A FUNCTIONAL DIVE INTO ROOT CAUSES OF AUTOIMMUNITY: THE GUT MICROBIOME'S INFLUENCE ON IMMUNE REGULATION

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KBMO DIAGNOSTICS

About Jo Gamble BA (HONS) DIP CNM IFMCP ABAAHP Fellow ICT

- Trailblazing **Functional Medicine Practitioner** and leading voice in integrative health.
- Began as a behavioral therapist, supporting children with autism and complex needs.
- Inspired by her daughter's diagnosis with multiple auto-immune diseases to explore root causes of illness.
- **First UK graduate of the Institute for Functional Medicine** (2013); earned a **Fellowship in Integrative Cancer** (2013)
- Almost two decades **of clinical expertise**, combining evidence-based science with a "no stone unturned" approach.
- **International lecturer, mentor, and advocate for holistic health**, inspiring better quality of life for practitioners and individuals alike.

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WHAT TO EXPECT

- Autoimmunity doesn't begin with a faulty immune system — it begins with a story
- The gut microbiome: your largest immune organ
- Intestinal permeability, dysbiosis, and the loss of immune tolerance
- The Th17/Treg axis: where balance tips into disease
- Diet, stress, and environmental triggers as root cause drivers
- The KBMO Gut Barrier Panel: testing the spectrum of gut dysfunction
- Translating the science into clinical action

PART 1

SETTING THE SCENE — THE AUTOIMMUNE EPIDEMIC

THE AUTOIMMUNE BURDEN IS ACCELERATING

Autoimmune diseases now affect an estimated 4–5% of the global population, with incidence rising across all developed nations.

Unlike infectious diseases, autoimmunity cannot be explained by genetics alone - the human genome has not changed, but our environment, diet, and microbiome have changed profoundly.

The rise of IBD, systemic lupus erythematosus, rheumatoid arthritis, multiple sclerosis, and type 1 diabetes over recent decades points firmly toward lifestyle and environmental drivers.

Emerging research from 2024–2026 confirms that altered gut microbial communities — characterised by reduced diversity, loss of key protective species, and increased barrier permeability — may constitute the pivotal driver of this escalating prevalence.

4-5%

of the global population is now affected by autoimmune diseases, with incidence rising rapidly.

AUTOIMMUNITY IS NOT A SINGLE EVENT — IT IS A PROCESS

STAGE 1

Silent Autoimmunity

Elevated antibodies.
No symptoms.
No tissue loss.

STAGE 2

Autoimmune Reactivity

Elevated antibodies.
Symptoms present.
No measurable tissue destruction.

STAGE 3

Autoimmune Disease

Elevated antibodies.
Symptoms present.
Measurable tissue destruction.

The **window of opportunity** lies in Stages 1 and 2 — before irreversible tissue damage occurs. Functional medicine intervenes here, identifying root causes before the immune system reaches the point of no return.

70%

of the body's immune cells reside in the gut, concentrated in the GALT and lamina propria.

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THE GUT IS THE LARGEST IMMUNE ORGAN IN THE BODY

The intestinal epithelium — a single layer of cells — forms the critical interface between the external environment and the internal immune system.

This layer must simultaneously allow nutrient absorption while acting as a selective barrier against pathogens, toxins, and undigested food antigens.

The gut microbiome, with a gene pool approximately 150 times larger than the human genome, sits at the heart of this interface, educating, calibrating, and regulating immune responses throughout the body.

Citation: Wang X et al. Front Immunol. 2024. DOI: 10.3389/fimmu.2024.1365554

PART 2

THE MICROBIOME - DIVERSITY, DYSBIOSIS, AND IMMUNE CONSEQUENCES

MICROBIAL DIVERSITY IS THE FOUNDATION OF IMMUNE HEALTH

A healthy gut microbiome is characterised above all by diversity — a rich ecological community of bacteria, fungi, and viruses that collectively maintain metabolic function, barrier integrity, and immune homeostasis.

The microbiome produces short-chain fatty acids (SCFAs), modulates T cell differentiation, shapes antibody production, and regulates the innate immune response through pattern recognition receptors.

