketogenic, carnivore without reintroduction
- Repeated antibiotic exposure, even years prior
- Travel-related GI illness without post-course rehabilitation
- Long-term low fibre intake — common in health-conscious patients eating quality but not diverse food
- Ultra-processed food reliance
- Chronic stress physiology or burnout

Clinical patterns

- Fluctuating IBS-type symptoms that respond partially but never fully resolve
- Gradual narrowing of food tolerance over months
- Mood and GI overlap — anxiety alongside gut symptoms
- Relapse with food reintroduction
- Persistent fatigue despite normal standard pathology

Clinical question: "Has your diet, stress levels, travel history, or antimicrobial exposure changed significantly over the last 1–2 years?"

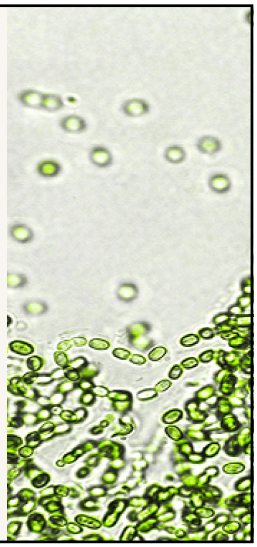
17

Reading the starved microbiome pattern

The characteristic cluster progression:

→ Butyrate producers ↓ — the leading signal, almost always first
→ IPA-producing microbes ↓ — tryptophan pathway begins to fail
→ Microbial richness stagnates, then falls
→ Mucin degradation ↑ — mucus layer loses protective substrate
→ Hexa-LPS ↑ — gram-negative organisms fill the vacated space
→ sIgA ↑ — mucosal immune system escalates

Read the cluster, not the single marker.
A borderline-low marker trending downward over multiple reports is more clinically important than a single low result in isolation. Direction is the data. The value is context.



18

Diversity, richness, functional markers & substrate species

Markers & what they tell you

Richness (200–250 optimal)

- Number of distinct species. Track direction across timepoints, not the single value.

Diversity

- Species count & relative balance. Persistent below-average scores with no upward trend indicate failed recovery.

Faecal pH (optimal 5.70–7.30)

- Measurable confirmation of fermentation capacity. Rising pH confirms carbohydrate fermentation has failed & protein fermentation dominates.

Calprotectin

- Active mucosal inflammation marker only — not a marker of microbial recovery. Normal calprotectin does not mean recovered ecosystem.

19

Which species need which substrates

Key species	Primary substrate	Clinical food sources
Faecalibacterium prausnitzii	Resistant starch, arabinogalactan	Cooled cooked potato, green banana flour, legumes
Roseburia / *Eubacterium rectale*	Resistant starch, inulin-type fructans	Green banana flour, legumes, cooked-cooled grains
Bifidobacterium spp.	Inulin, FOS, GOS	Chicory root, onion, leek, Jerusalem artichoke, GOS supplement
Akkermansia muciniphila	Polyphenols, mucin substrate	Pomegranate, berries, green tea, olive oil

This is why ≥30 plant foods per week is recommended — different species require different substrates. Diversity of substrate sustains diversity of organisms.

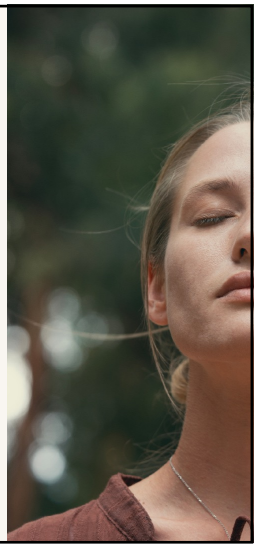
20

Clinical interpretation hierarchy

When interpreting a set of reports, work through this order:

- Red Flags first
- Trajectory over time — direction of change across reports before the isolated value
- Functional output — SCFA pathways and faecal pH confirming fermentation status
- Immune activity — sIgA persistence signals ongoing mucosal pressure
- Diversity and richness — in context of functional & immune markers
- Species-level interpretation — who is driving the pattern

Direction of change across reports is more clinically valuable than the isolated value itself.



21

What recovery actually looks like

A staged trajectory — symptoms shift first, the ecosystem rebuilds last.

Weeks 4–8 **01**

EARLY

Symptoms shift

Symptoms stabilise, transit improves, energy begins to return.

A positive signal — but the microbiome has not yet recovered.

Months 3–6 **02**

INTERMEDIATE

Function returns

sIgA trends downward, fibre tolerance widens, SCFA production begins to recover.

Retest now — confirm the functional trajectory is on course.

Months 6–12+ **03**

LATE

Ecosystem rebuilds

Diversity and richness improve; resilience to variation and illness normalises.

True recovery — longer in severely depleted patients.

