ANTI-INFLAMMATORIES

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

Separating proprietary gut-brain sachets and supplement marketing from the dietary fibres, fermented foods, and circadian habits that measurably reduce systemic inflammation in humans.

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A proprietary probiotic strain name printed on a daily sachet is not an established intervention for systemic inflammation, mood, or intestinal permeability.

Chronic low-grade inflammation—characterised by persistent, modest elevations in circulating acute-phase proteins and cytokines—is an established driver of vascular endothelial dysfunction, insulin resistance, and biological ageing. In consumer wellness, anti-inflammatory interventions are frequently reduced to isolated botanical extracts or single-strain gut-brain probiotic sachets marketed for mood and gut barrier integrity. Human clinical trials tell a different story. While single-strain supplements show inconsistent effects on systemic inflammatory markers in healthy and metabolically at-risk adults, structural changes in diet composition, microbial fermentation substrates, visceral adiposity, and circadian meal timing reliably downregulate innate immune activation.

What it is

Pharmaceutical anti-inflammatories block specific enzymatic or receptor pathways—such as non-steroidal anti-inflammatory drugs (NSAIDs) inhibiting cyclooxygenase-1 and -2 (COX-1/COX-2), corticosteroids suppressing pro-inflammatory gene transcription via the glucocorticoid receptor, or targeted biologics neutralising cytokines like tumour necrosis factor-alpha (TNF-alpha) and interleukin-1beta (IL-1beta). In preventive medicine, an anti-inflammatory lifestyle and dietary pattern refers to non-pharmacological habits that lower chronic, sterile, low-grade systemic inflammation (inflammaging). Unlike acute inflammation, which resolves after clearing a pathogen or repairing tissue injury, sterile chronic inflammation is sustained by metabolic and environmental inputs. The primary drivers in midlife adults are visceral adipose tissue expansion, intestinal dysbiosis with impaired mucosal barrier function, physical inactivity, and circadian misalignment. Non-pharmacological interventions target these upstream inputs using whole-food matrices: marine omega-3 polyunsaturated fatty acids (eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]), fermentable prebiotic fibres, live fermented foods, polyphenol-rich plants, regular aerobic and resistance exercise, and consolidated sleep and feeding windows. A clear evidentiary boundary separates these reproducible physiological levers from commercial wellness products. Consumer gut-brain and anti-inflammatory sachets frequently lead with proprietary bacterial strain names or isolated botanical extracts, implying direct effects on systemic inflammation, intestinal permeability ('leaky gut'), or mood. Outside of specific clinical indications—such as defined probiotic strains used to reduce antibiotic-associated diarrhoea in meta-analyses of randomised trials—isolated probiotic sachets lack consistent human trial evidence for lowering systemic inflammatory markers like hs-CRP or IL-6 in free-living adults.

Why it is used

Signaling along the interleukin-6 (IL-6) to C-reactive protein (CRP) pathway is causally implicated in human

atherothrombosis, as demonstrated by large prospective cohort studies and Mendelian randomisation analyses. Proof of principle in humans was established by the CANTOS randomised controlled trial (10,061 patients with prior myocardial infarction and hs-CRP of 2.0 mg/L or higher followed for a median of 3.7 years), which showed that pharmacologically blocking IL-1beta with canakinumab reduced recurrent major adverse cardiovascular events by 15% without altering low-density lipoprotein (LDL) cholesterol levels. Beyond vascular disease, persistent elevations in circulating TNF-alpha, IL-6, and IL-1beta impair insulin receptor substrate-1 (IRS-1) signaling in skeletal muscle and liver, accelerate neurodegenerative microglial activation, and promote skeletal muscle catabolism. For adults without established cardiovascular disease, pharmaceutical biologics carry unacceptable fatal infection risks and high costs. Dietary and behavioural interventions are used instead to lower the inflammatory burden alongside metabolic and vascular risk factors. Furthermore, because white blood cell differentials, serum albumin, and hs-CRP are core inputs in multi-system biological age algorithms (such as phenotypic age calculations tracked in LetsGo BioAge assessments), resolving low-grade inflammation directly improves both clinical risk profiles and composite biological age scores.