When diversity is lost — through antibiotics, ultra-processed diets, chronic stress, or environmental toxins — the ecosystem collapses, and with it, immune regulation.

Every autoimmune disease studied to date shows a consistent signature:

REDUCED MICROBIAL DIVERSITY

DYSBIOSIS HAS A DISTINCT SIGNATURE IN AUTOIMMUNE DISEASE

BACTERIA	ROLE	STATUS IN AUTOIMMUNITY
Faecalibacterium prausnitzii	Butyrate producer; anti-inflammatory	**Consistently depleted**
Akkermansia muciniphila	Mucin layer integrity; gut barrier	**Reduced in RA, MS, SLE**
Bacteroides fragilis	PSA → Treg induction; IL-10	**Depleted**
Bifidobacterium spp.	Immune education; Treg support	**Reduced**
Prevotella copri	Molecular mimicry with citrullinated proteins	**Elevated in RA**
Ruminococcus gnavus	Pro-inflammatory; LPS-like activity	**Elevated in SLE flares**

Citation: Zeng L et al. Research (Wash DC). 2026 | Han EJ et al. Clin Rev Allergy Immunol. 2025

PART 3

THE LEAKY GUT - GATEWAY TO IMMUNE DYSREGULATION

INTESTINAL PERMEABILITY IS THE BRIDGE BETWEEN DYSBIOSIS AND AUTOIMMUNITY

The integrity of the intestinal barrier depends on tight junction proteins - **occludin, claudin, and ZO-1** - which seal the spaces between epithelial cells.

Zonulin, identified by Dr Alessio Fasano, is the primary physiological regulator of these tight junctions. When zonulin is upregulated - by gluten, dysbiosis, stress, or infection - tight junctions open.

This allows bacterial fragments, undigested food antigens, and LPS to translocate into the bloodstream. This is the exact moment the immune system shifts from tolerance to reactivity.

Critically, elevated intestinal permeability has been shown to:

PRECEDE THE CLINICAL ONSET OF AUTOIMMUNE DISEASE

Citation: Mu Q et al. Front Immunol. 2017 | Fasano A. Clin Gastroenterol Hepatol. 2012

LPS: THE MOLECULAR MATCH THAT LIGHTS THE AUTOIMMUNE FIRE

Lipopolysaccharide (LPS) is a structural component of the outer membrane of gram-negative bacteria. In a healthy gut, LPS remains confined to the intestinal lumen.

When the gut barrier is compromised, LPS translocates into systemic circulation - a state known as **metabolic endotoxaemia**.

Even low-grade, chronic elevations of circulating LPS activate TLR4 receptors, triggering NF-κB signalling and a cascade of pro-inflammatory cytokines. This chronic, low-grade inflammatory state is now recognised as a central driver of autoimmune progression, metabolic disease, and neuroinflammation.

THE CYTOKINE CASCADE

LPS translocation triggers the release of:

TNF-α
IL-1β
IL-6

Citation: Candelli M et al. Int J Mol Sci. 2021 | Zeng L et al. 2026

THE KBMO GUT BARRIER PANEL: TESTING THE SPECTRUM OF GUT DYSFUNCTION

KBMO's Gut Barrier Panel recognises that leaky gut occurs across a spectrum — from early dysbiosis to frank intestinal permeability. Four gatekeeper markers are measured, each assessed via **IgG 1–4/C3d** and **IgA 1 and 2**:

MARKER	CLINICAL SIGNIFICANCE
CANDIDA	Dysbiosis precursor; early warning signal of gut ecology disruption
ZONULIN	Tight junction regulation; elevated = compromised barrier function
OCCLUDIN	Tight junction structural integrity; elevated antibodies = breakdown
LPS	Gram-negative bacterial translocation; elevated = leaky gut + endotoxaemia

The panel removes the guesswork. Providers can now **test, not guess** — identifying the precise stage of gut dysfunction and tailoring treatment accordingly.

PART 4

THE TH17/TREG AXIS - WHERE THE IMMUNE SYSTEM LOSES ITS BALANCE

THE BALANCE BETWEEN TOLERANCE AND ATTACK

At the heart of autoimmune pathogenesis lies a critical imbalance between two T cell populations.

