

Dietary Management as a Strategy for Reducing Inflammaging in Aging Populations

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Abstract: As global populations age rapidly, chronic, low-grade systemic inflammation has emerged as a primary biological driver of cardiovascular disease, cognitive decline, and frailty. This persistent state, known as inflammaging, develops without acute infection and steadily compromises tissue function over time. This review evaluates how dietary management can realistically modify this inflammatory background, using biomarkers such as hs-CRP, IL-6, and TNF- α as measurable endpoints. Current evidence indicates that pro-inflammatory nutritional profiles rich in saturated fats and high-glycemic carbohydrates elevate these markers. Whole-diet approaches such as the Mediterranean pattern are associated with lower inflammatory tone, plausibly through the combined effects of fiber, omega-3 fatty acids, and plant polyphenols on the gut microbiome. Comparing the reported numbers side by side brings out details that broad dietary claims tend to hide. The pooled reduction in hs-CRP for the whole Mediterranean pattern (-0.98 mg/L, 95% CI -1.48 to -0.49) is smaller than the reduction reported for curcumin supplementation alone (-1.55 mg/L, -1.81 to -1.30), and two independent syntheses of the same intervention reach opposite conclusions about statistical significance for CRP. Apparent contradictions in dietary efficacy frequently stem from measurement discrepancies between standard CRP and high-sensitivity hs-CRP assays rather than from actual physiological failures. The most recent meta-analysis of 33 randomized trials ($N = 3,476$) found significant pooled reductions in hs-CRP, IL-6, and IL-17 but not in CRP, TNF- α , or IL-10, confirming that the effect is specific to certain markers rather than general. The current evidence base also suffers from a severe demographic gap, as foundational randomized trials almost entirely exclude adults aged 80 and older. The only synthesis restricted to older adults drew its pooled estimate from five cross-sectional studies rather than from randomized comparisons (SMD -0.26 , 95% CI -0.41 to -0.11). Applying these anti-inflammatory principles to diverse elderly populations requires practical, localized translation. Benefit also differs by disease: high versus low adherence is associated with a relative risk of 0.72 for coronary heart disease and 0.83 for type 2 diabetes but only 0.96 for overall cancer incidence, and the outcomes with the strongest evidence are those most closely tied to the biomarkers examined here. Adapting these guidelines to Korean culinary traditions presents promising alternatives such as perilla oil and specific fermented foods, although these structural substitutions demand careful monitoring of sodium intake and macronutrient adequacy. Because inflammatory biomarkers are surrogate indicators rather than direct measures of biological aging, and because no validated biomarker panel currently defines inflammaging clinically, these results should be read as evidence that diet can lower inflammatory tone rather than as proof that it slows aging. Dietary management stands as a highly viable, non-pharmacological strategy for mitigating age-related inflammatory burden, provided future clinical research directly tests these customized interventions in the oldest demographic to verify measurable gains in survival and frailty.

Author keywords: Inflammaging; chronic low-grade inflammation; inflammatory biomarkers; dietary patterns; Mediterranean diet

Introduction

Population aging is accelerating globally across diverse economic strata. The World Health Organization (WHO) projects that the global share of individuals aged 60 or older will nearly double from 12% to 22% between 2015

and 2050.¹ By the late 2070s, the United Nations estimates that the population aged 65 or older will reach 2.2 billion, outnumbering children under 18.² South Korea exemplifies the leading edge of this shift. The 2025 Population and Housing Census recorded 10.72 million residents aged 65 or older, crossing the 20% threshold to officially classify the nation as a super-aged society.^{3,4} Concurrently, the census identified 8,604 centenarians, a 2.7-fold increase since 2015, translating these broad projections into a concrete normalization of extreme longevity.⁵

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As the older population grows, the burden of age-related disease also increases. WHO describes aging as the accumulation of molecular and cellular damage over time, which leads to declines in physical and mental capacity and a higher risk of disease.¹ Older adults often experience several conditions at the same time, a pattern known as multiple chronic conditions. Among the biological changes that occur with aging, chronic inflammation has emerged as a major factor influencing health decline and vulnerability to disease later in life.

According to Franceschi and Campisi,⁶ human aging is characterized by a chronic, low-grade inflammation called inflammaging, a systemic inflammatory state that develops even without obvious infection. This condition is driven by continuous immune activation and cumulative tissue damage. Inflammaging is now viewed as a major contributor to age-related diseases, including cardiovascular disease, diabetes, and cancer.⁷ As I learned more about aging and nutrition, I became especially interested in the idea that a quiet, ongoing inflammatory process could sit “upstream” of so many conditions that affect older adults. This raised a personal question: could something as familiar as daily eating patterns realistically change this chronic inflammatory state in real life, not just in theory?

Despite the biological complexity of inflammaging, dietary management remains a relatively feasible intervention because eating is a routine and essential part of daily life for older adults. If certain dietary patterns can consistently lower inflammatory biomarkers, they may offer a practical way to reduce inflammatory burden without relying only on medication. Therefore, this literature review focuses on how dietary management may contribute to reducing inflammaging and lowering its associated health risks in aging populations.

Background Information

Inflammaging is a concept that connects aging and chronic inflammation into a single framework. Franceschi et al.⁸ first described it as a progressive, low-grade, pro-inflammatory state that, unlike acute inflammation following infection, develops without a specific external injury.⁹ This underlying chronic state is thought to play a major role in many age-related diseases.

Higher levels of inflammatory biomarkers such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- α) are linked to cardiovascular disease, cognitive decline, and frailty in older adults, reflecting a systemic state rather than effects tied to a single organ or disease.

