CLINICAL NUTRITION GUIDE · CELLULAR ENERGY & LONGEVITY

The 30-Day Cellular Reset Meal Plan

Step-by-step nutrition plan to reboot mitochondrial energy — rebuilding the machinery that powers every cell in your body.

Format Four-phase, 30-day structured protocol

Scope Diet · Fasting · Exercise · Targeted nutrients · Biomarkers

Includes Sample daily menus, 8 recipes, supplement dosing table, shopping template

Evidence labels Human trials separated from animal and mechanistic data

Educational document. Not medical advice. See disclaimer inside.

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Medical disclaimer. This document is provided for general educational purposes only. It is not medical advice, diagnosis, or treatment, and it does not create a clinician–patient relationship. Nothing here should be used to diagnose, treat, cure, or prevent any disease. Nutrition, fasting, exercise, and supplementation carry individual risks. **Do not begin a fast, a structured elimination diet, or any supplement regimen without first consulting a qualified healthcare provider** — particularly if you are pregnant or breastfeeding, under 18, over 65 and frail, taking prescription medication, or managing a diagnosed mitochondrial disorder, diabetes, kidney or liver disease, an eating disorder, or a cardiac condition. Individuals with primary mitochondrial disease must **not** fast or initiate restrictive protocols without direct medical supervision, because fasting can precipitate metabolic decompensation in these populations. Supplement doses cited here reflect ranges reported in the published literature and consumer guidance; they are not personal prescriptions. Lab biomarkers should be ordered and interpreted by a licensed clinician.

How to read the evidence labels. Throughout this guide, claims are marked with an evidence tier. HUMAN indicates data from controlled human trials or human cohort studies. ANIMAL indicates the finding comes mainly from rodent or invertebrate models. MECHANISTIC indicates a plausible pathway inferred from cell-culture or biochemical work, without outcome data in humans. Where a number is a supplement dose from the literature, it is labelled as such. Where a food or menu item appears, it is a practical application of a dietary principle — *not* a verbatim entry from a published 30-day protocol. See the note in Section 3.

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Executive Summary

Mitochondria are not passive batteries. They are the body's energy-conversion, signalling, and quality-control network — and their capacity measurably declines with age. A 30-day structured reset does not "reverse" ageing, but it can remove several of the most tractable insults that suppress mitochondrial function: chronic post-prandial glycaemic load, systemic low-grade inflammation, nutrient scarcity, physical inactivity, disrupted circadian rhythm, and the absence of fasting intervals long enough to trigger cellular housekeeping.

Why a 30-day reset, and why now

Age-related mitochondrial decline is driven by a combination of factors that dietary and behavioural change can plausibly influence: accumulation of oxidative damage to mitochondrial DNA, a fall in the efficiency of electron transport, reduced mitochondrial biogenesis (the creation of new mitochondria), impaired mitophagy (the selective recycling of damaged mitochondria), and blunted signalling through the AMPK and sirtuin–PGC-1α axes that govern the whole system. Diet is one of the few levers that touches every one of these simultaneously — it supplies biosynthetic precursors and cofactors, modulates oxidative stress and inflammation, and determines the frequency and magnitude of the post-prandial signals that switch cells between energy storage and energy production.

THE FOUR MECHANISMS THIS PLAN TARGETS

Biogenesis — building new mitochondria, largely through the PGC-1α transcriptional programme, driven by exercise, cold exposure, fasting, and polyphenols. **Mitophagy** — the selective clearance of damaged mitochondria, driven primarily by fasting and exercise in mammalian models. **Dynamics** — the balance of fission and fusion that governs mitochondrial quality and network architecture. **Bioenergetics** — the actual efficiency of oxidative phosphorylation, which depends on micronutrient cofactors (B vitamins, CoQ10, iron-sulphur clusters, magnesium) and on the lipid composition of the inner membrane.

WHAT THE 30 DAYS ACTUALLY CHANGE

Structured elimination of refined sugars, industrial seed oils, and ultra-processed foods reduces the dietary drivers of inflammation and glycaemic volatility. A graduated eating window (introduced in Week 2) creates repeated 16-hour fasting intervals. Progressive Zone 2, HIIT, and resistance work supply the strongest known endogenous stimulus for mitochondrial biogenesis. A defined set of micronutrients and phytonutrients closes gaps that are hard to close with food alone on a real-world schedule.

WHAT YOU SHOULD AND SHOULD NOT EXPECT

Expect, in the first two weeks, changes in sleep stability, energy consistency through the afternoon, and reduced post-meal sluggishness — these are the most commonly reported early changes and are consistent with improved glycaemic control. Expect, in weeks three to six, subjective improvements in mental clarity and reduced joint or skin inflammation. Do *not* expect a change in a laboratory mitochondrial-function test, a measurable change in VO_2 max beyond what training alone produces in four weeks, or a guaranteed "biological age" reversal. No published 30-day protocol has demonstrated whole-body mitochondrial replacement in humans.

SCOPE OF EVIDENCE IN THIS DOCUMENT

Human randomised controlled trial data exist for Mediterranean-style diets and mitochondrial function, for time-restricted eating and metabolic markers, for exercise modality comparisons, for urolithin A, for creatine and cognition, and for several nutrient–mitochondrial relationships. Much of the most enthusiastic mechanistic story — mitophagy induction by spermidine, NAD^+ restoration by NMN, PGC-1α activation by resveratrol — rests on animal or cell-culture work, sometimes with human trials that failed to replicate the animal effect. This document flags that distinction everywhere it matters rather than blending the two.

SECTION 01

The Science of Mitochondrial Rejuvenation

What mitochondria actually do

Every nucleated cell in your body contains hundreds to thousands of mitochondria. Their most familiar job is oxidative phosphorylation: the conversion of the chemical energy in food into ATP, the universal currency that fuels muscle contraction, nerve firing, protein synthesis, ion pumping, and thermogenesis. But energy conversion is only part of the brief.

Mitochondria are also the central node of several signalling networks. They buffer cytosolic calcium, regulate apoptosis (programmed cell death), generate and quench reactive oxygen species (ROS) that act as signalling molecules, participate in steroid and haem synthesis, house part of the iron–sulphur cluster assembly machinery, and communicate bidirectionally with the nucleus through retrograde signalling that influences gene expression. Mitochondrial dysfunction is therefore not simply "low energy" — it propagates into immune dysregulation, insulin resistance, neurodegeneration, and loss of proteostasis.

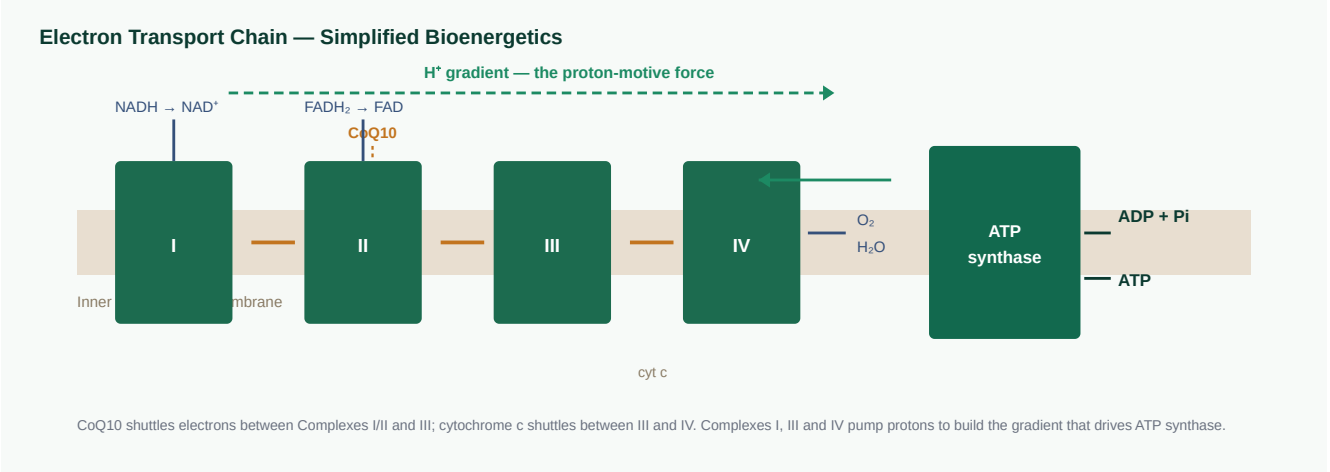


Figure 1. Simplified electron transport chain. Dietary and supplemental factors act at multiple points: B vitamins supply the NADH/$FADH_2$ precursors, CoQ10 and iron–sulphur clusters are structural electron carriers, magnesium is required for ATP hydrolysis and synthesis reactions, and membrane phospholipid composition (influenced by omega-3 intake) affects how efficiently the whole assembly operates.

