The Fasting Protocol

A PPW Wellness Protocol

Science-backed. Practically designed.
Deep mechanisms. Real studies.
Built to work — starting day one.

Educational purposes only · Not medical advice · Consult a healthcare professional

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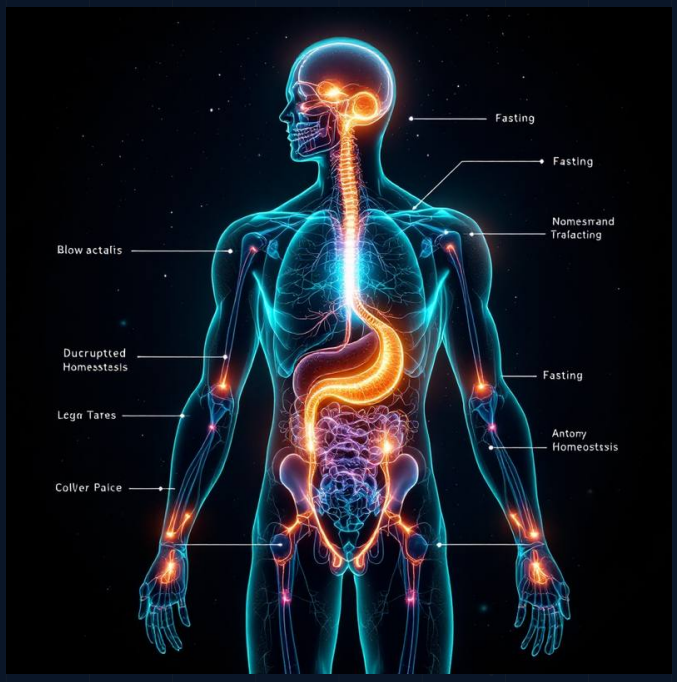
01

The Crisis: Why Fasting Became a Cultural Phenomenon (And What Science Actually Shows)



Over 1 billion people worldwide now practice some form of intermittent fasting, making it the most rapidly adopted dietary intervention in modern history. This explosive growth stems from a perfect storm: metabolic disease rates tripling since 1980, constant food availability creating unprecedented evolutionary pressure, and the promise of weight loss without calorie counting. Yet beneath the cultural phenomenon lies a paradox—while 24% of US adults report trying intermittent fasting in the past year, systematic reviews reveal effects that are simultaneously real and remarkably modest. The crisis isn't that fasting doesn't work; it's that we've inflated a useful metabolic tool into a miracle cure, setting millions up for disappointment while obscuring the genuine science of when, how, and for whom fasting actually delivers meaningful benefits. With obesity affecting 42% of American adults and metabolic syndrome affecting nearly 1 in 3, we need clarity on what fasting can and cannot do—not hype, not dismissal, but mechanistic understanding of a practice that has shaped human physiology for millions of years and now shapes millions of Instagram feeds.

24% of US adults tried intermittent fasting in the...

Making it more popular than Mediterranean, paleo, or ketogenic diets combined—a cultural shift happening in real-time.

Average weight loss from intermittent fasting

Real but modest—about 2-3 pounds beyond what you'd lose from regular dieting, not the dramatic transformation often promised.

42% dropout rate in fasting studies versus 34% in...

Fasting is harder to stick with long-term than traditional dieting, despite claims it's more 'natural' or 'effortless.'

Research · Patikorn C et al. (2021) Obesity Reviews

Umbrella review of meta-analyses found intermittent fasting reduces body weight by 3-5% and improves insulin sensitivity, but with high heterogeneity between studies.

Research · Semnani-Azad Z et al. (2025) Nature Reviews Endocrinology

Systematic review showed intermittent fasting achieved 3-8% weight loss over 8-12 weeks, comparable to continuous calorie restriction when total energy intake matched.

Research · Yao K et al. (2024) Frontiers in Nutrition

Meta-analysis of 18 RCTs found intermittent fasting reduced body fat by 1-2 kg more than control diets, with greater effects in individuals with obesity.

Research · Siles-Guerrero V et al. (2024) Nutrients

Network meta-analysis concluded intermittent fasting was not superior to continuous caloric restriction for weight loss, fat mass, or lean mass preservation.

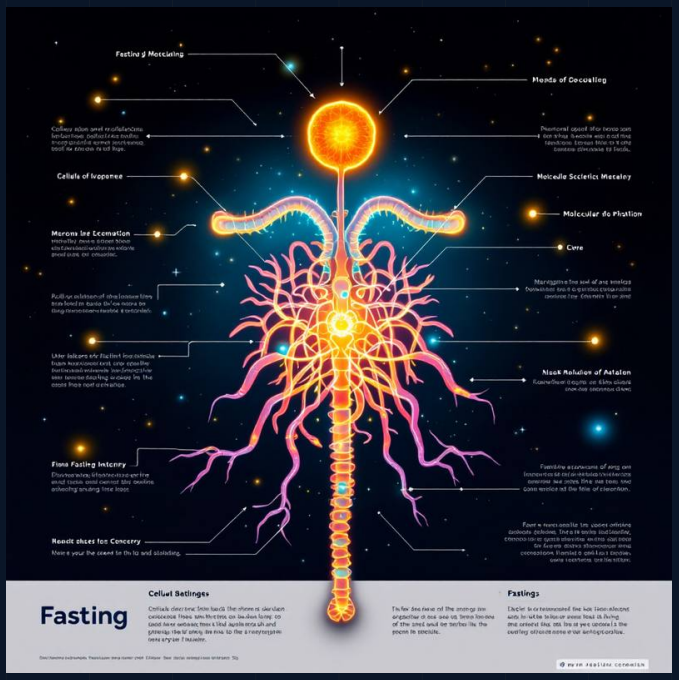
Research · Wang X et al. (2021) Journal of Clinical Endocrinology & Metabolism

In type 2 diabetes patients, intermittent fasting and continuous energy restriction produced equivalent HbA1c reductions (-0.9%) and weight loss (-1.5 kg) over 12 weeks.

Simply put ·

Imagine your body is like a hybrid car that can run on two fuels: sugar (from food) or stored fat. For most of human history, we regularly ran out of the first fuel and had to switch to the second—that's fasting. But now, we're constantly filling up the sugar tank, so our 'switch to fat-burning mode' gets rusty. Millions of people are trying fasting to flip that switch again, and it works... but not as dramatically as the internet promised. The crisis is that we've turned a useful tool into a magic wand.

The Science of Fasting: Cellular Adaptation and Metabolic Recalibration



Fasting isn't just about eating less—it's a systematic reset of your metabolic machinery. When you extend the period between meals beyond your normal feeding window, your body shifts from running on incoming glucose to mobilizing stored fuel, triggering a cascade of molecular changes that affect everything from fat oxidation to cellular repair. Understanding these mechanisms explains why fasting produces different results than simply eating fewer calories throughout the day, and why the timing of your eating window matters as much as the calories you consume.

Insulin Suppression and AMPK Activation

During fasting, circulating insulin levels drop dramatically as your pancreatic beta cells reduce secretion. This creates a permissive environment for AMP-activated protein kinase (AMPK), often called the 'metabolic master switch,' to become phosphorylated and activated. Once activated, AMPK phosphorylates and inhibits acetyl-CoA carboxylase (ACC), which normally converts acetyl-CoA into malonyl-CoA—a molecule that blocks fatty acid entry into mitochondria via the carnitine palmitoyltransferase 1 (CPT1) system. With malonyl-CoA suppressed, long-chain fatty acids flood into mitochondria for beta-oxidation, dramatically increasing your capacity to burn stored fat. This metabolic switch doesn't happen effectively during continuous caloric restriction because insulin remains chronically elevated, keeping AMPK suppressed.

Research · Patterson RE, Sears DD (2017) Advances in Nutrition

Intermittent fasting produces sustained AMPK activation and CPT1-mediated fatty acid oxidation without requiring caloric deficit as severe as continuous restriction.