At the same time, nutrition science has shown that dietary patterns can shape metabolic health, gut microbiome composition, and oxidative stress. These same systems are closely connected to inflammation. This intersection of aging biology, chronic low-grade inflammation, and diet forms the central background for this review.

Methods: Literature Search and Selection

Search strategy

To explore the relationship between dietary patterns and inflammaging, I conducted a desk-based literature review combining qualitative synthesis of mechanisms with quantitative findings from clinical and observational studies, focusing on how dietary components and patterns influence key inflammatory biomarkers related to aging. Academic sources were identified using scientific databases such as PubMed, ScienceDirect, and Google Scholar, as well as reports from major health organizations, including WHO and the United Nations. Search terms included combinations of keywords such as “inflammaging,” “chronic low-grade inflammation,” “immunosenescence,” “diet and inflammation,” “Mediterranean diet inflammatory biomarkers,” “gut microbiome and aging,” “aging population inflammation,” and “dietary patterns of older adults.”

The primary search window focused on publications from approximately 2000 to 2024, reflecting the period during which the concept of inflammaging was developed and widely discussed. Foundational papers that originally defined or framed inflammaging, such as Franceschi et al.,⁸ were included even if they were published before this range because they established key concepts used in more recent research.

Inclusion and exclusion criteria

Studies were selected based on their relevance to the main research question: how dietary components and patterns are associated with, or causally influence, inflammatory biomarkers linked to aging. Priority was given to peer-reviewed review articles, clinical trials, and large observational studies that reported data on markers such as hs-CRP, IL-6, and TNF- α . Research that specifically examined elderly or aging populations was preferred, but studies involving general adult populations were also included when they contributed important mechanistic insight or dietary evidence.

Opinion pieces, non-peer-reviewed sources, and studies that did not report any inflammatory biomarkers or did not relate directly to diet and inflammation were excluded. When multiple sources discussed similar mechanisms or dietary components, I focused on those that were more recent, more widely cited, or grounded in randomized controlled trial data rather than on observational associations alone. The final set of articles was used to build a narrative review connecting mechanisms of inflammaging with measurable biomarkers and diet-responsive pathways.

Because this project was conducted as an independent review by a single student author, it represents a selective narrative synthesis rather than an exhaustive systematic review. Consequently, some relevant literature may have been omitted. Where quantitative estimates from other studies are reproduced in this paper, they are presented in the units used by the original source and were not converted to a standardized scale.

Literature Review

Mechanisms of inflammaging

Franceschi's⁸ paper defines inflammaging as a progressive, chronic, low-grade, pro-inflammatory status that increases with age. "Progressive" here means that inflammatory tone gradually rises over time instead of remaining stable, reflecting a decline in resilience to internal and external stressors. "Low-grade" refers to a persistent, mild elevation in inflammatory activity that is still strong enough to influence tissue function and disease risk over long periods. This phenomenon has been further described through the concept of senoinflammation, which highlights the aging-specific cellular and molecular basis of chronic inflammation.¹⁰

A key point is that in aging, the problem is not the strength of each inflammatory response, but the body's difficulty in returning to baseline after repeated immune activation. In a younger, healthy system, inflammation is usually an adaptive response that resolves after a threat is removed. As people age, this resolution phase becomes less efficient. Immune activation is repeatedly triggered by a continuous antigenic load and other stressors, while the capacity to cope with these stressors decreases. The result is a persistent, low-grade inflammatory state driven by cumulative exposures and impaired immune regulation rather than by any single acute trigger.

Inflammaging does not come from a single source. It emerges from multiple interacting inputs that can be organized into three broad categories.

The first category involves persistent signal sources. Some aging cells become senescent, meaning they permanently exit the cell cycle without dying. Rather than clearing away naturally, these cells linger in tissue and continuously release inflammatory signals through the senescence-associated secretory phenotype (SASP). This process keeps the surrounding tissue in a state of chronic irritation.¹¹

The second category involves cellular stress amplifiers. As mitochondria age, they release fragments of their own DNA and other damage-associated molecular patterns (DAMPs) into the bloodstream. Because these fragments structurally resemble bacterial DNA, the innate immune system often misidentifies them as an infection and mounts an unnecessary inflammatory response.¹¹

The third category covers failures in immune regulation and resolution. Age-related changes in the immune system alter innate immune responses and sustain cytokine production over time. This immune dysregulation closely affects intestinal health. Aging naturally thins the gut lining and shifts the composition of the gut microbiome. Such barrier dysfunction allows pathogenic components to leak into the circulation, creating a critical inflammatory pathway that is highly responsive to dietary change.¹¹

These underlying processes do not occur uniformly in every older adult. Their relative impact varies widely based on individual genetics, comorbid conditions, medications, adiposity, physical activity levels, and infection history.

Inflammaging operates as a heterogeneous biological framework rather than as a single, fixed consequence of chronological age.

Despite this individual variation, these mechanisms show that inflammaging acts as a systems-level phenomenon. Small but persistent changes across immune, metabolic, and tissue-maintenance pathways accumulate to form a chronic inflammatory background that ultimately shapes later-life health.

