# Functional Medicine Approach to Fibromyalgia

Gluten Sensitivity, Dysbiosis & KBMO FIT Testing in Clinical Practice

Understanding Primary versus Secondary Fibromyalgia through the through the Lens of Gut Health, SIBO & Functional Medicine


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ibs clinics

# Clinical Differences: Primary versus Secondary Fibromyalgia

## Primary Fibromyalgia Presentation

- Sleep architecture dysregulation and non-restorative sleep sleep patterns
- Trauma history (physical or psychological) frequently present
- Central nervous system overactivation and sensitisation
- Gradual improvement trajectory with multi-modal approaches

## Secondary Fibromyalgia Presentation

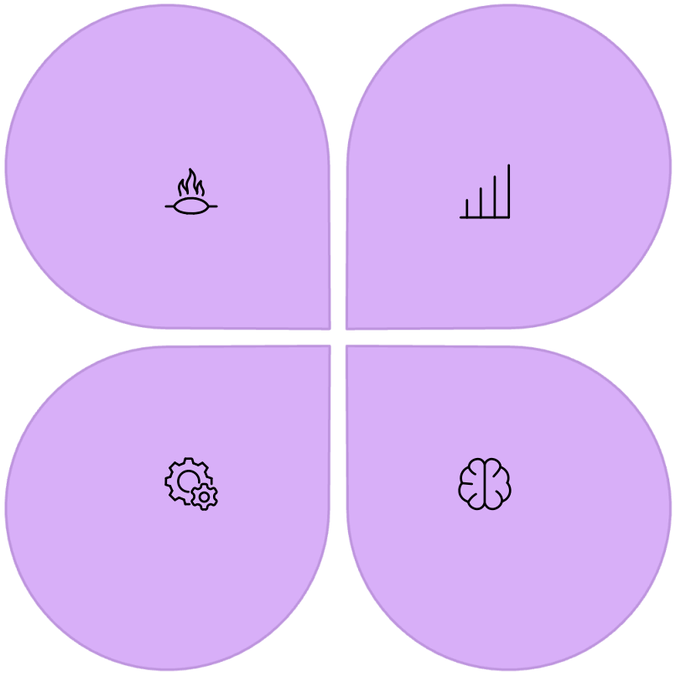
- Gut inflammation and digestive symptomatology prominent
- Food antigens driving systemic inflammatory responses
- Dysbiosis patterns evident on comprehensive testing
- Dramatic response to dietary modification and gut healing protocols

The key clinical distinction lies in treatment response velocity. Secondary fibromyalgia responds dramatically to targeted dietary changes, whilst primary fibromyalgia demonstrates more gradual improvement requiring comprehensive central sensitisation protocols.

# The Gut–Immune–Pain Axis

## Peripheral Inflammation

Gut-derived inflammatory mediators and food antigens trigger systemic immune activation



## Cytokine Production

Pro-inflammatory cytokines cross blood-brain barrier, sensitising central pain processing pathways

## Secondary Fibromyalgia

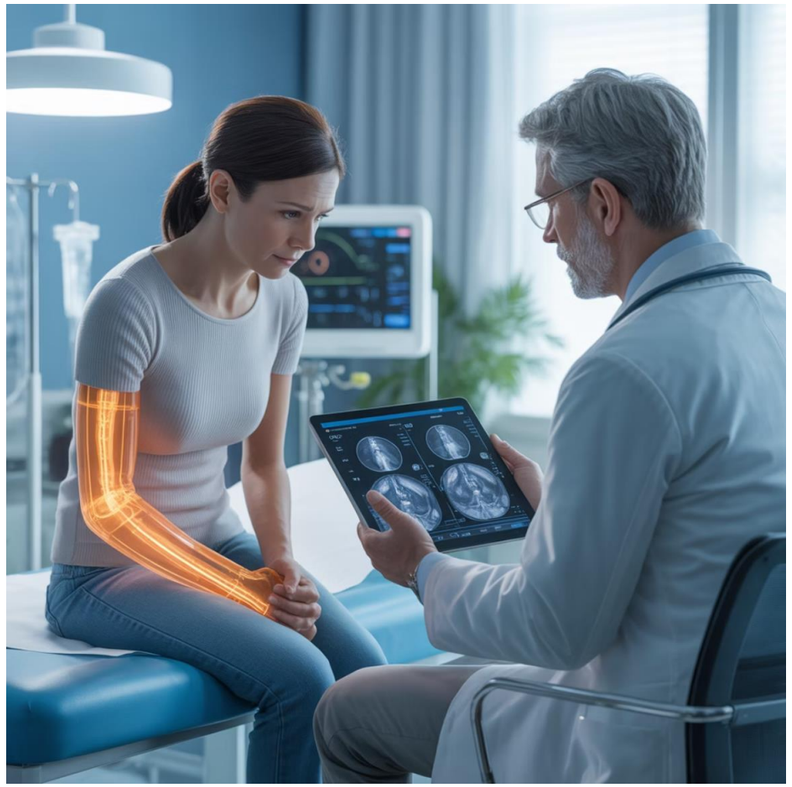
This mechanism explains how gut-driven inflammation can manifest as fibromyalgia fibromyalgia symptomatology