How it helps

Marine omega-3 fatty acids (EPA and DHA) alter cell membrane phospholipid pools in human immune cells by displacing arachidonic acid. This membrane shift reduces the substrate available for COX and lipoxygenase (LOX) enzymes to synthesise pro-inflammatory eicosanoids (prostaglandin E2 and leukotriene B4) and increases the synthesis of specialised pro-resolving mediators (resolvins, protectins, and maresins) that actively terminate inflammatory cascades. In meta-analyses of human randomised controlled trials, Mediterranean-style diets rich in marine omega-3s, extra-virgin olive oil polyphenols, and plant fibres reduce hs-CRP by roughly 0.5 to 1.0 mg/L in adults with elevated starting point risk. Dietary fibre diversity and fermented foods modulate the gut-immune axis through microbial metabolite production. When commensal colonic bacteria ferment soluble fibres, resistant starches, and polyphenols, they generate short-chain fatty acids (SCFAs)—primarily acetate, propionate, and butyrate. In human colonic mucosa and preclinical models, butyrate serves as the primary fuel for colonocytes, strengthens tight-junction protein assembly (claudins and occludins), binds to G-protein-coupled receptors (GPR41, GPR43, GPR109A), and inhibits histone deacetylases (HDACs). This epigenetic inhibition suppresses nuclear factor kappa B (NF-kappaB)—the master transcriptional regulator of TNF-alpha, IL-6, and IL-1beta—and promotes the differentiation of anti-inflammatory regulatory T (Treg) cells. Lowering intestinal permeability reduces the translocation of bacterial lipopolysaccharide (LPS, or endotoxin) into the portal and systemic circulation, blunting Toll-like receptor 4 (TLR4) activation on vascular endothelial cells and hepatic Kupffer cells. Human clinical trial evidence shows that increasing dietary fibre alone does not lower inflammation in every adult. In a 10-week randomised controlled trial of 36 healthy adults, a diet high in fermented foods (6 servings daily of yogurt, kefir, fermented cottage cheese, kimchi, vegetable brine drinks, or kombucha) steadily increased gut microbiota diversity and decreased 19 circulating inflammatory proteins, including IL-6, IL-10, and IL-12b. In contrast, the high-fibre intervention arm (increasing intake by roughly 20 grams per day) showed mixed inflammatory responses; participants with lower starting point microbial diversity lacked the carbohydrate-active enzymes required to degrade the sudden fibre load, resulting in gastrointestinal distress and unchanged or modestly increased inflammatory markers. Fermented foods supplied postbiotic metabolites and lactic acid bacteria that lowered immune activation first, creating a more permissive environment for subsequent fibre fermentation. Physical activity and visceral fat loss alter adipokine and myokine signaling in human tissues. Visceral adipose tissue is infiltrated by pro-inflammatory M1 macrophages that secrete TNF-alpha and IL-6, stimulating hepatic synthesis of CRP. Caloric restriction and visceral fat loss directly reduce M1 macrophage density in human adipose biopsies. Simultaneously, contracting skeletal muscle releases acute pulses of myokines, including muscle-derived IL-6, which acts in an anti-inflammatory manner in humans by stimulating IL-1 receptor antagonist (IL-1ra) and IL-10 while inhibiting TNF-alpha production. Circadian alignment regulates innate immune trafficking through core clock genes (BMAL1, CLOCK, PER, CRY)

