ANTI-INFLAMMATORIES

Do anti-inflammatory diets and lifestyle habits actually lower hs-CRP?

Separating whole-diet human trial evidence, visceral fat mechanics, and circadian eating patterns from single-ingredient wellness marketing.

LetsGo Editorial · LetsGo Editorial · 9 November 2026 · 12 min read

Marketing for anti-inflammatory wellness focuses heavily on isolated botanical extracts and proprietary probiotic sachets, yet human randomized trials show that systemic inflammatory markers respond primarily to energy balance, visceral adipose tissue volume, dietary fibre diversity, and sleep-wake timing.

Chronic, low-grade sterile inflammation—termed inflammaging—is characterized by persistent two- to four-fold elevations in circulating cytokines and acute-phase proteins such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-alpha), and high-sensitivity C-reactive protein (hs-CRP). Unlike acute inflammation, which resolves after clearing a pathogen or healing an injury, chronic sterile inflammation damages vascular endothelium, impairs insulin signaling in skeletal muscle, and accelerates atherosclerotic plaque instability. Consumer wellness products frequently claim that single culinary spices, alkalizing waters, or proprietary probiotic strains switch off these pathways. When tested in human randomized controlled trials (RCTs), single food extracts rarely move systemic cytokines unless they replace ultra-processed calorically dense foods or correct a clinical deficiency. By contrast, comprehensive dietary patterns, fermented food intake, aerobic and resistance training, and circadian alignment reproducibly lower circulating inflammatory proteins in adults aged 40 to 65. Clinical trial evidence demonstrates a clear distinction between adding an isolated compound and changing an upstream pattern: lowering hs-CRP via an isolated anti-inflammatory agent without addressing visceral adiposity or apolipoprotein B (ApoB) does not automatically translate into lower cardiovascular event rates, whereas lowering hs-CRP through a Mediterranean dietary pattern or loss of visceral fat improves both the biomarker and hard clinical outcomes.

What it is

Nuclear factor kappa B (NF-kB) and the NLRP3 inflammasome sit at the center of the innate immune activation that anti-inflammatory dietary and behavioral exposures aim to downregulate. When activated by metabolic stressors—such as oxidized lipoproteins, advanced glycation end-products, free fatty acids, or bacterial lipopolysaccharide (LPS) translocating across a compromised intestinal barrier—NF-kB triggers the transcription of pro-inflammatory cytokines including IL-1beta, IL-6, and TNF-alpha. Hepatocytes respond to circulating IL-6 by synthesizing and releasing CRP into systemic circulation. Nutritional and lifestyle interventions target the upstream inputs to these pathways. In human nutrition science, the most validated anti-inflammatory pattern is the Mediterranean diet and patterns scored by the Dietary Inflammatory Index (DII). These patterns emphasize extra-virgin olive oil (rich in the phenolic compound oleocanthal), marine omega-3 polyunsaturated fatty acids (eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]), diverse fermentable fibres from legumes, vegetables, and whole grains, and polyphenol-rich berries and greens, while minimizing refined starches, added sugars, trans fats, and processed meats. Beyond food composition, anti-inflammatory physiology depends on three non-dietary pillars established in human studies: skeletal muscle

contraction (which releases myokines that counter TNF-alpha), maintenance of low visceral adipose tissue mass, and circadian alignment of sleep and feeding windows. Eating heavy meals during the biological night—such as at 1 a.m. during shift work or travel—and sleeping on an irregular schedule act as direct pro-inflammatory stimuli in human laboratory trials.

