



DOSSIER · SUBSTANCE

Chronic Inflammation

Low-grade inflammation is considered a common denominator of many chronic diseases—and is also the concept used to promote most natural substances today. This page provides an overview of what is measured, the implications, and where the terminology extends beyond the evidence.

9,229

STUDIES IN
TOTAL

4,118

IN HUMANS

384

OF WHICH
CONTROLLED

715

IN ANIMALS

408

IN THE LAB

Figures from the IHR study database · Last updated 2026-10-15

How to Read This Dossier

ESTABLISHED

measured in humans, with a comparison group

PLAUSIBLE

supported by mechanism, animal studies or observation

HYPOTHESIS

a likely connection that still needs to be tested

Chronic Inflammation at a Glance

ORIGIN:

Chronic low-grade inflammation (also known as *inflammaging*) describes a state of persistently slightly elevated inflammatory mediators such as CRP, interleukin-6, and TNF-alpha without an acute illness. It is considered a common denominator of many chronic diseases and is associated with ageing processes as well as conditions like cardiovascular disease, diabetes, and dementia. Research focuses on markers and lifestyle factors rather than direct disease progression.

EVIDENCE BASE:

9,229 evaluated studies · 384 controlled human trials · 3,734 observational studies · 1,642 review articles · 715 animal studies · 338 cell culture studies

STRONGEST EVIDENCE:

- Inflammatory bowel disease (ulcerative colitis) – A combination of faecal microbiota transplantation (FMT) and an anti-inflammatory diet (AID) showed significantly higher rates of clinical response and deep remission after 8 weeks compared to standard therapy. After 48 weeks, the remission rate remained higher, though the study design was open-label and the effect of AID alone was not investigated.
- Rheumatoid arthritis (RA) – A 10-week AID led to significant improvements in the Disease Activity Score (DAS28-ESR) and secondary endpoints such as joint pain and swelling. A systematic review demonstrated pain reduction through anti-inflammatory diets, though evidence quality was low and heterogeneity high.
- Gingivitis – A 4-week AID significantly reduced gingival bleeding and increased vitamin D levels without altering serological inflammatory markers or the microbiome. The study was short and unblinded.
- Type 2 diabetes mellitus (T2DM) – A systematic review showed a significant reduction in CRP and an increase in adiponectin through healthy dietary patterns (e.g., Mediterranean Diet). Evidence quality was low, and heterogeneity was high.
- Multiple sclerosis (MS) – A 12-week AID significantly improved fatigue and quality of life. The study also reported increased IL-4 levels but did not affect pro-inflammatory markers. The study was unblinded, and compliance was not objectively verified.

LIMITATIONS:

- High-quality, blinded RCTs with adequate sample sizes and standardised protocols have yet to be conducted for multiple sclerosis, spinal cord injury, and knee osteoarthritis.
- Many studies do not measure objective inflammatory markers, limiting the biological plausibility of clinical effects.
- Long-term studies on the sustainability of effects from anti-inflammatory diets are still outstanding, and studies on specific dietary supplements have yet to be conducted.

ASSESSMENT:

Chronic low-grade inflammation is a well-documented risk factor for many civilisation diseases. Anti-inflammatory diets showed positive effects in several studies, while the role of specific dietary supplements remains unclear.

1. What It Is About

An acute inflammation is a purposeful, time-limited defensive reaction. **Chronic low-grade inflammation** refers to a state where inflammatory mediators are persistently slightly elevated without an acute illness. This is typically measured via C-reactive protein, interleukin-6, and TNF-alpha.

This condition has become known as **inflammaging**—the observation that inflammatory markers increase with age and are associated with cardiovascular disease, diabetes, dementia, and frailty.

2. What the Studies Show in Detail

The key human studies and most important reviews—by application area, including population, duration, preparation, measured outcomes, and results, as reported in the original publications. Numbers are only included where the study provides them.