## Central Sensitisation

Amplified pain signalling throughout nervous system, creating widespread pain from peripheral inputs

Understanding this bidirectional communication illuminates why addressing gut inflammation can rapidly resolve widespread pain in secondary pain in secondary fibromyalgia presentations.

# Patient Overview, Ms E, age 34 years old



## Presenting History

Female adult presenting with chronic widespread pain since childhood, accompanied by accompanied by irritable bowel syndrome, persistent insomnia, significant brain fog, and fog, and progressive hair loss.

## Iatrogenic Factors

Extensive history of antibiotic use throughout adolescence and adulthood, combined with combined with daily antacid consumption, created profound microbiome disruption and disruption and digestive impairment.

## Lifestyle Stressors

Pattern of overtraining coupled with insufficient caloric intake generated significant hypothalamic-pituitary-adrenal axis stress and metabolic dysfunction, further compromising gut barrier integrity.

# The Perfect Storm: Converging Risk Factors

## Frequent Antibiotics

Repeated courses throughout development led to microbiome depletion, reduced diversity, and loss of beneficial commensal organisms

## Daily Antacid Use

Chronic acid suppression impaired protein digestion, reduced mineral absorption, and created small intestinal bacterial overgrowth risk

## Low Caloric Intake

Insufficient energy provision resulted in micronutrient deficiency, impaired gut barrier maintenance, and compromised immune function

## Excess Training Load

Overtraining without adequate recovery increased intestinal permeability through through physiological stress and reduced splanchnic blood flow

# Comprehensive Testing Strategy

01

## SIBO Breath Testing

Three-hour hydrogen and methane breath test to test to assess small intestinal bacterial overgrowth overgrowth and identify fermentation patterns patterns

02

## Comprehensive Stool Analysis

Full digestive function assessment including markers of inflammation, immune function, digestive capacity, and microbial balance

03

## KBMO FIT Testing

Food inflammation test measuring antigen load load across 132 foods, plus intestinal permeability permeability markers and candida antibody assessment

This multi-layered approach enables identification of overlapping gut dysfunctions whilst avoiding the common pitfall of single-mechanism attribution in mechanism attribution in complex inflammatory cases.

# Metaxplore GI Plus - Comprehensive Stool Test Findings

1

## Low Secretory IgA of <149.56 (500-2000 ug/g)

Impaired mucosal immunity indicating compromised first-line defence against pathogens pathogens and reduced capacity to maintain oral tolerance to food antigens

2

## Reduced Pancreatic Elastase of 179.64 (>200 ug/g)

Low elastase levels suggesting reduced digestive capacity, likely secondary to chronic antacid use impairing pancreatic enzyme activation and secretion

3

## Depleted Butyrate Producers

Significant reduction in butyrate-producing bacteria elevating gut inflammation risk and inflammation risk and compromising colonocyte energy metabolism and barrier function barrier function

4

## Mild Calprotectin Elevation of 81.27 (<50 ug/g)

Modest inflammatory marker elevation, likely attributable to NSAID use for pain pain management rather than inflammatory bowel disease

5

## Microbial Diversity of 4.17 (+0.22)

Diminished ecosystem diversity indicating dysbiosis and reduced microbial resilience, resilience, compromising metabolic flexibility and immune regulation

6

## Microbial Richness of 213.00 (+0.34)

Decreased species richness reflecting impoverished gut microbiome with limited limited functional capacity and vulnerability to pathogenic colonization

# SIBO Test Findings, Ms E

## Breath Test Results Interpretation

Testing revealed methane of 17ppm at 140 minutes and so and so meets diagnostic criteria for Intestinal Methanogen Methanogen Overgrowth IMO.

Hydrogen levels were 16ppm at baseline rising to 24ppm at 100 minutes not diagnostic for H2 SIBO.

Collectively CH4 and H2 met diagnostic criteria for SIBO at SIBO at >15ppm.