Why mitochondria decline with age

Several processes converge on the ageing mitochondrion. Mitochondrial DNA (mtDNA) sits close to the electron transport chain — the primary site of ROS production — and lacks the histone packaging and some of the repair machinery available to nuclear DNA, so it accumulates oxidative lesions over decades. Clonal expansion of damaged mtDNA, reduced expression of PGC-1α and its downstream targets, declining NAD^+ availability, impaired fission–fusion cycling, and a measurable fall in mitophagic flux all contribute. The result is a population of mitochondria that produce less ATP per unit of oxygen, emit more ROS, and are less able to respond to exercise or fasting signals.

This is the point at which diet becomes relevant. Dietary patterns do not overwrite this biology, but they determine the substrate load, the inflammatory tone, the antioxidant and cofactor supply, and the frequency of the fasting–feeding cycles that tell cells which mode to run. Reviews of dietary effects on mitochondrial function consistently describe the same productive levers: reduced refined carbohydrate and industrial fat intake, higher intake of polyphenol-rich plant foods, adequate protein, and eating patterns that create defined fasting windows.

The four pillars of mitochondrial health

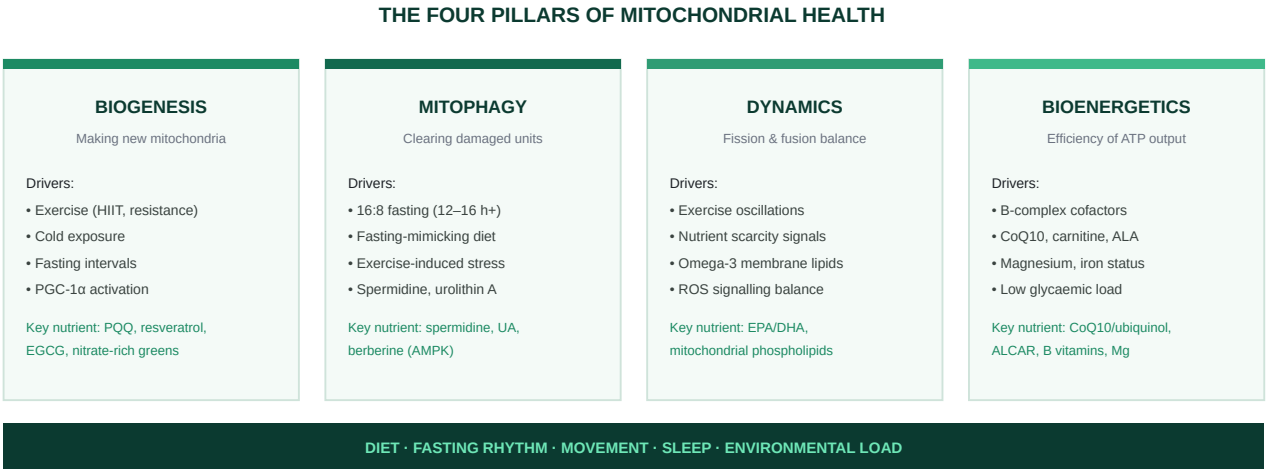


Figure 2. The four pillars are interdependent: biogenesis without mitophagy accumulates defective units, and mitophagy without biogenesis depletes the network. A useful plan therefore stimulates all four within the same 30-day window rather than maximising any single one.

1. Biogenesis

Mitochondrial biogenesis is the synthesis of new mitochondrial mass. It is coordinated largely by PGC-1α, a transcriptional coactivator that switches on both nuclear-encoded mitochondrial genes and the factors that drive mtDNA replication. In human skeletal muscle, exercise — particularly high-intensity interval work and resistance training — is the most consistently demonstrated stimulus. Cold exposure and fasting are additional inputs, and a number of dietary compounds are proposed to act on the same pathway. Reviews of diet and mitochondrial function describe polyphenol-rich patterns and caloric oscillation as the dietary components most plausibly linked to biogenesis signalling.

2. Mitophagy

Mitophagy is the selective autophagic removal of mitochondria that have lost membrane potential or accumulated damage. In cell and animal models, nutrient deprivation is the canonical trigger: the AMPK–mTOR axis shifts toward catabolism, and damaged organelles are engulfed and delivered to lysosomes. In humans, the timing question is important — autophagic flux begins to rise within roughly 12 to 16 hours of fasting, with deeper responses reported at 24 to 48 hours. This is why a 16:8 eating window is the plan's default and why longer fasts are framed as optional and clinician-supervised rather than routine.

3. Dynamics

Mitochondria constantly fuse and divide. Fusion allows content mixing and complementation of damaged components; fission isolates damaged segments for mitophagy and supports distribution during cell division. The balance is governed by large GTPase machinery (MFN1/2, OPA1 for fusion; DRP1 for fission). Chronic nutrient excess tends to favour a fragmented, less efficient network, whereas exercise and intermittent nutrient scarcity promote a more connected, functional network. Dietary fat quality — specifically the availability of long-chain omega-3 fatty acids for membrane phospholipid incorporation — plausibly influences this through membrane biophysical properties.

4. Bioenergetics

Bioenergetics is the efficiency with which the chain converts substrate into ATP. It depends on an adequate supply of cofactors: the B vitamins that form NAD, FAD, coenzyme A and thiamine pyrophosphate; CoQ10 as the mobile electron carrier; iron for

iron–sulphur clusters and haem; magnesium for ATP chemistry; and carnitine for shuttling long-chain fatty acids into the matrix for β-oxidation. It also depends heavily on substrate load — repeated large glycaemic excursions drive ROS production and reduce the proportion of energy derived from efficient fatty-acid oxidation.

> **TRANSLATING THE FOUR PILLARS INTO A MEAL PLAN**
>
> Biogenesis and bioenergetics are the pillars most directly addressed by nutrition. Mitophagy is addressed primarily by *timing* (the eating window) rather than by foods. Dynamics is addressed by overall dietary pattern and fat quality. A plan that only changes what you eat, without changing when you eat or how you move, leaves two of the four pillars largely untouched.

SECTION 02

The Mitochondrial Reset Dietary Framework

Three core principles

Principle 1 — Low glycaemic load

Frequent large post-prandial glucose excursions are associated with increased oxidative stress, reduced fatty-acid oxidation, and impaired mitochondrial efficiency. The reset therefore builds meals around protein, fibre-rich non-starchy vegetables, and fats, with carbohydrate concentrated in low-glycaemic whole forms — berries, legumes, and limited whole grains. The aim is not ketosis by default (a ketogenic pattern is a legitimate option with its own evidence base, including for some aspects of mitochondrial function, but it is not required and is harder to sustain for many people). The aim is to flatten the curve.

Principle 2 — Anti-inflammatory pattern

Chronic low-grade inflammation suppresses mitochondrial biogenesis and promotes fission. The dietary levers with the strongest human evidence for reducing inflammatory tone are a Mediterranean-style pattern rich in vegetables, olive oil, fish, and nuts; adequate omega-3 intake; and removal of ultra-processed foods, refined sugar, and industrial seed oils high in omega-6 linoleic acid. Mediterranean-diet adherence has been directly associated with better mitochondrial function and biogenesis markers in human studies, making it the most defensible foundation for this plan.

Principle 3 — Nutrient density

Mitochondria are enzyme machines, and enzymes need cofactors. A diet that is adequate in energy but poor in micronutrients — a common pattern in high-ultra-processed-food diets — can be calorically sufficient and mitochondrially underpowered. The reset prioritises organ meats and shellfish (B12, CoQ10, carnitine, iron, selenium, copper), dark leafy greens and cruciferous vegetables (folate, magnesium, sulforaphane precursors), berries (anthocyanins, ellagitannins), nuts and seeds (magnesium, spermidine, vitamin E), fatty fish (EPA/DHA, CoQ10, astaxanthin), and fermented foods (B vitamins, spermidine from aged cheese).

The Mito Food Plan structure

The Mito Food Plan, developed in the functional-medicine literature and adopted by mitochondrial disease advocacy organisations as a general dietary framework, structures intake around a high volume of colourful plant foods alongside adequate high-quality protein and generous healthy fats. The plan's central quantitative anchor is a target of **8 to 12 servings of fruits and vegetables per day**, weighted toward non-starchy vegetables, with protein at every meal and fats used deliberately rather than incidentally.