In health, these populations exist in **dynamic equilibrium**.

In autoimmunity, this balance tips: **Tregs are depleted or dysfunctional, and Th17 cells dominate** - driving chronic inflammation, tissue damage, and loss of self-tolerance.

REGULATORY T CELLS (TREGS)

Defined by the transcription factor Foxp3. Maintain peripheral tolerance, suppress excessive immune activation, and prevent the immune system from attacking self-tissue.

TH17 CELLS

Drive pro-inflammatory responses, producing IL-17A, IL-17F, and IL-22. Essential for defence against extracellular pathogens.

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Citation: Dominguez-Villar M, Hafler DA. Nature Reviews Immunology | Kim CH. Nature Immunol. 2023

THE MICROBIOME DIRECTLY PROGRAMMES THE TH17/TREG BALANCE

The gut microbiome is not a passive bystander — it actively programmes T cell differentiation.

Butyrate-producing bacteria (Faecalibacterium prausnitzii, Roseburia, Clostridium clusters IV and XIVa) produce butyrate, which acts as an HDAC inhibitor, driving Foxp3 expression and Treg induction. Bacteroides fragilis polysaccharide A (PSA) stimulates IL-10 production by CD4+ Tregs.

Conversely, **segmented filamentous bacteria (SFB)** and dysbiotic species drive excessive RORγt+ Th17 activation.

When butyrate-producing species are lost — as consistently observed in autoimmune dysbiosis — the Treg programme collapses, and Th17-driven inflammation is unleashed.

TREG INDUCTION

Driven by butyrate producers and B. fragilis PSA. Promotes immune tolerance and suppresses inflammation.

TH17 ACTIVATION

Driven by dysbiotic species and SFB. Promotes pro-inflammatory responses and tissue damage.

Citation: Wang X et al. Front Immunol. 2024 | Kim CH. Nature Immunol. 2023 | Frontiers in Immunology 2025

MOLECULAR MIMICRY: WHEN THE IMMUNE SYSTEM CONFUSES SELF WITH NON-SELF

Molecular mimicry occurs when a microbial antigen shares sufficient structural similarity with a host self-antigen to trigger cross-reactive immune responses.

The immune system, primed against the pathogen, inadvertently attacks the body's own tissue. This mechanism is implicated across multiple autoimmune conditions.

The gut - as the primary site of microbial exposure - is where this process begins.

RHEUMATOID ARTHRITIS

Prevotella copri peptides mimic citrullinated proteins → anti-CCP antibodies

HASHIMOTO'S THYROIDITIS

Yersinia enterocolitica and H. pylori cross-react with thyroid antigens

MULTIPLE SCLEROSIS

HHV-6 U24 protein mimics myelin basic protein

TYPE 1 DIABETES

Cow's milk albumin cross-reacts with islet cell antigen-1

Citation: Zeng L et al. 2026 | Clinic Rev Allerg Immunol. 2016

PART 5

ROOT CAUSE DRIVERS - DIET, STRESS, AND THE ENVIRONMENT

FOOD IS INFORMATION - AND SOMETIMES THE WRONG MESSAGE

The modern Western diet - characterised by ultra-processed foods, refined sugars, seed oils, and low dietary fibre - is profoundly destructive to microbial diversity.

Dietary fibre is the primary substrate for SCFA production; its absence starves butyrate-producing bacteria and collapses immune regulation.

Gluten activates zonulin, opening tight junctions and increasing antigen translocation. **Dairy proteins** (particularly bovine albumin) cross-react with islet cell antigens. **Lectins** in grains and legumes may adversely influence enterocyte and lymphocyte structure and function.

THE KBMO FIT TEST

Measuring IgG 1–4 and C3d against 176 foods.

Identifies the specific dietary antigens driving chronic immune activation in each individual patient.