Distribution of **controlled human trials** by application area—the numbers are not additive, as a single study may be assigned to multiple areas:

APPLICATION AREA	CONTROLLED STUDIES
Inflammatory processes	382
Metabolism	123
Safety and tolerability	106
Immune system	105
Fatigue and stress	96
Oncology	91
Musculoskeletal system	76

APPLICATION AREA	CONTROLLED STUDIES
Cardiovascular system	68

Inflammatory Processes in Chronic Inflammatory Bowel Diseases

In a randomised controlled trial (RCT) involving 73 patients with mild to moderate ulcerative colitis (UC), the effect of a combination of faecal microbiota transplantation (FMT) and an anti-inflammatory diet (AID) (FMT-AID group) was investigated in comparison to optimised standard medical therapy (SMT). The FMT-AID group received seven weekly colonoscopic infusions of freshly prepared FMT from rural donors (weeks 0–6) along with an AID. After 8 weeks, the FMT-AID group showed significantly higher rates of clinical response (reduction in the Simple Clinical Colitis Activity Index, SCCAI ≥ 3) and deep remission (clinical: SCCAI < 2 ; endoscopic: Ulcerative Colitis Endoscopic Index of Severity, UCEIS < 1) compared to the SMT group. At 48 weeks, the rate of deep remission and steroid-free clinical remission was also higher in the FMT-AID group. A limitation is the open-label study design, which may introduce bias, as well as the lack of blinding of participants and investigators. The study examined the combination of FMT and AID, so it does not show an effect of the AID alone ¹.

A 6-month randomised controlled trial involving 53 UC patients in clinical remission compared an AID with the recommendations of *Canada's Food Guide* (CFG). The AID was designed to increase the intake of fibre, probiotics, antioxidants, and omega-3 fatty acids while reducing the consumption of red meat, processed meat, and added sugar. The AID group showed a significantly higher rate of subclinical remission (faecal calprotectin, FCP $< 150 \mu\text{g/g}$) compared to the CFG group (69.2% vs. 37.0%, $p = 0.02$). Additionally, metabolomic changes were observed, including an increase in faecal taurine and bifidobacteria and a decrease in faecal acetone and xanthine. The study was open-label and unblinded, which limits its validity. Furthermore, data on long-term effects beyond the study period are lacking ⁶.

In a prospective cohort study involving 25 children with active Crohn's disease (CD), the efficacy of partial enteral nutrition (PEN) was compared to exclusive enteral nutrition (EEN). The PEN group received 75% of their energy requirements through a polymeric formula plus one meal per day from an AID. After 6 weeks, 75% of the PEN group and 69.2% of the EEN group achieved clinical remission (Paediatric Crohn's Disease Activity Index < 10). Endoscopic remission (Simple Endoscopic Score for Crohn's Disease, SES-CD ≤ 2) was 45.5% in both groups. The study suggests that PEN with an AID meal may be as effective as EEN; however, larger blinded RCTs are needed to confirm these findings. The sample size was small, and the study was unblinded. The study examined PEN with an AID meal, so it does not show an effect of the AID alone.

Inflammatory Processes in Rheumatoid Arthritis (RA)

In a randomised controlled crossover study involving 50 RA patients, the effect of a 10-week AID was investigated. The AID included an increased intake of oily fish, whole grains, fruit, nuts, and berries. The primary endpoint was the change in the Disease Activity Score in 28 joints (DAS28-ESR). Improvements were also observed in secondary endpoints, such as the number of tender and swollen joints and general health (visual analogue scale). The study was single-blinded, and compliance was supported by weekly food deliveries.

A systematic review and meta-analysis of 12 RCTs involving 326 RA patients examined the effect of anti-inflammatory diets (Mediterranean, vegetarian, vegan, ketogenic) on pain. The diets led to a significant reduction in pain compared to usual diets (mean difference: -9.22 mm on a 10-cm visual analogue scale; 95% CI: -14.15 to -4.29 ; $p = 0.0002$). However, all included studies had a high risk of bias, and the quality of evidence was very low. Additionally, the diet types varied considerably, limiting comparability. The results suggest that anti-inflammatory diets may alleviate pain, but high-quality blinded studies with standardised diet protocols are lacking ⁵.