that govern the rhythmic expression of pro-inflammatory cytokines in human monocytes and macrophages. Controlled human laboratory crossover studies (typically 8 to 14 days in healthy adults) demonstrate that sleep restriction, shift-work schedules, and eating during the biological night desynchronise peripheral clocks in the liver and gut from the central suprachiasmatic nucleus. This misalignment acutely elevates postprandial glucose, free fatty acids, and circulating hs-CRP and IL-6. Clinical outcome trials demonstrate that lowering hs-CRP via an isolated supplement does not guarantee tissue protection if the upstream metabolic driver remains intact. High-dose antioxidant vitamins or isolated botanical extracts transiently scavenge reactive oxygen species or modestly blunt NF-kappaB in cell culture, yet large human randomised trials of isolated antioxidant vitamins have failed to reduce cardiovascular events or mortality. Conversely, the PREDIMED randomised controlled trial (7,447 adults at high cardiovascular risk followed for a median of 4.8 years) demonstrated that a Mediterranean diet supplemented with extra-virgin olive oil or mixed nuts lowered both circulating inflammatory markers (IL-6, ICAM-1, VCAM-1, hs-CRP) and major adverse cardiovascular events by roughly 30%. The clinical outcome improved because the whole-food matrix addressed endothelial function, lipid oxidation, and postprandial lipaemia simultaneously.

Who should try it

Adults with starting point hs-CRP persistently between 1.0 and 3.0 mg/L (moderate vascular risk) or above 3.0 mg/L (elevated risk) on two separate fasting blood draws taken at least two weeks apart—in the absence of acute infection or tissue trauma—are the primary candidates for structured anti-inflammatory dietary and circadian interventions. The guide is also indicated for individuals with features of insulin resistance or metabolic syndrome: elevated fasting insulin, fasting triglycerides above 150 mg/dL (1.7 mmol/L), elevated apolipoprotein B (ApoB), or an increased waist-to-height ratio (above 0.5), as well as those with subclinical gastrointestinal complaints (bloating, irregular transit) where commercial probiotics have failed, or frequent circadian disruption from late-night work and dining schedules. Trade-offs, side effects, interactions, and contraindications require direct management: - Fermented foods and histamine: Fermented vegetables, aged dairy, and kombucha contain biogenic amines (histamine and tyramine). In individuals with histamine intolerance, mast cell activation disorders, or those taking monoamine oxidase inhibitors (MAOIs), fermented foods trigger headaches, flushing, palpitations, or gastrointestinal symptoms. - Rapid fibre escalation: Abruptly increasing fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs) or insoluble fibre in adults with irritable bowel syndrome (IBS), small intestinal bacterial overgrowth (SIBO), or stricturing inflammatory bowel disease (Crohn's disease) causes osmotic bloating, gas, and cramping. Fibre diversity must be titrated over several weeks, starting with low-FODMAP soluble fibres before expanding plant variety. - Botanical and concentrated supplements: Concentrated curcumin, high-dose omega-3 fish oils, and ginger extracts inhibit platelet aggregation and interact with cytochrome P450 enzymes. They increase bleeding risk when combined with antiplatelet agents (aspirin, clopidogrel) or anticoagulants (warfarin, apixaban, rivaroxaban) and must be paused prior to surgical procedures. - Cost and opportunity cost: Spending monthly budgets on proprietary probiotic sachets, NAD+ precursors, or unstandardised herbal blends diverts financial resources and adherence capacity from the four reproducible interventions: replacing ultra-processed foods with polyphenol- and omega-3-rich whole foods, titrating fermented foods and soluble fibre, eliminating late-night caloric intake, and building aerobic and resistance training volume.

Measurements that show improvement

High-sensitivity C-reactive protein (hs-CRP) is the primary clinical blood marker of systemic IL-6 and hepatic inflammatory signaling, with a target decrease toward <1.0 mg/L (and optimally <0.5 mg/L) over an 8- to 12-week intervention window. Because hs-CRP is an acute-phase reactant that spikes up to 100-fold during a viral upper respiratory infection, dental procedure, or within 48 hours of unaccustomed eccentric exercise, a