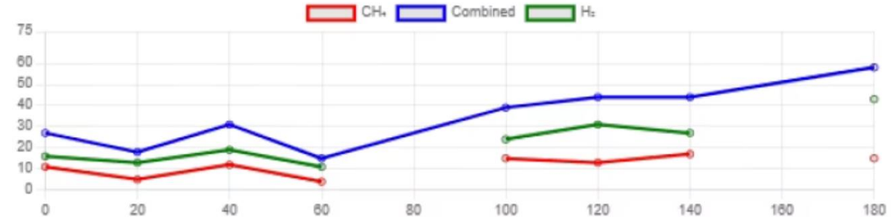
**Summary Report of Hydrogen and Methane Breath Analysis with Carbon Dioxide Correction**

| Gas Analysed | Patient Result 0-100 mins | Expected difference 0-100 mins |
|---|---|---|
| Increase in Hydrogen ($H_2$) | 13 | < 20 |
| Increase in Methane ($CH_4$) | 11 | < 12 |
| Increase in Combined $H_2$ & $CH_4$ | 24 | < 15 |

| Analysis of data suggests: |
|---|
| Results indicate small intestinal bacterial overgrowth |
| Intestinal Methanogenic Overgrowth (IMO) |

Small Intestinal Bacterial Overgrowth (SIBO) Hydrogen and Methane Breath after Lactulose consumption

| Number | Expected Location | Interval | ppm $H_2$ | ppm $CH_4$ | Combined | % $CO_2$ | $fCO_2$[1] |
|---|---|---|---|---|---|---|---|
| 1 | Small Intestine | Baseline | 16 | 11 | 27 | 3.4 | 1.6 |
| 2 | | 20 min | 13 | 5 | 18 | 3.4 | 1.6 |
| 3 | | 40 min | 19 | 12 | 31 | 3.2 | 1.7 |
| 4 | | 60 min | 11 | 4 | 15 | 3.0 | 1.8 |
| 5 | | 80 min | | | | 0.0 | 1.7 |
| 6 | | 100 min | 24 | 15 | 39 | 3.2 | 1.7 |
| 7 | Transition | 120 min | 31 | 13 | 44 | 3.0 | 1.8 |
| 8 | Large Intestine | 140 min | 27 | 17 | 44 | 3.3 | 1.7 |
| 9 | | 160 min | | | | o.o | NaN |
| 10 | | 180 min | 43 | 15 | 58 | 3.2 | 1.7 |



# KBMO FIT Test Findings: The Game-Changer

| List of Restricted Foods: | |
|---|---|
| 4+ Reactions: | Wheat, Gliadin<br>Wheat, Gluten<br>Wheat, Whole |
| 3+ Reactions: | Potato, Sweet |
| 2+ Reactions: | Egg Yolk<br>Egg White<br>Turmeric<br>Yeast, Brewer's<br>Shrimp |

| Gut Barrier Panel | | | | |
|---|---|---|---|---|
| | IgG1-4+C3d | | IgA1-2 | |
| | | Cut off | | Cut off |
| Candida | Positive |  | Negative |  |
| Zonulin | Positive |  | Negative |  |
| Occludin | Positive |  | Negative |  |
| LPS | Negative |  | Negative |  |

# Phase 1: Intestinal Barrier Healing

### Remove Reactive Foods

Eliminate all identified food antigens based on FIT results, with particular focus on strict gluten avoidance and removal of moderate reactors

### Support Mucosal Immunity

Implement targeted supplementation to restore secretory IgA function and strengthen mucosal immune defences at epithelial surfaces

### Reduce Inflammatory Load

Deploy anti-inflammatory nutrients and botanicals to decrease systemic inflammatory burden and support and support tissue repair processes

### Introduce Digestive Support

Restore digestive capacity through betaine HCl, digestive enzymes, and bile support to address hypochlorhydria hypochlorhydria and maldigestion

# Immediate Patient Action

> *Upon receiving her FIT test results, the patient implemented complete complete gluten removal before the follow-up consultation, demonstrating remarkable self-efficacy and engagement with her with her health journey.*

This autonomous action, driven by clear objective evidence, exemplifies the power of comprehensive testing to catalyse behavioural change. The patient's immediate response demonstrated understanding of the gut-immune-pain connection and commitment to addressing root causes rather than solely managing symptoms.

Her proactive approach set the foundation for the remarkable clinical clinical improvements that followed over subsequent weeks.

# Phase 2: Targeted Antimicrobial Intervention

## Primary Objectives

Implementation of antimicrobials and nutrients specifically targeting candida overgrowth and dysbiotic bacterial populations identified through comprehensive stool analysis.

## Predictable Die-Off Response

Week three saw the onset of Herxheimer reactions as microbial cell wall components released endotoxins. These manifested as transient itching, fatigue intensification, and mild headaches.

## Patient Support Strategies

Managed die-off symptoms through enhanced detoxification support, hydration, fibre, and reassurance that symptoms indicate therapeutic progress.