Citation: Cordain L et al. Br J Nutr. 2000 | Whalen KA et al. J Nutr. 2016

CHRONIC STRESS IS A DIRECT DRIVER OF GUT PERMEABILITY

The gut-brain-immune axis is bidirectional and profoundly sensitive to psychological stress. Chronic stress activates the HPA axis, elevating **cortisol** and **corticotropin-releasing hormone (CRH)**.

CRH directly increases intestinal permeability; cortisol alters gut motility, reduces secretory IgA, and shifts microbiota composition - depleting Lactobacillus and Bifidobacterium species.

Mast cell activation, triggered by stress, further disrupts tight junctions. The result: a stress-induced leaky gut that allows LPS and food antigens to enter systemic circulation, activating the immune system in a cycle of chronic, low-grade inflammation.

The Clinical Reality:

ADDRESSING THE HPA AXIS IS NOT OPTIONAL IN AUTOIMMUNE MANAGEMENT - IT IS ESSENTIAL.

Citation: Madison AA et al. Biol Psychiatry. 2024 | Beurel E et al. Gut Microbes. 2024

ENVIRONMENTAL TRIGGERS COMPLETE THE AUTOIMMUNE TRIAD

Autoimmunity requires three converging factors. No single factor is sufficient alone to drive the disease process.

The Autoimmune Triad:

Genetic Susceptibility

Compromised Gut Barrier

Environmental Trigger

INFECTIONS

EBV, H. pylori, Campylobacter jejuni, Candida - driving molecular mimicry and epitope spreading.

XENOBIOTICS

Heavy metals (mercury, lead), pesticides, BPA, pharmaceutical agents - alter DNA methylation, induce Th17 activation.

MEDICATIONS

NSAIDs, PPIs, antibiotics - directly increase gut permeability and disrupt the microbiome ecosystem.

EARLY LIFE FACTORS

C-section delivery, formula feeding, antibiotic exposure in infancy - suboptimal microbiome seeding with lifelong immune consequences.

Citation: Clinic Rev Allerg Immunol. 2016 | Zeng L et al. 2026

PART 6

THE FUNCTIONAL MEDICINE FRAMEWORK

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FROM ROOT CAUSE TO CLINICAL ACTION

THE 5R FRAMEWORK: A SYSTEMS APPROACH TO GUT RESTORATION

STEP	ACTION	CLINICAL TOOLS
REMOVE	Eliminate dietary triggers, pathogens, dysbiotic organisms	KBMO FIT Test , elimination diet, antimicrobials
REPLACE	Restore digestive capacity	Digestive enzymes, HCl, bile acids
REINOCULATE	Restore beneficial microbiota	Lactobacillus, Bifidobacterium, Akkermansia, fermented foods
REPAIR	Heal the gut barrier	L-glutamine, zinc carnosine, collagen, butyrate, DGL
REBALANCE	Address lifestyle drivers	Stress management, sleep, HPA axis support, exercise

The **KBMO Gut Barrier Panel** guides the depth and sequencing of this protocol - identifying whether the patient is at the dysbiosis stage (Candida), permeability stage (Zonulin/Occludin), or systemic endotoxaemia stage (LPS).

THE FIT TEST: IDENTIFYING THE FOOD-IMMUNE BURDEN

The KBMO Food Inflammation Test (FIT) measures **IgG 1–4 and C3d immune complexes** against 176 foods and additives.

The inclusion of C3d - a complement activation marker - amplifies the inflammatory signal and identifies foods driving a more significant immune response than IgG alone would reveal.

Chronic food-driven immune activation maintains a state of intestinal inflammation, perpetuates gut permeability, and sustains the antigen load that fuels autoimmune reactivity.

Removing identified trigger foods reduces the immune burden, allows the gut barrier to heal, and creates the conditions for immune tolerance to be restored.

The Clinical Reality:

THIS IS NOT SYMPTOM MANAGEMENT — THIS IS ROOT CAUSE RESOLUTION.

EMERGING THERAPIES: THE MICROBIOME AS MEDICINE

The most compelling evidence from 2024–2026 research positions the microbiome not merely as a target of intervention but as a therapeutic agent in its own right.

The microbiome is not just a target - it is the therapy.