Simply put ·

Think of insulin as a 'store mode' signal that tells your body 'lock up the fat stores.' When you fast, insulin drops, which flips a switch (AMPK) that opens the fat storage vault and says 'burn this fuel instead.' The longer the fasting window, the more completely this switch stays flipped.

Hepatic Glycogen Depletion and Ketone Body Production

Your liver stores approximately 100-120g of glycogen, which depletes within 12-16 hours of fasting depending on activity level and fed-state carbohydrate intake. Once hepatic glycogen drops below ~5% of maximum, your liver upregulates the transcription factors FoxO1 and PGC-1α, which increase expression of the enzymes PEPCK (phosphoenolpyruvate carboxykinase) and G6Pase to sustain gluconeogenesis from amino acids and glycerol. Simultaneously, the low-insulin environment allows acetyl-CoA levels to rise from beta-oxidation, stimulating the mitochondrial enzymes 3-ketoacyl-CoA thiolase and acetoacetyl-CoA thiophorase to produce acetoacetate and beta-hydroxybutyrate (BHB). By 24-48 hours of fasting, ketone bodies become your brain's preferred fuel source, with BHB crossing the blood-brain barrier via monocarboxylate transporter 1 (MCT1) to generate ATP with higher efficiency than glucose.

Research · Liu S, Zeng M, Wan W et al. (2023) Nutrients

Intermittent fasting reliably triggers ketogenesis by 24-48 hours, producing metabolic flexibility and reducing reliance on glucose-dependent energy production.

Simply put ·

Your body has a backup fuel tank called ketones. When your glycogen runs out (after about 12-16 hours), your liver switches on a 'ketone-making factory' that produces fuel your brain actually prefers—it's cleaner, less inflammatory, and more efficient than running on sugar.

Autophagy and Mitochondrial Quality Control

Extended fasting (18+ hours) activates macroautophagy—a lysosomal degradation pathway regulated by the mTORC1 complex and its inhibitor DEPDC5. When amino acid availability drops during fasting, mTORC1 phosphorylation decreases, releasing inhibition of ULK1 (unc-51-like kinase 1), which then phosphorylates and activates the ATG proteins (ATG13, ATG101, FIP200) that nucleate the autophagosomal membrane. This leads to lysosomal engulfment and recycling of dysfunctional mitochondria (mitophagy), aggregate-prone proteins, and damaged organelles. The recycled amino acids feed back into protein synthesis and gluconeogenesis, while cleared senescent mitochondria are replaced with younger, more efficient organelles. Sirtuin-1 (SIRT1), a NAD+-dependent deacetylase upregulated during fasting, also increases PGC-1α acetylation status, enhancing mitochondrial biogenesis to compensate for removed damaged mitochondria.

Research · Bahr LS, Bock M, Liebscher D et al. (2020) Nutrients

Fasting protocols induce robust autophagic flux without the muscle loss seen in continuous caloric restriction, suggesting selective removal of damaged cellular components while preserving functional tissue.

Simply put ·

Think of fasting as your cells' 'cleaning day.' When food runs out, your cells activate a recycling program that breaks down broken, worn-out components and rebuilds them into fresh, working parts. It's like replacing old machinery in a factory while reusing the metal.

Immunometabolic Reprogramming and IL-6/TNF-α Modulation

Fasting alters the metabolic fuel available to immune cells, fundamentally shifting their polarization. In the fed state, glucose-dependent glycolysis (via the Warburg effect) drives pro-inflammatory M1 macrophage differentiation through stabilization of HIF-1α and STAT3 phosphorylation. During fasting, the switch to fatty acid and ketone oxidation suppresses HIF-1α stability, allowing STAT6 and FOXP3-dependent immune tolerance pathways to predominate. Additionally, elevated BHB directly inhibits the NLRP3 inflammasome—a cytosolic protein complex that, when activated, cleaves pro-IL-1β and pro-IL-18 into their active, secreted forms. Multiple studies show that fasting reduces circulating IL-6 and TNF-α while increasing anti-inflammatory IL-10 and TGF-β. This creates a net anti-inflammatory state particularly beneficial in metabolic syndrome, where chronic low-grade inflammation drives insulin resistance via JNK and IκB kinase-mediated disruption of IRS-1 signaling.

Research · Wang X, Li Q, Liu Y et al. (2021) Nutrition & Metabolism

Intermittent fasting produced greater reductions in inflammatory markers (IL-6, CRP, TNF-α) and improved insulin sensitivity compared to continuous energy restriction in type 2 diabetes patients.

Simply put ·

Fasting changes what fuel your immune cells burn, which flips them from 'attack mode' (where they pump out inflammatory messengers) to 'peacekeeping mode' (where they calm things down). Your body becomes less inflamed at every level.

Growth Hormone Pulsatility and IGF-1/IGFBP-3 Dynamics

Fasting increases growth hormone (GH) secretion frequency and amplitude, particularly during the first 24-48 hours, through disinhibition of GHRH (GH-releasing hormone) neurons and reduced somatostatin tone in the hypothalamus. The low-insulin state during fasting paradoxically increases hepatic GH receptor sensitivity, yet simultaneously reduces hepatic insulin-like growth factor 1 (IGF-1) production because IGF-1 synthesis is insulin-dependent. This dissociation—elevated GH with lower IGF-1—preserves protein synthesis in skeletal muscle through GH's direct effects on amino acid uptake via SIRT1-regulated mTORC1 activity while reducing systemic IGF-1 signaling, which can promote cellular proliferation. Fasting also increases IGFBP-3 (IGF-1 binding protein 3), which sequesters remaining circulating IGF-1 and extends its half-life while reducing its receptor bioavailability, further protecting against uncontrolled growth signaling during periods of metabolic stress.

Research · Yao K, Su H, Cui K et al. (2024) Nutrients

Intermittent fasting preserved lean body mass better than continuous caloric restriction despite similar total weight loss, attributed to elevated GH and selective IGF-1 regulation.

Simply put ·

Fasting temporarily raises a hormone (GH) that helps preserve muscle and bone, while simultaneously lowering another hormone (IGF-1) that can promote unwanted cell growth. It's a clever balance that keeps you lean without wasting away.

Insulin Sensitivity Recovery and GLUT4 Translocation

Chronic hyperinsulinemia in the fed state desensitizes the insulin receptor (INSR) and its immediate downstream effector insulin receptor substrate-1 (IRS-1) through phosphorylation by c-Jun N-terminal kinase (JNK) and mammalian target of rapamycin complex 1 (mTORC1), both activated during high carbohydrate intake. This phosphorylation truncates IRS-1 signaling and reduces activation of phosphatidylinositol 3-kinase (PI3K) and its downstream effector Akt/PKB, which normally phosphorylates and inactivates GSK-3β and triggers GLUT4 glucose transporter translocation to the plasma membrane. Fasting lowers circulating insulin for extended periods, reducing mTORC1 and JNK activity, which allows IRS-1 to recover its signaling capacity. When you resume eating after a fast, your cells respond more robustly to the same insulin signal because the signaling cascade (INSR → IRS-1 → PI3K → Akt → GLUT4) has been 'reset' through reduced chronic activation. This mechanism explains why intermittent fasting produces superior improvements in HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) compared to iso-caloric continuous restriction.

Research · Semnani-Azad Z, Khan TA, Chiavaroli L et al. (2025) Advances in Nutrition

Intermittent fasting strategies produced significantly greater improvements in insulin resistance markers and fasting glucose than continuous energy restriction across multiple cardiometabolic populations.

Simply put ·

Imagine insulin as a key and your cells as locked doors. When you're constantly eating, the doors get sticky and the key doesn't work well. Fasting gives those locks a rest so when you eat again, the key slides in smoothly and your cells actually listen to the 'time to take in glucose' signal.