THE MITO FOOD PLAN — DAILY STRUCTURE

Component	Target	Rationale for mitochondrial function
Vegetables & fruit	8–12 servings/day, majority non-starchy	Polyphenols, folate, magnesium, nitrate, fibre for glycaemic control and gut microbiota. Colour variety maximises phytonutrient range.
Protein	~1.2–1.6 g/kg body weight, spread across meals	Supplies carnitine, CoQ10, B12, iron, zinc, sulphur amino acids; preserves lean mass during any caloric deficit; supports glutathione synthesis.

Fats	Olive oil, avocado, nuts, oily fish; MCT oil optional	Membrane phospholipid composition; fat-soluble vitamin absorption (A, D, E, K, CoQ10); MCTs provide an efficient, carnitine-independent fuel.
Carbohydrate	Largely from vegetables, berries, legumes; whole grains limited	Keeps post-prandial insulin response modest and preserves access to fatty-acid oxidation between meals.
Hydration & electrolytes	Water; sodium, potassium, magnesium during fasting	Prevents the headache, cramping, and fatigue that cause most early fasting abandonment.

Foods to emphasise

- **Wild-caught oily fish** — salmon, sardines, mackerel, anchovies, herring. EPA/DHA for membrane incorporation; CoQ10; selenium; vitamin D.
- **Grass-fed and pasture-raised meat** — beef, lamb, poultry, eggs. B12, carnitine, CoQ10, iron, choline.
- **Organ meats, in moderation** — liver and heart are among the densest dietary sources of CoQ10, B12, folate, and copper.
- **Non-starchy vegetables** — leafy greens, broccoli, cauliflower, cabbage, peppers, courgette, asparagus, mushrooms (especially crimini and shiitake for NAD precursors and ergothioneine).
- **Berries** — blueberries, raspberries, blackberries, strawberries. Anthocyanins and ellagitannins; lowest glycaemic load among fruits.
- **Nuts and seeds** — walnuts (ellagitannins, ALA), sunflower seeds (NAD precursors, vitamin E), pumpkin seeds (magnesium, zinc), almonds.
- **Extra-virgin olive oil** — oleocanthal, hydroxytyrosol, monounsaturated fat; the backbone of the Mediterranean evidence base.
- **MCT oil** — caprylic and capric acid provide rapid, carnitine-independent ketone substrate; useful around training or during the fasting window.
- **Fermented and cultured foods** — aged cheese, kefir, yoghurt, sauerkraut, kimchi. Spermidine, B vitamins, and gut microbial diversity.
- **Green tea, coffee, turmeric, and dark chocolate** — EGCG, chlorogenic acids, curcumin, flavanols; all concentrated polyphenol sources.

Foods to avoid or minimise

ELIMINATION LIST AND THE REASON BEHIND EACH

Category	Why it is removed in Phase 1
Ultra-processed foods	High in refined starch, industrial fat, emulsifiers, and additives; consistently associated with worse metabolic markers and higher inflammatory load. Delivers calories without cofactors.
Refined sugars & sweetened drinks	Large, rapid glycaemic excursions and fructose-driven hepatic lipid accumulation — both directly impair mitochondrial oxidative capacity.
Industrial seed oils	High linoleic acid content; the modern dietary omega-6:omega-3 ratio is far from the range on which the enzymatic pathways evolved. Replacing them with olive oil and animal fats is one of the highest-yield swaps.
Fried and repeatedly heated foods	Oxidised lipid species and advanced glycation end-products increase systemic oxidative burden.

Gluten and dairy (for some)	Not universally necessary. Removed during Phase 1 as an elimination trial and reintroduced individually in Week 4 to identify genuine sensitivity — a standard elimination–rechallenge method, not a claim that gluten is intrinsically harmful to mitochondria.
Alcohol	Disrupts sleep architecture and mitochondrial respiration in hepatocytes; removed for the duration of the reset.

A NOTE ON THE EVIDENCE BEHIND DIETARY FRAMEWORKS

The Mito Food Plan is a clinical framework, not a randomised trial intervention. Its components are each supported to varying degrees — the Mediterranean pattern and omega-3 intake have the strongest human data, while the specific "8–12 servings" target is a practical operationalisation rather than a validated threshold. The framework is included here because the underlying components are defensible, not because the plan as a package has been tested against a control in a trial.

SECTION 03

The 30-Day Meal Plan Structure

IMPORTANT NOTE ON THE SAMPLE MENUS

The daily menus in this section are **synthesised from the dietary principles described above** — a low-glycaemic, Mediterranean-adjacent, nutrient-dense pattern plus a graduated eating window. They are **not** reproduced from any single published 30-day protocol, and no published 30-day mitochondrial meal plan with day-by-day menus was identified during the research for this document. Treat each menu as a template to be adapted to your preferences, culture, budget, and any clinician-directed restrictions. Portion sizes are intentionally omitted because they depend on body size, activity, and goal.

How the four weeks fit together

The sequence is deliberate. Week 1 removes the inputs that suppress mitochondrial function, without simultaneously adding a fasting stressor — this makes the transition tolerable and isolates the effect of the dietary change. Week 2 adds the timing variable. Week 3 adds the exercise variable. Week 4 consolidates all three and tests reintroductions. Each week builds on the one before; do not skip to Week 3.

WEEK 1 · DAYS 1–7

Foundation Phase — Elimination and Preparation

Focus: Remove inflammatory foods, establish a consistent eating window, and introduce the key nutrient categories. No fasting beyond a normal overnight period this week — the goal is to make the dietary change stick before adding a timing stressor.

Practical targets for Week 1:

- Eliminate refined sugar, sweetened drinks, ultra-processed snacks, seed oils, fried food, and alcohol.
- Reach 8 servings of vegetables and fruit daily, with at least 5 of them non-starchy vegetables.
- Put protein at all three meals — a palm-sized portion minimum.
- Set a consistent first-meal and last-meal time with a 12-hour overnight gap (e.g. 8 a.m. to 8 p.m.), so Week 2's 16:8 is a 2-hour extension rather than a 4-hour leap.
- Begin a 10-minute post-meal walk after the largest meal.
- Start the core supplement base only if your clinician agrees (see Section 4).

BREAKFAST
Three-egg omelette with spinach, mushrooms, and turmeric; half an avocado; a small handful of blueberries; black coffee or green tea.

LUNCH
Grilled salmon salad — mixed leaves, cucumber, cherry tomatoes, red onion, walnuts, olive oil and lemon dressing; a small portion of chickpeas.

AFTERNOON SNACK
Greek yoghurt with sunflower seeds and cinnamon; or a boiled egg with sauerkraut.

DINNER
Grass-fed beef or lentil bolognese over courgette ribbons; roasted broccoli and red pepper with olive oil; a side of sautéed kale with garlic.

EVENING
Herbal tea. Kitchen closed 12 hours before tomorrow's breakfast.

WEEK 2 · DAYS 8–14

Activation Phase — Mitophagy Induction

Focus: Introduce a 16:8 time-restricted eating window and deliberately increase polyphenol-rich foods. The 16-hour fast is where nutrient scarcity begins to shift the cell toward catabolism and autophagic flux; the polyphenol load supplies the phytonutrient pressure that complements it.

Practical targets for Week 2:

- Move to a 16:8 window — for example, first meal at 11 a.m., last meal finished by 7 p.m. Water, black coffee, and unsweetened tea are permitted outside the window.
- Add 2–3 polyphenol "pulses" per day: berries, green tea, dark chocolate (85%+), turmeric with black pepper, pomegranate, or extra-virgin olive oil.
- Keep protein constant — do not let the shorter window reduce total protein intake.
- Add two 20–30 minute Zone 2 sessions and one short interval session (Section 6).
- Expect an adjustment period of 3–5 days: mild hunger, irritability, or headache are common and usually resolve. Persistent symptoms mean the window is too aggressive — shorten it.

11:00 — FIRST MEAL
Breaking the fast with protein and fat first: smoked salmon, avocado, and two boiled eggs with rocket; a bowl of raspberries and walnuts; green tea.

14:30 — LUNCH
Chicken thigh and roasted vegetable bowl — cauliflower, broccoli, red pepper, red onion, olive oil, tahini; side of kimchi.

17:00 — SNACK
Handful of almonds and 85% dark chocolate; or pomegranate seeds with pumpkin seeds.

18:45 — DINNER
Sardines or mackerel with a large chopped salad, olives, capers, and lemon; a small sweet potato; steamed asparagus with olive oil.

