

BIOME GUIDE PROTOCOL

Metabolic Health Microbiome Protocol

Evidence-based prebiotic, probiotic, and dietary strategies
to support blood sugar, liver health, and metabolic balance

Covers: Metabolic Syndrome | Type 2 Diabetes | NAFLD/MASLD | Insulin Resistance

Based on 16 systematic reviews, meta-analyses, and RCTs from PubMed | March 2026

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What This Protocol Addresses

Metabolic syndrome is a cluster of interconnected conditions that dramatically increase your risk of heart disease, stroke, and type 2 diabetes. It includes insulin resistance, elevated blood sugar, abdominal obesity, high triglycerides, and low HDL cholesterol. An estimated 1 in 3 adults in the U.S. meets the criteria for metabolic syndrome, and many more are on the trajectory toward it.

This protocol targets the **gut-metabolic axis**, the communication network between your intestinal microbiome and the metabolic processes that control blood sugar, fat storage, inflammation, and liver function. Research now shows that specific patterns of gut dysbiosis are not just associated with metabolic disease but actively drive it through at least five distinct biological pathways.

Five Pathways from Gut Dysbiosis to Metabolic Disease

Pathway	How It Works	Metabolic Impact
Metabolic Endotoxemia (LPS)	Gram-negative bacteria overgrow, increasing circulating LPS. LPS crosses a leaky gut barrier into portal circulation.	TLR4-mediated chronic inflammation in liver, fat, and muscle tissue. Drives insulin resistance and NAFLD progression.
SCFA Deficiency	Depleted butyrate producers (Faecalibacterium, Roseburia) reduce colonic butyrate levels.	Impaired GLP-1 and PYY secretion from L-cells. Reduced insulin release, increased appetite, weakened gut barrier.
Bile Acid Dysregulation	Altered bacterial BSH activity disrupts the primary-to-secondary bile acid ratio.	Impaired FXR and TGR5 receptor signaling. Disrupted hepatic glucose production and lipid metabolism.
BCAA Overproduction	Enriched Prevotella copri and Bacteroides vulgatus increase branched-chain amino acid biosynthesis.	Elevated serum BCAAs activate mTOR pathway, promoting insulin resistance.
TMAO Pathway	Microbial metabolism of choline and L-carnitine produces TMA, oxidized to TMAO in the liver.	Elevated TMAO linked to cardiovascular risk, atherosclerosis, and impaired glucose tolerance.

Why this matters for you: These five pathways explain why metabolic syndrome, type 2 diabetes, and fatty liver disease so often travel together. They share the same upstream microbial trigger: a gut ecosystem depleted of protective, butyrate-producing bacteria and overrun with inflammatory, LPS-producing species. Fixing the gut is not just treating a symptom of metabolic disease. It is addressing a root cause.

Evidence base: This protocol draws on 16 systematic reviews, meta-analyses, and randomized controlled trials published in Nature Medicine, Nature Communications, BMJ Open, Advances in Nutrition, and other leading peer-reviewed journals. Every recommendation includes the specific study, sample size, and measured outcomes.

Your Metabolic Microbial Fingerprint

Across dozens of studies comparing the gut microbiome of people with metabolic syndrome, type 2 diabetes, and NAFLD to healthy controls, a consistent pattern of dysbiosis emerges. This is your metabolic microbial fingerprint: the specific bacterial imbalances that correlate with and contribute to metabolic dysfunction.

Depleted Taxa (Protective Bacteria You Need More Of)

Bacterium	What It Does	Where It Is Depleted
Akkermansia muciniphila	Maintains mucin layer, stimulates GLP-1 for insulin sensitization, reduces endotoxemia. The protein Amuc_1100 on its outer membrane activates TLR2, improving metabolic signaling.	T2D, MetS, obesity, NAFLD
Faecalibacterium prausnitzii	Major butyrate producer. Anti-inflammatory via IL-10 induction and NF-kB suppression. Butyrate feeds colonocytes and strengthens tight junctions.	T2D, NAFLD, MetS
Roseburia intestinalis	Butyrate producer. SCFA-mediated insulin signaling. Negatively correlated with fasting glucose in human studies.	T2D, MetS
Bifidobacterium spp.	Gut barrier maintenance. Produces acetate and lactate that cross-feed butyrate producers. Competes against pathogens for colonization.	T2D, NAFLD, MetS
Coprococcus spp.	SCFA producer with anti-inflammatory properties. Part of the Ruminococcaceae family consistently depleted in liver disease.	NAFLD
Eubacterium rectale	Butyrate producer, primary fermenter of resistant starch alongside Ruminococcus bromii.	T2D