THE PRINCIPLE

Symptoms improve before the microbiome recovers — every time.

CLINICAL TRAP

Close the case on symptoms alone, and you will always close it too early.

22

When to test & retest

When to test

- Persistent or incompletely resolving GI symptoms
- Post-antibiotic or post-infectious presentations
- Restrictive dietary history
- Chronic fatigue with GI history and normal standard pathology

When NOT to test

- During an acute GI flare — blood in stool distorts microbial reads
- Within 4–6 weeks of antibiotic course
- Within 2 weeks of colonoscopy prep

Retesting framework

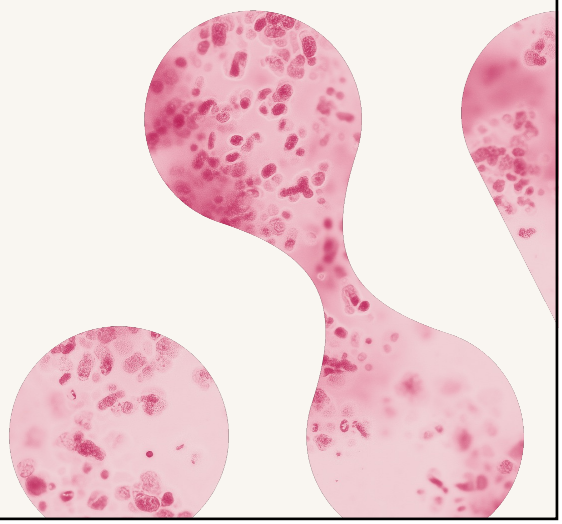
- 8–12 weeks — SCFA and inflammatory trajectory
- 3–6 months — diversity and richness recovery
- Continue until improvement is sustained across consecutive reports

Retesting is not monitoring — it is clinical decision-making.
Every retest is a decision point: continue, escalate, or recalibrate.

23

microba events

Management framework 004



CLINICAL CONVERSATIONS
004

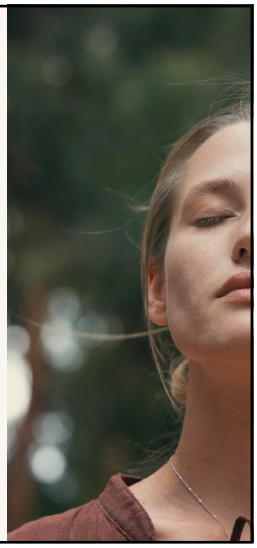
EXPECT BETTER
MICROBA.COM

24

Clinical rehabilitation framework

Step 1: Meet the patient

- Validate current effort — doing the right things and not fully recovering is frustrating
- Reduce fear around food — reintroduction is part of the therapy
- Set realistic expectations — months, not weeks, confirmed by data not symptoms

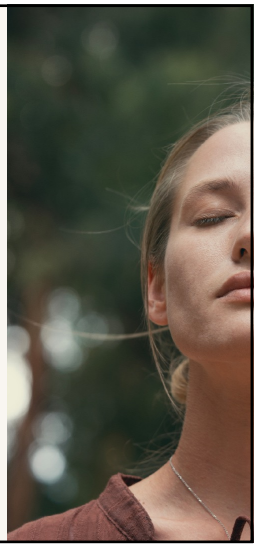


25

Clinical rehabilitation framework

Step 2: Rebuild substrate diversity

- Start low & go slow — gradual fibre escalation to avoid intolerance
- Target >30 plant foods per week
- Resistant starch — cooled cooked potato, green banana flour, legumes, oats
- Inulin & FOS sources — chicory root, Jerusalem artichoke, garlic, onion, leek
- Polyphenols — berries, pomegranate, green tea, extra virgin olive oil
- Fermented foods daily — yoghurt, kefir, kimchi, sauerkraut
- Omega-3 rich proteins — oily fish minimum 3x per week

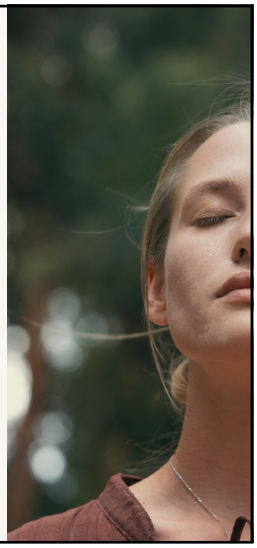


26

Clinical rehabilitation framework

Step 3: Targeted supplementation – map to markers

- Butyrate formally LOW — targeted dietary resistant starch, broad prebiotic substrate, & Bifidobacterium probiotics to stimulate endogenous butyrate production
- Richness depleted — high-strain diversity probiotic plus broad prebiotic substrate
- Hexa-LPS & TMA elevated — anti-inflammatory lipid therapy
- sIgA persistently elevated — mucosal immune modulation, barrier repair nutrients