FECAL MICROBIOTA TRANSPLANTATION (FMT)

Demonstrated early clinical promise in type 1 diabetes, SLE, and multiple sclerosis - restoring microbial diversity, increasing Treg populations, and reducing inflammatory markers.

PRECISION PROBIOTICS

Akkermansia muciniphila and Faecalibacterium prausnitzii are emerging as next-generation probiotics, with mechanistic evidence for gut barrier restoration and Th17/Treg rebalancing.

MEDITERRANEAN DIET

Rich in polyphenols, diverse plant fibres, and fermented foods -inversely associated with autoimmune risk and directly increases SCFA-producing bacterial populations.

Citation: Han EJ et al. Clin Rev Allergy Immunol. 2025 | Seida I et al. Autoimmun Rev. 2024 | Zeng L et al. 2026

THE CLINICAL PICTURE: CONNECTING THE DOTS

Every patient with an autoimmune condition carries a unique story - but the underlying biology follows a recognisable pattern.

Reduced microbial diversity leads to depleted butyrate producers, which collapses Treg induction and tips the Th17/Treg axis toward inflammation. Dysbiosis drives intestinal permeability, allowing LPS, food antigens, and microbial peptides to enter systemic circulation.

Molecular mimicry, epitope spreading, and chronic TLR activation follow. **Stress amplifies every step.**

The result is a self-perpetuating cycle of immune dysregulation - one that conventional medicine manages with immunosuppression, but functional medicine seeks to interrupt at its root.

DIVERSITY LOSS 01

Depleted butyrate producers collapse Treg induction.

PERMEABILITY 02

Dysbiosis opens tight junctions; LPS & antigens translocate.

IMMUNE DYSREGULATION 03

Th17 dominance and chronic TLR activation take over.

AUTOIMMUNITY 04

Molecular mimicry and epitope spreading drive tissue attack.

Case study

Meet Sarah.

Sarah is a 32 year old professional woman who presented to me with recurrent and persistent mouth ulcers and a history of an anal fissure.

She had a great deal of emotional trauma from childhood, albeit her awareness of trauma was good, it was clear there was a lot of internalisation

She had seen a functional practitioner in the past who had used l-glutamine with no improvement and was referred to me from a functional dentist who had ruled out a dental route to her symptoms.

There is a significant family history of auto immunity with her brother being hospitalized for many months with an unknown auto immune disease and her sister being coeliac.

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Case study

Sarah had seen rheumatologists, immunologists, gastroenterologist and no diagnosis had been made.

Whilst coeliac was ruled out, I did suggest a gluten free diet along with bone broth addition.

She had previously done a stool test which showed low diversity but nothing else significant.

Her diet was good, albeit she ate gluten again.

No alcohol.

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Supplement	Where to Buy	Dosage	Additional Information
Theanine	Natural Dispensary: TR-SA508-HI	1 twice daily	To calm nervous system
Mucin	Natural Dispensary: INV-MUC90C	3 capsules daily e.g. 2 morning 1 night time	Has been formulated for the mucosal layer of the gastro intestinal Tract
Proflora	https://www.amritanutrition.co.uk/products/proflora-4r-30-vcapsules	1 daily	To support gut restoration
Repairvite	https://www.amritanutrition.co.uk/products/repairvite-k-60-171-3g	1 scoop daily	To support gut lining

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Immune Status (MeGeMIT)