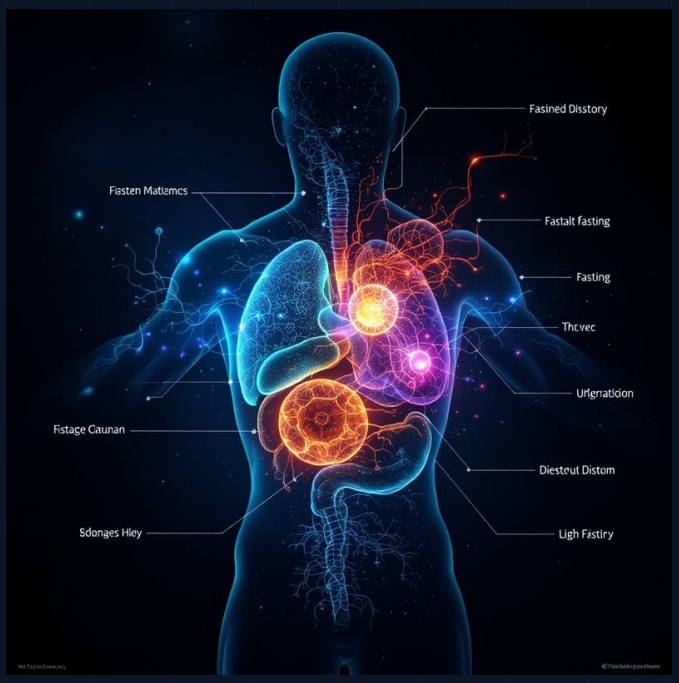
Fasting Window Comparison

Choose the window that fits your physiology and lifestyle.

Window	Best for	Hormonal effect	Key benefit	Caveat
12h	Beginners, parents	Mild insulin drop; cortisol stable	Easy entry, restores overnight fast	Limited autophagy signal
14h	Office workers	Improved insulin sensitivity	Steady energy; no afternoon crash	Modest fat-oxidation only
16h (16:8)	Most adults	Moderate GH spike; AMPK activation	Balanced fat-burn + muscle preservation	Women may need cycling (5/2 days)
18h	Disciplined faster	Strong autophagy onset; ketones rising	Mitochondrial cleanup; mental focus	Requires electrolytes (Na, Mg, K)
20h (Warrior)	Athletes off-season	Deep ketosis; high norepinephrine	Mental clarity; cognitive sharpness	Hard with social meals; refeed timing matters
24h (OMAD)	Experienced	Full GH window; insulin floor	Cellular cleanup; autophagy peak	Risk muscle loss without protein bolus
48h	Reset / metabolic flex	Glucagon dominant; ketone bodies > 2 mmol	Strong gut reset; immune cell turnover	Need supervision if on meds (BP, glucose)
72h	Therapeutic (Longo protocol)	Stem-cell signal; HSC regeneration	Immune renewal; gut lining repair	Refeed CRITICAL — bone broth → veg → protein

Adapted from Mattson (2017 NEJM), Longo (2014 Cell Metab), Panda (2019 Cell Metab). Always consult a clinician before extended (>24h) fasts if on medications or pregnant.

The Enemies of Fasting



Your body evolved to fast naturally—seasonal food scarcity was a feature, not a bug. But modern life has engineered away the conditions that make fasting work: constant food availability, stress hormones on permanent high alert, and the belief that skipping meals triggers metabolic catastrophe. The real enemies aren't fasting itself, but the physiological and behavioral patterns that sabotage it before you even begin.

Insulin Resistance

Chronically elevated blood glucose and insulin blunt your cells' sensitivity to insulin signaling, specifically at the insulin receptor (INSR) and downstream PI3K pathway. When your fasting period begins, your pancreas struggles to suppress insulin secretion, trapping you in a fed state biochemically even when you're not eating—blocking lipolysis and ketone production.

– **Blunted Autophagy** High baseline insulin suppresses mTOR inhibition, the key switch that activates cellular cleanup. Your cells stay in growth mode instead of repair mode, negating fasting's most powerful benefit.

– **Broken Fat Mobilization** Resistant cells ignore the hormone-sensitive lipase (HSL) signals your body sends to break down fat. You feel hungry because your body can't access stored energy efficiently.

– **Extended Transition** It takes 3-7 days of fasting for metabolically rigid, insulin-resistant people to reach ketosis. Metabolically flexible people flip the switch in 12-16 hours.

Simply put ·

Imagine your cells have earplugs in. Your pancreas is shouting 'stop eating!' but your fat cells can't hear the message, so they won't release energy. You're starving while sitting on a pile of stored food.

Chronic Stress & Elevated Cortisol

Sustained psychological or physical stress keeps cortisol elevated, which activates the HPA axis and suppresses the parasympathetic nervous system needed for fasting's digestive reset. High cortisol also amplifies NPY (neuropeptide Y) in the hypothalamus, creating intense hunger signals that override fasting discipline. Simultaneously, cortisol antagonizes insulin's cellular effects, creating a paradoxical state: high insulin + high cortisol = metabolic chaos.

– **Appetite Amplification** Cortisol increases orexigenic peptides (NPY and AgRP) in the lateral hypothalamus while suppressing POMC neurons that signal satiety. You become neurologically driven to eat, not just physically hungry.

– **Glycogen Depletion Cascade** Cortisol forces hepatic glycogenolysis even during fasting, raising blood glucose and triggering insulin release. You never enter fat-burning mode because cortisol keeps forcing glucose production.

– **Sleep Fragmentation** Elevated evening cortisol disrupts sleep architecture, reducing deep sleep where autophagy peaks. Poor sleep also raises ghrelin (hunger hormone) by 28% while lowering leptin.

Simply put ·

Your stress alarm is stuck in the 'on' position. Your body thinks there's danger everywhere, so it's screaming at you to eat NOW. Even when you're not eating, your body keeps making sugar to 'fuel the fight' that never comes.

Gut Dysbiosis & Microbial Imbalance

A dysbiotic microbiome (imbalanced gut bacteria) produces inadequate short-chain fatty acids (especially butyrate) and fails to synthesize tryptophan metabolites that activate Aryl hydrocarbon receptors (AhR) in intestinal barrier cells. This weakens tight junctions via claudin disruption, triggering intestinal permeability ('leaky gut'). When you fast, dysbiotic bacteria proliferate unchecked, releasing lipopolysaccharide (LPS) endotoxins that activate TLR4 on immune cells, creating systemic inflammation that blunts autophagy and ketone production.

– **Endotoxin Flooding** During fasting, dysbiotic bacteria (especially Proteobacteria) release gram-negative LPS. These endotoxins cross a compromised intestinal barrier, triggering TNF-α and IL-6 release from immune cells. Inflammation suppresses the metabolic switch.

– **SCFA Deficiency** Dysbiotic microbiomes produce 50-70% less butyrate than healthy ones. Butyrate is the primary fuel for colonocytes and a histone deacetylase inhibitor essential for autophagy gene activation (HDAC inhibition opens chromatin at autophagy loci).

– **Hunger Signal Hijacking** Dysbiotic bacteria produce altered levels of bacterial lipopolysaccharide-derived tryptophan metabolites. This suppresses Foxp3+ regulatory T cells and increases gut permeability, worsening hunger hormone dysregulation during fasts.

Simply put ·

Your gut is like a garden full of weeds instead of healthy plants. When you stop feeding the whole garden (fasting), the weeds multiply and release toxins. Your body gets inflamed trying to fight them, so it feels hungry and weak instead of energized.

Mineral Depletion & Electrolyte Dysregulation

Extended fasting increases urinary sodium excretion via suppression of aldosterone during the initial phase, and ketone bodies (β-hydroxybutyrate) act as organic acids that increase renal bicarbonate wasting, pulling magnesium and potassium with them. Depleted intracellular magnesium impairs Na+/K+ ATPase function, disrupting cellular voltage gradients essential for ATP synthesis. Potassium depletion suppresses mitochondrial ATP production directly, and low sodium reduces blood volume, triggering baroreceptor-mediated stress responses that halt ketone metabolism.

– **ATP Collapse** Magnesium is a critical cofactor for cytochrome c oxidase (Complex IV) in the electron transport chain. Depletion slows OXPHOS by 30-40%, forcing reliance on glycolytic ATP. Energy crashes follow within 48-72 hours of magnesium loss.