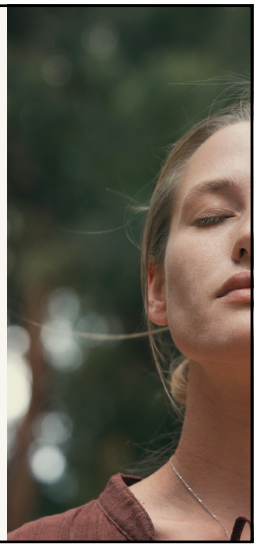


27

Clinical rehabilitation framework

Step 4: Retest & adjust

- Protocol is a clinical hypothesis — the retest confirms whether it is correct
- If markers are not moving as expected, recalibrate protocol along with dietary adherence — not the patient's effort



28

Fibre has daily targets. Prebiotics do not.

Three reference points clinicians should know — and the gap between them.

FIBRE - AI

25–30g/day

Women 25 g · Men 30 g
NHMRC Adequate Intake — based on adequate laxation.

FIBRE - SDT

28–38g/day

Women 28 g · Men 38 g
NHMRC Suggested Dietary Target — to reduce chronic disease risk.

PREBIOTICS

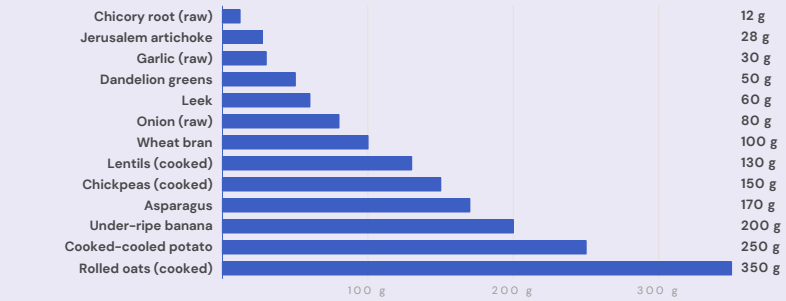
No formal RDI

ISAPP: ≥3 g/day; ~5 g/day target
No governing body has set an intake target. ISAPP offers the only working figure.

29

What is 5g of prebiotics

Grams of food required to deliver ~5 g of prebiotic fibre, ordered by efficiency.

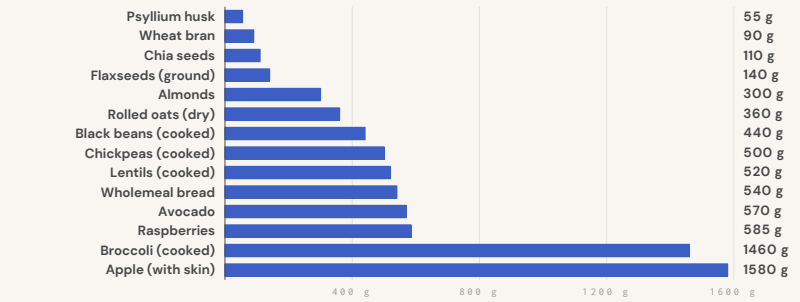


Values approximated from Moshfegh 1999, Van Loo 1995, Muir 2009, Costabile et al. — content varies with cultivar, season, & preparation.

30

What is 38g of fibre

Grams of food required to deliver the NHMRC SDT of 38 g — the upper target for chronic disease prevention.



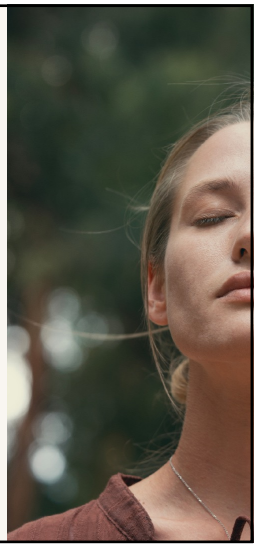
Values from USDA SR & FSANZ AUSNUT 2011-13. SDT for men = 38 g/day; women = 28 g/day. Scale servings to patient.

