

Review

Red Meat Consumption and Rheumatic Diseases: A Comprehensive Narrative Review

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Abstract

Dietary factors influence chronic inflammation and immune regulation, potentially affecting the onset and activity of rheumatic disease. Red meat, a major source of protein, heme iron, and saturated fat, has been linked to metabolic and inflammatory disorders, yet its role in rheumatic diseases remains unclear. To comprehensively evaluate the relationship between red-meat consumption and major rheumatic diseases—including rheumatoid arthritis (RA), gout, systemic lupus erythematosus (SLE), and other inflammatory or degenerative conditions—integrating data from systematic reviews, meta-analyses, cohort studies, and clinical trials. A literature search was conducted in PubMed, Embase, and Web of Science through April 2025. Eligible studies included randomized trials, observational cohorts, and meta-analyses assessing red-meat intake in relation to rheumatic outcomes. Data were synthesized narratively due to heterogeneity. Whenever available, we considered whether studies between distinguished processed and unprocessed red meat and key confounders (e.g., smoking and adiposity). Across eight high-quality studies ($n \approx 5300$ participants) and three meta-analyses, evidence for RA was neutral: large cohorts and feeding trials found no association between red-meat intake and disease incidence or activity. However, high consumption (≥ 100 g/day) was linked to 6-year earlier RA onset in one cohort, particularly among smokers or individuals with higher body mass index. For gout and hyperuricemia,



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pooled analyses confirmed increased risk with high meat intake (odds ratio 1.27-1.32). In contrast, intake of fresh red meat was inversely associated with hip osteoarthritis requiring arthroplasty (hazard ratio = 0.94 per weekly serving). Data for SLE, spondyloarthritis, and psoriatic arthritis were limited or indirect through Mediterranean-type diet adherence. Red meat exerts disease-specific effects in rheumatology. Restriction is strongly warranted in gout and hyperuricemia, while moderate intake of unprocessed meat appears unlikely to meaningfully increase RA risk or short-term inflammatory activity when embedded in anti-inflammatory dietary patterns. For SLE and other rheumatic diseases, evidence isolating red-meat exposure remains limited and heterogeneous, and conclusions should be interpreted cautiously—particularly regarding processed meat and cooking-related pro-inflammatory exposures. Further longitudinal and interventional research is needed to clarify dose thresholds and mechanistic pathways.

Keywords

Red meat consumption; rheumatoid arthritis; gout; systemic lupus erythematosus; inflammation; diet and rheumatic diseases

1. Introduction

Dietary exposures can modulate low-grade systemic inflammation, oxidative stress, and immune function, all of which are central to the pathophysiology of rheumatic diseases [1]. Red meat has attracted particular attention because it concentrates nutrients and bioactive compounds with potentially divergent effects: high-quality protein, heme iron, zinc, and B vitamins, alongside saturated fat, cholesterol, purines, and advanced glycation end products (AGEs) [2]. These constituents may influence inflammatory signaling and innate immune activation (for example, via Toll-like receptor pathways), as well as redox balance [3].

Epidemiological studies linking red meat to rheumatic conditions have produced mixed results. Early case-control and cohort observations entertained a potential adverse association with inflammatory polyarthritis, while later prospective cohorts often reported null findings for incident RA. In contrast, biochemical and epidemiological evidence consistently implicates purine-rich meats in the risk of gout. For SLE and other autoimmune rheumatic diseases, existing research has largely assessed dietary patterns rather than individual food exposures, complicating attribution to red meat alone.

Mechanistic lines of evidence illustrate possible pathways. Heme iron and lipid peroxidation products can amplify oxidative stress. AGEs may prime innate immune responses, and meat processing can introduce nitrosamines and sodium, which impact vascular and inflammatory tone [2, 3]. Conversely, lean unprocessed red meat can deliver essential amino acids and micronutrients that support musculoskeletal integrity. The net effect likely depends on meat type (fresh versus processed), portion size, and the surrounding dietary pattern. Alterations in gut microbiota composition may also mediate inflammatory responses to meat-derived compounds.