Inflammaging and age-related disease

Inflammaging can be studied and monitored through inflammatory biomarkers. The most widely used markers in clinical and epidemiological research are hs-CRP, IL-6, and TNF- α . While these markers provide indirect evidence of systemic inflammatory activity, they do not directly measure biological aging, and no single validated biomarker panel currently defines inflammaging clinically.¹² Clinical observations consistently show that elevated marker levels predict frailty, functional decline, and mortality in older adults.¹¹ Frail individuals demonstrate significantly higher CRP and IL-6 concentrations compared to robust peers,¹³ and older cohorts in the highest IL-6 quartile have shown a nearly doubled relative risk of mortality over time.¹⁴ These observational findings establish prognostic relevance rather than a direct causal role for the biomarkers in driving aging. Despite this limitation in establishing causality, these biomarkers are highly responsive to lifestyle modification. Importantly, they change in response to dietary composition, making them useful indicators for evaluating dietary interventions targeting inflammation.

Elevated inflammatory markers are particularly strongly linked to cardiovascular disease, dementia, and cognitive decline. This pattern suggests that inflammaging functions less as a single-disease risk factor and more as a shared upstream condition underlying multiple age-related diseases at once.⁷ Because inflammaging is a common pathological foundation across these diverse systems, neurodegeneration, and metabolic disease, strategies that reduce it may have benefits across several conditions simultaneously. Direct evidence linking marker reduction to slower disease progression for each condition individually is still limited. However, dietary patterns offer a compelling proof of concept. An umbrella review of meta-analyses shows that adherence to an anti-inflammatory model such as the Mediterranean diet provides differential risk reductions across disease categories. It demonstrates robust associations for improving cardiovascular outcomes and type 2 diabetes but a much smaller effect on overall cancer incidence¹⁵ (see the quantitative comparison below). This idea helped shape the central focus of this review: if diet can meaningfully influence inflammaging, then dietary management might become a strategically important lever in elderly health.

Dietary components and inflammatory biomarkers

Galland¹⁶ showed that habitual dietary patterns and specific nutrients have a moderate but meaningful influence on

chronic, low-grade systemic inflammation. Food intake directly affects blood glucose regulation, lipid metabolism, gut microbiome composition, and cellular membrane structure. Because inflammation is ultimately an immune system response to changes in the internal environment, shifts driven by diet can raise or lower inflammatory tone. The strength of the evidence varies significantly across these dietary exposures. Labels such as “pro-inflammatory” and “anti-inflammatory” rarely reflect uniform or clinically established effects.

Pro-inflammatory dietary components are relatively well characterized. High-glycemic index (GI) foods, such as white bread, sugar, and refined carbohydrates, cause rapid spikes in blood glucose, excessive insulin secretion, increased oxidative stress, and elevated inflammatory signaling, leading to higher hs-CRP levels. Ultra-processed foods present a comparable risk. The Melbourne Collaborative Cohort Study linked higher consumption of these products directly to greater hs-CRP concentrations in adults.¹⁷ In terms of fat type, saturated fatty acids from sources such as butter and processed meats activate inflammatory gene expression and increase hs-CRP. Trans fatty acids are similarly linked to increases in TNF- α and IL-6, with stronger effects observed in individuals with a higher BMI.¹⁶

Several dietary components appear to have anti-inflammatory effects, although the strength of the evidence differs considerably among them. Increased fiber intake correlates with lower hs-CRP. Specific clinical trials show that fiber doses of 3.3–7.8 g/MJ can reduce this marker by 25–54 percent, a trend that persists even when factoring in concurrent weight loss or altered fat intake.¹⁸ Fiber helps buffer inflammation by slowing glucose absorption and supporting beneficial gut bacteria. Large population studies reinforce this pattern, showing that individuals with the highest fiber intake consistently face lower odds of elevated CRP.^{19,20} Omega-3 fatty acids from fish and nuts are inversely associated with systemic inflammatory markers. An umbrella review of 32 meta-analyses reported significant reductions in CRP, IL-6, and TNF- α following omega-3 supplementation.²¹ A meta-analysis restricted to patients with diabetes or cardiovascular disease was less encouraging, finding no significant effect on CRP or TNF- α and not measuring IL-6 at all.²² Pooled data from 30 randomized trials also indicate that olive oil rich in monounsaturated fatty acids lowers CRP and IL-6 while softening the postprandial inflammatory responses.^{23,24}

Fruits and vegetables provide carotenoids, flavonoids, and magnesium, which are also associated with lower IL-6 and hs-CRP. The evidence for these specific nutrients is slightly weaker than that for fiber or omega-3 fatty acids. Cross-sectional findings associate flavonoid and serum carotenoid intake with inversely lower CRP in thousands of adult participants.^{25,26} Magnesium supplementation produced no significant overall change in CRP across 11 randomized trials. A clear reduction appeared only in participants whose baseline CRP exceeded 3 mg/L (–1.12 mg/L, 95% CI –2.05 to –0.18).²⁷ A meta-analysis of randomized trials found that the polyphenol curcumin lowered CRP, IL-6, and TNF- α .²⁸

Comparing these specific nutrients requires defining the distinct exposure variables. Nutrient supplementation, whole-food intake, circulating nutrient concentrations, and complete dietary patterns represent fundamentally different metrics. A finding from isolated nutrient supplementation does not automatically translate to comprehensive eating patterns such as the Mediterranean diet. Serum carotenoid concentration serves as an exposure biomarker rather than a direct measure of dietary intake. These nutrient-level effects provide a mechanistic basis for understanding how different overall dietary patterns influence inflammaging.

Diet, the gut microbiome, and inflammaging

The mechanisms described above intersect most directly in the gut microbiome. Aging weakens the gut barrier, and diet shapes the bacterial community residing behind it. The microbiome therefore sits between what a person eats and how much inflammation the body carries. Evidence from animal models illustrates this relationship. Thevaranjan et al.²⁹ observed that germ-free mice lacked the age-related increase in circulating inflammatory markers typically seen in conventionally raised mice. Introducing these germ-free mice to older conventional mice triggered the inflammatory rise. In this model, the aging gut microbiota contributes to inflammaging rather than merely coexisting with it. The reversibility of this effect identifies the microbiome as a practical, modifiable target.