31

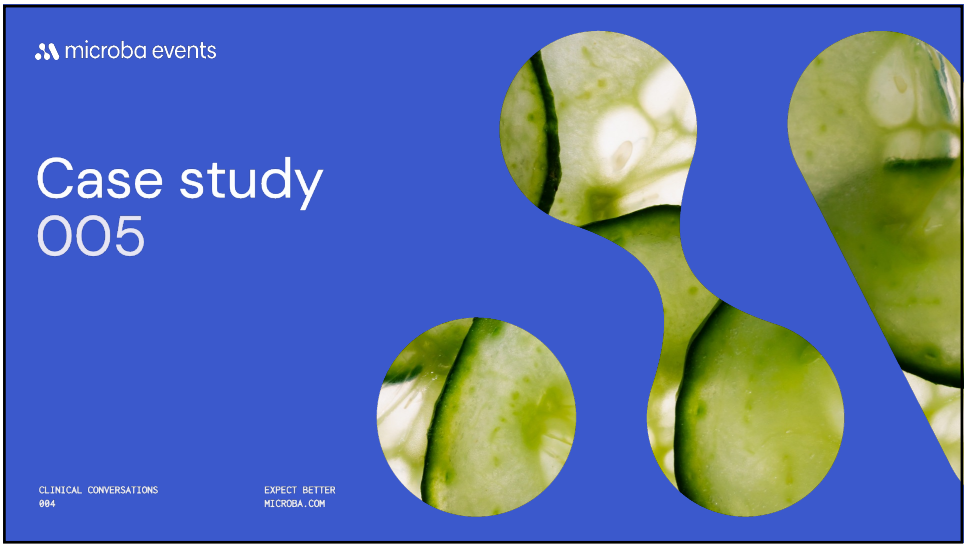
Common reasons for microbiome rehabilitation failure

- Fibre introduced too aggressively — symptoms triggered, patient stops
- Treatment stopped at first symptom improvement — the single most common reason for relapse
- Probiotics without prebiotic substrate — seeding without feeding
- Ongoing restrictive eating — fermentable fibre avoidance prevents diversity rebuilding
- Excessive fear of symptoms — reinforces restriction
- Lack of retesting — management defaults to symptoms
- Under-eating overall

The goal is ecological rebuilding, not temporary symptom suppression.



32



33

Patient overview: Four report timeline

JUNE 2023
PHASE 1
Stress-driven functional dysbiosis
Five years of chronically low fibre intake. Gut-brain axis dominant. Depleted baseline with early immune activation.
Complete symptom resolution following treatment.

2023 – 2025
THE GAP
Cumulative insult — no monitoring
Overseas travel, dietary disruption, doxycycline tx (2 wks, malaria prevention), binder use, acute GI infection requiring hospitalisation.
No pathology taken.

OCTOBER 2025
PHASE 2
Post-infectious collapse
Post-infectious dysbiosis confirmed. Pathobionts detected, barrier dysfunction, hexa-LPS climbing, butyrate further depleted.
Rehabilitation commenced. Symptoms improved.

FEBRUARY 2026
PHASE 3
Incomplete recovery
GI symptoms largely resolved. Microbiome at its lowest point across the case.
Protocol recalibrated in response to data.

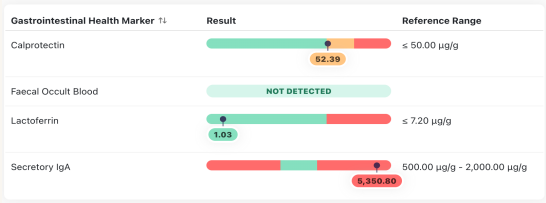
MAY 2026 · REPORT 4
PHASE 4
Early ecosystem recovery signals
Richness 118 species. Propionate pathway active; butyrate producers still depleted. Protocol continues.
sIgA Optimal ✓ Richness Low
TMA High pH High Butyrate ↓
Propionate ↑

34

Report 1: June 2023
Functional dysbiosis

Clinical presentation

- Alternating loose stools and constipation
- High fat and high glycaemic food intolerance
- Stress-exacerbated GI symptoms — gut-brain axis involvement
- Burnout physiology and high exercise load
- Long-term low fibre intake (~5 years)
- Prior ad hoc antimicrobial use



35

Report 1: June 2023
Functional dysbiosis

Key findings

- Richness: 139 species — borderline low
- sIgA: elevated — mucosal immune activation
- Calprotectin: borderline — colonoscopy returned normal
- BCAA producers: mildly elevated

Interpretation

- Gut-brain axis dysregulation driving functional, stress-responsive bowel pattern
- Early immune activation without overt IBD
- Low diversity with early metabolic imbalance

Intervention & outcome

- PHGG, multi-strain probiotic, Saccharomyces boulardii, fermented foods
- Stress response and nervous system regulation — introduced as primary treatment pillar
- Outcome: complete symptom resolution, normalised bowel pattern

Clinical decision point: Symptoms resolved completely. No retest was conducted. The baseline deficit — borderline richness, elevated sIgA — remained unconfirmed.