Enriched Taxa (Inflammatory Bacteria to Reduce)

Bacterium	Why It Is Harmful	Where Enriched
Escherichia / Shigella	Major LPS producers. Drive metabolic endotoxemia and TLR4-mediated systemic inflammation. Directly contribute to insulin resistance.	T2D, NAFLD
Fusobacterium	Pro-inflammatory, degrades mucin layer, disrupts gut barrier. Enriched as protective bacteria decline.	T2D, NAFLD
Ruminococcus (select spp.)	Some species degrade the mucin layer, increasing permeability. Context-dependent: not all Ruminococcus are harmful.	T2D

NAFLD-Specific Finding

A meta-analysis of 54 studies (n=8,894) found that NAFLD patients have significantly reduced alpha diversity (Shannon index: SMD = -0.36, $p < 0.001$; Chao1: SMD = -0.42, $p = 0.001$), with specific depletion of the Ruminococcaceae family and Coprococcus, and enrichment of Fusobacterium and Escherichia. This pattern worsens with disease progression from simple steatosis to NASH.

Prebiotic Strategy: Feeding Your Depleted Bacteria

Prebiotics are the non-digestible fibers that selectively feed beneficial gut bacteria. For metabolic health, the prebiotic strategy is designed to replenish the specific taxa identified in the microbial fingerprint above. Each prebiotic targets different bacterial populations, which is why diversity of fiber sources matters more than any single supplement.

Prebiotic	Primary Taxa Fed	Metabolic Evidence	Dose
Resistant Starch (Type 2/3)	Roseburia, E. rectale, R. bromii, Faecalibacterium	Meta-analysis (13 RCTs, n=428): fasting insulin SMD -0.72 (p=0.001), FBG SMD -0.26 (p=0.035), HbA1c SMD -0.43 (p=0.005), LDL SMD -0.35 (p=0.008)	Start 10g/day, titrate to 20-30g/day
Inulin / FOS	Bifidobacterium (strongest), Faecalibacterium, Lactobacillus	Highly bifidogenic. Fermentation produces acetate/lactate that cross-feed butyrate producers.	Start 3-5g/day, titrate to 10-15g/day
Polyphenols	Akkermansia muciniphila	Ellagitannins and proanthocyanidins selectively increase Akkermansia. Berberine (a plant alkaloid) increases Akkermansia in both animal and human studies.	Pomegranate, cranberry, blueberry, green tea daily
GOS	Bifidobacterium (strongest bifidogenic), Lactobacillus	Highly selective for Bifidobacterium. Produces minimal gas compared to inulin, making it ideal for sensitive individuals.	2.5-5g/day

The Resistant Starch Advantage: Of all prebiotics studied for metabolic health, resistant starch has the strongest direct evidence for improving insulin sensitivity, blood glucose, and lipid parameters. The easiest way to increase resistant starch intake is to cook and then cool starchy foods: potatoes, rice, and pasta that have been cooked and refrigerated overnight contain 2-3x more resistant starch than freshly cooked versions. Green banana flour (1-2 tablespoons in smoothies) is another concentrated source.

Probiotic Strain Evidence

Unlike many conditions where probiotic evidence is limited to a handful of small trials, metabolic health has an unusually deep evidence base. Multiple large meta-analyses, including one covering 105 RCTs with 6,826 participants, confirm that specific probiotic formulations meaningfully improve glycemic control, liver enzymes, and body composition markers.

Akkermansia muciniphila (Pasteurized)

Study: Depommier et al. (2019), Nature Medicine. Randomized, double-blind, placebo-controlled pilot trial. N=32 completers. Overweight/obese, insulin-resistant adults with metabolic syndrome. 3-month intervention with 10^{10} cells/day.

Outcome	Pasteurized vs. Placebo	P-value
Insulin sensitivity	Improved ~30%	p = 0.002
Fasting insulin	Reduced 34%	p = 0.006
Total cholesterol	Decreased 8.68%	p = 0.02
GGT (liver enzyme)	Reduced ~24%	p = 0.009
Plasma LPS (endotoxemia)	Significantly decreased	p < 0.05
Body weight	Trended down -2.27 kg	p = 0.091 (trend)

Why Pasteurized Outperforms Live

A surprising finding: pasteurized (heat-killed) *Akkermansia* outperformed live bacteria on most endpoints. The reason is the protein Amuc_1100, embedded in the outer membrane. Heat treatment exposes this protein, which then activates TLR2 signaling to improve metabolic parameters. This makes the pasteurized form both more effective and more shelf-stable. *Akkermansia* supplements are now commercially available in pasteurized form (e.g., Pendulum *Akkermansia*).