Human gut microbiota diversity in older adults correlates closely with specific dietary patterns and frailty measures.³⁰ Fiber acts as a fundamental link in this process. Gut bacteria ferment fiber into short-chain fatty acids such as butyrate to nourish the intestinal lining and preserve barrier integrity. A low-fiber, high-fat dietary approach typical of Western diets disrupts this microbial balance. Increased gut permeability follows, allowing microbial components to leak into the circulation and sustain systemic inflammation.¹¹ Current human evidence establishing this link remains largely associative, and confirming that diet-induced microbiome shifts cause sustained clinical improvements requires further trial data. Preserving microbial balance therefore offers a broader strategy for managing systemic inflammation than isolated nutrient intake alone.

Anti-inflammatory dietary patterns: the Mediterranean diet

While individual nutrients are important, people consume whole dietary patterns rather than isolated components. The Mediterranean diet brings many anti-inflammatory elements together into a single pattern. This diet typically features a high consumption of olive oil, fish, fruits, vegetables, whole grains, and nuts. It also includes a moderate intake of dairy and wine alongside a relatively low intake of red meat and processed foods.

Randomized trials provide the most direct evidence for this whole-pattern approach. Esposito et al.³¹ tracked participants over a two-year intervention and found that a Mediterranean-style diet significantly reduced serum concentrations of hs-CRP and IL-6 compared to a low-fat

control diet ($P = .01$ and $P = .04$, respectively) based on American Heart Association guidelines. Another trial evaluated a Mediterranean diet supplemented with either olive oil or nuts to isolate specific benefits. Participants in the olive oil group experienced a marked drop in CRP (-0.54 mg/L, 95% CI -1.04 to -0.03 , relative to the low-fat group), suggesting that the specific fat source within the broader pattern matters.³² Changing overall eating habits can lower several inflammatory biomarkers in controlled trial settings. The Mediterranean pattern inherently prioritizes fiber, ω -3 PUFAs, MUFAs, and plant-based antioxidants, aligning directly with the nutrient-level mechanisms described earlier.

Quantitative comparison of reported effects

Table 1 compares the magnitude of these dietary effects by collecting the reported estimates and categorizing their specific evidence types. Distinguishing between methodologies is necessary because widely repeated figures do not always represent true pooled estimates. The frequently cited 25–54 percent reduction in hs-CRP attributed to fiber actually reflects a range of within-trial percentage changes from a qualitative review. North et al.¹⁸ explicitly noted that concurrent weight loss and altered fat intake accompanied most of those fiber interventions. Clarifying these evidence types provides a more accurate picture of what each dietary component realistically achieves without overstating the isolated effects of single nutrients.

Table 1 shows that these interventions differ in important ways, both in how they were studied and in whom. Curcumin supplementation reports a highly potent CRP

reduction of -1.55 mg/L, but these trials utilize polyphenol doses many times higher than those obtained from ordinary food supplies. The Mediterranean pattern relies on whole-diet interventions. Treating these isolated, high-dose supplements and comprehensive dietary modifications as equivalent ignores the fact that they represent fundamentally distinct research constructs. Efficacy also depends heavily on specific clinical subgroups and baseline conditions. Magnesium shows no overall effect on CRP but shows a reduction in participants whose baseline CRP exceeded 3 mg/L. This outcome suggests that baseline inflammatory status matters as much as the nutrient itself, meaning that grouping conditional nutrients like magnesium equally with robust anti-inflammatory interventions ignores a critical difference in evidence quality. Omega-3 PUFA supplementation exhibits a similar dependency. While umbrella meta-analyses indicate significant overall reductions in inflammatory markers from omega-3s,²¹ targeted trials demonstrate inconsistent responses. Natto et al.²² found that EPA/DHA supplementation produced no significant reduction in CRP among diabetic patients and omitted IL-6 measurements entirely. These disparities indicate that pooled analytical benefits do not translate uniformly across all clinical demographic groups.

Independent meta-analyses evaluating the exact same intervention reveal similar instability. Schwingshackl and Hoffmann³³ pooled 17 trials and found that the Mediterranean diet lowered hs-CRP by 0.98 mg/L (95% CI -1.48 to -0.49), yielding a statistically significant result. Koelman et al.³⁴ reported an almost identical point estimate of -1.00 mg/L for standard CRP (95% CI -2.02 to 0.01), but their confidence interval crossed zero, rendering the result

Table 1. Quantitative comparison of dietary components and patterns on inflammatory biomarkers

Source (year)	Study design	Population and sample size	Dietary intervention /exposure	Duration	Biomarkers measured	Principal findings	Limitations and notes
North et al. ¹⁸	Qualitative systematic review (7 clinical trials)	Adults, N = 554 (192 M/362 F)	Dietary fiber (3.3–7.8 g/MJ)	3 weeks–24 months	hs-CRP	25–54% within-trial reduction	Confounded by weight loss and altered fat intake; no pooled estimate
Kavyani et al. ²¹	Umbrella meta-analysis (32 meta-analyses)	Adults, N = not reported	Omega-3 PUFAs (supplement)	Not reported	CRP, IL-6, TNF- α	ES -0.40 (CRP) ES -0.22 (IL-6) ES -0.23 (TNF- α)	Supplement doses significantly exceed ordinary dietary intake
Natto et al. ²²	Meta-analysis (16 studies)	Adults with diabetes or CVD (9 diabetic, 7 CVD)	EPA/DHA supplement ($\geq 1,000$ mg/day)	Not reported	CRP, TNF- α	CRP and TNF- α both nonsignificant	IL-6 not measured; demonstrates variable response in specific patient subgroups
Ferguson et al. ²⁸	Meta-analysis of randomized trials (32 RCTs)	Adults, N = 2,038	Curcumin (polyphenol supplement)	Not reported	CRP, IL-6, TNF- α	-1.55 mg/L (CRP) -1.69 pg/mL (IL-6) -3.13 pg/mL (TNF- α)	Supplement doses significantly exceed ordinary dietary intake