36

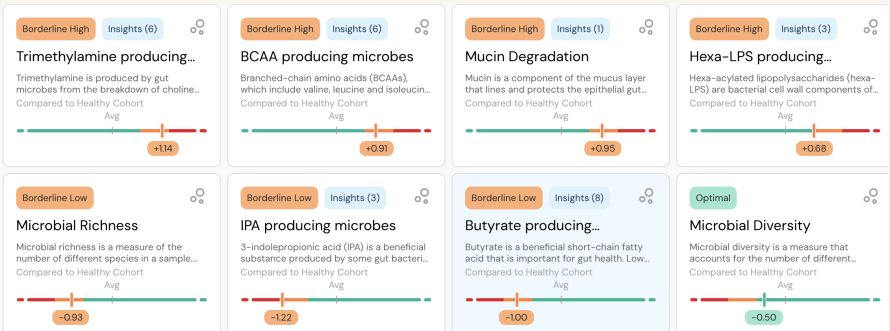
Report 2: October 2025
Post-infectious collapse

What happened between the reports - the gap

- Multiple overseas trips — significant dietary disruption
- Doxycycline 2-week course — documented Bifidobacterium-depleting effect, landed on already borderline-low richness
- Binders use during travel
- Acute GI infection — hospitalisation, IV fluids only, no PCR taken
- No microbiome monitoring throughout

37

Report 2: October 2025
Post-infectious collapse



38

Report 2: October 2025
Post-infectious collapse

Key findings

- Richness: 146 species — borderline low (numerical increase, not clinically meaningful)
- Aeromonas spp. detected — indeterminate, consistent with acute GI history
- Dominant pathobiont: Bacteroides vulgatus
- ↑ hexa-LPS, ↑ Bilophila wadsworthia, ↑ E. coli (flexneri)
- ↑ mucin degradation, ↓ butyrate producers, ↑ TMA producers
- sIgA persistently elevated — uninterrupted since Report 1
- Calprotectin: normal

Interpretation

- Post-infectious dysbiosis — barrier dysfunction, endotoxin dominance, persistent immune activation
- Functional fatigue emerging despite normal standard pathology

39

Report 2: October 2025
Post-infectious collapse

Management - phase 2

- Barrier support first — L-glutamine, targeted probiotics, fermented foods
- Graded fibre reintroduction — GOS and PHGG at low starting doses
- Omega-3 fatty acids — anti-inflammatory LPS burden
- Mediterranean dietary pattern

40

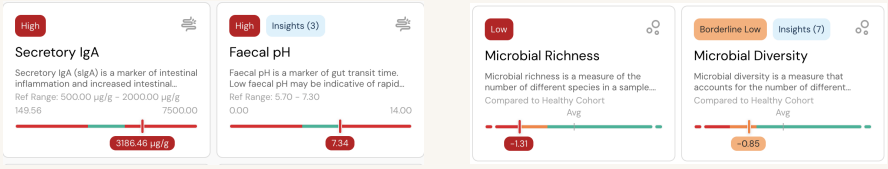
Report 3: February 2026
Incomplete recovery

Clinical status

- GI symptoms largely resolved
- Persistent fatigue — morning fatigue and afternoon energy crashes
- Mild ongoing intolerance to legumes and high-fibre loads
- Fully compliant with protocol

41

Report 3: February 2026
Incomplete recovery



42

Report 3: February 2026
Incomplete recovery

Key findings

- Richness: 130 species — lowest recorded, below 2023 baseline
- Butyrate producers: formally LOW — lowest value across all three reports
- Hexa-LPS: HIGH — first time across the case
- TMA: HIGH — first time across the case
- Faecal pH: above range — alkaline shift confirming SCFA fermentation has failed
- sIgA: persistently elevated — three years of continuous mucosal immune activation
- *E. coli dysenteriae* elevated — strain shift from flexneri in Report 2
- Propionate producers: borderline high — compensatory shift, slower transit

43

Report 3: February 2026
Incomplete recovery

Interpretation

- Chronic post-infectious dysbiosis phenotype
- Persistent immune activation despite symptom improvement
- Incomplete barrier repair — low butyrate state
- Fatigue driven by gut-immune-mitochondrial axis interaction

44

Phase 3 protocol

Each intervention mapped directly to the specific marker deficit identified in February 2026.

Target marker	Intervention	Product/approach	Dose
Elevated sIgA — mucosal immune activation	Multi-strain probiotic	LGG, BB-12, Saccharomyces boulardii	1 daily
Richness 130 species — depleted diversity	High-strain diversity probiotic	30B CFU, multi-strain	1 daily
Richness + butyrate — substrate support	Broad prebiotic blend	Oats 2g, RS65 green banana 2g, 2'-fucosyllactose 1g, GOS 1g, PHGG 1g, pomegranate ext 0.5g	7.5g twice daily
Transit — elevated propionate, slow transit	Spore + PHGG synbiotic	B. coagulans IS2, B. subtilis HU58, PHGG	1 tablespoon with food
Hexa-LPS HIGH, TMA HIGH	Anti-inflammatory lipid therapy	High-strength omega-3	4020mg total daily
Barrier dysfunction — sIgA + low butyrate	Gut barrier repair nutrients	L-glutamine, turmeric, zinc carnosine, quercetin	1 scoop 2 per day