Why it is used

High-sensitivity C-reactive protein (hs-CRP) is an independent predictor of future myocardial infarction, stroke, and cardiovascular mortality in large human cohort studies, even when low-density lipoprotein cholesterol (LDL-C) is pharmacologically controlled. Interventional trials show that patients with residual inflammatory risk (defined in clinical trials as an hs-CRP persistently at or above 2.0 mg/L) experience higher rates of recurrent cardiovascular events. While pharmaceutical trials (such as CANTOS using the IL-1beta monoclonal antibody canakinumab, and COLCOT and LoDoCo2 using low-dose colchicine) proved the causal role of the IL-1beta to IL-6 to CRP pathway in human atherosclerosis, lifestyle modification remains the primary intervention for lowering subclinical inflammation in individuals without established cardiovascular disease. In metabolic tissue, pro-inflammatory cytokines like TNF-alpha and IL-6 phosphorylate insulin receptor substrate-1 (IRS-1) at serine residues rather than tyrosine residues in skeletal muscle and liver cells, directly blunting intracellular insulin signaling and driving compensatory hyperinsulinemia. Lowering tissue-level inflammation restores insulin sensitivity and reduces hepatic de novo lipogenesis. In consumer markets, anti-inflammatory habits are frequently sought for non-specific complaints including cognitive fatigue, bloating, irritable bowel symptoms, or mood fluctuations. Commercial products heavily promote single-strain probiotic sachets for these gut-brain and inflammatory symptoms, but human meta-analyses show that isolated probiotic strains rarely alter systemic plasma cytokines or resolve complex functional bowel disorders on their own. Reproducible clinical improvements in gut barrier function and systemic inflammatory tone come instead from whole-diet substrate changes, live fermented foods, and consistent sleep timing.

How it helps

Colonic fermentation of indigestible plant polysaccharides into short-chain fatty acids (SCFAs) is the primary gut-mediated mechanism lowering systemic inflammation in humans. Humans lack the endogenous enzymes to cleave complex plant polysaccharides (pectins, inulins, resistant starches, beta-glucans). When these fibres reach the colon, commensal bacteria ferment them into acetate, propionate, and butyrate. In human colonic tissue and in vitro models, butyrate serves as the primary fuel source for colonocytes, strengthens tight-junction proteins (claudins and occludins), and acts as a histone deacetylase (HDAC) inhibitor. This epigenetic action upregulates forkhead box P3 (FOXP3)+ regulatory T cells (Tregs) and suppresses NF-kB activation in mucosal immune cells. Stronger tight junctions reduce metabolic endotoxemia—the paracellular leakage of gram-negative bacterial endotoxin (LPS) into portal circulation after meals, which otherwise binds Toll-like receptor 4 (TLR4) on circulating monocytes and hepatic Kupffer cells. Microbiome remodeling via live fermented foods operates distinctly from isolated fibre intake. A 10-week randomized controlled trial in 36 healthy adults compared a high-fibre diet against a diet rich in fermented foods (yogurt, kefir, fermented cottage cheese, kimchi, vegetable brine drinks, and kombucha). The fermented-food group demonstrated a steady increase in gut microbiota alpha-diversity and statistically significant reductions in 19 circulating inflammatory proteins, including IL-6, IL-10, and IL-12b, alongside reduced activation of four types of immune cells (effector memory CD4+ T cells, classical monocytes, non-classical monocytes, and B cells). Participants in the high-fibre arm who had low starting point microbial diversity did not lower their inflammatory markers over the 10-week window, demonstrating in humans that live fermented foods remodel microbial community structure more rapidly than isolated fibre when starting point diversity is depleted. Eicosanoid switching and specialized pro-resolving mediators (SPMs) govern membrane-derived lipid signaling. Marine omega-3 fatty acids (EPA and DHA) found