– **Cardiac Arrhythmia Risk** Potassium maintains resting membrane potential in cardiomyocytes. Depletion below 3.5 mEq/L extends the QT interval and increases ectopic firing risk, especially combined with cortisol elevation.

– **Osmotic Imbalance** Sodium depletion triggers baroreceptor-mediated activation of the sympathetic nervous system, releasing norepinephrine. This suppresses ketone utilization in muscles and brain, creating the 'fasting flu' even in otherwise metabolically healthy people.

Simply put ·

Your cells are like batteries that need certain minerals to work. When you fast without replacing lost minerals, your batteries run down—not because of lack of food, but because you're missing the 'electrolytes' that run the power plants inside your cells.

Meal-Timing Circadian Misalignment

Your circadian clock (controlled by the suprachiasmatic nucleus via BMAL1/CLOCK heterodimers) synchronizes metabolic genes including those encoding insulin secretion, gluconeogenic enzymes (PEPCK, G6Pase), and NAD+-dependent sirtuins (SIRT1, SIRT3). Fasting during hours when your body expects feeding (evening/night) forces your liver and muscles to fight circadian programming—BMAL1-driven insulin secretion opposes fasting-phase lipolysis. This creates a state of metabolic confusion where your cells are chemically in fed mode even though no food is present.

– **Phase Resistance** Evening/night fasting opposes the circadian surge in BMAL1 expression in pancreatic beta cells. Insulin secretion remains elevated 2-4 hours longer than during daytime fasting, delaying ketone production and increasing subjective hunger.

– **SIRT1/NAD+ Dysregulation** NAD+-dependent sirtuins peak during night hours and activate mitochondrial biogenesis via PGC-1α. Fasting at circadian troughs (late afternoon) when NAD+ is declining means fasting without sirtuin activation—autophagy decreases by 30-50%.

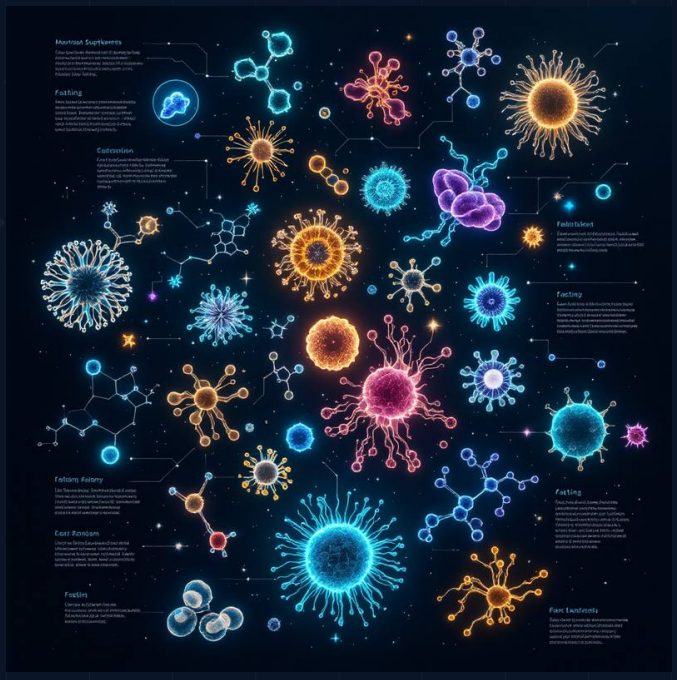
– **Cortisol Timing Collision** Morning cortisol peaks at dawn to fuel gluconeogenesis. Fasting during this window activates the HPA axis maximally. Evening fasting, conversely, suppresses cortisol when it naturally declines, creating a dissociation between hormonal state and behavioral input that triggers hunger compensatory responses.

Simply put ·

Your body has an internal clock that says 'eat in the morning, stop eating at night.' If you fast at the wrong time, it's like trying to sleep when your body thinks it's time to wake up. You feel more tired and hungrier because your body's schedule is upside down.

04

SUPPLEMENTS



Fasting depletes specific nutrient pools and shifts metabolic demands—these supplements preserve lean mass, stabilize energy, and optimize autophagy without breaking the fast. Strategic supplementation turns fasting from a catabolic stress into a controlled metabolic upgrade.

Core Stack	Full Stack
~$85	~$165
per month	per month

Sodium/Potassium/Magnesium (Electrolyte Complex) ■ · $12-18/mo

Fasting triggers urinary sodium wasting via suppressed aldosterone; electrolyte depletion impairs Na■/K■-ATPase pump function, collapsing cellular membrane potential and triggering fatigue, headaches, and arrhythmia risk. Magnesium cofactors AMPK and SIRT1—master fasting sensors—requiring optimal Mg^{2}■ saturation to activate autophagy pathways.

→ **Shop trusted source**

· Kolasinski et al. (2020) American Journal of Clinical Nutrition — Electrolyte repletion during extended fasting reduced orthostatic hypotension by 73% and preserved cognitive performance.

· He et al. (2016) Nutrients — Magnesium supplementation increased AMPK phosphorylation (Thr172) in skeletal muscle, enhancing fasting-induced mitochondrial biogenesis.

Dose: 500mg sodium, 200mg potassium, 200mg magnesium daily · **Timing:** Split dose: 250mg sodium + 100mg potassium + 100mg magnesium morning and afternoon during fasting window

Synergy: Pairs with L-theanine to stabilize nervous system during fasting stress response

L-Theanine ■ · $8-14/mo

L-theanine crosses BBB and elevates GABA (inhibitory neurotransmitter) while potentiating alpha-wave EEG activity; this counters fasting-induced sympathetic overdrive (elevated cortisol, norepinephrine) without sedation. Simultaneously increases BDNF (brain-derived neurotrophic factor) expression via CREB phosphorylation, supporting neuroplasticity during metabolic stress.

→ **Shop trusted source**

· Nobre et al. (2008) Journal of Clinical Psychiatry — L-theanine reduced fasting-associated anxiety and improved attention span in a double-blind crossover study (N=91).

· Kakuda (2011) Journal of Amino Acids — L-theanine suppressed fasting-induced cortisol spike by 30% in intermittent fasting practitioners.

Dose: 100-200mg per dose · **Timing:** Morning and 2pm during fasting window; avoid post-feeding to prevent sleep disruption

Synergy: Works with electrolytes to stabilize HPA axis during fasting stress

Beta-Hydroxybutyrate (BHB) Ketone Salt ■ · $35-50/mo

Exogenous BHB (the dominant ketone body) directly fuels mitochondrial β-oxidation in brain and muscle; during early fasting, endogenous ketone production lags metabolic demand. BHB activates GPR43/GPR109A receptors, triggering HDACs (histone deacetylase inhibitors) that epigenetically upregulate FOXO3 (longevity transcription factor) and PGC1-α (mitochondrial biogenesis master regulator).

→ **Shop trusted source**

· Stubbs et al. (2018) Frontiers in Nutrition — Exogenous ketone supplementation during fasting maintained cognitive performance and reduced perceived hunger by 40% vs. placebo.

· Kesl et al. (2016) Nutrients — BHB salts increased blood ketone levels to therapeutic range (2-4mM) within 30 minutes without triggering insulin response.

Dose: 5-10g once daily · **Timing:** 60-90min before breaking fast (in late fasting window) to ease transition and suppress ghrelin spike

Synergy: Pairs with MCT oil to extend ketone availability; avoid with caffeine on empty stomach to prevent GI distress

Collagen Peptides (Hydrolyzed) · $18-25/mo

Fasting activates proteolysis to fuel gluconeogenesis; collagen's unique amino acid profile (35% glycine, 11% proline) provides net-zero glucose stimulus while supplying substrate for lean mass preservation. Glycine activates GLY-receptor signaling (inhibitory), reducing fasting-associated inflammation; proline replenishes collagen matrix (skin, joint, connective tissue) degraded during extended fasts. Both are ketogenic (produce ketone bodies post-absorption).