FASTING WINDOW
19:00 → 11:00 (16 hours). Water, electrolytes, and unsweetened tea only.

WEEK 3 · DAYS 15–21

Biogenesis Phase — Building New Mitochondria

Focus: Exercise–nutrition synergy and PGC-1α activating foods. This is the week where training load rises and where nutrient timing around training becomes deliberate. Exercise is the strongest endogenous biogenesis stimulus available; the nutritional job is to supply the cofactors and substrates that allow adaptation to occur, and to avoid blunting the adaptive signal with excessive post-exercise carbohydrate.

Practical targets for Week 3:

- Three resistance sessions and two HIIT sessions (Section 6), separated by at least 48 hours per modality.
- Prioritise PGC-1α-linked foods: green tea (EGCG), turmeric with piperine (curcumin), resveratrol-containing foods such as red grapes and berries, cruciferous vegetables (sulforaphane), and nitrate-rich greens (rocket, spinach, beetroot).
- Keep the 16:8 window, and consider training in the fasted state for Zone 2 once or twice — but not for HIIT or heavy resistance work.
- Increase total daily protein slightly to support training adaptation.
- Introduce one cold exposure per week if tolerated (Section 7).

07:30 — FASTED ZONE 2
35–45 minutes brisk walking or easy cycling, water only.

09:30 — FIRST MEAL
Frittata with kale, mushrooms, and sweet potato; fermented kimchi; a small bowl of mixed berries; matcha or green tea.

13:00 — LUNCH
Grass-fed steak or tempeh with a large rocket, beetroot, and walnut salad; olive oil; a portion of lentils.

16:30 — PRE-TRAINING SNACK
MCT oil in coffee or a small handful of macadamias, if training in the late afternoon.

18:30 — DINNER
Baked cod with turmeric and lemon; roasted cauliflower with cumin; sautéed spinach; a small serving of quinoa if training load is high.

FASTING WINDOW
19:30 → 09:30 (14–16 hours, adjusted to training).

WEEK 4 · DAYS 22–30

Optimization Phase — Integration and Maintenance

Focus: Convert 30 days of structure into a sustainable pattern. This week includes the reintroduction test for foods removed in Week 1, and, for appropriate candidates under clinician guidance, consideration of a fasting-mimicking diet day or a single 24-hour fast.

Practical targets for Week 4:

- Hold the 16:8 window on at least 5 days per week; allow a 12-hour window on the other 2. Flexibility is what makes the pattern survive month two.
- Reintroduce one eliminated food group at a time, three days apart, and track symptoms. This is how you determine whether gluten or dairy removal was actually necessary for you.
- Optional and clinician-supervised only: one fasting-mimicking diet block or a single 24-hour fast (Section 5) — **not** appropriate for anyone with a mitochondrial disorder, diabetes on glucose-lowering medication, a history of disordered eating, or pregnancy.
- Re-run baseline labs at the end of this period if your clinician ordered them (Section 8).
- Write down the three habits you will keep and the two you will drop. Maintenance beats intensity.

11:00 — FIRST MEAL

Bone broth or miso soup; two poached eggs on sautéed greens; avocado; walnuts; green tea.

14:00 — LUNCH

Large Mediterranean bowl — grilled chicken or chickpeas, cucumber, tomato, olives, feta (if reintroduced and tolerated), oregano, olive oil, lemon; a slice of sourdough only if reintroduction confirms tolerance.

17:30 — SNACK

Apple with almond butter, or kefir with flaxseed.

18:45 — DINNER

Wild salmon with roasted Brussels sprouts and sweet potato; a large mixed salad; a small glass of pomegranate juice or a bowl of berries.

AFTER DAY 30

Retain the eating window, the protein-at-every-meal rule, the 8+ vegetable servings, and two resistance sessions per week. Everything else is optional.

SECTION 04

Key Nutrients and Supplements

> Food first, always. The table below consolidates the compounds most discussed in the mitochondrial literature, with the dose ranges reported in that literature and the strength of evidence behind each. Doses are informational, drawn from published reviews, institutional fact sheets, and clinical guides — they are not recommendations for any individual, and several of these compounds interact with prescription medication.

MITOCHONDRIAL NUTRIENTS — DOSING, MECHANISM, AND EVIDENCE TIER

Compound	Reported dose	Proposed mechanism	Food sources	Evidence
NAD⁺ precursors (NMN / NR)	**250–500 mg/day**	Replenish the NAD⁺ pool consumed by sirtuins, PARPs, and CD38; substrate for mitochondrial Complex I via NADH.	Crimini mushrooms, nutritional yeast, green peas, beef, sunflower seeds, tomatoes	HUMAN ANIMAL — human trials show NAD⁺ rises; outcome data still limited
CoQ10 / Ubiquinol	**100–300 mg/day** up to 1200 mg in therapeutic use	Mobile electron carrier between Complexes I/II and III; lipid-phase antioxidant. Take with dietary fat.	Organ meats, oily fish, beef, soy, spinach, broccoli	HUMAN — best established in statin-associated depletion and heart failure
PQQ (pyrroloquinoline quinone)	**10–20 mg/day**	Proposed activator of PGC-1α and mitochondrial biogenesis; redox cycling cofactor.	Parsley, green pepper, kiwi, tofu, natto, breast milk	ANIMAL MECHANISTIC — small human trials only
Alpha-lipoic acid	**300–600 mg/day** R-ALA preferred	Cofactor for pyruvate dehydrogenase and α-ketoglutarate dehydrogenase; regenerates glutathione, vitamins C and E.	Spinach, broccoli, yams, potatoes, red meat, organ meats	HUMAN — strongest data in diabetic neuropathy
L-Carnitine / Acetyl-L-Carnitine	**500–3000 mg/day**	Shuttles long-chain fatty acids into the mitochondrial matrix for β-oxidation; ALCAR also supports acetyl-CoA supply to the brain.	Red meat, dairy, chicken; endogenous synthesis requires vitamin C, iron, B6, niacin	HUMAN — established in deficiency states and secondary deficiency
Omega-3 (EPA / DHA)	**1–3 g/day combined EPA+DHA**	Incorporation into mitochondrial membrane phospholipids (cardiolipin environment); resolution of inflammation via resolvins and protectins.	Salmon, sardines, mackerel, anchovies, herring; algal oil	HUMAN — membrane incorporation documented in human trials
Spermidine	**1.2–3 mg/day supplement** wheat germ 24–35 mg per 100 g	Polyamine that induces autophagy in model systems; proposed cardioprotective and neuroprotective effects.	Wheat germ, soybeans, aged cheese, mushrooms, legumes, natto	ANIMAL MECHANISTIC — human outcome trials lacking

Urolithin A	**1000 mg/day supplement**	Gut-microbial metabolite of ellagitannins; stimulates mitophagy and improves muscle endurance markers in human trials.	Pomegranate, walnuts, berries (ellagitannins — conversion depends on individual microbiome)	HUMAN — randomised trials with muscle biomarker endpoints
B-complex (B1, B2, B3, B5, B6, B7, B9, B12)	**RDA-level to moderate doses**	Precursors of NAD/FAD, coenzyme A, thiamine pyrophosphate, PLP, biotin carboxylases; one-carbon metabolism for mtDNA methylation.	Organ meats, eggs, legumes, leafy greens, whole grains, nutritional yeast	HUMAN — deficiency effects well documented
Magnesium (glycinate or malate)	**300–400 mg elemental**	Required for ATP synthesis and hydrolysis, for polymerase function, and for mitochondrial membrane stability.	Leafy greens, pumpkin seeds, almonds, dark chocolate, legumes	HUMAN ANIMAL — deficiency impairs mitochondrial function
Creatine (monohydrate)	**3–5 g/day maintenance** 20 g/day loading for brain saturation	Phosphocreatine buffer for ATP regeneration; supports cognitive performance under stress and sleep deprivation.	Red meat, herring, salmon (dietary intake far below supplemental doses)	HUMAN — extensive RCT data, including brain outcomes
Resveratrol	**Variable; typically 150–500 mg/day in trials**	Proposed SIRT1 activation with downstream PGC-1α upregulation; also AMPK-adjacent effects.	Red grapes, red wine (not recommended here), berries, peanuts	ANIMAL MECHANISTIC — human results inconsistent
Curcumin (with piperine)	**Bioavailable formulations; piperine required**	NF-κB inhibition, reducing inflammatory tone that suppresses biogenesis; Nrf2 activation.	Turmeric root (poor oral bioavailability without piperine or a lipid formulation)	HUMAN — anti-inflammatory markers; mitochondrial outcomes indirect
EGCG (green tea)	**322 mg/day acceptable daily intake** 600 mg NOAEL from supplements	AMPK activation, proposed PGC-1α upregulation, antioxidant and anti-inflammatory effects.	Brewed green tea (a cup supplies well under 322 mg; supplement doses are far higher)	HUMAN — safety data established; high-dose hepatotoxicity risk with extracts
Berberine	**500 mg, 2–3× daily**	AMPK activation; improves insulin sensitivity and glycaemic control, indirectly supporting mitochondrial substrate handling.	Not a practical food source at therapeutic doses (Goldenseal, barberry — medicinal plants)	HUMAN — glycaemic endpoints; significant drug-interaction profile