in fatty fish incorporate into cellular phospholipid membranes, partially displacing arachidonic acid (an omega-6 fatty acid). While arachidonic acid is metabolized by cyclooxygenase (COX) and lipoxygenase (LOX) enzymes into potent pro-inflammatory eicosanoids (prostaglandin E2 and leukotriene B4), EPA and DHA serve as precursors for less inflammatory 3-series prostaglandins and 5-series leukotrienes, as well as specialized pro-resolving mediators—resolvins, protectins, and maresins. Identified in human blood and inflammatory exudates, SPMs actively terminate the inflammatory cascade by halting polymorphonuclear neutrophil infiltration and stimulating macrophage efferocytosis (phagocytosis of apoptotic cellular debris) without causing immunosuppression. Concurrently, oleocanthal in extra-virgin olive oil inhibits COX-1 and COX-2 enzymes in vitro in a dose-dependent manner structurally analogous to ibuprofen; in human dietary trials, its systemic effect is modest and accrues over months of daily intake. Visceral adipose reduction and exercise-induced myokine release directly alter circulating IL-6 kinetics. Visceral fat is infiltrated by M1-polarized pro-inflammatory macrophages that form crown-like structures around hypertrophic, hypoxic adipocytes, accounting for roughly 30% of circulating IL-6 in resting overweight humans. Caloric restriction or dietary quality shifts that shrink visceral adipose tissue remove this direct portal source of cytokines. Conversely, skeletal muscle contraction during exercise releases acute bursts of muscle-derived IL-6 into systemic circulation. Unlike chronically elevated macrophage-derived IL-6, acute exercise-induced myokine IL-6 acts anti-inflammatorily in humans by stimulating the release of IL-1 receptor antagonist (IL-1ra) and IL-10, and by inhibiting endotoxin-stimulated TNF-alpha production. Evidence quality and effect sizes: Meta-analyses of RCTs evaluating the Mediterranean diet across thousands of adults over 3 to 24 months demonstrate average reductions in hs-CRP of roughly 0.5 to 1.0 mg/L and statistically significant reductions in IL-6 compared with control diets. In RCTs of overweight and obese adults, intentional weight loss of 5% to 10% of total body weight consistently lowers hs-CRP by 1.0 to 2.0 mg/L. By contrast, RCTs of isolated culinary spices in dietary quantities or commercial multi-strain probiotic sachets show no reliable effect size on plasma hs-CRP in non-diseased human populations.

Who should try it

Adults with persistently elevated starting point hs-CRP between 1.0 and 3.0 mg/L (or above 3.0 mg/L after acute infection is excluded) derive the largest absolute risk reduction from anti-inflammatory nutritional and behavioral interventions. Clinical return is particularly pronounced in four overlapping profiles: - Adults with cardiometabolic discordance: Individuals presenting with elevated hs-CRP alongside elevated ApoB, hypertriglyceridemia, low HDL-C, or fasting hyperinsulinemia. - Individuals with central adiposity: Adults with a waist-to-height ratio above 0.50 or elevated visceral adipose tissue mass on a Dual-Energy X-ray Absorptiometry (DXA) scan, even when total body mass index (BMI) falls within the normal range (18.5 to 24.9 kg/m2). - Shift workers and frequent transmeridian travelers: Adults exposed to circadian misalignment, irregular sleep onset, and biological-night eating windows, which induce acute postprandial insulin resistance and daytime IL-6 and hs-CRP elevations in controlled human trials. - Adults with metabolic osteoarthritis: Individuals in whom lowering both systemic adipokine/cytokine load and mechanical joint stress jointly improves pain and function scores in human RCTs. Trade-offs, side effects, and contraindications: - Gastrointestinal tolerance: Rapidly increasing fermentable fibres (galacto-oligosaccharides in legumes, fructans in alliums and wheat, inulin) or fermented foods frequently triggers luminal gas distension, flatulence, and osmotic diarrhea, particularly in individuals with irritable bowel syndrome (IBS) or small intestinal bacterial overgrowth (SIBO). Fermentable substrates (FODMAPs) require gradual titration over 4 to 8 weeks. - Histamine intolerance: Aged and fermented foods (kimchi, sauerkraut, kefir, kombucha, aged cheeses) contain high concentrations of biogenic amines, specifically histamine and tyramine. In adults with reduced intestinal diamine oxidase (DAO) enzymatic activity, these foods provoke headaches, cutaneous flushing, nasal congestion, tachycardia, or gastrointestinal cramping. - Botanical and supplement interactions: Whole culinary foods carry minimal drug interaction risk, but concentrated anti-inflammatory extracts (high-concentration curcuminoids, concentrated marine omega-3 capsules, ginger extracts, or bromelain) exert antiplatelet Effects by inhibiting thromboxane A2 synthesis or platelet aggregation, increasing