45

Report 3: February 2026
Incomplete recovery

Phase 3 | Dietary strategy

Mediterranean high-diversity pattern
DAILY

>30 plant foods per week
WEEKLY TARGET

Resistant starch daily
Cooled cooked potato, green banana flour, legumes

Polyphenol-rich foods
Berries, pomegranate, green tea, olive oil

Fermented foods rotation
DAILY

Omega-3 rich proteins
Oily fish minimum 3× per week

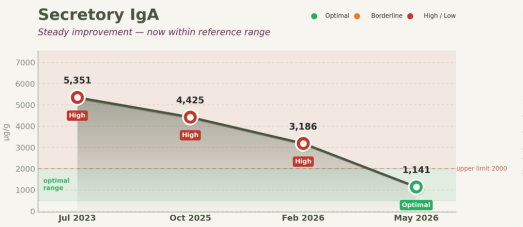
Strict reduction of ultra-processed foods & artificial sweeteners

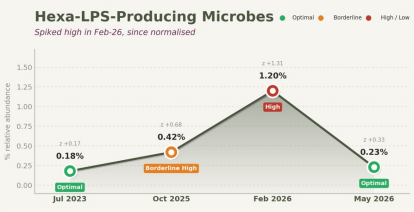
Hydration 2–2.5 L daily
DAILY

46

Report 4: Inflammation & barrier — resolving

The diagnostic markers that drove the case are now within range



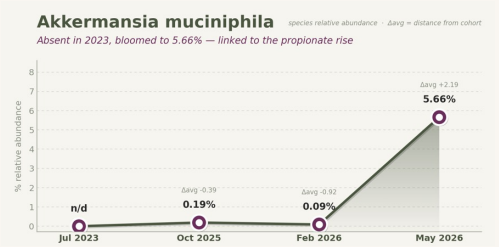


Both inflammatory and barrier markers normalised by May 2026. Secretory IgA fell from 5351 to 1141 µg/g — optimal for the first time across all four reports, signalling that mucosal immune activation has resolved. Hexa-LPS producers spiked to a High 1.20% in Feb 2026, then returned to an optimal 0.23%, confirming the endotoxin burden is controlled.

47

Report 4: Akkermansia bloom — regeneration

One constructive counter-signal within a depleted ecosystem



Absent in 2023, now 5.66%

Akkermansia muciniphila was undetected in 2023 and bloomed to 5.66% by May 2026 (Δ +2.19 from the cohort mean).

As a mucin-associated keystone and propionate producer, its emergence is consistent with recovery of the mucin layer and tracks the propionate rise. Within an otherwise depleted functional ecosystem, this is a genuinely constructive signal — and consistent with the polyphenol strategy.

48

Report 4: May 2026
Early recovery signals

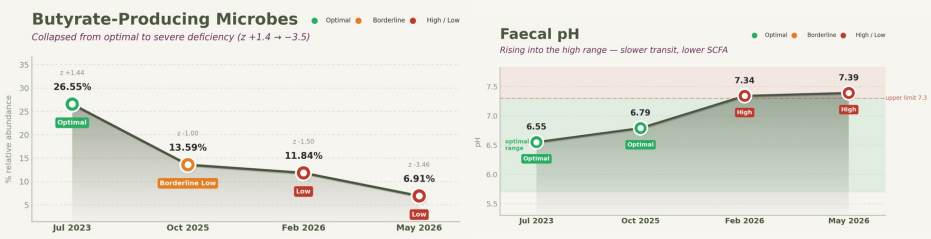
Clinical interpretation – early recovery trajectory

- Propionate pathway active — the ecosystem is beginning to rebuild SCFA capacity, starting with propionate before butyrate
- Akkermansia emergence — mucin layer activity recovering, polyphenol strategy having effect
- Fibre-fermenting Bacteroides species rising — dietary substrate strategy taking hold
- Butyrate producers still depleted — the most important producers (Agathobacter, Agathobaculum) remain below average

Interpretation: This is early-stage ecosystem recovery. Propionate before butyrate is the expected sequence. The protocol is working but butyrate restoration requires continued substrate and dietary support.