Notes on the most important entries

NAD^+ precursors

NAD^+ declines with age and with chronic metabolic stress, and it is the substrate for sirtuins and for the DNA-repair enzymes PARP1/2. Oral nicotinamide mononucleotide (NMN) and nicotinamide riboside (NR) reliably raise circulating and, in some tissues, intracellular NAD^+ in humans. What remains uncertain is whether raising NAD^+ translates into measurable mitochondrial or clinical benefit — human trials reporting hard outcomes are still small and short. Food sources contribute meaningful amounts but generally far below supplemental doses; crimini mushrooms, nutritional yeast, green peas, beef, sunflower seeds, and tomatoes are the commonly cited dietary contributors.

CoQ10

CoQ10 is the only fat-soluble electron carrier in the chain, and its reduced form (ubiquinol) also acts as a lipid-phase antioxidant. Tissue levels fall with age, and statin therapy reduces circulating CoQ10 by inhibiting the shared mevalonate pathway. Absorption is poor and highly fat-dependent — take it with a meal containing fat. The 100–300 mg/day range is standard; the Linus Pauling Institute's monograph and mitochondrial-disease clinical guidance describe doses up to 1200 mg/day in therapeutic contexts, typically under specialist supervision. **CoQ10 can reduce warfarin efficacy** because of structural similarity to vitamin K — see Section 9.

PQQ, ALA, and carnitine

PQQ is the most speculative entry on the list. Rodent and cell studies suggest it activates PGC-1α and increases mitochondrial density, but human data are thin and mostly from small short trials. Alpha-lipoic acid has a genuine biochemical role as a cofactor for two mitochondrial dehydrogenases and as a glutathione-recycling agent; its strongest human evidence is in diabetic peripheral neuropathy at 600 mg/day. Carnitine is rate-limiting for fatty-acid entry into mitochondria; the NIH Office of Dietary Supplements fact sheet covers deficiency states, the endogenous synthesis pathway, and the fact that vegetarians obtain very little from the diet. Note that acetyl-L-carnitine is the form with better blood–brain barrier penetration, relevant to cognitive endpoints.

Urolithin A and spermidine

These are the two most interesting mitophagy-directed compounds. Urolithin A is not present in food — it is produced by gut bacteria from ellagitannins in pomegranate, walnuts, and berries, and only a subset of people carry the microbiome capable of efficient conversion. Supplementation with 1000 mg/day has been tested in randomised human trials with muscle endurance and mitochondrial biomarker endpoints. Spermidine induces autophagy in model organisms and is abundant in wheat germ (24–35 mg per 100 g), soybeans, aged cheese, and mushrooms; a supplemental dose of 1.2–3 mg/day is commonly cited. Human outcome trials are still awaited for both, so treat the enthusiasm with proportionate caution.

EGCG dose safety

The 322 mg/day figure is the acceptable daily intake for EGCG; 600 mg/day is the no-observed-adverse-effect level used in safety assessments. These thresholds matter because concentrated green-tea extracts at high doses have been linked to hepatotoxicity, whereas brewed tea at normal consumption levels has not. If you use a green tea extract, check the EGCG content on the label rather than assuming a capsule is equivalent to a cup of tea.

SUPPLEMENT INTERACTIONS TO KNOW BEFORE YOU START

CoQ10 — reduces warfarin effect (monitor INR). **Berberine** — inhibits CYP3A4 and CYP2D6; raises blood levels of many drugs including statins, immunosuppressants, and some antihypertensives; may cause hypoglycaemia with glucose-lowering medication. **ALA** — may lower blood glucose; additive with insulin or sulfonylureas. **EGCG extracts** — hepatotoxicity risk at high doses. **NMN/NR** — long-term human safety data limited; avoid in pregnancy and in anyone with a history of hormone-sensitive cancer without specialist advice. **Curcumin** — may potentiate anticoagulants. **Magnesium** — separates from thyroid medication and certain antibiotics by at least 2 hours. Bring this entire list to your pharmacist before starting anything on it.

SECTION 05

Intermittent Fasting and Time-Restricted Eating

> Fasting is the timing lever. It does not add nutrients; it removes them for long enough that the cell switches from storage to maintenance. In animal and cell models this switch is the most reliable known trigger for mitophagy. In humans, the metabolic consequences of time-restricted eating are well documented; the mitophagy-specific consequences are inferred rather than directly measured.

The 16:8 protocol

A 16:8 protocol confines all eating to an 8-hour window and leaves a 16-hour fasting period. For most people a window of roughly 11:00–19:00 or 12:00–20:00 works best because it preserves a normal family dinner and avoids eating immediately before bed. During the fasting window, water, black coffee, and unsweetened tea are permitted; anything with calories — including milk, sweeteners that elicit an insulin response, and most "zero-calorie" creamers — is not.

Protocols and adherence data for time-restricted eating have been reviewed extensively. The practical summary is that 16:8 is well tolerated by healthy adults, produces modest reductions in caloric intake and body weight in free-living conditions, and improves some glycaemic and lipid markers — but the magnitude of benefit is often attributable substantially to the reduction in late-night eating rather than to the fasting state per se. That distinction is honest and does not diminish the protocol's usefulness.

The fasting-mimicking diet

The fasting-mimicking diet (FMD) is a 5-day, low-calorie, low-protein, high-unsaturated-fat protocol designed to produce fasting-like metabolic and immune effects while still providing food. In the published formulation, the macronutrient profile is engineered to keep protein and sugar low enough to suppress the nutrient-sensing pathways that fasting suppresses, while unsaturated fat supplies most of the energy. It has been studied in humans for effects on body composition, metabolic markers, and — in a notable trial — on markers of biological age and immune function. Work in Alzheimer's-model and clinical populations has also examined FMD in combination with standard therapy.

The FMD is **not** a casual intervention. It is a structured, clinician-supervised protocol. It is contraindicated in pregnancy, type 1 diabetes, advanced kidney or liver disease, frailty, and — critically — in mitochondrial disease, where the metabolic shift can precipitate crisis.

Autophagy and mitophagy timeline

APPROXIMATE PHASES OF A FAST — HUMAN INFERENCE FROM ANIMAL AND HUMAN METABOLIC DATA

Elapsed fasting	What is reasonably established	What is inferred or uncertain
0–4 h	Post-absorptive state; insulin falls, glucagon rises.	—
4–12 h	Glycogenolysis supplies glucose; transition toward lipolysis begins.	Autophagic flux likely begins to rise but is not measurable in routine clinical practice.
12–16 h	Ketone production rises measurably; AMPK activation, mTOR suppression.	**Early autophagy/mitophagy** — inferred largely from rodent and cell data; human hepatic mitophagy cannot be directly assayed.

16–24 h	Ketones become a meaningful fuel for brain and muscle; insulin very low.	Deeper autophagic response; magnitude in humans is unknown and varies by individual.
24–48 h	Glycogen largely depleted; pronounced ketosis.	**Deeper mitophagy** reported in model systems. Should not be attempted without supervision.
> 48 h	Increasing risk of electrolyte disturbance, refeeding issues, lean-mass loss.	Not part of this plan. Specialist territory only.

WHICH PROTOCOL TO ACTUALLY USE

For a healthy adult, the risk–benefit balance favours **16:8 daily** as the default, with optional longer fasts only under supervision. The deeper mitophagy seen at 24–48 hours in animal models is real in those models; whether the incremental human benefit justifies the adherence cost and the safety risk is not established. Consistency at 16:8 for 30 days is almost certainly worth more than one heroic 48-hour fast.