# Clinical Outcomes at Four Months

**75%**

### Global Improvement

Overall symptom reduction across across all presenting complaints complaints

**100%**

### Sleep Restoration

Complete normalisation of sleep quality and architecture

**80%**

### Pain Reduction

Significant decrease in widespread widespread pain intensity and frequency

**85%**

### IBS Resolution

Marked improvement in bowel function and abdominal symptoms symptoms

**90%**

### Cognitive Function

Near-complete resolution of brain brain fog and mental clarity restoration

**70%**

### Energy & Hair

Substantial energy improvement improvement and visible hair regrowth

# Three-Week Response: Rapid Clinical Improvement

1

**Pain Reduction**

Significant decrease in knee pain and abdominal discomfort, indicating reduced systemic and visceral inflammation

2

**Sleep Restoration**

Rapid normalisation of sleep architecture with improved sleep onset, maintenance, and morning refreshment

3

**Inflammatory Weight Loss**

3–4 kg reduction representing resolution of inflammatory inflammatory oedema rather than fat or lean tissue loss loss

4

**Hair Regrowth Initiated**

Early signs of hair regrowth indicating improved nutrient nutrient status and reduced inflammatory burden on follicles follicles

5

**Appetite Normalisation**

Restoration of healthy appetite signals suggesting vagal nerve vagal nerve function recovery and improved gut-brain communication

# Phase 3: Microbiota Restoration & Resilience

## Spore-Based Probiotics

Introduce resilient Bacillus species to re-establish beneficial bacterial populations and competitive exclusion mechanisms

## Saccharomyces Boulardii

Deploy beneficial yeast for ongoing antifungal support and immune modulation, particularly targeting residual candida

## PHGG Prebiotic Support

Provide partially hydrolysed guar gum to selectively feed beneficial bacteria without exacerbating SIBO tendencies

## Butyrate Supplementation

Supply direct butyrate to support colonocyte metabolism, enhance barrier function, and promote anti-inflammatory signalling

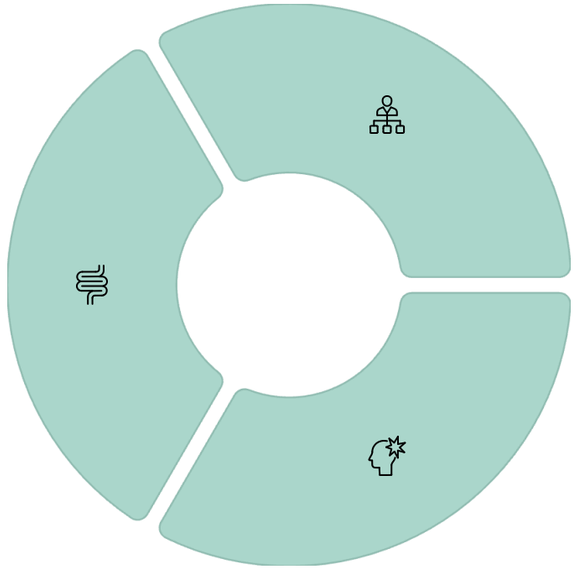
## Dietary Diversification

Gradually reintroduce diverse plant foods to build long-term microbiome resilience and metabolic flexibility

# Root Cause Hypotheses

## Gut Dysfunction

- SIBO contributing to visceral hypersensitivity and pain amplification
- Increased intestinal permeability enabling immune activation activation
- Food sensitivities driving systemic inflammatory cascades
- Hypochlorhydria causing maldigestion and downstream dysbiosis dysbiosis



## Immune Activation

- Chronic low-grade inflammation - antigenic exposure
- Impaired SIgA compromising mucosal defence
- Cytokine production driving central sensitisation
- Loss of oral tolerance mechanisms

## Pain Amplification

- Peripheral inflammation sensitising central pain pathways
- Viscero-somatic convergence amplifying widespread pain
- Neuroimmune interactions perpetuating pain cycles
- Gut-brain axis dysfunction maintaining sensitisation

# Summary - Why FIT Testing Was Decisive

### Identified True Inflammatory Trigger

Revealed gluten as the primary driver rather than focusing exclusively on exclusively on SIBO or dysbiosis

### Explained Multi-System Symptoms

Connected widespread pain, neurological symptoms, and gut dysfunction through unified mechanism

### Validated Patient Experience

Provided objective confirmation of subjective symptoms, empowering patient engagement and adherence

### Prevented Treatment Misdirection

Avoided exclusive SIBO focus that would have missed the primary primary pathogenic mechanism

# Thank you for listening!

### Questions?

What aspects of this case would you like to explore further?

### Clinical Reflections

How does this case challenge or confirm your current approach to fibromyalgia?

### FIT Testing Experience

How do you currently apply FIT testing in your clinical practice? What barriers or successes have you encountered?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