Leucocytes	5.5		4 - 11	/nl
Lymphocytes	1600		1200 - 2300	/µl
T3 T-Lymph abs	1317		1000 - 1700	/µl
T3 T-Lymph rel	↑ 82		67 - 80	%Lympho
T4 T-Helper abs	689		500 - 1000	/µl
T4 T-Helper rel	43		35 - 45	%Lympho
CD8-cells abs	476		350 - 800	/µl
CD8 cells rel	30		20 - 38	%Lympho
CD4/CD8 Ratio	1.40		1.2 - 2.2	Ratio
T8 cytotox abs	423		260 - 550	/µl
T8 cytotox rel	↑ 26.4		15 - 25	%Lympho
T8 suppr abs	63		< 300	/µl
T8 suppr rel	4.0		< 13	%Lympho
RatioT8zyt/T8sup	↑ 6.68		1.5 - 4	Ratio
T3 activated abs	81		< 170	/µl
T3 activated rel	5		< 10	%Lympho
NK1 CD8-CD57+abs	51		< 160	/µl
NK1 CD8-CD57+rel	3		< 8	%Lympho
NK2 3+16+56+ abs	68		< 120	/µl
NK2 3+16+56+rel	5		< 5	%Lympho
NK3 3-16+56+abs	95		< 430	/µl
NK3 3-16+56+rel	6		< 19	%Lympho
B-cells abs	181		140 - 305	/µl
B-cells rel.	11		7 - 14	%Lympho
CD5+B-cells abs	19			/µl
CD5+B-cells rel	10		< 30	%B-Zell.
CD80+Bcells abs	15		4 - 27	/µl
CD80+Bcells rel	8		3 - 10	%B-Zell.
T cells regulatory	8		5 - 10	%CD4
TH17 cells abs	139			/µl
TH17 cells rel	↑ 20		5 - 17	%CD4