As butyrate falls, faecal pH rises

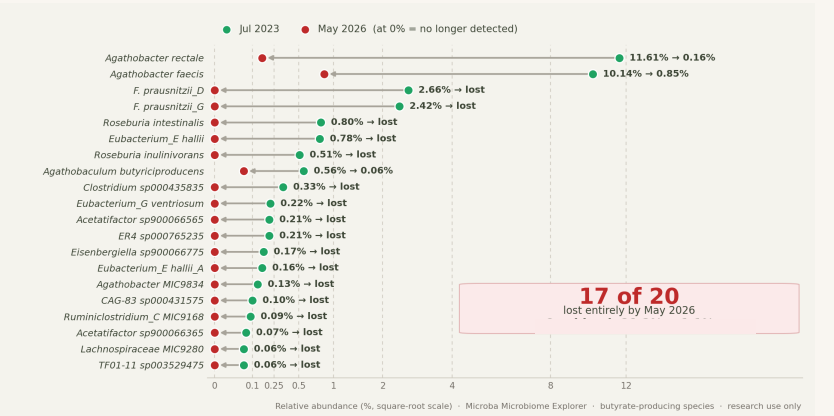
Butyrate is a short-chain fatty acid that acidifies the colon



As butyrate-producing microbes decline, faecal pH rises. Butyrate is a short-chain fatty acid; it is acidic and acidifies the bowel. Without sufficient butyrate, the bowel becomes more alkaline — shifting faecal pH from an optimal 6.55 into the high range at 7.39 as butyrate producers collapsed from 26.6% to 6.9%.

Twenty butyrate producers in decline

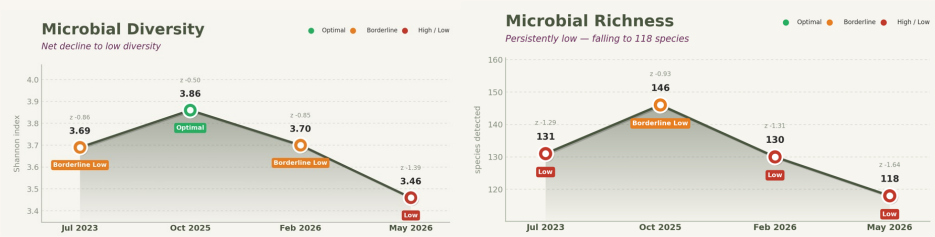
The most-depleted producers across all four reports



51

Microbial diversity and richness

Both ecological measures peaked in 2025, then declined to low



The ecosystem has narrowed over three years. Shannon diversity rose to 3.86 in Oct 2025, then fell to a low 3.46, and richness followed the same arc — 131 → 146 → 130 → 118 named species. Fewer species and lower evenness mean less functional redundancy and resilience, consistent with the loss of butyrate-producing taxa.

52

Longitudinal trends — all four reports

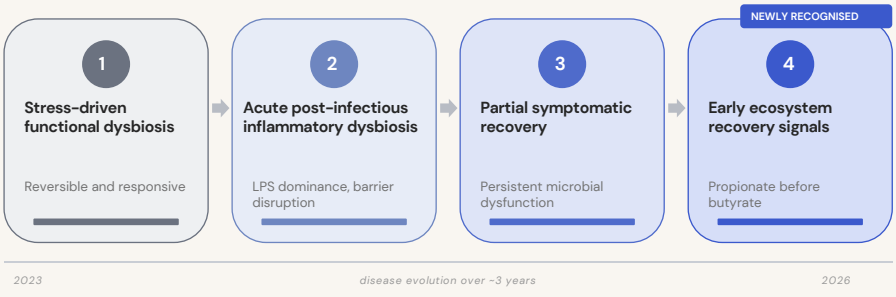
MARKER	REPORT 1 JUL 2023	REPORT 2 OCT 2025	REPORT 3 FEB 2026	REPORT 4 MAY 2026	TREND
Microbial richness	BORDERLINE LOW 139	BORDERLINE LOW 146	LOWEST POINT 130	LOW 118	Rebuilding underway; richness lags diversity — recovery expected
Butyrate producers	FUNCTIONAL RESERVE	BORDERLINE	FORMALLY LOW	STILL LOW 6.91% (−3.46 DFA)	Last to recover in the ecological sequence; direct butyrate support continuing
sIgA	ELEVATED	ELEVATED	ELEVATED	OPTIMAL 1141.43 µg/g	First time in range across all four reports; mucosal immune activation resolved
Hexa-LPS / pathobionts	OPTIMAL	ELEVATED *B. vulgatus dominant*	HIGH *E. coli dysenteriae*	OPTIMAL 0.23%	LPS burden controlled — omega-3 & dietary strategy effective
TMA producers	NOT FLAGGED	BORDERLINE HIGH	HIGH	HIGH 6.08% (+1.68 DFA)	Elevated but addressed; omega-3 & Mediterranean diet
Faecal pH	OPTIMAL	OPTIMAL	ABOVE RANGE	ABOVE RANGE 7.39	Will normalise as butyrate producers recover — expected sequencing
Propionate producers	NOT FLAGGED	NOT FLAGGED	BORDERLINE HIGH	ACTIVE 11.29% (+1.74 DFA)	SCFA pathway rebuilding — propionate precedes butyrate in recovery
Akkermansia muciniphila	NOT FLAGGED	NOT FLAGGED	NOT REPORTED	EMERGING 5.66% (+2.19 DFA)	Polyphenol strategy working — mucin layer activity recovering
Pathobionts	ABSENT	DOMINANT *B. vulgatus*	ELEVATED *E. coli dysenteriae*	NOT REPORTED	Reducing
Blautia spp.	–	–	–	DOMINANT *Massiliensis 11.66%, Wexlerae 8.73%*	Propionate producers active; ecosystem rebuilding from the substrate up