single elevated reading cannot be interpreted in isolation. starting point and follow-up trends require confirmation with two fasting measurements separated by 2 to 4 weeks under stable recovery conditions. Additional biomarkers and physiological metrics verify upstream resolution over 8 to 12 weeks: - Glycoprotein acetylation (GlycA): An NMR spectroscopy blood marker reflecting the concentration and glycosylation state of multiple acute-phase proteins (alpha-1-acid glycoprotein, haptoglobin, alpha-1-antitrypsin, alpha-1-antichymotrypsin, and transferrin). Direction of improvement: decrease. GlycA exhibits lower week-to-week intra-individual biological variability than hs-CRP and correlates independently with cardiometabolic risk in large human cohort studies. - Fasting insulin, HbA1c, and fasting triglycerides: Markers of adipose and hepatic insulin resistance, which drive sterile metabolic inflammation. Direction of improvement: decrease in fasting insulin, HbA1c, and triglycerides (target <100 mg/dL or <1.1 mmol/L). - Apolipoprotein B (ApoB): Vascular wall inflammation is driven by the subendothelial retention and oxidation of ApoB-containing lipoproteins. Direction of improvement: decrease. - Omega-3 Index: The percentage of EPA and DHA in red blood cell membranes, reflecting 90 to 120 days of dietary intake. Direction of improvement: increase toward 8% or higher. - Resting heart rate (RHR) and nocturnal heart rate variability (HRV): Autonomic nervous system markers. Vagus nerve efferent activity mediates the cholinergic anti-inflammatory pathway, suppressing macrophage cytokine release via the alpha-7 nicotinic acetylcholine receptor. Direction of improvement: lower nocturnal RHR (particularly when late-night meals and alcohol are eliminated) and higher or more stable nocturnal HRV. - Body composition (DEXA visceral adipose tissue mass or waist-to-height ratio): Direction of improvement: reduction in visceral adipose tissue (VAT) mass and a waist circumference below 0.5 of standing height.

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 1 to 3 servings daily of live fermented foods (plain kefir, live-culture unsweetened yogurt, kimchi, or unpasteurised sauerkraut) for 4 weeks prior to aggressive fibre escalation; pause immediately if histamine-related flushing, headaches, or gastrointestinal symptoms occur.
- Increase soluble and prebiotic fibre intake gradually by 3 to 5 grams per week across legumes, intact whole grains, nuts, seeds, and berries until reaching at least 30 grams daily, holding the dose steady if osmotic bloating occurs.
- Consume oily fish (sardines, mackerel, salmon, anchovies) 2 to 3 times per week and use extra-virgin olive oil as the primary culinary fat for 12 weeks to raise the red blood cell Omega-3 Index toward 8% or higher.
- Terminate all caloric intake at least 3 hours before sleep onset to align peripheral hepatic and intestinal clocks with the central circadian pacemaker, tracking nocturnal resting heart rate for a 2 to 5 bpm reduction.
- Retest fasting hs-CRP (confirmed with two draws 2 weeks apart, at least 48 hours after heavy exercise), fasting insulin, and ApoB after 8 to 12 weeks, aiming for an hs-CRP below 1.0 mg/L and a waist-to-height ratio below 0.5.

Safety note

Do not discontinue prescribed anti-inflammatory, anticoagulant, or immunosuppressive medications in favour of dietary changes or supplements without consulting the prescribing physician. Individuals with inflammatory bowel disease, histamine intolerance, or taking blood-thinning medications require individualised medical

guidance before introducing concentrated botanicals, high-dose omega-3 supplements, or high-FODMAP/fermented food guides.

Disclaimer

This article is for educational and informational purposes only and does not constitute medical advice, diagnosis, or treatment. Always seek the advice of a qualified physician or other licensed healthcare provider with any questions regarding a medical condition, biomarker interpretation, or before starting any new dietary, supplement, or exercise guide.

Reducing chronic low-grade inflammation in midlife does not require proprietary gut-brain sachets or unverified botanical stacks. Human randomised trials and mechanistic studies consistently point to four high-leverage interventions: shifting the dietary matrix toward marine omega-3s, polyphenols, and gradually titrated soluble fibres; incorporating live fermented foods to expand microbial diversity and downregulate cytokines like IL-6; reducing visceral adiposity through regular aerobic and resistance training; and aligning meal timing and sleep with the endogenous circadian clock. Tracking hs-CRP, fasting insulin, waist-to-height ratio, and nocturnal resting heart rate over 8 to 12 weeks provides an objective readout of whether these habits are resolving inflammatory risk.