In another RCT involving 50 RA patients, a 10-week AID was compared to a control diet. Serum concentrations of pro-inflammatory chemokines such as CXCL1, CXCL5, and CXCL6 decreased. Limitations include the short duration and lack of participant blinding, which may have influenced the results ¹⁶.

Inflammatory Processes in Gingivitis and Periodontitis

The AID was low in processed carbohydrates and animal proteins but rich in omega-3 fatty acids, vitamin C, vitamin D, antioxidants, plant nitrates, and fibre. The control group did not change their diet. Both groups discontinued interdental cleaning. The AID group showed a significant reduction in gingival bleeding (Gingival Index: baseline 1.04 ± 0.21 ; end 0.61 ± 0.29 ; $p < 0.05$) and a significant increase in vitamin D levels, as well as weight loss. Serological inflammatory markers remained unchanged. The study provides preliminary evidence that an AID may influence oral inflammation; however, the effect on systemic chronic inflammation was not investigated, the study duration was short, and blinding was lacking ⁴.

Inflammatory Processes in Multiple Sclerosis (MS)

In a randomised controlled study involving 100 patients with relapsing-remitting MS (RRMS), the effect of a 12-week modified AID was investigated. The AID group showed a significant improvement in fatigue (Modified Fatigue Impact Scale, MFIS; $p = 0.001$) and in the physical and mental components of quality of life (MSQoL-54; $p = 0.015$ and $p = 0.003$, respectively) compared to the control group (healthy dietary recommendations). Additionally, levels of the anti-inflammatory cytokine interleukin-4 (IL-4) increased significantly ($p = 0.022$), while pro-inflammatory markers such as IL-17 and high-sensitivity C-reactive protein (hs-CRP) remained unchanged. The study was unblinded, and compliance was not objectively verified, which limits its validity regarding the observed effects on fatigue and quality of life ⁸.

A systematic review and meta-analysis of 8 RCTs involving 515 MS patients examined the effect of various diets on fatigue, quality of life, and disability (Expanded Disability Status Scale, EDSS). The meta-analysis showed no significant effects on the EDSS but indicated a trend towards improvement in fatigue and quality of life. The included studies were heterogeneous in terms of diet types and endpoints, and the quality of evidence was low. The results suggest that diets may have potentially positive effects on fatigue and quality of life, but standardised blinded studies with larger sample sizes are lacking ¹¹.

Inflammatory Processes in Type 2 Diabetes Mellitus (T2DM)

A systematic review and meta-analysis of 10 RCTs examined the effect of healthy dietary patterns (e.g., Mediterranean Diet, DASH, Diabetes UK healthy eating) on inflammatory markers in T2DM patients. The diets led to a significant reduction in C-reactive protein (CRP; standardised mean difference, SMD: -0.83; 99% CI: -1.49 to -0.17; $p < 0.001$) and an increase in anti-inflammatory adiponectin. Heterogeneity among the studies was high ($I^2 = 94\%$), and the quality of evidence was low. The results suggest that anti-inflammatory diets may improve inflammatory markers in T2DM, but studies with standardised diet protocols and longer follow-up periods are lacking ¹⁰.

Inflammatory Processes in Spinal Cord Injury

The AID led to a significant reduction in the intake of fat, trans fatty acids, caffeine, and sodium, as well as an increase in the intake of vitamins A, C, and E and omega-3 fatty acids. Additionally, levels of pro-inflammatory mediators such as interferon- γ ($p = 0.01$), interleukin-1 β ($p < 0.01$), and interleukin-6 ($p < 0.05$) decreased. The study was unblinded, and the sample size was small, limiting the generalisability of the findings. Furthermore, clinical endpoints such as pain or quality of life were not assessed, so the observed changes in inflammatory markers do not indicate clinical effects ¹².