Multi-Strain Probiotics for Glycemic Control

Key meta-analyses:

Rittiphairoj et al. (2021), Advances in Nutrition: 28 RCTs, n=1,947 T2D patients. Probiotics reduced fasting blood glucose by -12.99 mg/dL (95% CI: -23.55 to -2.42, p=0.016). Greater reductions in patients with FBG >130 mg/dL and those not on insulin therapy. Common effective strains: *L. acidophilus*, *L. casei*, *B. lactis*, *B. longum*, *S. thermophilus*.

Koutnikova et al. (2019), BMJ Open: 105 RCTs, n=6,826. In T2D subgroup: fasting glucose -0.66 mmol/L, HbA1c -0.28%, insulin -1.66 mU/L, HOMA-IR -1.05 (all significant). In overweight subgroup:

body weight -0.94 kg, waist circumference -1.31 cm, visceral fat -6.30 cm². Most effective species: *B. breve*, *B. longum*, *L. acidophilus*, *L. casei*.

Multi-Strain Probiotics for NAFLD / Fatty Liver

Zhou et al. (2023), Medicine: 21 RCTs, n=1,037 NAFLD patients. Treatment duration 8-56 weeks.

Outcome	Effect vs. Placebo	P-value
ALT (liver enzyme)	-8.52 IU/L (95% CI: -12.59 to -4.46)	p < 0.00001
AST (liver enzyme)	-6.82 IU/L (95% CI: -10.16 to -3.49)	p < 0.00001
GGT (liver enzyme)	-5.88 IU/L (95% CI: -6.59 to -5.16)	p < 0.00001
Hepatic steatosis grade	Significantly improved	p < 0.00001
Triglycerides	-17.30 mg/dL	p = 0.009
HDL-C	+3.37 mg/dL	p = 0.02

Duration Matters: The NAFLD meta-analysis found that treatment lasting 12 weeks or more showed superior improvements in ALT, GGT, triglycerides, and glucose compared to shorter courses. Plan for at least 3 months of consistent probiotic supplementation to see meaningful liver enzyme improvements.

Supporting Supplements

These supplements complement the prebiotic and probiotic core of the protocol by addressing specific metabolic pathways. Each has its own evidence base from randomized controlled trials and meta-analyses.

Berberine: The Dual-Action Metabolic Ally

Berberine is a plant alkaloid with a unique dual mechanism: it directly lowers blood glucose AND reshapes the gut microbiome by increasing Akkermansia muciniphila abundance. This makes it the single most synergistic supplement in this protocol.

Clinical evidence: A meta-analysis of 46 RCTs (n=4,158) found berberine significantly improved every major metabolic marker: FPG -0.89 mmol/L, HbA1c -0.75%, fasting insulin -2.05 uIU/mL, HOMA-IR -0.71, total cholesterol -0.64 mmol/L, triglycerides -0.50 mmol/L, LDL -0.86 mmol/L, and HDL +0.17 mmol/L (all p < 0.05).

Microbiome evidence: The PREMOTÉ trial (Nature Communications, n=409, 20 centers) showed berberine + probiotics reduced HbA1c by -1.04% in newly diagnosed T2D patients. Berberine inhibited harmful DCA biotransformation by Ruminococcus bromii and shifted the microbiome toward a more favorable composition.

Dose: 500mg 2-3x daily with meals (most common: 1.5g/day divided). Start at 500mg once daily and increase gradually to minimize GI side effects.

Important Safety Note: Berberine has additive glucose-lowering effects with metformin and other diabetes medications. If you are on prescription diabetes medications, work with your physician to monitor blood glucose when adding berberine, as medication doses may need adjustment. Do not combine berberine with metformin without medical supervision.

Additional Metabolic Support Supplements

Supplement	Evidence	Dose	Notes
Alpha-Lipoic Acid (ALA)	Meta-analysis of 24 RCTs: FBG SMD -0.54 (p=0.003), HbA1c SMD -1.22 (p=0.002), HOMA-IR SMD -0.76 (p<0.001), LDL SMD -0.44 (p=0.008), TG SMD -0.58 (p=0.006)	300-600mg/day	R-alpha-lipoic acid form preferred. May lower blood glucose; monitor if on diabetes meds.
Vitamin D3	Meta-analysis of 39 RCTs (n=2,982): significant reductions in FBG, HbA1c, HOMA-IR, and fasting insulin. Effects strongest in vitamin D-deficient patients.	2,000-4,000 IU/day	Test baseline 25(OH)D levels. Target 40-60 ng/mL. Take with fat-containing meal.