Serology IFT

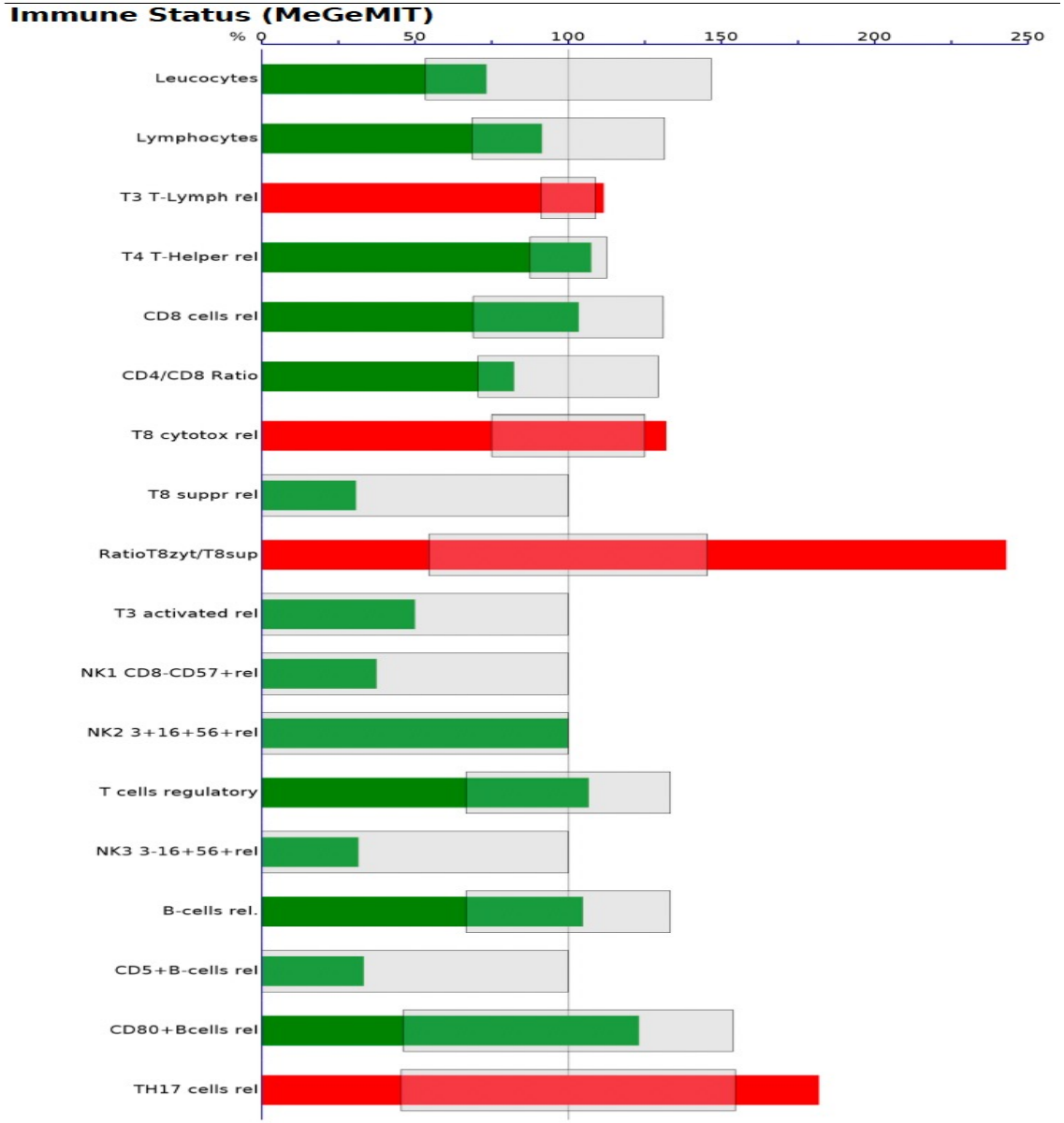
EBV antibody IFT

VCA-IgG	↑ 1:640		< 1:80	Titer
VCA-IgM	<1:10		< 1:10	Titer
Early IgG	↑ 1:40		< 1:20	Titer
EBNA IgG	↑ 1:40		< 1:20	Titer

VZV antibody IFT

VZV-IgG	↑ 1:640		< 1:40	Titer
VZV-IgA	<1:40		< 1:40	Titer

CMV antibody analogous to IFT



Supplement	Where to Buy	Dosage	Additional Information
Theanine	Natural Dispensary: TR-SA508-HI	1 twice daily	To calm nervous system
Mucin	Natural Dispensary: INV-MUC90C	3 capsules daily e.g. 2 morning 1 night time	Has been formulated for the mucosal layer of the gastro intestinal Tract
Proflora	https://www.amritanutrition.co.uk/products/proflora-4r-30-vcapsules	1 daily	To support gut restoration
Repairvite	https://www.amritanutrition.co.uk/products/repairvite-k-60-171-3g	1 scoop daily	To support gut lining
Mico-rei	Natural dispensary: HDT-416	2 daily	To support immune regulation
Immuncare	https://www.amritanutrition.co.uk/products/immucare-i-180-capsules	3 daily	To support immune regualation
Monolaurin	Natural dispensary: DFH-MLA120-INV	1 twice daily	To lower viral load
Cat's claw	https://naturaldispensary.co.uk/products/Cat_s_Claw_Capsules_100_s-3943-0.html	1 twice daily	To lower viral load

TEST	RESULTS			
Array 12 - Pathogen-Associated Immune Reactivity Screen **	**IN RANGE** (Normal)	**EQUIVOCAL***	**OUT OF RANGE**	**REFERENCE RANGE** (ELISA Index)
Porphyromonas gingivalis	<0.40			0.2 - 1.41
Streptococcus mutans	<0.40			0.4 - 1.91
Helicobacter pylori	<0.50			0.5 - 1.91
Campylobacter jejuni	0.25			0.5 - 2.41
Yersinia enterocolitica	0.20			0.2 - 1.31
Clostridium difficile	<0.20			0.2 - 1.31
Candida albicans	0.27			0.0 - 1.41
Rotavirus	<0.30			0.0 - 2.61
Entamoeba histolytica	<0.20			0.2 - 1.91
Giardia lamblia	0.26			0.2 - 1.61
Cryptosporidium	0.20			0.4 - 2.61
Blastocystis hominis	0.48			0.1 - 1.61
Human + Chlamydia HSP-60	<0.50			1.0 - 2.61
Chlamydias	0.19			0.0 - 1.81
Streptozymes	1.46			0.0 - 2.61
Streptococcal M Protein			1.93	0.1 - 1.41
Mycoplasmas	<0.20			0.2 - 1.81
Acinetobacter	0.51			0.3 - 2.21
Klebsiella	<0.30			0.0 - 1.71
Mycobacterium avium	0.21			0.2 - 1.51
Aspergillus	0.27			0.2 - 1.11
Penicillium	1.43			0.0 - 1.91
Stachybotrys chartarum	2.09			0.4 - 2.71
Citrullinated EBV	<0.30			0.3 - 1.11
CYP450, mimic Hepatitis C Peptide	0.49			0.1 - 1.71
Cytomegalovirus	<0.40			0.2 - 1.21
Human Herpesvirus-6			2.16	0.2 - 1.41
Borrelia burgdorferi	0.50			0.0 - 1.51
Babesia + Ehrlichia + Bartonella	0.30			0.1 - 0.91