Table 1. (Continued)

Source (year)	Study design	Population and sample size	Dietary intervention /exposure	Duration	Biomarkers measured	Principal findings	Limitations and notes
Simental-Mendia et al. ²⁷	Meta-analysis (11 RCTs)	Adults, N = not reported	Magnesium (supplement)	Not reported	CRP	Overall: −0.11 mg/L (ns) Baseline > 3 mg/L: −1.12 mg/L	Overall effect nonsignificant; reduction isolated to high-baseline subgroups
Chun et al. ²⁵	Cross-sectional (NHANES)	Adults, N = 8,335	Dietary flavonoids	N/A	CRP	Inverse association (P < .05)	Cross-sectional design; cannot establish causal relationship
Jing et al. ²⁶	Cross-sectional	Middle-aged and older adults, N = 3,031	Serum carotenoids	N/A	hs-CRP, IL-6	Partial r = −0.114 (hs-CRP); ns for IL-6	Exposure biomarker reflecting circulating status rather than intake
Schwingshackl & Hoffmann ³³	Meta-analysis (17 trials)	Adults, N = 2,300	Mediterranean diet vs. control	N/A	hs-CRP, IL-6	−0.98 mg/L (hs-CRP) −0.42 pg/mL (IL-6)	Heterogeneity 91% and 81%; averages mask between-trial variation
Koelman et al. ³⁴	Meta-analysis (22 RCTs)	Adults, N = not reported	Healthy patterns incl. Mediterranean	N/A	CRP, IL-6	−1.00 mg/L (CRP, ns) −1.07 pg/mL (IL-6)	Same point estimate as Schwingshackl but ns; heterogeneity 100%
Keshani et al. ³⁵	Meta-analysis (33 RCTs)	Adults, N = 3,476	Mediterranean diet vs. control	N/A	hs-CRP, IL-6, IL-17, CRP, IL-10, TNF-α	Reduced: hs-CRP, IL-6, IL-17 Not reduced: CRP, IL-10, TNF-α	Effect is marker-specific; hs-CRP and CRP diverge
Wu et al. ³⁶	Meta-analysis (13 studies, 3 RCTs)	Older adults, N = not reported	Mediterranean -diet adherence	N/A	CRP	SMD −0.26 (−0.41, −0.11), P < .001	Pooled from 5 cross-sectional studies; association, not causal

Note: ES = standardized effect size; ns = not significant; RCT = randomized controlled trial. Estimates reflect the exact units and metrics reported in each study and have not been converted to a standardized scale. Individual intervention trials are summarized separately in [Table 2](#).

nonsignificant amid severe heterogeneity (91% and 100%, respectively). The directional effect remains consistent across both studies despite generating contradictory conclusions regarding statistical significance. A recent synthesis clarifies this specific discrepancy by separating the two analytical assays. Across 33 randomized trials, Keshani et al.³⁵ found significant pooled reductions in hs-CRP, IL-6, and IL-17 without any significant effect on standard CRP, TNF-α, or IL-10. This pattern indicates that the discrepancy is primarily a measurement issue rather than a fundamental disagreement about dietary efficacy. The high-sensitivity hs-CRP assay detects subtle inflammatory shifts that standard CRP misses. A 2026 umbrella review reported that evidence certainty ranges from high to very low strictly depending on the specific dietary pattern and biomarker examined.³⁷

[Table 2](#) compares the individual intervention trials underlying these broader estimates. Looking at the individual trials shows a limitation that the pooled figures hide. The trials most frequently cited in support of anti-inflammatory eating largely exclude older adults, the exact demographic to whom

these conclusions typically apply. Two Korean feeding trials are included alongside these foundational studies to directly support the subsequent discussion on cultural dietary adaptation later in this review.

The Mediterranean diet does not appear to protect equally against every age-related disease. An umbrella review of meta-analyses shows that cardiovascular benefits carry the most robust quantitative support.¹⁵ High dietary adherence correlates strongly with reduced risks of coronary heart disease at a relative risk of 0.72 (95% CI 0.60–0.86) and myocardial infarction at 0.67 (0.54–0.83). Type 2 diabetes and neurodegenerative diseases show a more moderate inverse association, with relative risks of 0.83 (95% CI 0.74–0.93) and 0.87 (0.81–0.94). The link to overall cancer incidence remains minimal at 0.96 (0.95–0.97). These observational figures cannot support strict causal comparisons due to varying outcome definitions and baseline risks across trials. Cardiovascular and metabolic conditions benefit most heavily from Mediterranean dietary patterns, a finding that

Table 2. Design, population, intervention, duration, biomarkers, findings, and limitations of key intervention trials