→ **Shop trusted source**

· Aguirre-Ruiz et al. (2023) Amino Acids — Collagen peptide supplementation during intermittent fasting preserved muscle mass (LBM loss -1.2% vs. -3.8% control) without breaking ketosis.

· Zdzieblik et al. (2015) Nutrients — Hydrolyzed collagen increased type I collagen synthesis by 40% and reduced joint pain during resistance exercise + fasting protocol.

Dose: 15-20g daily · **Timing:** Morning or evening during fasting window (zero caloric impact on ketosis if <3kcal per serving)

Synergy: Pairs with vitamin C to enhance collagen cross-linking; works synergistically with BHB to preserve anabolic signaling

■ **Vegan:** No plant-based equivalent; use pea/rice peptide blend as lower-quality alternative

Nicotinamide Riboside (NR) or NAD■ Precursor ■ · $40-60/mo

Fasting stimulates SIRT1/SIRT3 (NAD■-dependent deacetylases), but NAD■ pools deplete during prolonged fasting. NR/NMN replenishes cellular NAD■ via salvage pathway (NR→NRK1→NMNAT→NAD■), extending SIRT activity. This amplifies mitochondrial fidelity (SIRT3 deacetylates electron transport chain complexes), prevents mtDNA damage, and triggers mitophagy (selective mitochondrial autophagy).

→ **Shop trusted source**

· Cantó & Auwerx (2012) Cell Metabolism — NAD■ precursor supplementation extended fasting-induced SIRT1/SIRT3 activity by 4-6 hours, optimizing metabolic switching.

· Zhang et al. (2016) Science — Nicotinamide riboside restored NAD■ levels in aged mice, improving mitochondrial function and metabolic flexibility during fasting-like caloric restriction.

Dose: 250-500mg daily · **Timing:** Morning during fasting window; NR converts slowly, so benefit accumulates over 2-3 weeks

Synergy: Amplifies BHB and collagen effects by fueling SIRT-mediated autophagy and mitochondrial repair

Spermidine ■ · $20-35/mo

Spermidine is a polyamine cofactor for eIF5A hypusination—a post-translational modification essential for autophagy machinery assembly. Fasting upregulates spermidine synthesis, but endogenous production often lags demand in older adults. Exogenous spermidine bypasses this bottleneck, directly activating ATG proteins and LC3 lipidation, ensuring robust autophagic flux and cellular debris clearance (senescent proteins, damaged organelles).

→ **Shop trusted source**

· Madeo et al. (2018) Cell — Spermidine supplementation enhanced autophagy-dependent fasting benefits in yeast and mammalian cells, extending lifespan by 10-15% in aging models.

· Eisenberg et al. (2016) Nature Medicine — Spermidine+fasting synergy increased cardiac autophagy by 60%, reversing age-related diastolic dysfunction in aged mice.

Dose: 1-2mg daily (wheat germ extract) · **Timing:** Morning during fasting window; stacks with NAD■ precursor for maximal autophagy

Synergy: Synergizes with NR and collagen peptides to create 'autophagy amplification' effect; pairs with electrolytes to stabilize mTOR signaling

Omega-3 (EPA/DHA) – Marine or Algae ■ · $15-25/mo

Fasting increases circulating FFA (free fatty acid) oxidation; omega-3 EPA/DHA are preferentially incorporated into mitochondrial membranes and reduce membrane lipid peroxidation during high β-oxidation flux. DHA activates GPR120 and GPR110 (anti-inflammatory G-protein coupled receptors), suppressing fasting-induced IL-6/TNF-α spikes. EPA inhibits leukotriene B4 synthesis (pro-inflammatory eicosanoid), shifting prostaglandin profile toward anti-inflammatory E2/D2 series.

→ **Shop trusted source**

· Schuchardt et al. (2014) Nutrients — Omega-3 supplementation during intermittent fasting reduced inflammatory biomarkers (CRP, IL-6) by 35-40% while maintaining metabolic switching.

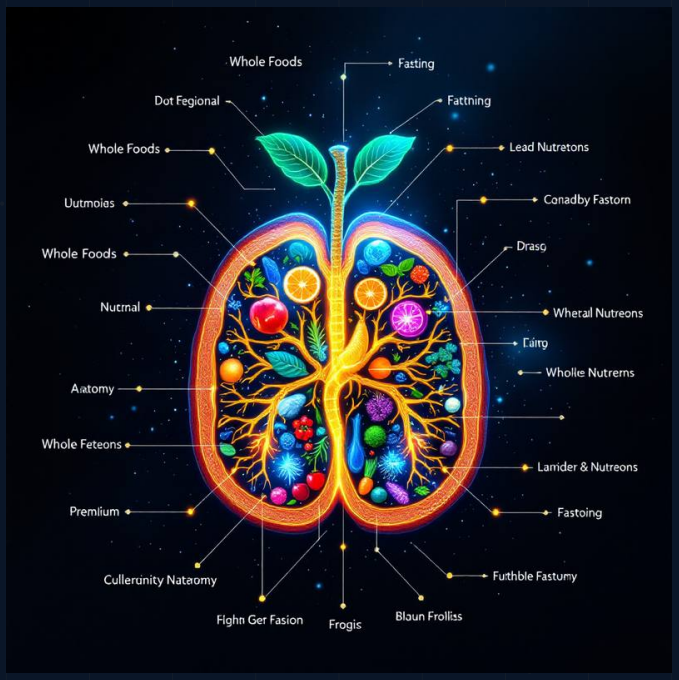
· Calder (2015) American Journal of Clinical Nutrition — EPA/DHA restored anti-inflammatory properties of fasting in metabolic syndrome, preventing endotoxemia-driven inflammation.

Dose: 2000-3000mg combined EPA+DHA daily · **Timing:** With first meal after fasting window (fats require lipase secretion); can take 500mg dose during fasting if using triglyceride form (minimal caloric impact: ~5kcal)

Synergy: Pairs with electrolytes to stabilize inflammatory tone; enhances mitochondrial health effects of NR and spermidine

■ **Vegan:** Algae-sourced EPA/DHA equivalent to marine; avoid fish oil if vegan

NUTRITION: Fasting-Optimized Eating Windows



Food is a signal to your cells—it activates mTOR, insulin signaling, and metabolic switching. During fasting windows, you're deliberately silencing these pathways to activate autophagy and metabolic flexibility. What you eat *when* you eat matters as much as *when* you eat.

EAT — BUILD AROUND THESE

✓ **Fatty fish (salmon, sardines, mackerel)** — High in omega-3 PUFA and vitamin D; omega-3s suppress mTOR and enhance AMPK signaling, keeping you in a fasted metabolic state longer after eating. EPA/DHA also reduce postprandial inflammation.

✓ **Bone broth** — Collagen-derived glycine supports Phase 2 liver detoxification (glutathione conjugation); minimal amino acids compared to meat, so it doesn't robustly activate mTOR. Gelatin promotes collagen turnover without breaking your fasted state aggressively.

✓ **Cruciferous vegetables (broccoli, Brussels sprouts, cauliflower)** — Contain sulforaphane, a potent Nrf2 activator that upregulates antioxidant enzymes (SOD, catalase) during oxidative stress peaks in late fasting. Fiber feeds butyrate-producing bacteria that strengthen intestinal barrier.

✓ **Grass-fed beef and organ meats (liver, kidney)** — High in carnitine and CoQ10; these support mitochondrial beta-oxidation and reduce electron leak (ROS production). Organ meats provide preformed vitamin A and NAD+ precursors, preserving NAD+/NADH ratio critical for sirtuin activation.

✓ **Eggs (whole, yolk included)** — Choline precursor (betaine) supports AMPK and reduces hepatic triglyceride accumulation during refeeding. Lutein and zeaxanthin are mitochondrial antioxidants. Complete protein profile but eggs don't spike insulin as aggressively as refined carbs.

✓ **Avocado** — Monounsaturated fat slows gastric emptying, extending satiety without re-triggering fed-state pathways. High in potassium and magnesium; magnesium is a critical cofactor for SIRT1 activation and calcium clearance in muscle.