* Reference ranges are calculated based on the mean +2 standard deviations (SD). Results >1 SD and <2 SDs above the mean are considered to be equivocal. An equivocal result

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List of Restricted Foods:	
4+ Reactions:	Brazilnut Spirulina
3+ Reactions:	Vanilla Halibut Wine, Red
2+ Reactions:	Egg Yolk Egg White Millet Black Bean Pinto Bean Yeast, Brewer's Chicken Turkey Oyster

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

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Supplement	Where to Buy	Dosage	Additional Information
Mucin	Natural Dispensary: INV-MUC90C	3 capsules daily e.g. 2 morning 1 night time	Has been formulated for the mucosal layer of the gastro intestinal Tract
Sodium butyrate	https://naturaldispensary.co.uk/products/Sodium_Butyrate_100_s-10015843-0.html	**1 three times daily**	**To support barriers and gut diversity**
Liver support	https://www.amritanutrition.co.uk/products/liver-support-120-capsules	**1 twice daily**	**To support the liver as we clear viruses**

Weeks 1-4:

Neem	https://www.amritanutrition.co.uk/products/melia-supreme-60-capsules-supreme-nutrition-products	**1 twice daily**	**To reduce bacterial and viral load**
Morinda	https://www.amritanutrition.co.uk/products/morinda-supreme-130-capsules-supreme-nutrition-products	**1 twice daily**	**To eliminate bacteria and virus and rebalance immunity**

Weeks 5-8

Houttuynia supreme	https://www.amritanutrition.co.uk/products/houttunyia-supreme-45g	**1 twice daily**	**Anti-viral**
Lauric select	https://www.amritanutrition.co.uk/products/lauric-select-600mg-90-vcapsules	**3 daily**	**To lower virus**

Weeks 9-12:

Vital guard	https://www.amritanutrition.co.uk/products/vital-guard-supreme-160-capsules-supreme-nutrition-products	**2 twice daily**	**Anti-microbial**
Berberine	https://www.amritanutrition.co.uk/products/berberine-select-500mg-120-vcapsules	**3 daily**	**Anti-microbial**

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Supplement	Where to Buy	Dosage	Additional Information
Sodium butyrate	https://naturaldispensary.co.uk/products/Sodium_Butyrate_100_s-10015843-0.html	**1 three times daily**	**To support barriers and gut diversity**
Multimessenger	https://www.amritanutrition.co.uk/products/multimessenger-formerly-transfer-factor-multi-immune-60-capsules	1 twice daily	To support immunity to fight infections
AVP	https://www.amritanutrition.co.uk/products/avp-120-capsules-makewell	2 twice daily	To ongoingly reduce viral load
Renew Gut	https://www.amritanutrition.co.uk/products/renew-gut-120-capsules-researched-nutritionals	4 before bed	To repair mucosal membranes

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KEY TAKEAWAYS

- **Autoimmunity is an ecological disease.** The rising prevalence is driven by environmental, dietary, and microbiome shifts, not genetics alone.
- **The microbiome programmes tolerance.** Depletion of butyrate producers collapses Treg induction, tipping the Th17/Treg axis toward chronic inflammation.
- **Permeability precedes onset.** A compromised gut barrier allows LPS and undigested antigens to translocate, acting as the molecular match that lights the autoimmune fire.
- **Molecular mimicry connects the dots.** Structural similarities between microbial/food antigens and host tissue direct the immune attack against self.
- **Test, don't guess.** The KBMO FIT Test and Gut Barrier Panel provide precise, actionable data to identify the specific root causes driving immune activation.
- **Root cause resolution is possible.** Functional medicine offers a systems-based approach (the 5R

Thank You

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