Safety considerations

- **Do not fast if you have a mitochondrial disorder.** Fasting increases reliance on fatty-acid oxidation and can overwhelm a compromised respiratory chain, precipitating lactic acidosis or metabolic crisis.
- **Diabetes on insulin or sulfonylureas** — hypoglycaemia risk; requires dose adjustment under medical supervision.
- **Pregnancy and breastfeeding** — not appropriate.
- **History of eating disorders** — structured fasting can reactivate restrictive patterns; do not use without psychological support.
- **Women with hypothalamic amenorrhoea or significant stress load** — fasting is an additional stressor; a 12-hour overnight fast may be the appropriate ceiling.
- **Medication timing** — some drugs require food. Never skip a dose to preserve a fast.
- **Electrolytes** — sodium, potassium, and magnesium during longer fasts prevent most of the symptoms that cause people to quit.

SECTION 06

Exercise Synergy

> Exercise is the single most powerful endogenous stimulus for mitochondrial biogenesis available, and it is not substitutable by any nutrient. The nutrition plan in this document exists largely to support and amplify the adaptation that training produces. Recent comparative work on high-intensity interval training versus moderate-intensity continuous training supports HIIT as a time-efficient stimulus for mitochondrial and cardiorespiratory adaptation, with the caveat that both modalities have distinct and complementary benefits.

HIIT protocol

Two well-studied formats are worth using:

- **The 4×4 protocol** — four intervals of 4 minutes at 85–95% of maximum heart rate, separated by 3 minutes of active recovery at around 60–70%. Total session ~40 minutes including warm-up and cool-down. This is among the most studied formats for VO_2 max and mitochondrial adaptation.
- **The 60-second protocol** — intervals of 60 seconds at greater than 90% of heart-rate reserve, with 60 seconds of rest, repeated 8–12 times. Shorter, more intense, and better suited to people with limited time or access to equipment.

Frequency: 2 sessions per week during Weeks 2–4. Do not stack HIIT on consecutive days. If you are new to interval training, spend two weeks building an aerobic base first.

Resistance training

Resistance training drives mitochondrial biogenesis in skeletal muscle through mechanical and metabolic signalling that is partly distinct from endurance work, and it preserves the muscle mass that is itself a determinant of metabolic health. Two to three full-body sessions per week, covering push, pull, hinge, squat, and carry patterns, at 6–12 repetitions per set, is sufficient for a 30-day block. Progressive overload — adding load or repetitions over the month — is what actually drives adaptation.

Zone 2 cardio

Zone 2 is the intensity at which you can hold a conversation but would prefer not to — roughly 60–70% of maximum heart rate, or the top of your aerobic base. It is the foundation of mitochondrial density in slow-twitch fibres and of fat-oxidation capacity. Target 2–3 sessions of 30–45 minutes per week. Zone 2 is also the modality best suited to fasted training, because it relies heavily on fat oxidation and does not demand the glycogen availability that HIIT does.

Timing exercise relative to meals and supplements

PRACTICAL TIMING FRAMEWORK

Session type	Fasted or fed?	Nutrition and supplement notes
Zone 2 (morning)	Fasted is acceptable and often preferred	Water and electrolytes. Break the fast afterwards with protein-forward meal. This is the easiest way to extend the overnight fast to 14–16 hours.
HIIT	Fed, or at least not deeply fasted	Performance and recovery both suffer in a fasted state at this intensity. Small protein-and-fat meal 90–120 minutes prior.

Resistance	Fed	Protein 25–40 g within a few hours post-session. Creatine timing is not critical — total daily intake matters more than timing.
CoQ10 / fat-soluble supplements	With a fat-containing meal	Absorption is fat-dependent. Do not take on an empty stomach during the fasting window.
Magnesium	Evening	Glycinate is well tolerated and may support sleep quality; separate from thyroid medication by 4 hours.

THE ADAPTATION SIGNAL

Much of the mitochondrial benefit of training comes from the transient stress itself — the AMPK activation, the ROS pulse, the calcium flux — not from the recovery. Blunting that signal with a large carbohydrate load immediately after every session is counterproductive for mitochondrial adaptation in a non-athlete. Protein and fat after training, carbohydrate timed around the hardest sessions, is the sensible default.

SECTION 07

Lifestyle Factors

Diet and exercise do not operate in isolation. Three lifestyle variables — sleep and circadian timing, thermal exposure, and chronic stress — modulate mitochondrial function directly, and a fourth, environmental toxin load, determines the oxidative burden the system must absorb.

Sleep and circadian rhythm

Mitochondria run on a circadian clock. Mitochondrial oxidative capacity, ROS production, and the expression of biogenesis regulators all oscillate across the day, and mistimed eating uncouples the metabolic clock from the central clock in the suprachiasmatic nucleus. The practical consequences are the reason the 16:8 window in this plan ends in the early evening rather than late at night: late-night eating produces a larger glycaemic and insulin response for the same food, and it disrupts the mitophagy window that sleep would otherwise open.

- Target 7–9 hours of sleep opportunity, with a consistent wake time — the wake time anchors the clock more strongly than the bedtime.
- Get bright light exposure within an hour of waking; dim light in the two hours before bed.
- Finish eating at least 2–3 hours before sleep.
- Keep the bedroom cool — around 18–19 °C is the commonly cited range — since core temperature decline is part of sleep onset.

Cold and heat exposure

Cold exposure activates brown adipose tissue and drives PGC-1α-dependent mitochondrial biogenesis in that tissue; heat exposure induces heat-shock proteins and mitochondrial chaperones. Both are hormetic stressors — the benefit comes from a moderate, transient stress followed by recovery. Practical and low-risk starting points: a 30–60 second cold finish to a normal shower, two to three times per week; and sauna or hot bath use of 15–20 minutes, if available and medically appropriate. Neither is essential, and neither should be attempted by anyone with cardiovascular disease, uncontrolled hypertension, or Raynaud's phenomenon without medical clearance.

Stress management

Chronic psychological stress elevates cortisol, which promotes gluconeogenesis, raises circulating glucose, and suppresses the anabolic and repair processes that mitochondrial maintenance depends on. The interventions with reasonable evidence for lowering physiological stress load are unglamorous: 10–20 minutes of daily breathwork or meditation, time in nature, resistance training, and adequate sleep. The point is not optimisation — it is reducing the allostatic load that competes with repair.

Toxin avoidance

Mitochondria are the primary site of xenobiotic metabolism and the primary target of its by-products. Practical reductions with a favourable effort-to-benefit ratio:

- Filter drinking water where contamination is a concern; avoid storing food in soft plastics, particularly when hot or fatty.
- Choose low-pesticide produce where budget allows, and wash produce thoroughly.

- Ventilate cooking areas — cooking fumes are a meaningful indoor particulate source.
- Avoid smoking and minimise alcohol; both have direct mitochondrial toxicity.
- Read labels on personal-care products for known sensitisers, though be sceptical of broad "detox" claims not backed by measurement.

SECTION 08

Tracking Progress and Biomarkers

> Track what you can measure reliably, and be honest that the most meaningful mitochondrial outcomes are not available in a standard lab panel. There is no routine clinical test for mitophagy or biogenesis. What you can measure are the downstream metabolic and inflammatory markers that mitochondrial function influences, plus your own subjective experience, which is a legitimate data source when tracked systematically.

Subjective measures — track weekly

SIMPLE WEEKLY SELF-ASSESSMENT (RATE 1–10, SAME DAY EACH WEEK)

Dimension	What to rate	Why it matters
Morning energy	How rested you feel on waking	Most sensitive early indicator of sleep and glycaemic improvement
Afternoon stability	Severity of the 2–4 p.m. energy dip	Proxy for post-prandial glycaemic volatility
Mental clarity	Focus, word-finding, task switching	Typically the last subjective measure to improve
Sleep quality	Sleep onset latency, night wakings, refreshment	Improves first in most people, sometimes within 7–10 days
Joint / skin inflammation	Stiffness, puffiness, rash, acne	Reflects systemic inflammatory tone
Hunger and satiety	Hunger intensity outside the eating window	Tracks fasting adaptation and protein adequacy

Objective measures — baseline and end of Week 4

LABORATORY BIOMARKERS WORTH DISCUSSING WITH YOUR CLINICIAN

Marker	Relevance	Notes
Fasting glucose	Baseline glycaemic control	Single value is noisy; pair with insulin.
Fasting insulin & HOMA-IR	Insulin sensitivity — the best single proxy for metabolic flexibility	HOMA-IR is calculated from glucose and insulin; the most likely marker to move in 30 days.
HbA1c	Three-month average glycaemia	Will not fully reflect a 30-day change; better as a longer-term tracker.
hs-CRP	Systemic inflammatory tone	Falls with weight loss and improved diet; sensitive to acute illness.
Lipid panel (with triglycerides/HDL ratio)	Triglyceride/HDL ratio is a useful insulin-resistance proxy	Often improves measurably within four weeks of carbohydrate reduction.
IGF-1	Growth-hormone axis; affected by protein intake, fasting, and sleep	Interpret with care — both high and low values have been framed as relevant.
Vitamin D, B12, ferritin, folate	Common deficiencies that impair mitochondrial function	Correct before attributing fatigue to "mitochondria".
Thyroid panel (TSH, free T4)	Rules out a common alternative cause of fatigue	Essential before concluding a plan is or isn't working.