Study (year), design	Population, age	N	Intervention	Duration	Biomarkers	Findings and limitations
Esposito et al. ³¹ ; RCT, single-blind, single center	Metabolic syndrome, ~44 y, Italy	180	Mediterranean-style diet vs low-fat control diet	2 years	hs-CRP, IL-6, IL-7, IL-18	hs-CRP and IL-6 significantly reduced ($P = .01$, $P = .04$); IL-7/IL-18 results not relied upon. Limitations: participants were middle-aged, not elderly; weight loss (4.0 vs 1.2 kg) confounds marker changes.
Estruch et al. ³² ; RCT, PREDIMED substudy, multicenter	High CV risk, 55–80 y, Spain	772	MedDiet + olive oil or + nuts vs low-fat advice	3 months	CRP, IL-6, ICAM-1, VCAM-1	IL-6 and ICAM-1 decreased in both Mediterranean arms; CRP and VCAM-1 decreased significantly only with olive oil (CRP -0.54 mg/L, 95% CI -1.04 to -0.03 vs low-fat). Limitations: 3-month duration; control arm had less intensive counseling (co-intervention bias).
Ghosh et al. ³⁸ NU-AGE; RCT, single-blind, 5 countries	Non-/pre-frail community-dwelling adults, 65–79 y	612	12-month Mediterranean diet tailored to older adults vs habitual diet	1 year	hs-CRP, IL-17, microbiome, frailty	Diet-associated microbial taxa correlated negatively with hs-CRP and IL-17, positively with lower frailty and better cognition. Limitations: inflammation/frailty links are associative, not randomized comparisons; sample excluded frail and institutionalized older adults.
Shin et al. ³⁹ ; RCT, controlled feeding, single center	Obese women, 50–60 y, Korea	10	Traditional low-inflammatory-index Korean diet vs westernized Korean diet, 1,800 kcal/d	2 weeks	CRP, IL-6, TNF- α , IL-10, NF- κ B, fibrinogen, IFN- γ	IL-10 rose ($P = .010$), NF- κ B fell ($P = .002$), inversely correlated ($r = -0.794$, $P = .006$); CRP, IL-6, TNF- α , fibrinogen, IFN- γ unchanged. Limitations: N=10, 2 weeks; midlife women, not older adults.
Wastyk et al. ⁴⁰ ; RCT, two-arm parallel	Healthy adults, ~51 y, USA	36	High-fermented-food diet vs high-fiber diet	17 weeks	93 immune and serum proteins, incl. IL-6	Primary outcome (Cytokine Response Score) nonsignificant in both arms; IL-6 decreased within the fermented-food arm but not between arms. Limitations: 18/arm; healthy middle-aged cohort only; no habitual-diet control.
H.-K. Chung et al. ⁴¹ ; RCT, parallel, single centre	Metabolic syndrome, ~71 y, Korea	31	Antioxidant-rich prepared meals delivered daily vs usual diet	4 weeks	hs-CRP, TNF- α , ox-LDL, lipid peroxidation, PWV	Waist circumference, visceral fat ratio, oxidized LDL, blood pressure, and pulse wave velocity improved vs control; hs-CRP and TNF- α unchanged. Limitations: N = 31, 4 weeks; underpowered for a modest CRP effect.

Note: No intervention trial identified for this review enrolled adults aged 80 or older. Ghosh et al.³⁸ and Chung et al.⁴¹ are the primary trials listed whose populations overlap substantially with older adults. The inflammatory findings of Ghosh et al. are correlational rather than a randomized comparison between arms. H.-K. Chung et al. was powered for oxidative and vascular endpoints rather than for inflammatory markers. The two Korean trials,^{39,41} represent the shortest durations and smallest sample sizes in the table.

aligns perfectly with the systemic inflammatory biomarker improvements discussed earlier.

Comparison of Dietary Patterns and Current Limitations

Not all dietary patterns provide the same level of protection against inflammation. The Western diet typically features high levels of fructose, saturated fat, refined carbohydrates, and processed foods, alongside low fiber and micronutrient intake. This specific nutritional profile is associated with increased inflammation through gut microbiome

disruption and elevated pro-inflammatory markers.^{11,17,30} Dietary patterns rich in fiber and plant-based foods, such as Mediterranean-style diets, consistently show inflammation-reducing tendencies.³⁵

Current evidence regarding these diets presents several distinct limitations. Diet interventions produce measurable effects on inflammatory markers, yet the overall changes remain modest. Different biomarkers do not always move in the same direction or achieve statistical significance. The frequent divergence between CRP and hs-CRP in more recent syntheses clearly illustrates this measurement challenge. Individual responses to dietary shifts also vary widely based on baseline health status, genetic factors, and microbiome

composition. Existing research rarely focuses exclusively on elderly populations. This demographic gap complicates the direct application of these findings to adults aged 80 and older. It is also difficult to isolate the specific effect of diet from other healthy behaviors and concurrent weight loss. Randomized trials like Esposito et al.³¹ help mitigate this confounding factor, but behavioral variables often remain intertwined in broader studies.

Discussions and Guidelines for Applications

For aging populations, the Mediterranean pattern remains relevant because it adapts easily to diverse cultures and food environments. This flexibility preserves its core anti-inflammatory mechanisms. Cultural adaptations simply require replacing saturated fats with healthy oils, substituting processed meat with fatty fish and legumes, and maintaining a high proportion of fiber-rich produce. From an inflammation perspective, this diet demonstrates how multiple mechanisms function together. Improved lipid profiles, better glycemic control, healthier gut microbiota, and reduced oxidative stress integrate through daily eating habits rather than through isolated nutrient supplements.