Supplement	Evidence	Dose	Notes
Omega-3 EPA/DHA	Well-established for triglyceride reduction. Supports resolution of hepatic inflammation in NAFLD. Anti-inflammatory via SPM pathway.	2-4g/day combined EPA+DHA	Higher EPA ratio for inflammation. Triglyceride-form fish oil or algae-based.
Chromium Picolinate (conditional)	Meta-analysis of 28 RCTs: FPG -19 mg/dL ($p=0.030$), HbA1c -0.71% ($p=0.004$), HOMA-IR -1.53 ($p<0.001$). Mixed results in non-deficient populations.	200-400 mcg/day	Best reserved for patients with poor glycemic control despite other interventions. Evidence is not universally strong.

Dietary Framework: The Metabolic Mediterranean Approach

The Mediterranean diet is the single best-studied dietary pattern for metabolic health, and the PREDIMED-Plus trial confirmed its benefits specifically in people with metabolic syndrome. What makes it relevant to this protocol is that its metabolic benefits are partly mediated through the gut microbiome: Mediterranean diet adherence increases *Faecalibacterium prausnitzii*, *Roseburia hominis*, *Akkermansia muciniphila*, and genes for butyrate production.

Daily Targets

Food Group	Daily Target	Microbiome / Metabolic Benefit
Extra virgin olive oil	2-4 tablespoons	Oleic acid + polyphenols. Anti-inflammatory, increases <i>Akkermansia</i> .
Non-starchy vegetables	5+ servings	Diverse fiber feeds diverse taxa. Cruciferous vegetables support hepatic detoxification.
Legumes	1 serving (1/2 cup)	Resistant starch + soluble fiber. Among the strongest bifidogenic whole foods.
Nuts and seeds	1 handful (30g)	Polyphenols + fiber. Walnuts specifically increase <i>Roseburia</i> and <i>Bifidobacterium</i> .
Fatty fish	2-3x per week	EPA/DHA for inflammation. Reduces hepatic lipogenesis in NAFLD.
Fermented foods	1-2 servings	Live cultures + postbiotics. Kefir, sauerkraut, kimchi, yogurt.
Whole grains	2-3 servings	Arabinoxylans feed <i>Bifidobacterium</i> and <i>Roseburia</i> . Prefer intact/minimally processed.

Foods to Minimize or Avoid

Ultra-processed foods: Disrupt Firmicutes-to-Bacteroidetes ratio, increase Proteobacteria (the phylum containing *Escherichia/Shigella*). These are the single biggest dietary driver of metabolic dysbiosis.

Added sugars and refined carbohydrates: Feed Enterobacteriaceae, reduce *Bifidobacterium*, and directly spike blood glucose independent of microbiome effects.

Excessive red meat: Increases TMAO production via L-carnitine metabolism. Limit to 1-2 servings per week maximum.

Artificial sweeteners: Emerging evidence that saccharin, sucralose, and aspartame disrupt the gut microbiome and may impair glucose tolerance. Use stevia or monk fruit if a sweetener is needed.

Metabolic Reset Smoothie (Daily)

- 1 tablespoon green banana flour (resistant starch)
- 1/2 cup frozen blueberries (polyphenols for Akkermansia)
- 1/4 cup plain kefir (live cultures)
- 1 tablespoon ground flaxseed (fiber + omega-3 ALA)
- 1/2 teaspoon cinnamon (insulin-sensitizing properties)
- 1 scoop unflavored collagen or plant protein
- 1 cup unsweetened almond milk or water

Blend until smooth. This delivers resistant starch, polyphenols, live cultures, and fiber in one convenient daily dose. Best consumed as breakfast or mid-morning snack to maximize the second-meal effect on blood glucose.

The Two-Phase Protocol

Phase 1: Metabolic Reset (Weeks 1-6)

The first six weeks focus on reducing metabolic endotoxemia, rebuilding depleted beneficial taxa, and establishing the dietary foundation. Start conservatively with prebiotics to avoid GI discomfort, and titrate doses upward as your gut adapts.