When to expect results

WEEK 1–2 · SLEEP AND ENERGY STABILITY

The earliest and most consistently reported changes are sleep-related: faster sleep onset, fewer night wakings, and a less dramatic afternoon energy dip. These track glycaemic stabilisation and the removal of late-night eating. Do not expect mental clarity yet, and do not judge the plan on how you feel on day 4 — the adaptation dip is real.

WEEK 3–6 · MENTAL CLARITY AND REDUCED INFLAMMATION

Subjective cognitive sharpness, reduced joint stiffness, and clearer skin are the changes most often described in the third to sixth week. Objectively, this is when fasting insulin, triglycerides, and hs-CRP are most likely to show movement. Note that a 30-day plan ends before this window closes — the point of the final week is to carry the pattern forward long enough to see it.

BEYOND 6 WEEKS · FITNESS AND BODY COMPOSITION

Changes in VO_2 max, muscle mitochondrial density, and body composition are measurable on a 6–12 week horizon at minimum, not 30 days. Be sceptical of any protocol promising documented mitochondrial change in four weeks.

SECTION 09

Special Considerations and Cautions

MITOCHONDRIAL DISEASE — DO NOT FAST WITHOUT MEDICAL SUPERVISION

People with primary mitochondrial disease have a genetically compromised respiratory chain. Fasting shifts reliance toward fatty-acid oxidation and ketogenesis, which can overwhelm a limited oxidative capacity and precipitate lactic acidosis, hypoglycaemia, rhabdomyolysis, or a metabolic crisis. **Fasting, ketogenic diets, fasting-mimicking diets, and heat or cold stress are all potential triggers and must not be undertaken without direct specialist supervision.** Nutrition in mitochondrial disease is a clinical intervention, typically guided by a metabolic dietitian and often involving a "mitochondrial cocktail" of cofactors (commonly CoQ10, riboflavin, thiamine, L-carnitine, alpha-lipoic acid, and vitamin E) prescribed at doses and combinations determined by the treating team — not self-assembled from a consumer guide. Advocacy organisations such as MitoAction and the Australian mitochondrial foundation publish patient-oriented nutrition guidance that should be used instead of this document if you have a diagnosed mitochondrial disorder.

Drug–supplement interactions

CLINICALLY SIGNIFICANT INTERACTIONS TO RAISE WITH YOUR PRESCRIBER

Supplement	Interacts with	Effect
CoQ10	Warfarin	Structural similarity to vitamin K reduces anticoagulant effect; INR must be monitored.
Berberine	Statins, immunosuppressants, antihypertensives, glucose-lowering drugs	CYP3A4/CYP2D6 inhibition raises drug levels; additive hypoglycaemia risk.
Alpha-lipoic acid	Insulin, sulfonylureas	Additive glucose lowering — risk of hypoglycaemia.
Omega-3 (high dose)	Anticoagulants, antiplatelets	Additive bleeding tendency at gram-plus doses.
Curcumin	Anticoagulants, some chemotherapy agents	Potentiates anticoagulation; may alter drug metabolism.
Green tea extract (EGCG)	Iron supplements, some beta-blockers	Chelates iron; hepatotoxicity risk at high extract doses.
Magnesium	Levothyroxine, tetracyclines, fluoroquinolones, bisphosphonates	Chelation reduces absorption — separate by at least 2–4 hours.
NMN / NR	Limited data	Long-term safety unknown; avoid in pregnancy and hormone-sensitive cancer without specialist advice.
Creatine	Nephrotoxic drugs	Generally safe in healthy kidneys; caution with pre-existing renal impairment.

Who should consult a doctor first

- Anyone with a diagnosed or suspected mitochondrial disorder.
- Anyone taking prescription medication — particularly anticoagulants, glucose-lowering agents, immunosuppressants, thyroid hormone, or antihypertensives.
- Type 1 diabetes, or type 2 diabetes on insulin or sulfonylureas.

- Pregnancy, planned pregnancy, or breastfeeding.
- History of eating disorders or disordered eating.
- Chronic kidney disease, liver disease, or a history of kidney stones.
- Cardiovascular disease, uncontrolled hypertension, or arrhythmia (relevant to HIIT, sauna, and cold exposure).
- Anyone over 70, frail, or with unintentional weight loss.
- Anyone with a diagnosed autoimmune or inflammatory condition.
- Adolescents and children — this plan is written for adults.

SECTION 10

Sample Recipes and Shopping List

These recipes are built to fit the framework: protein-forward, low glycaemic load, polyphenol-rich, and quick enough to survive a working week. Each serves two unless stated.

Eight mitochondrial-supportive recipes

1. Turmeric and spinach frittata

Six eggs, a large handful of spinach, 100 g sliced mushrooms, half a red onion, one teaspoon of ground turmeric, a pinch of black pepper, two tablespoons of olive oil. Sauté onion and mushrooms in oil until soft, add turmeric and pepper, wilt the spinach, pour over beaten eggs, and cook on low until set — or finish under a grill. *Why it fits:* choline and B12 from eggs, iron and folate from spinach, curcumin plus piperine for bioavailability, and a near-zero glycaemic load.

2. Sardine and white bean salad

Two tins of sardines in olive oil, one tin of cannellini beans (rinsed), a handful of parsley, half a red onion, lemon juice, capers, and three tablespoons of extra-virgin olive oil. Combine and allow to sit for ten minutes. *Why it fits:* one of the highest EPA/DHA-plus-CoQ10 combinations available, with legume fibre blunting the glycaemic response.

3. Miso and bone-broth soup with greens

500 ml bone broth or vegetable stock, one tablespoon of white miso, a thumb of grated ginger, pak choi or spinach, spring onion, and a soft-boiled egg. Warm the broth, whisk in the miso off the heat, add greens and egg. *Why it fits:* a gentle way to break a 16-hour fast — protein, glycine, and electrolytes without a glucose spike.

4. Walnut and berry breakfast bowl

150 g full-fat Greek yoghurt or coconut yoghurt, 80 g mixed berries, 20 g walnuts, one tablespoon of ground flaxseed, cinnamon. *Why it fits:* ellagitannins for urolithin A production, omega-3 ALA, polyphenols, and a low glycaemic load relative to cereal-based breakfasts.

5. Grass-fed beef and broccoli stir-fry

300 g thinly sliced grass-fed beef, one head of broccoli, a red pepper, garlic, ginger, tamari, and a teaspoon of toasted sesame oil. Sear the beef hot and fast, remove, then stir-fry the vegetables and combine. *Why it fits:* carnitine, CoQ10, iron, and B12 from beef; sulforaphane precursors from broccoli.

6. Roasted cauliflower with tahini and pomegranate

One head of cauliflower in florets, olive oil, cumin, salt; roasted at 200 °C for 25 minutes. Dress with tahini thinned with lemon and water, and scatter with pomegranate seeds and parsley. *Why it fits:* ellagitannins from pomegranate, sulphur compounds from cruciferous vegetable, and a very low glycaemic load.

7. Green tea and matcha fat bombs (no-bake)

100 g coconut oil, 40 g almond butter, two teaspoons of matcha, a pinch of salt; blended, poured into small moulds, and chilled. *Why it fits:* a practical way to take fat-soluble cofactors (CoQ10) alongside a polyphenol source, and a useful tool during the eating window when hunger is high.

8. Lentil and kale soup with olive oil

200 g green lentils, one onion, two carrots, two garlic cloves, a bunch of kale, a litre of stock, three tablespoons of olive oil, lemon. Simmer until the lentils are tender, stir the kale through at the end. *Why it fits:* folate, magnesium, and fibre; a plant-protein anchor meal that keeps glycaemic response flat.