bleeding risk when combined with warfarin, direct oral anticoagulants (apixaban, rivaroxaban), clopidogrel, or aspirin. Concentrated curcumin extracts formulated with piperine (a UDP-glucuronosyltransferase and CYP3A4 inhibitor) are also associated with idiosyncratic drug-induced liver injury (DILI) in human pharmacovigilance registries. - Opportunity cost: Allocating financial resources to proprietary gut-brain probiotic sachets, alkaline waters, or exotic antioxidant powders diverts capital and adherence capacity away from whole-food fibre diversity, visceral fat loss, sleep regularity, and eliminating biological-night caloric intake.

Measurements that show improvement

Fasting high-sensitivity C-reactive protein (hs-CRP) measured via venous blood draw is the primary clinical standard for quantifying systemic inflammatory tone. Because low-grade inflammation is asymptomatic until structural tissue damage accumulates, interventions should be evaluated using serial blood biomarkers, body composition imaging, and autonomic wearables: 1. High-sensitivity C-reactive protein (hs-CRP): Low cardiovascular risk is defined clinically as below 1.0 mg/L (with optimal starting point readings below 0.5 to 0.7 mg/L). Because hepatic CRP synthesis spikes 10- to 100-fold during an acute viral upper respiratory infection, occult dental infection, or within 24 to 48 hours of heavy eccentric resistance training, any single reading above 3.0 mg/L requires a repeat fasting test 2 to 4 weeks later when asymptomatic. Direction of improvement: decrease. 2. GlycA (Glycoprotein Acetylation): A nuclear magnetic resonance (NMR) spectroscopy blood assay that quantifies the concentration and glycosylation state of mobile N-acetyl methyl groups on multiple acute-phase proteins (predominantly alpha-1-acid glycoprotein, haptoglobin, alpha-1-antitrypsin, and transferrin). In human cohort studies, GlycA demonstrates lower intra-individual biological variability week-to-week than hs-CRP and independently predicts incident cardiovascular events and type 2 diabetes. Optimal values generally sit below 350 to 400 umol/L depending on the laboratory reference assay. Direction of improvement: decrease. 3. Fasting Insulin, Triglycerides, and HbA1c: Because cytokine-mediated serine phosphorylation of IRS-1 directly impairs insulin clearance and signaling, resolving visceral and hepatic inflammation lowers fasting insulin (optimal target below 6 to 8 uIU/mL) and fasting triglycerides (target below 80 to 100 mg/dL), followed over 8 to 12 weeks by reductions in HbA1c. Direction of improvement: decrease. 4. Apolipoprotein B (ApoB): Replacing dietary saturated fatty acids and refined starches with monounsaturated fats, polyunsaturated fats, and viscous soluble fibres (beta-glucan, psyllium, pectin) upregulates hepatic LDL receptors and lowers circulating ApoB-containing lipoprotein particles (target below 60 to 80 mg/dL depending on starting point risk), reducing the subendothelial particle retention that initiates vascular inflammation. Direction of improvement: decrease. 5. Visceral Adipose Tissue (VAT) Mass and Waist-to-Height Ratio: Quantified directly via DXA body composition scan (targeting VAT mass below 0.5 kg or VAT area below 100 cm2) or tracked anthropometrically via waist circumference measured horizontally at the iliac crest or umbilicus divided by height. Reducing the waist-to-height ratio below 0.50 directly correlates with reduced portal vein IL-6 flux. Direction of improvement: decrease. 6. Nocturnal Resting Heart Rate (RHR) and Heart Rate Variability (HRV): Efferent vagus nerve activity regulates systemic cytokine release via the cholinergic anti-inflammatory pathway, wherein acetylcholine binds alpha-7 nicotinic acetylcholine receptors (alpha7nAChR) on macrophages to inhibit TNF-alpha release. In human observational cohorts, higher cardiac vagal tone—reflected in higher nocturnal root mean square of successive differences (RMSSD) HRV and lower nocturnal RHR—inversely correlates with plasma hs-CRP and IL-6. Finishing caloric intake at least 3 hours before sleep onset typically lowers nocturnal RHR by 2 to 5 beats per minute and raises nocturnal HRV within 72 hours. Direction of improvement: RHR decrease, HRV increase.