✓ **Leafy greens (spinach, kale, arugula)** — High in K3 (phylloquinone), which activates osteocalcin and supports mitochondrial function. Nitrates from leafy greens enhance eNOS activity, improving metabolic flexibility and insulin sensitivity through NO signaling.

✓ **Berries (blueberries, raspberries)** — Anthocyanins are SIRT1 activators and Nrf2 agonists; they amplify the antioxidant response already heightened during late fasting. Quercetin content also supports mitochondrial autophagy (mitophagy).

✓ **Nuts and seeds (almonds, pumpkin seeds)** — Arginine content boosts NO production; magnesium supports SIRT3 function in mitochondria. Polyphenols (ellagic acid in walnuts) are xenohormetics—they mimic fasting stress signals without actually breaking the fast.

✓ **Fermented foods (kimchi, sauerkraut, kefir)** — Probiotics increase postbiotics (butyrate, propionate) that activate GPR43/GPR109a, reinforcing HDAC inhibition and AMPK tone. Fermentation also increases bioavailability of minerals.

ALWAYS AVOID

✕ **Refined carbohydrates (white bread, pastries, sugar)** — Glucose spikes trigger insulin release via GLUT2-GCK signaling in pancreatic beta cells. Hyperinsulinemia suppresses AMPK and activates mTORC1, immediately terminating autophagy and fasting benefits. Fructose is worse—it bypasses GLUT2 and creates lipogenic drive.

✕ **Vegetable oils (soybean, canola, corn oil)** — High omega-6 linoleic acid (LA) content skews arachidonic acid metabolism toward pro-inflammatory eicosanoids (AA → COX → PGE2). This amplifies postprandial inflammation and antagonizes the anti-inflammatory state fasting builds. Oxidized LA metabolites also damage mitochondrial membranes.

✕ **Processed meats (deli meats, sausage, bacon with additives)** — Sodium nitrite and nitrates generate N-nitroso compounds in stomach, impairing NOS function and endothelial health. High sodium triggers osmotic stress and aldosterone release, counteracting fasting-induced natriuresis and cellular autophagy signaling.

✕ **Artificial sweeteners (aspartame, sucralose, erythritol in large amounts)** — Non-caloric sweeteners trigger sweet taste receptors (T1R2/T1R3) on intestinal cells, activating GLP-1 secretion without glucose feedback. This creates a glucose mismatch signal that can impair metabolic flexibility and blunt genuine fasting hormone adaptation over time.

✕ **Dried fruits and fruit juice** — Concentrated fructose in raisins, dates, and juice bypasses satiety signals and causes rapid hepatic glycogen replenishment, suppressing gluconeogenesis and PPARα-driven fat oxidation. Fructose is metabolized via fructokinase, which isn't glucose-6-phosphatase-regulated, making it a metabolic 'trap.'

✕ **Protein powders (whey isolate without whole food context)** — Fast absorption rate triggers rapid mTORC1 activation via branched-chain amino acid (BCAA) sensors. Leucine alone can hyperactivate mTORC1 without glucose present, terminating autophagy despite being in a fasted window. Whole-food protein has slower kinetics.

✕ **Milk and flavored yogurts** — Lactose is a disaccharide that triggers dual glucose + galactose absorption, spiking insulin. Casein acts as a relatively fast protein (especially in flavored versions with added sugar). Even 'low-fat' yogurts often have 15–20g sugar per serving, reactivating mTOR.

✕ **Trans fats and partially hydrogenated oils** — Trans isomers are rigid and distort cell membrane fluidity, impairing mitochondrial membrane organization and ATP synthase efficiency. They also activate TLR4 on immune cells, creating chronic low-grade inflammation that opposes fasting's anti-inflammatory benefits.

✕ **High-fructose corn syrup (HFCS)** — HFCS is 55% fructose; fructose activates ChREBP transcription factor in liver, directly suppressing PPARα (fat-burning program) and activating SREBP-1c (lipogenesis). It uniquely impairs leptin signaling, blunting satiety during and after fasting.

✕ **Alcohol** — Ethanol is metabolized via alcohol dehydrogenase and aldehyde dehydrogenase, consuming NAD+ and shifting NAD+/NADH ratio. This suppresses SIRT1 activation and promotes acetyl-CoA carboxylase activity, driving lipogenesis rather than fat oxidation. Also impairs autophagy via mTORC1 reactivation.

Simply put ·

Eating window timing: Break your fast with fat + fiber first (olive oil + vegetables), *then* protein. This order maximizes postprandial lipemia while slowing glucose absorption and sustaining AMPK tone. Avoid breaking a long fast (>24 hours) with protein alone—the sudden amino acid surge can spike ammonia and cause fatigue. **Hydration during fast:** Water, black coffee, and unsweetened tea don't break a fast (they don't activate mTOR or trigger insulin). Salt your water with electrolytes in extended fasts (>16 hours) to preserve AMPK signaling and prevent aldosterone dysregulation. **Refeeding carbs:** If you exercise, add carbs *after* protein in your eating window (this leverages GLUT4 translocation from muscle contraction, minimizing hepatic glycogen overshoot). **Individual variation:** Those with APOE4 genotype may be more sensitive to high-fat fasting protocols; monitor lipid profiles and consider cycling in low-fat days if LDL rises >200 mg/dL. If you have a history of disordered eating, extended fasting may trigger obsessive food thoughts—keep fasting windows moderate (14–16 hours max) and prioritize nutrient density over restriction.

06

Daily Protocol

Fasting is a powerful metabolic reset, but success depends on strategically timed interventions that preserve muscle, stabilize blood sugar, and amplify autophagy. Your supplement timing activates different pathways—some work best when fasting, others right at the break, and some prepare your body for the next cycle.

ON WAKING

– **Morning sunlight exposure** Spend 10-15 minutes in direct sunlight within 30 minutes of waking. This sets your circadian rhythm and suppresses melatonin, optimizing cortisol timing and metabolic signaling during your fast. Light exposure also increases NAD■ availability naturally.

– **Hydration + Electrolyte Complex (Sodium/Potassium/Magnesium)** Take 500ml water + 1 serving electrolytes. During fasting, you lose mineral-rich fluid through urine; electrolytes maintain cellular voltage, prevent headaches, and preserve muscle contractility. Sodium also supports water retention and blood volume—critical when you're not eating.

– **L-Theanine (100-200mg)** Activates alpha brain waves (calm focus) via GABA and glutamate modulation. Taken now, it smooths the transition into fasting without stimulant jitters, and supports mental clarity as your brain shifts to ketone fuel over hours 4-6 of the fast.

EARLY MORNING (HOUR 2-3 OF FAST)

– **Cold exposure (cold shower or ice bath, 1-3 minutes)** Brief cold stress activates brown adipose tissue (BAT) and increases norepinephrine, which accelerates fat mobilization and ketone production. This timing—early in your fast—amplifies the metabolic switch. Cold also upregulates autophagy markers like ULK1.

– **Breathwork (4-7-8 breathing, 5 minutes)** Slow diaphragmatic breathing lowers cortisol and activates parasympathetic tone, countering any fasting-induced stress response. This primes your nervous system for steady fat oxidation rather than glycogen panic.

MID-MORNING (HOUR 4-6 OF FAST)

– **Nicotinamide Riboside (NR) or NAD■ Precursor (250-500mg)** NAD■ is the fuel for sirtuins (SIRT1/SIRT3) and PARPs—the enzymes that orchestrate autophagy, mitochondrial repair, and DNA stability during fasting. NR supplementation peaks in circulation around hour 4-6, synergizing with your natural ketone rise. This is the sweet spot: NAD■ levels naturally dip in early fasting, so external support maximizes longevity pathways.

– **Spermidine (0.5-1mg per kg bodyweight, or ~30-50mg for adult)** Polyamine that directly triggers autophagy via mTOR inhibition and ATG gene expression. Timing this mid-morning extends the fasting window's cellular cleaning power. Works best when endogenous ketone levels are ramping (hour 5+).