Category	Intervention	Details
Prebiotics	Resistant starch	Start 10g/day (1 Tbsp green banana flour), titrate to 20g/day by week 3. Cooled rice/potatoes count toward total.
	GOS	2.5g/day. Gentle and well-tolerated from day 1.
Probiotics	Multi-strain formula	<i>L. acidophilus</i> + <i>L. casei</i> + <i>B. lactis</i> + <i>B. longum</i> . 10-20 billion CFU/day with food.
	<i>Akkermansia muciniphila</i>	Pasteurized, 10 ¹⁰ cells/day if available (e.g., Pendulum brand).
Supplements	Berberine	Start 500mg 1x/day with a meal, increase to 500mg 2x/day by week 2.
	Alpha-lipoic acid	300mg/day.
	Vitamin D3	4,000 IU/day with fat-containing meal. Test baseline 25(OH)D.
	Omega-3	2g EPA+DHA/day.
Diet	Mediterranean foundation	Eliminate ultra-processed foods and added sugars. Emphasize EVOO, vegetables, legumes, fatty fish 2-3x/week.

Phase 2: Metabolic Optimization (Weeks 7-12+)

With the foundation set, Phase 2 deepens microbial diversity, optimizes metabolic biomarkers, and establishes long-term sustainable habits. The prebiotic repertoire expands to include more fiber types, feeding a wider range of beneficial taxa.

Category	Intervention	Details
Prebiotics	Resistant starch	Maintain 20-30g/day from food + supplement sources.
	Add inulin/FOS	Start 3g/day, titrate to 8-10g/day. Chicory root coffee is a convenient source.
	Polyphenol-rich foods	Daily pomegranate, cranberry, blueberries, green tea to boost <i>Akkermansia</i> .

Category	Intervention	Details
Probiotics	Continue multi-strain + Akkermansia	Reassess at 12 weeks. May taper if biomarkers improving and diet is prebiotic-rich.
Supplements	Berberine	Increase to 500mg 3x/day if tolerated and glycemic targets not met.
	Alpha-lipoic acid	Increase to 600mg/day if tolerated.
	Vitamin D3	Adjust based on retest of 25(OH)D. Target 40-60 ng/mL.
	Omega-3	Increase to 3-4g/day if triglycerides remain elevated.
	Chromium (conditional)	200-400 mcg/day chromium picolinate if glycemic targets not met.
Diet	Full Mediterranean + fermented foods	Add kefir, sauerkraut, kimchi (1-2 servings/day). Maximize fiber diversity. Daily Metabolic Reset Smoothie.

Monitoring Your Progress

Metabolic health is one of the most measurable conditions you can track. Unlike many gut-health protocols where progress is subjective, metabolic markers provide objective data to confirm that the protocol is working.

Core Biomarkers (Test Every 3 Months)

Biomarker	What It Measures	Target
HbA1c	Average blood glucose over 2-3 months	< 5.7% (normal), < 6.5% (pre-diabetic range)
Fasting glucose	Morning blood sugar after 8-12 hour fast	< 100 mg/dL (optimal < 90)
Fasting insulin	Insulin resistance indicator (more sensitive than glucose alone)	< 10 uIU/mL (optimal 2-6)
HOMA-IR	Calculated insulin resistance score (fasting glucose x fasting insulin / 405)	< 1.0 (optimal), < 2.0 (acceptable)
ALT / AST / GGT	Liver enzymes reflecting hepatic inflammation and fat accumulation	ALT < 25 (women) / < 33 (men), AST < 25/33, GGT < 30
Triglycerides	Blood fat level linked to insulin resistance	< 150 mg/dL (optimal < 100)
HDL-C	Protective cholesterol	> 40 mg/dL men, > 50 mg/dL women (optimal > 60)
Waist circumference	Visceral fat indicator (more predictive than BMI)	< 40 in. men, < 35 in. women

Advanced Testing (Optional)

Advanced biomarkers to discuss with your healthcare provider:

25(OH)D (Vitamin D): Test at baseline and after 3 months of supplementation. Target 40-60 ng/mL.

hs-CRP: High-sensitivity C-reactive protein measures systemic inflammation. Target < 1.0 mg/L.

LPS-binding protein: Marker of metabolic endotoxemia (bacterial LPS translocation).

Stool microbiome test: Tracks changes in Akkermansia, Faecalibacterium, Roseburia, and overall diversity.

Liver ultrasound or FibroScan: For NAFLD patients, measures hepatic steatosis grade and fibrosis.

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Disclaimer

This information is for educational purposes only and is not medical advice.

Consult your healthcare provider before starting any new supplement regimen, especially if you are taking medications for diabetes, blood pressure, or cholesterol.

These protocols are based on published clinical research; individual results may vary.

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If you are currently managing type 2 diabetes, NAFLD, or metabolic syndrome with prescription medications, do not discontinue or modify doses without consulting your physician.