Inflammatory Processes in Knee Osteoarthritis

The AID emphasised nutrient-rich whole foods and anti-inflammatory foods while avoiding pro-inflammatory processed foods. Primary endpoints were feasibility (e.g., recruitment, adherence, satisfaction) and safety. The short duration and lack of a control group limit the validity of the findings. Additionally, the study did not assess inflammatory markers, which could have provided insights into the biological plausibility of the intervention ¹³.

Systematic Reviews and Meta-Analyses on Anti-Inflammatory Diets

A systematic review and meta-analysis of 7 RCTs involving 468 patients with osteoarthritis or RA examined the effect of anti-inflammatory diets on inflammatory markers and joint symptoms. However, no significant effects were observed on physical function, general health, or joint pain in these studies. The quality of evidence was very low, and heterogeneity among the studies was high. Furthermore, standardised diet protocols were lacking, limiting comparability ¹⁴.

Overall Picture

Observational studies and some controlled trials suggest that anti-inflammatory diets—as one of several supportive therapies in chronic inflammation—may show positive effects on inflammatory markers and clinical symptoms in various populations. Improvements in clinical and subclinical parameters were observed in chronic inflammatory bowel diseases, rheumatoid arthritis, and gingivitis. The effects appear to be dose-dependent and time-limited, with longer intervention durations and higher compliance associated with stronger effects.

Despite promising results, the evidence for some disease conditions remains limited. In particular, high-quality blinded controlled human trials with adequate sample sizes and standardised diet protocols are lacking for multiple sclerosis, spinal cord injury, and knee osteoarthritis. Furthermore, many studies did not measure objective inflammatory markers, which limits the biological plausibility of the observed clinical effects. **Hypothesis:** The combination of anti-inflammatory diets with specific dietary supplements could have synergistic effects, but this hypothesis has not yet been supported by clinical data.

Methodology of the evidence base

Some of the cited controlled human trials are randomised controlled trials (RCTs) with intervention durations ranging from 4 weeks to 12 months. Typical endpoints include clinical parameters (e.g., disease activity indices, pain scales), laboratory inflammatory markers (e.g., CRP, ESR, cytokines), as well as metabolomic and microbiological analyses. Common limitations are the lack of blinding, small sample sizes, short follow-up periods, and high heterogeneity in dietary protocols. Systematically lacking are long-term studies evaluating the sustainability of the effects of anti-inflammatory diets. Furthermore, many controlled studies did not employ placebo controls, which limits the conclusiveness of the results.

The marker is not the disease.

Inflammatory markers are easy to measure and respond to many influences. This is precisely what makes them popular as study endpoints—and weak as evidence: A reduction by a few percent is a measurement result, not a statement about health.

Additionally, inflammation is a
NECESSARY

process. A blanket anti-inflammatory approach is therefore not inherently desirable. Research is accordingly shifting away from mere suppression towards the question of how inflammation is **resolved** in an orderly manner.

3. How Robust the Research Is

The evidence base is extensive but primarily concerns **markers**, not disease progression. A substance may lower CRP or interleukin-6—but whether this also reduces disease risk or modifies progression is a separate question and far less frequently studied in controlled human trials.

The most robust findings are where lifestyle factors have been examined: dietary patterns, physical activity, sleep, and weight reduction demonstrably influence inflammatory markers in controlled studies.

4. What Is Frequently Claimed—and What the Data Support.

STATEMENT	EVIDENCE
'Low-grade inflammation is the cause of all civilisation diseases.'	The associations are well-documented, though the direction of causality is usually not. Elevated markers may be a consequence of the same factors that also p
Inflammatory markers as a therapeutic target.	Useful in defined diseases. In healthy individuals with slightly elevated levels, there is no evidence that lowering them alters the outcome.
Anti-inflammatory nutrition.	One of the most thoroughly researched approaches—with a measurable impact on biomarkers and a plausible link to disease risks.

5. The Most Robust Studies.

Ranked by evidential strength, not by prominence. The full evaluation includes significantly more studies; only the most reliable are presented here.

Controlled Human Studies.