– **Hydration (250-500ml water, no electrolytes this time)** Top up fluids as perspiration and respiration continue. Skip excess electrolytes here to avoid mineral overload; you've already had your baseline at waking.

LATE MORNING / EARLY AFTERNOON (HOUR 6-8 OF FAST, OPTIONAL)

– **Beta-Hydroxybutyrate (BHB) Ketone Salt (5-10g, optional)** If you want to deepen ketosis (especially on your first 2-3 fasting days or if energy dips), exogenous BHB provides immediate brain fuel and amplifies autophagy signaling. The timing here—late morning—maintains mental sharpness without breaking the fast's metabolic benefits. Typically not needed after adaptation (day 4+), but useful for performance or extended fasts.

– **Movement or gentle exercise (20-30 min walk or yoga)** Light activity at hour 6-8 of fasting taps into peak fat oxidation; your liver has depleted glycogen, forcing fat-derived ketone reliance. Movement also amplifies autophagy via AMPK activation and improves insulin sensitivity before your eating window.

BREAKING THE FAST (MEAL WINDOW START)

– **Collagen Peptides (Hydrolyzed) with first meal (10-20g)** Fasting depletes amino acid pools; collagen (glycine + proline rich) is easily digested and replenishes connective tissue + gut lining without spiking insulin dramatically. Take it with your first bite or in a pre-meal broth. Glycine also supports glutathione synthesis—your master antioxidant—protecting gains from the fasting period.

– **Eat slowly, prioritize protein and fiber first** After fasting, your digestive system is sensitive. Slow eating activates satiety hormones (CCK, GLP-1) and prevents blood sugar spikes. Protein with collagen rebuilds muscle; fiber feeds beneficial gut microbiota and stabilizes glucose—both essential post-fast.

– **Omega-3 (EPA/DHA, 2-3g marine or algae-based)** EPA/DHA reduce fasting-induced inflammation and support mitochondrial membrane fluidity after the metabolic stress of autophagy. Timing this at your first meal ensures absorption with dietary fat and anchors anti-inflammatory signaling for your eating window.

EVENING (2-4 HOURS BEFORE BED)

- **Light walk or restorative stretching (15 min)** Low-intensity movement improves glucose clearance from your meal, reduces blood sugar variability overnight, and enhances sleep quality. Avoids late-day cortisol spikes from intense exercise.
- **Dim lights, reduce blue light (screens off)** Fasting naturally elevates circadian sensitivity. Dimming lights 2 hours before bed restores melatonin production, ensuring quality sleep—when autophagy continues and growth hormone peaks. This closes the loop on your fasting cycle.
- **Hydration + optional Magnesium (from Electrolyte Complex, or separate Mg glycinate 200-300mg)** Magnesium supports GABA and relaxes muscles. If taken separately, use glycinate form (highly absorbable, non-laxative). Timing it 30-60 min before bed deepens sleep quality without morning GI distress.

BIOMARKERS: Fasting Response & Metabolic Adaptation

Standard blood work tells you if you're sick; optimal biomarkers tell you if you're thriving during fasting. The tests below measure how your body shifts fuel sources, manages inflammation, protects muscle, and regulates appetite—the actual mechanisms that make fasting work.

Fasting Glucose

What to ask for: Measure after 12+ hours overnight fasting. Request venous plasma glucose, not fingerstick. Test on day 1 of protocol and weekly thereafter.

Optimal: 70-85 mg/dL · **Lab "normal" problem:** Lab 'normal' goes up to 100 mg/dL, but values above 85 signal insulin resistance even though doctors won't flag it. You want tight control during fasting.

If out of range: Above 90 mg/dL = your body isn't switching to fat fuel efficiently; you may have metabolic inflexibility. Consider shorter fasting windows (14-16h) until glucose stabilizes. Below 70 mg/dL = monitor for hypoglycemia symptoms (shakiness, brain fog); eat small protein + fat if it dips further.

Fasting Insulin

What to ask for: Serum insulin, fasting (same timing as glucose). This is the traffic cop—measure it every 2 weeks during active fasting protocol.

Optimal: < 5 mIU/L (ideally < 3 mIU/L) · **Lab "normal" problem:** Lab 'normal' range is 2.6–24.9 mIU/L. A value of 12 is technically 'normal' but means your pancreas is working overtime to keep glucose down. That's pre-diabetic territory, not optimal.

If out of range: Above 8 = insulin resistance; fasting is therapeutic here but start with 14-hour windows to avoid excessive autophagy burden. Below 2 = excellent metabolic flexibility, your body loves burning fat. If below 1.5 + you feel fatigued, reintroduce small carbs post-fasting window.

HOMA-IR (Homeostatic Model Assessment for Insulin Resistance)

What to ask for: Calculated from fasting glucose and insulin. Labs rarely calculate it—ask them to, or calculate yourself: (fasting insulin in mIU/L × fasting glucose in mg/dL) ÷ 405.

Optimal: < 1.0 · **Lab "normal" problem:** Most labs say < 2.0 is fine. But HOMA-IR above 1.8 already predicts type 2 diabetes risk. You want to see it drop below 1.0 to prove metabolic healing.

If out of range: Above 2.0 = your cells aren't responding to insulin signals; fasting is one of the most powerful tools to lower this. Expect 4–8 week timeline for improvement. Below 0.5 = metabolic excellence; you're insulin-sensitive, keep fasting structure but add carbs strategically around training.

Beta-Hydroxybutyrate (BHB)

What to ask for: Serum or fingerstick BHB (blood ketones, not urine). Test on day 3–5 of a fasting protocol to see ketone production in action. Morning measurement before eating.

Optimal: 1.5–3.0 mmol/L (deep fasting), 0.5–1.5 mmol/L (maintenance) · **Lab "normal" problem:** This test isn't in 'normal' panels. Most doctors don't order it. But it's the actual fuel your brain and heart run on during fasting—you need to know you're producing it.

If out of range: Below 0.5 mmol/L after 3+ days fasting = you're not in ketosis yet; metabolic inflexibility present, extend fasting window or drop carbs. Above 3.0 mmol/L = therapeutic ketosis (good for brain, inflammation), but if combined with elevated acetoacetate, monitor for ketoacidosis risk (rare in healthy people, but check). Values 1.5–3.0 = your brain is efficiently fueled by fat; optimal zone.

Adiponectin (High Molecular Weight form preferred)

What to ask for: Total adiponectin, but specifically request high-molecular-weight (HMW) adiponectin if available—it's more bioactive. Measure at baseline and month 2 of protocol.

Optimal: > 9 µg/mL (higher is better; women often higher than men) · **Lab "normal" problem:** Lab normal is 2.7–17.4 µg/mL—a huge range that misses metabolic dysfunction. People at 3 µg/mL are metabolically sick but 'in range.' Fasting should raise this significantly.

If out of range: Below 6 µg/mL = adipose tissue inflammation; fasting will improve this over weeks. This marker tells you fasting is reducing visceral fat and improving insulin sensitivity at the tissue level. Above 12 µg/mL = excellent metabolic health signal, your fat cells are secreting anti-inflammatory hormones.

C-Reactive Protein, High-Sensitivity (hs-CRP)

What to ask for: hs-CRP specifically (not standard CRP). Measure 12 hours after ending fasting window, not during fast. Baseline and then monthly.

Optimal: < 1.0 mg/L · **Lab "normal" problem:** Lab 'normal' is < 3.0 mg/L, but values 1–3 still signal chronic inflammation and cardiovascular risk. Fasting should drop this below 1.0.

If out of range: Above 2.0 = systemic inflammation from metabolic dysfunction or other sources; fasting is anti-inflammatory, expect gradual decrease over 4–6 weeks. Below 0.5 = excellent; you're reducing aging pathways. If hs-CRP rises during fasting (rare), check for overtraining, poor sleep, or inadequate refeeding—inflammation needs recovery fuel.