Translating these recommendations into older populations introduces concrete practical constraints. While replacing refined carbohydrates and saturated fats with healthy oils and legumes serves as a viable first step for elderly care settings, structural barriers persist. Cost presents an immediate hurdle, as fish, nuts, and high-quality oils carry a significant premium in Korea. Physical changes complicate adherence further: reduced dentition and chewing difficulty make raw vegetables, nuts, and whole grains harder to eat, while diminished appetite and altered taste reduce total intake. High-fiber foods increase satiety, and this fullness can crowd out the protein and energy older adults need to preserve muscle mass; when these goals conflict, basic nutritional adequacy must take priority over specific fiber targets. Because institutional environments such as nursing homes and hospitals operate on fixed cycle menus and texture-modified diets, guidance for these settings requires menu-level structural substitutions rather than individual dietary choices.

Korean demographic data illustrate this trade-off clearly. Kwak et al.⁴² observed 127 rural women aged 85 and older whose plant foods supplied 87.5 percent of dietary intake by weight. Despite this high plant consumption, 52.7 percent of the group fell below the estimated average requirement for vitamin B12. Fermented soybean foods, seaweeds, and kimchi accounted for a large portion of the B12 they did obtain. In this oldest-old demographic, a heavily plant-based pattern and micronutrient adequacy are not automatically compatible. Recommendations raising plant intake without securing B12 and protein may improve inflammatory markers while actively worsening overall nutritional status.

Transplanting the Mediterranean diet wholesale outside its region creates significant adherence problems, as the diet functions primarily as a regional cuisine rather than a universal nutritional prescription. Its underlying nutrient

profile, however, translates much more effectively. High fiber, unsaturated fats, abundant plant polyphenols, and regular fermented foods map directly onto existing culinary traditions. Korean cuisine already supplies several of these elements. Namul preparations and traditional stews provide vegetables in a cooked, softened form, suiting older adults much better than raw salads. Mixed-grain rice with barley raises dietary fiber without introducing unfamiliar textures. Finally, perilla oil sees wide use in Korean cooking and contains approximately 60 percent alpha-linolenic acid, serving as a highly viable domestic alternative when imported olive oil remains cost-prohibitive.

Korean population data support this transition at the pattern level. Among 14,209 adults in the 2015–2017 Korea National Health and Nutrition Examination Survey (KNHANES), a higher rate of Korean food consumption was inversely associated with C-reactive protein in women. Participants in the highest tertile of flavonoid intake were less likely to have CRP above 3 mg/L (OR 0.59, 95% CI 0.42–0.83). Onion, radish, soybean-paste stew, cheonggukjang, and kimchi were among the leading identified flavonoid sources.⁴³ In a larger KNHANES analysis of 17,984 adults, the highest quartile of fermented food intake correlated with lower odds of elevated hs-CRP among adults aged 20–39 and 40–64 (OR 0.70, 95% CI 0.54–0.90 and OR 0.82, 0.69–0.97, respectively).⁴⁴ Both cross-sectional analyses demonstrate that the pattern-level signal reported for Mediterranean diets is detectable in a Korean population eating traditional food, although they cannot establish causal CRP reduction.

That signal often disappears when individual foods are tested experimentally. A four-week randomized trial comparing 100 g/day of kimchi with an equal weight of raw radish found that TNF- α and IL-6 declined in both arms. There was no significant difference between them.⁴⁵ An eight-week randomized, double-blind trial of cheonggukjang supplementation reported no change in hs-CRP or IL-6.⁴⁶ A meta-analysis of 25 randomized trials evaluating alpha-linolenic acid found no significant pooled effect on CRP, IL-6, or TNF- α .⁴⁷ This fatty acid makes perilla oil an attractive olive oil substitute, yet isolated supplementation shows null results. The fermented-food arm of Wastyk et al.⁴⁰ is often cited to support kimchi and doenjang, yet the prespecified primary outcome of that trial was null. Their fermented-food intervention contained substantial dairy rather than traditional Korean ferments, failing to establish an effect specific to Korean food. The case for Korean adaptation rests entirely on the comprehensive dietary pattern rather than on any single food component. Substituting perilla oil for olive oil represents a plausible nutrient-level swap rather than a demonstrated anti-inflammatory intervention.

The critical qualification concerns older adults specifically. In the same national survey, the inverse association between fermented-food intake and hs-CRP was completely absent among adults aged 65 and over.⁴⁴ Sodium intake significantly modified the relationship in this age group, applying to both total dietary sodium and sodium contributed directly by fermented foods. The favorable association was evident at lower sodium intakes but attenuated as

sodium rose. Because high sodium intake acts as a counter-vailing factor, it must be managed using low-salt preparation techniques. Seasoning *namul* with perilla powder and sesame rather than excessive soy sauce or salt offers a practical solution. This trade-off appears specifically in the target demographic of this review, providing a firmer reason to monitor sodium than general cardiovascular arguments usually supply.

Sodium is not the only compositional caveat. Biogenic amines accumulate during protein-rich fermentation. Nine out of the 36 artisanal *doenjang* samples exceeded 500 mg/kg of histamine, and roughly a third exceeded the 1,000 mg/kg total amine guideline.⁴⁸ *Gochujang* samples consistently fell well below every reference threshold.⁴⁹ These compounds are heat-stable and resist removal during ordinary cooking.⁵⁰ While exposure at condiment-level intakes is not a practical concern for the general population, the European Food Safety Authority places the no-adverse-effect tyramine dose at 600 mg per meal for healthy individuals and at just 6 mg per meal for people taking classical monoamine oxidase inhibitors.⁵¹ Age itself does not raise susceptibility. This specific caution applies only to the subset of older adults taking such medication. Consequently, dietary guidance should name specific fermented foods rather than broadly endorsing the entire category.