Products and services

These LetsGo products and services sit next to this subject. They are not a prescription. A link is not evidence that the product causes the change discussed above. - [LetsGo supplement shelf](/supplements) — Bottles

LetsGo stocks for members. Read the label before the first order. A link is not a dose. - [LetsGo programmes](/programs) — Coached programmes. Not a clinic menu and not a promise of a result.

Practical takeaways

- Consume 30 or more distinct plant foods per week (legumes, vegetables, whole grains, nuts, seeds, herbs, spices) for 12 weeks to supply diverse fermentable polysaccharides for colonic butyrate synthesis.
- Eat 2 to 4 servings daily of unsweetened, live fermented foods (plain kefir, yogurt, kimchi, sauerkraut) for at least 10 weeks; stop or halve the dose if histamine-related flushing, headaches, or persistent bloating occur after a 2-week titration.
- Replace butter and refined seed oils with extra-virgin olive oil as the primary culinary lipid and consume low-mercury marine fish (salmon, sardines, mackerel, anchovies) 2 to 3 times per week to raise cell membrane EPA and DHA.
- Terminate all caloric intake at least 3 hours before sleep onset and keep daily wake and sleep onset times within a 30- to 45-minute window seven days a week; track nocturnal RHR for a 2 to 5 bpm reduction within 7 days.
- Reduce waist-to-height ratio below 0.50 (or reduce body weight by 5% to 10% if overweight) over 12 to 24 weeks to shrink visceral adipose macrophage infiltration.
- Repeat a fasting venous blood draw for hs-CRP, fasting insulin, triglycerides, and ApoB at the 12-week mark—ensuring 48 to 72 hours of rest from heavy eccentric exercise and complete absence of viral or dental infection—targeting an hs-CRP below 1.0 mg/L.

Safety note

Persistently elevated hs-CRP above 3.0 mg/L, and especially above 10.0 mg/L, warrants medical evaluation to rule out acute or occult infection, autoimmune rheumatologic disease, inflammatory bowel disease, or malignancy before attributing the elevation solely to lifestyle factors. Individuals taking anticoagulant or antiplatelet medications should consult a physician before using concentrated botanical or omega-3 supplements.

Disclaimer

This article is for educational and informational purposes only and does not constitute medical advice, diagnosis, or treatment. Always consult a qualified physician or healthcare provider regarding laboratory interpretation, medical conditions, or changes to your nutrition, exercise, or medication regimen.

Lowering chronic sterile inflammation in midlife does not depend on proprietary gut-brain sachets or isolated botanical extracts. Human randomized trials consistently demonstrate that systemic inflammatory tone is governed by visceral adipose volume, colonic fermentation of diverse plant fibres into short-chain fatty acids, live fermented food intake, marine omega-3s and olive oil polyphenols, regular skeletal muscle contraction, and circadian alignment of sleep and feeding windows. Tracking objective biomarkers including hs-CRP, GlycA, fasting insulin, ApoB, and visceral fat mass allows adults and their physicians to verify whether dietary and behavioral changes are resolving inflammatory risk.