Master weekly shopping list template

WEEKLY TEMPLATE — ADJUST QUANTITIES TO HOUSEHOLD SIZE

Category	Items
Protein	Oily fish ×3 (salmon, sardines, mackerel); white fish ×1; grass-fed beef or lamb ×2; pasture-raised chicken ×2; eggs ×18; tinned sardines ×3
Vegetables (non-starchy)	Spinach, rocket, kale, broccoli, cauliflower, cabbage, courgette, peppers, mushrooms (crimini/shiitake), asparagus, onions, garlic, ginger
Fruit	Blueberries, raspberries, blackberries, strawberries, pomegranate, lemons, avocado ×4, kiwi
Fats	Extra-virgin olive oil, coconut oil, MCT oil, tahini, almond butter, olives, walnuts, almonds, pumpkin seeds, sunflower seeds, flaxseed
Fermented & pantry	Kimchi or sauerkraut, kefir or Greek yoghurt, miso, tamari, apple cider vinegar, lentils, chickpeas, cannellini beans, tinned tomatoes, turmeric, black pepper, cumin, cinnamon, matcha, green tea, 85% dark chocolate
Optional supplements	Only what your clinician has approved — see Section 4 for dose ranges and interactions

SECTION 11

Conclusion and Long-Term Maintenance

> Thirty days is long enough to change what you eat, when you eat, and how you train. It is not long enough to rebuild a mitochondrial network. The value of the reset is that it establishes the four inputs — nutrient density, fasting rhythm, exercise stimulus, and recovery — in a sequence that a person can actually sustain, and produces early enough feedback to make continuing worthwhile.

The five takeaways worth keeping

1. **Timing is a nutrient.** A 16:8 window is the most accessible lever for the mitophagy pillar, and it costs nothing.
2. **Exercise is not optional.** No supplement in Section 4 substitutes for the biogenesis signal that HIIT and resistance training provide. Nutrition supports the adaptation; it does not cause it.
3. **Remove before you add.** Eliminating refined sugar, seed oils, and ultra-processed food does more for mitochondrial function than any single supplement on the list.
4. **Match the evidence to the claim.** Urolithin A, omega-3, creatine, and CoQ10 have meaningful human data. PQQ, resveratrol, and spermidine are largely animal and mechanistic stories. Neither group is worthless, but they are not equivalent.
5. **Measure, don't assume.** Fasting insulin, HOMA-IR, hs-CRP, and the triglyceride/HDL ratio will tell you whether the plan is working. Subjective energy ratings will tell you whether you can keep doing it.

Beyond day 30

The maintenance version of this plan is roughly 70% of the intensity. Keep the eating window on most days, keep protein at every meal, keep the 8-plus vegetable servings, and keep two resistance sessions and one or two interval sessions per week. Relax the strict elimination — most people find that two or three "off-plan" meals a week do not undo the metabolic adaptations, while a full return to the previous pattern erases them within weeks. Re-run the Section 8 labs at three and six months. If you want to repeat a full 30-day reset, once or twice a year is a reasonable rhythm.

The honest framing is this: mitochondrial capacity is one of the more plastic systems in the body, and diet, fasting, and exercise are the three strongest inputs available to you. They are also inexpensive, unpatentable, and — unlike most of what is sold in the longevity market — supported by a substantial body of human data. The 30-day structure exists to get you started, not to finish the job.

SECTION 12

References

Sources are listed alphabetically by lead author or issuing organisation. Inline citations throughout this document link to the same items. Where a source is a consumer-facing guide rather than peer-reviewed literature, that is noted.

1. Analytical Science Journals (Wiley). Curcumin bioavailability, NF-κB inhibition, and piperine co-administration. analyticalsciencejournals.onlinelibrary.wiley.com
2. EatingWell. *30-Day MIND Diet Plan* — practical meal-planning template for cognitive-supportive eating. eatingwell.com
3. Get Healthspan. *Fasting-Mimicking Diet: what it is and how to do it* (consumer guide). gethealthspan.com
4. Goldman Laboratories. *Dietary sources of NAD+ precursors* (technical note).
5. Jinfiniti Precision Medicine. *Mitochondrial supplements guide* — dosing ranges and mechanisms. jinfiniti.com
6. Linus Pauling Institute, Oregon State University. *Coenzyme Q10*, Micronutrient Information Center. lpi.oregonstate.edu
7. MitoAction. *The mitochondrial cocktail* — patient and clinician guidance. mitoaction.org
8. Mito Foundation (Australia). *Nutrition and mitochondrial disease* — clinical nutrition guide. mito.org.au
9. Mount Sinai Health System. *30-Day Meal Plan* (patient education PDF). mountsinai.org
10. NIH Office of Dietary Supplements. *Carnitine — Health Professional Fact Sheet*. ods.od.nih.gov
11. Rupa Health. *The Mito Food Plan: a comprehensive guide* — clinical framework for mitochondrial nutrition. rupahealth.com
12. Superpower. *Spermidine foods guide* — dietary spermidine content (consumer guide). superpower.com
13. USC Leonard Davis School of Gerontology, Longevity Institute. *Fasting-mimicking diet and markers of biological age*. longevity.usc.edu
14. PMC3961091. Omega-3 fatty acid incorporation into mitochondrial membranes and effects on membrane function. pmc.ncbi.nlm.nih.gov/articles/PMC3961091
15. PMC4262417. Berberine, AMPK activation, and metabolic regulation. pmc.ncbi.nlm.nih.gov/articles/PMC4262417
16. PMC6485371. Magnesium and mitochondrial function. pmc.ncbi.nlm.nih.gov/articles/PMC6485371
17. PMC7138948. Resveratrol, SIRT1, and PGC-1α signalling. pmc.ncbi.nlm.nih.gov/articles/PMC7138948
18. PMC7749612. Fasting, autophagy, and mitophagy. pmc.ncbi.nlm.nih.gov/articles/PMC7749612
19. PMC7774245. Mitochondrial cocktail dosing in mitochondrial disease. pmc.ncbi.nlm.nih.gov/articles/PMC7774245
20. PMC9176307. High-intensity interval training protocols and physiological adaptation. pmc.ncbi.nlm.nih.gov/articles/PMC9176307
21. PMC9542544. Effects of diet on mitochondrial function. pmc.ncbi.nlm.nih.gov/articles/PMC9542544
22. PMC9762714. Ketogenic diet and mitochondrial function. pmc.ncbi.nlm.nih.gov/articles/PMC9762714
23. PMC9858309. Ellagitannins, urolithins, and gut microbial metabolism. pmc.ncbi.nlm.nih.gov/articles/PMC9858309
24. PMC10346988. EGCG safety, acceptable daily intake, and hepatotoxicity thresholds. pmc.ncbi.nlm.nih.gov/articles/PMC10346988
25. PMC10377404. Fasting-mimicking diet in Alzheimer's disease models and clinical contexts. pmc.ncbi.nlm.nih.gov/articles/PMC10377404
26. PMC10721691. Creatine supplementation and brain function. pmc.ncbi.nlm.nih.gov/articles/PMC10721691
27. PMC11205942. Nicotinamide mononucleotide (NMN) supplementation: human trial review. pmc.ncbi.nlm.nih.gov/articles/PMC11205942
28. PMC11505271. Alpha-lipoic acid: mechanisms and clinical evidence. pmc.ncbi.nlm.nih.gov/articles/PMC11505271
29. PMC11541945. Pyrroloquinoline quinone (PQQ) and mitochondrial biogenesis. pmc.ncbi.nlm.nih.gov/articles/PMC11541945
30. PMC11990101. Mediterranean diet and mitochondrial function. pmc.ncbi.nlm.nih.gov/articles/PMC11990101
31. PMC12043657. HIIT versus moderate-intensity continuous training: mitochondrial outcomes. pmc.ncbi.nlm.nih.gov/articles/PMC12043657
32. PMC12476002. Dietary interventions for mitochondrial dysfunction. pmc.ncbi.nlm.nih.gov/articles/PMC12476002
33. PMC12899804. Time-restricted eating protocols and metabolic outcomes. pmc.ncbi.nlm.nih.gov/articles/PMC12899804
34. Singh, A. et al. *Urolithin A supplementation and muscle endurance: a randomised controlled trial*. Nature Aging. nature.com/articles/s43587-025-00996-x

End of document. This guide synthesises published reviews, institutional fact sheets, and consumer-facing clinical frameworks. Its sample menus and recipes are original constructions that apply the principles described, not reproductions of a validated protocol. Doses are literature-derived ranges, not personal prescriptions. If you take prescription medication, have a diagnosed mitochondrial disorder, or are uncertain whether fasting or supplementation is appropriate for you, consult a qualified healthcare professional before beginning any part of this plan.