Given substantial patient interest in nutrition, the absence of disease-specific guidance, and the heterogeneity of published results, we undertook a comprehensive narrative synthesis of red-meat

consumption across rheumatic diseases. We summarize the best available evidence for RA, gout/hyperuricemia, SLE, and other rheumatic conditions, highlight consistencies and discrepancies, and delineate priorities for future research and clinical practice.

2. Methods

We conducted a structured narrative review consistent with best practices for narrative syntheses in biomedical journals [4]. PubMed, Embase, and Web of Science were searched from 2000 to April 2025 using the terms: (“red meat” OR “beef” OR “pork” OR “processed meat”) AND (“rheumatoid arthritis” OR “gout” OR “hyperuricemia” OR “systemic lupus erythematosus” OR “psoriatic arthritis” OR “ankylosing spondylitis” OR “osteoarthritis” OR “vasculitis” OR “scleroderma” OR “systemic sclerosis” OR “myositis” OR “Sjögren”). Reference lists of eligible articles and pertinent reviews were hand-searched to identify additional studies. Screening was performed in two stages (title/abstract screening followed by full-text assessment). We included studies published in English.

Eligibility criteria included systematic reviews/meta-analyses, randomized controlled trials, prospective and retrospective cohorts, and case-control studies in humans that quantified red-meat exposure (fresh and/or processed) and reported at least one rheumatology-relevant outcome: disease incidence, clinical activity indices (e.g., DAS28, SLEDAI, BASDAI), flares, joint replacement for osteoarthritis (OA), and mechanistic biomarkers (e.g., C-reactive protein [CRP], interleukin-6 [IL-6], serum urate). We excluded animal studies, ecological analyses, non-original reports, and interventions where red meat was inseparable from a composite dietary pattern. For clarity, “composite dietary patterns” refers to dietary indices or multi-component dietary interventions in which red meat intake was not evaluated as an independent exposure (e.g., Mediterranean diet score, Western dietary patterns, dietary inflammatory indices, or overall diet-quality scores), preventing isolation of red meat-specific associations.

Two reviewers independently extracted study design, population, sample size, exposure definition, comparator, outcomes, effect size, and major findings. Given heterogeneous designs and endpoints, we did not attempt a meta-analysis. Instead, we prioritized high-level syntheses and large cohorts, then contextualized with smaller or mechanistic studies. Risk of bias was assessed qualitatively, considering the dietary assessment method, confounder adjustment, and representativeness, aligned with general appraisal guidance [4].

3. Results

3.1 Rheumatoid Arthritis

A prospective study reported no association between red meat consumption and incident RA, alongside a modest protective signal for fish [5]. Indeed, the large Nurses’ Health cohort corroborated null findings for total protein, iron, and red meat in relation to incident RA [5]. Conversely, the EPIC-Norfolk nested case-control analysis observed that higher intake of red and processed meats was associated with increased risk of inflammatory polyarthritis (odds ratio roughly twofold) [6]. Cross-sectional data from China suggested that daily intake of 100 g or more of red meat was linked to earlier onset of RA (about six years), particularly among smokers or individuals with higher body mass index [7]. Notably, smoking and elevated BMI are established independent risk factors for RA and may act as confounders and/or effect modifiers; therefore,

these subgroup findings should be interpreted cautiously, particularly in cross-sectional designs, and in light of multivariable adjustment where reported [7]. Importantly, randomized crossover meal studies in women with RA found no short-term inflammatory penalty after red-meat meals compared with fish or soy meals—postprandial IL-6 and triglyceride responses were similar across meal types [8, 9]. These short-term mechanistic findings inform postprandial inflammatory responses but do not exclude potential long-term effects of habitual dietary patterns on RA development.

Overall, discrepancies across studies may be explained by differences in study design (cross-sectional vs. prospective cohorts vs. nested case-control), outcome definitions (incident RA vs. inflammatory polyarthritis or earlier onset), exposure characterization (total vs. processed vs. unprocessed red meat), and the extent of adjustment for major confounders such as smoking, adiposity, and overall dietary patterns. In aggregate, current evidence for RA remains mixed at the study level but leans toward neutrality in higher-level syntheses and large cohorts.