Korean feeding trials measuring inflammatory biomarkers exist, but they are small, short, and largely null. A two-week randomized controlled feeding study tested a traditional Korean diet with a low dietary inflammatory index in ten women aged 50–60. This intervention raised anti-inflammatory IL-10 and lowered NF- κ B while leaving CRP, IL-6, and TNF- α unchanged.³⁹ A four-week randomized trial of antioxidant-rich prepared meals in 31 Korean adults with a mean age near 71 improved oxidative and vascular measures. It produced no significant change in hs-CRP or TNF- α .⁴¹ No accurately powered randomized trial has tested a Korean-adapted anti-inflammatory diet in adults aged 65 or older using circulating inflammatory markers as a prespecified primary endpoint.

The underlying evidence base is fundamentally younger than the population it seeks to treat. Esposito et al.³¹ studied adults with a mean age near 44, and Wastyk et al.⁴⁰ studied a cohort with a mean age of 51. Only Estruch et al.³² and Ghosh et al.³⁸ studied populations overlapping substantially with older adults. No trial reviewed here enrolled adults over 80. A synthesis restricted explicitly to older adults confirms this gap.³⁶ It identified only 13 acceptable-quality studies, including just three randomized trials. Their pooled inverse association between Mediterranean-diet adherence and CRP derived entirely from five cross-sectional studies rather than from randomized comparisons (SMD -0.26 , 95% CI -0.41 to -0.11 , $P < .001$). The NU-AGE trial stands as the most relevant experimental evidence. It shows that a Mediterranean diet adapted for older adults can shift the gut microbiome in ways that track with lower hs-CRP and reduced frailty. These relationships, however, remain associative. Because the participants were non-frail or pre-frail community dwellers, the trial does not establish that the diet actively lowers inflammation in the frailest older adults.

Senescent cell burden, mitochondrial damage, and barrier deterioration are significantly further advanced at 80 than at 44. A dietary change may act on a much smaller share of the total inflammatory input in advanced age. Whether the effect shrinks, holds, or grows with age remains an open empirical question. These limitations indicate that dietary management represents one modifiable lever alongside physical activity and medical care, rather than a standalone solution for reversing biological aging.

Recommendations for Future Work

Future research should prioritize dietary interventions that target elderly populations through extended follow-up periods and simultaneous measurement of multiple inflammatory biomarkers. Despite adults aged 80 and older carrying the highest burden of inflammaging-related disease, very few intervention studies focus specifically on this demographic. Closing this gap is essential to determine whether anti-inflammatory benefits observed in midlife generalize to the oldest populations. Methodologically, trials need to prespecify primary inflammatory outcomes rather than indiscriminately reporting large biomarker panels. Researchers must also account for intervention intensity, adherence, and concurrent weight change, as variations in counseling and weight loss confound several major trials reviewed here.

Beyond biomarker shifts, studies should investigate whether dietary modifications translate into measurable improvements in frailty, cognitive function, and survival. Culturally adapted dietary patterns also require direct, rigorous evaluation. Any trial assessing a Korean-adapted anti-inflammatory diet against a control must strictly monitor sodium intake, protein adequacy, cost, and texture. This framework establishes a much stronger basis for clinical guidelines than extrapolating from Mediterranean or fermented-food trials conducted in fundamentally different contexts. Existing Korean feeding trials demonstrate the need for these methodological upgrades. Future studies require adequate statistical power for prespecified endpoints, durations exceeding the current two- to four-week standard, and enrollment explicitly restricted to adults aged 65 and older.

Conclusions

This review supports the view that inflammaging functions as a shared upstream condition underlying many major age-related diseases. Cellular senescence, mitochondrial dysfunction, and compromised immune regulation drive this chronic state. Biomarkers such as hs-CRP, IL-6, and TNF- α serve as measurable indicators of these inflammatory shifts. Current evidence indicates that dietary composition actively influences these markers. Pro-inflammatory diets high in saturated fats, trans fats, and refined carbohydrates tend to elevate them. Conversely, diets rich in omega-3 fatty acids, fiber, and plant-based antioxidants correlate with reduced inflammatory tone.

The Mediterranean diet integrates these protective components into a practical nutritional pattern. Clinical data support this broad approach, although actual physiological effects remain highly specific. For example, while a two-year randomized intervention led by Esposito et al.³¹ demonstrated significant reductions in hs-CRP and IL-6, the latest comprehensive meta-analysis clarifies the limits of this dietary impact.³⁵ The diet appears to lower targeted markers rather than uniformly suppressing the entire inflammatory panel.

While dietary control can modify baseline inflammatory levels, lowering a surrogate biomarker does not automatically equate to decelerated biological aging. Confirming genuine shifts in long-term disease progression requires extensive follow-up data. Despite this clinical limitation, adjusting community meals and senior care nutrition toward anti-inflammatory principles offers a practical strategy for aging populations. Formulating evidence-based clinical recommendations ultimately requires dedicated intervention research explicitly focused on adults aged 80 and older over extended durations.

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Declaration on the Use of Artificial Intelligence

In preparing this manuscript, the author used an AI assistant as a supplementary tool to refine language for clarity, assist in verifying reported numerical values, and format citations. All scientific content, study selection, data extraction, literature synthesis, interpretations, and conclusions were conceived and executed independently by the author, who assumes full and complete responsibility for the final content of this article.

Supplementary Materials

None.

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