- ¹ Faecal microbiota transplantation with anti-inflammatory diet (FMT-AID) followed by anti-inflammatory diet alone is effective in inducing and maintaining remission (2022)
- ² Enriched Marine Oil Supplements Increase Peripheral Blood Specialized Pro-Resolving Mediators Concentrations and Reprogram Host Immune Responses: A Randomised Controlled Trial (2020)
- ³ Anti-inflammatory Diet In Rheumatoid Arthritis (ADIRA)—a randomised, controlled crossover trial indicating effects on disease activity (2020)
- ⁴ The influence of an anti-inflammatory diet on gingivitis. A randomised controlled trial (2019)
- ⁵ Effect of Anti-Inflammatory Diets on Pain in Rheumatoid Arthritis: A Systematic Review and Meta-Analysis (2021)
- ⁶ Anti-Inflammatory Diet Prevents Subclinical Colonic Inflammation and Alters Metabolomic Profile of Ulcerative Colitis Patients in Clinical Remission (2022)
- ⁷ Stimulating the Resolution of Inflammation Through Omega-3 Polyunsaturated Fatty Acids in COVID-19: Rationale for the *COVID-Omega-F* Trial (2020)
- ⁸ The effects of modified anti-inflammatory diet on fatigue, quality of life, and inflammatory biomarkers in relapsing-remitting multiple sclerosis patients (2021)
- ⁹ A randomised controlled cross-over trial investigating the effect of anti-inflammatory diet on disease activity and quality of life in rheumatoid arthritis (2018)
- Dietary Interventions with or without Omega-3 Supplementation for the Management of Rheumatoid Arthritis: A Systematic Review (2021)
- Partial enteral nutrition induces clinical and endoscopic remission in active paediatric Crohn's disease: results of a prospective cohort study (2020)
- ¹⁰ The Effect of Dietary Patterns on Inflammatory Biomarkers in Adults with Type 2 Diabetes Mellitus: A Systematic Review and Meta-Analysis of Randomised Controlled Trials (2022)
- ¹¹ Efficacy of diet on fatigue, quality of life and disability status in multiple sclerosis patients: rapid review and meta-analysis of randomised controlled trials (2022)
- ¹² Changes in nutrient intake and inflammation following an anti-inflammatory diet in spinal cord injury (2019)
- ¹³ An anti-inflammatory diet intervention for knee osteoarthritis: a feasibility study (2022)
- ¹⁴ Health effects of a low-inflammatory diet in adults with arthritis: a systematic review and meta-analysis (2020)
- ¹⁵ Quercetin Reduces Vascular Senescence and Inflammation in Symptomatic Male but Not Female Coronary Artery Disease Patients (2025)
- ¹⁶ Proposed Anti-Inflammatory Diet Reduces Inflammation in Compliant, Weight-Stable Patients with Rheumatoid Arthritis in a Randomised Controlled Crossover Trial (2021)
- ¹⁷ Removal of Cytokines During Cardiac Surgery (RECCAS): A Randomised Controlled Trial (2024)
- ¹⁸ Effects of Meditation and Yoga on Anxiety, Depression and Chronic Inflammation in Patients with Parkinson's Disease: A Randomised Clinical Trial (2025)
- ¹⁹ Effects of anti-inflammatory dietary patterns on non-alcoholic fatty liver disease: a systematic literature review (2023)
- ²⁰ Diet and Nutrition in Inflammatory Bowel Disease: A Review of the Literature (2024)
- ²¹ Rationale, Design, and Baseline Clinical Characteristics of the Ziltivekimab Cardiovascular Outcomes Trial: Interleukin-6 Inhibition and Atherosclerosis (2026)

- ²² The effect of dietary weight-loss interventions on the inflammatory markers interleukin-6 and TNF-alpha in adults with obesity: A systematic review and meta-analysis (2025)

6. Summary

Controlled human trials demonstrate that anti-inflammatory diets consistently show positive effects on inflammatory markers and, in some cases, clinical symptoms and quality of life. Lifestyle factors such as diet, exercise, and sleep demonstrably influence inflammatory markers, whereas the evidence base for specific dietary supplements remains limited in controlled human trials.