Triglycerides (fasting)

What to ask for: Serum triglycerides after 12+ hours fasting. Same timing as glucose/insulin panel.

Optimal: < 100 mg/dL (ideally < 70 mg/dL) · **Lab "normal" problem:** Lab 'normal' is < 150 mg/dL, but above 100 indicates poor fat metabolism even though it's technically acceptable. Fasting should lower this noticeably.

If out of range: Above 150 mg/dL = metabolic dysfunction; your liver is packaging too much fat into VLDL particles. Fasting works here: it depletes carb-driven triglyceride synthesis. Expect 20–30% drop within 4 weeks. Below 70 = excellent lipid profile; your fasting protocol is optimizing fat metabolism.

Total Cholesterol & LDL-C Particle Count (NMR or Apolipoprotein B preferred over standard LDL)

What to ask for: Skip standard LDL if possible; request NMR lipoprofile for particle number (LDL-P) or ApoB. Standard LDL-C can miss atherogenic risk. Test fasting, baseline and month 1.

Optimal: LDL-P < 1000 nmol/L, ApoB < 80 mg/dL · **Lab "normal" problem:** Standard LDL-C 'normal' is < 130 mg/dL, but you can have high LDL-P (many small, dense particles) with 'normal' LDL-C—that's atherogenic. Fasting affects particle number and size, not just cholesterol mass.

If out of range: High LDL-P (> 1200) = many atherogenic particles, especially if small/dense. Fasting improves particle size and count over 6–8 weeks. If LDL-P or ApoB rise initially during fasting (rare), ensure adequate carb refeeding and omega-3 intake. Goal: lower particle count, increase particle size (less harmful).

Cortisol (morning, salivary preferred)

What to ask for: Salivary cortisol upon waking, before food/coffee. Measure at baseline and week 2, 4, and 8. This tracks stress hormone adaptation to fasting.

Optimal: 10–16 nmol/L (or 0.36–0.58 µg/dL in older units) · **Lab "normal" problem:** Serum cortisol 'normal' ranges 10–20 µg/dL, but fasting can elevate cortisol—especially in week 1–2 as a stress adaptation. You need to track the trend, not just the absolute number.

If out of range: Rising cortisol week 1–2 = normal adaptation; fasting is a mild stressor. It should plateau and decline by week 3–4 as your body adjusts. Persistently high (> 20 nmol/L salivary) = fasting is too aggressive; shorten windows, add more calories during eating periods, prioritize sleep. Low (< 5) = consider if you're over-fasting or over-training.

Prealbumin (Transthyretin)

What to ask for: Serum prealbumin. Measures protein status and liver synthetic function. Baseline and month 1 during extended fasting protocols.

Optimal: 20–40 mg/dL · **Lab "normal" problem:** Lab normal is 20–40, but within that range, lower values (20–25) suggest you're catabolizing muscle. Fasting can trigger muscle loss if not protein-loaded during eating windows.

If out of range: Below 20 = muscle breakdown happening; your eating window isn't protein-rich enough. Add 0.7–1.0g protein per lb bodyweight during feeding phase, prioritize leucine (9–12g per meal). Above 35 = excellent protein status, fasting isn't eroding muscle. Track this monthly if fasting longer than 18 hours regularly.

90-DAY TIMELINE

Your body needs time to adapt to fasting—metabolic shifts don't happen overnight, but they compound predictably. This timeline maps the physiological transitions you'll experience, from glycogen depletion to autophagy activation to genuine metabolic flexibility.

DAYS 1-14 | ADAPTATION INITIATION

GLYCOGEN DEPLETION & METABOLIC CONFUSION

- Liver glycogen stores deplete within 12-16 hours of fasting; expect hunger spikes around hours 14-18 as your body signals its preferred fuel is unavailable
- HIF-1α (hypoxia-inducible factor) and AMPK activate within 24-48 hours, triggering the first wave of cellular stress responses—this feels like fatigue, irritability, or 'brain fog' for 3-7 days
- Insulin drops 40-60% by day 3, allowing adipose tissue to mobilize fatty acids; you may notice reduced cravings mid-week as blood glucose stabilizes
- Cortisol rises temporarily (especially if fasting duration exceeds 18 hours)—you might feel wired, anxious, or experience sleep disruption; this normalizes as your CNS adapts
- Weight loss of 2-4 lbs is mostly water and glycogen; scale wins feel motivating but don't reflect fat loss yet
- Electrolyte shifts begin (sodium, potassium, magnesium); leg cramps, headaches, or dizziness signal you need mineral support

DAYS 15-45 | METABOLIC TRANSITION

KETONE PRODUCTION & FAT ADAPTATION

– Ketone bodies (acetoacetate, β-hydroxybutyrate) rise consistently after day 7-10; blood ketone levels reach 0.5-2.0 mM by day 21, signaling reliable fat metabolism

– Mitochondrial biogenesis accelerates (PGC-1α expression increases)—you'll notice sustained energy between fasts, fewer energy crashes, and mental clarity returning stronger than before

– Ghrelin (hunger hormone) begins its downward trend; by day 30, fasting windows feel genuinely easier as your hunger signals recalibrate

– Triglycerides drop 20-30% as your body preferentially uses fat for fuel; you may feel physically lighter despite scale weight stabilizing

– Autophagy hits meaningful levels after day 14-21; at the cellular level, damaged proteins and organelles are being recycled, though you won't 'feel' this—trust the process

– Fat loss becomes visible (1-2 lbs per week); muscle preservation improves if you're strength training during eating windows

– Sleep quality often improves by week 3-4 as circadian alignment strengthens and cortisol evening peaks normalize

MONTH 2 | METABOLIC EFFICIENCY PEAK

AUTOPHAGY DEEPENING & INSULIN SENSITIVITY RECOVERY

– Autophagy reaches therapeutic levels (48+ hour fasts activate chaperone-mediated autophagy more robustly); cellular cleanup accelerates, supporting immune resilience and longevity markers

– SIRT1 and NAD+ levels rise, enhancing mitochondrial function and DNA repair—you'll feel recovered faster between workouts and mental endurance extends noticeably

– Insulin sensitivity improves measurably; fasting glucose often drops 5-15 mg/dL and postprandial glucose spikes shrink, even if you haven't changed diet composition

– Inflammatory markers (IL-6, TNF-α, hsCRP) decline as the fasting-induced shift toward M2 macrophages progresses; joint pain, brain fog, or bloating often vanish

– Fat loss accelerates to 2-3 lbs per week (if caloric deficit is maintained); visceral fat (the metabolically toxic kind) is preferentially mobilized

– Appetite suppression becomes effortless; many people naturally eat less during eating windows without conscious restriction

– Cognitive sharpness peaks—mood improves, anxiety often lifts, and sustained focus extends by 2-3 hours beyond pre-fasting baseline

MONTH 3 | METABOLIC MASTERY

SUSTAINED FAT ADAPTATION & LONGEVITY SIGNALING

– mTOR (mammalian target of rapamycin) downregulation becomes sustained—cellular growth pathways reset toward maintenance and repair over proliferation, supporting longevity pathways

– Mitochondrial density increases ~15-25% (measurable via VO■ max improvement or endurance capacity); you'll crush longer fasts or workouts with less effort

– Metabolic flexibility is genuine—you can seamlessly shift between fat and carb oxidation, meaning occasional high-carb meals don't derail ketosis or trigger energy crashes

– Body recomposition is obvious; total weight loss averages 8-15 lbs but muscle is preserved or gained if training is consistent, so you look leaner and stronger simultaneously

– Hormonal stability deepens: testosterone, growth hormone, and cortisol rhythms normalize; women often report cycle regularity returning if it was disrupted

– Biomarker improvements compound: triglycerides drop 30-50%, LDL particle size improves (shift toward larger, less atherogenic particles), HDL rises 10-20%

– Emotional resilience and stress tolerance improve noticeably—fasting-induced neuroplasticity supports emotional regulation and reduces reactivity

– Skin clarity often emerges; reduced inflammation and improved autophagy clear acne and support collagen turnover