● Optimal / favourable ● Borderline / transitional ● Out of range / adverse ● Not assessed

53

A four-phase evolution

The three-phase model now extends to a fourth — early recovery



54

Clinical lessons from the case

- Recovery is nonlinear — the trend across multiple reports is the signal; any single report is a snapshot
- Symptom trajectory and microbiome trajectory can diverge — the data is the only way to know which direction the ecosystem is actually moving
- Report 1 showed borderline diversity and depleted keystone species, yet the patient was discharged on negative colonoscopy and symptom resolution. Given the dietary history, a clear protocol for microbiome rehabiliation could have been established. As well as a repeat test 3-6 months later.
- Retesting changes management — in this case it changed the entire therapeutic approach at every stage
- Barrier repair takes time — sustained support is required, not a short course
- Low resilience magnifies every future insult — building resilience protects future trajectory
- Early recovery shows propionate before butyrate — expect this sequence; continue protocol until butyrate producers recover

55

Key learnings

Eight learnings, four shifts in practice.

01 RECOGNISE

Spot the starved-microbiome picture early

- Define starved microbiome — substrate absence, not pathogen presence.
- Catch early clinical signals before symptoms force investigation.

02 INTERPRET

Read the marker cluster, not isolated values

- Sequence the cluster — butyrate, then IPA, then richness, then LPS & sIgA.
- Connect dietary recommendations to specific species and substrates.

03 MANAGE

Rebuild the gut in the right order

- Support in sequence after insult — barrier first, then substrate, then diversity.
- Apply staged rehabilitation mapped to markers — not generic supplementation.

04 COMMUNICATE

Translate findings into realistic expectations

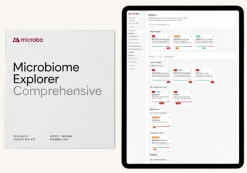
- Distinguish symptom resolution from genuine recovery — confirm with data.
- Frame longitudinal findings to support realistic expectations and compliance.

THE THROUGH-LINE

Treat substrate, sequence, and structure — not symptoms alone.

56

Our Clinical Range



Microbiome Explorer Essentials

Insights about the microbiome's impact on systemic health.

- Intestinal permeability
- Autoimmunity concerns
- Mental health issues
- Hormonal issues
- Systemic inflammation

Panels: Microbiome Profile (mNGS)

Microbiome Explorer Extended

Insights about the microbiome's impact on systemic health.

- Intestinal permeability
- Autoimmunity concerns
- Mental health issues
- Hormonal issues
- Systemic inflammation

Panels Microbiome Profile (mNGS) GI Health Panel (ELISA)

Microbiome Explorer Comprehensive

Insights about the microbiome's impact on systemic health.

- Intestinal permeability
- Autoimmunity concerns
- Mental health issues
- Hormonal issues
- Systemic inflammation

Panels Microbiome Profile (mNGS) GI Health Panel (ELISA) Pathogen Panel (PCR)

57

Supporting resources

DOWNLOAD

The Usual Suspects Species Guide

Beneficial and detrimental metabolites and their associated species

Download

GUIDE

Prebiotic Guide

Make the connection between beneficial gut species and the prebiotics that nurture them.

Download

GUIDE

Polyphenols Clinical Guide

Discover the plant power of polyphenols and their interaction with health and disease.

Download

GUIDE

Low FODMAP Prebiotic Guide

Discover some well-tolerated, low FODMAP, prebiotic fibres that may help manage gut symptoms.

Download

58

microba events

Questions & Answers 006

CLINICAL CONVERSATIONS
004

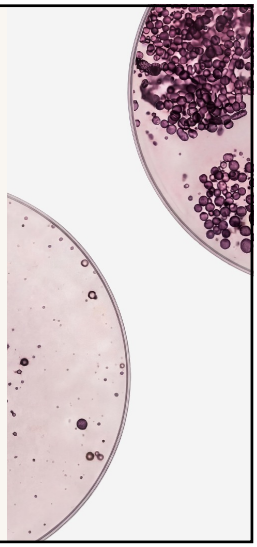
EXPECT BETTER
MICROBA.COM

59

microba events

Thank you

We develop world-leading microbiome research & technology, delivering tests that go deeper & broader & insights that improve people's lives.
Because better is possible.



CLINICAL CONVERSATIONS
004

EXPECT BETTER
MICROBA.COM

61