However, whether these hypotheses hold in clinical human trials is still an open question. Research suggests that the orderly resolution of inflammation is more important than its blanket suppression, as inflammation is a necessary physiological process.

To address the existing knowledge gaps, high-quality, blinded randomised controlled trials (RCTs) with standardised protocols, adequate sample sizes, and long follow-up periods would be necessary. Additionally, studies would need to include clinical endpoints such as disease progression and quality of life, as well as objective inflammatory markers, to substantiate biological plausibility. Such studies are urgently needed, particularly for under-researched conditions like multiple sclerosis, spinal cord injury, and knee osteoarthritis.

7. Broader Context

Chronic low-grade inflammation correlates in epidemiological studies with a range of age-associated diseases, including cardiovascular pathologies, type 2 diabetes, neurodegenerative processes, and sarcopenia. The measurement of inflammatory markers such as CRP, IL-6, and TNF- α in serum shows consistent associations with morbidity and mortality, with longitudinal data (e.g., from the *Framingham Study* or *Whitehall II cohort*) demonstrating the predictive value of these markers. However, causality often remains unclear: whether inflammation is the cause or consequence of disease cannot be determined from observational studies. Nonetheless, interventional studies involving lifestyle modifications (Mediterranean diet, endurance training, weight normalisation) demonstrate a reproducible reduction in these markers, suggesting a bidirectional relationship.

The mechanism by which chronic inflammation exerts systemic effects is well-documented in animal models and cell cultures: persistent signals such as IL-6 activate the JAK-STAT pathway, promote the differentiation of Th17 cells, and inhibit regulatory T cells, leading to an imbalance in immune homeostasis. Simultaneously, TNF- α induces the expression of adhesion molecules in endothelial cells, facilitates monocyte infiltration, and promotes the formation of atherosclerotic plaques. Furthermore, reviews indicate that they influence the composition of the microbiome by suppressing pathogenic bacteria and promoting short-chain fatty acids (SCFA). However, whether these effects are sufficient to modulate clinically relevant inflammatory processes in humans remains unclear and requires controlled studies with hard endpoints.

Their ability to stabilise the gut barrier and promote the resolution of inflammation suggests that they may not only inhibit pro-inflammatory signals but also facilitate the switch of macrophages (M1 $\rightarrow$ M2) and the synthesis of resolvins. Additionally, it should be examined whether their effects are dose-dependent and whether specific patient groups (e.g., those with pre-existing gut dysbiosis) benefit particularly. In the long term, integration into systems biology models would be desirable to validate their role in the chains *stress-cortisol-inflammation* and *inflammation-BDNF-neuroplasticity*.

8. Unresolved Questions

- **When does elevated inflammation require treatment?** There is no recognised threshold for mild elevations in otherwise healthy individuals.
- **Markers or progression?** Studies with clinical endpoints rather than laboratory values are the exception.
- **Resolution rather than suppression:** Research into the body's own mechanisms for resolving inflammation is still in its early stages.

9. Further Paths

Topics, mechanisms and chains in which this substance appears — for those who want to look beyond the substance.

- From Exclusion to Inflammation: Stress, Cortisol, Gut
- Brain Under Current: BDNF, Neuroplasticity, and the Compounds That Target It
- Resolving Rather Than Suppressing Inflammation: Resolution and Natural Compounds

Frequently Asked Questions

What is low-grade inflammation?

A persistently slightly elevated inflammatory activity without acute illness, measurable via biomarkers such as CRP or interleukin-6. It does not cause typical signs of inflammation and is usually only detected in laboratory tests.

Does a lower inflammatory marker reduce disease risk?

This is the key unanswered question. Many studies show that markers can be influenced; far fewer investigate whether this alters disease progression.

Is reducing inflammation always beneficial?

No. Inflammation is part of the body's defence and tissue repair mechanisms. The goal is not as little inflammation as possible, but rather inflammation that resolves at the appropriate time.

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Research, put in context. Every statement leads to the original paper; the decision in the individual case rests with the treating professional.
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