3.2 Gout and Hyperuricemia

Two recent meta-analyses consistently associated higher red-meat intake with greater risk of gout and hyperuricemia, with pooled odds ratios in the 1.27-1.32 range [10, 11]. Earlier comprehensive reviews that included diet patterns similarly highlighted meat as a contributor to gout risk [12]. Together, these results support guideline-consistent advice to reduce intake of red and processed meats in the management of hyperuricemia and gout.

3.3 Systemic Lupus Erythematosus

No systematic reviews or clinical trials have been identified to date regarding the relationship between red meat intake and disease activity or flare outcomes in SLE. Evidence is primarily indirect via Mediterranean-style or anti-inflammatory dietary patterns that tend to limit red/processed meat, with reported improvements in inflammatory markers or symptom domains in autoimmune cohorts [12, 13]. Because pattern-level effects confound single-food attribution, SLE-specific conclusions regarding red meat remain tentative and underscore research gaps.

A summary of the main findings from systematic reviews and meta-analyses is presented in Table 1.

Table 1 Systematic reviews and meta-analyses on red-meat consumption and major rheumatic diseases.

Disease	Author, Year [Ref]	Study type	Main findings
Rheumatoid arthritis	Benito-Garcia E et al., 2007 [5]	Prospective cohort	No association between red-meat intake and incident RA.
Rheumatoid arthritis	Pattison DJ et al., 2004 [6]	Prospective cohort	High red-meat consumption is associated with increased risk of inflammatory polyarthritis (OR $\approx$ 2.3 for the highest quartile).
Rheumatoid arthritis	Jin J et al., 2021 [7]	Cross-sectional	Higher meat intake is linked to earlier RA onset by $\sim$ 6 years.
Rheumatoid arthritis	Hulander E et al., 2024 [8]	Randomized cross-over trial	No postprandial inflammatory difference vs fish/soy meals.
Rheumatoid arthritis	Lindqvist HM et al., 2024 [9]	Metabolomic study	Minimal changes in serum metabolites after red-meat meal.
Gout/Hyperuricemia	Chi X et al., 2024 [10]	Systematic review + meta-analysis	High meat intake increases the risk of gout/hyperuricemia (OR 1.27-1.32).
Gout/Hyperuricemia	Lee S et al., 2024 [11]	Cohort (HEXA Study, Korea)	Red and processed meat are associated with a higher incidence of hyperuricemia (HR 1.35 per SD increase).
Gout/Hyperuricemia	Li R et al., 2018 [12]	Meta-analysis	Red-meat intake linked to gout (OR 1.29 [1.16-1.44]) and hyperuricemia (OR 1.24 [1.04-1.48]).
Psoriatic arthritis/Psoriasis	Ford AR et al., 2018 [14]	Systematic review	Low-energy, anti-inflammatory diet beneficial; meat restriction part of guidelines.
Ankylosing spondylitis	Macfarlane TV et al., 2018 [15]	Systematic review	No consistent evidence; some reports suggest a benefit from lower meat intake.

CID: chronic inflammatory diseases, HR: hazard ratio, OR: odds ratio, RA: rheumatoid arthritis, SLE: systemic lupus erythematosus.

3.4 Other Rheumatic Conditions

Dietary research in ankylosing spondylitis (AS) and psoriatic arthritis (PsA) is limited and heterogeneous. A systematic review of diet in AS conducted according to the PRISMA framework concluded that no specific dietary component, including red meat, consistently affected risk or activity [15]. In PsA and psoriasis, recommendations from the National Psoriasis Foundation emphasize weight loss in overweight/obese patients; the effects of red meat were not isolated in higher-level syntheses, though many proposed anti-inflammatory patterns advise minimizing processed meats [14]. For OA, a prospective Australian cohort reported an inverse association between fresh red-meat intake and risk of hip arthroplasty for OA (hazard ratio 0.94 per additional weekly serving), with no association for knee arthroplasty [16]. Finally, in a large Danish cohort examining multiple chronic inflammatory diseases (RA, PsA, psoriasis, IBD, MS), red-meat consumption was not associated with incident disease overall [17]. These findings suggest that, beyond gout, disease-specific links to red meat are sparse or inconsistent.

Additional observational and cohort data on other rheumatic and inflammatory conditions are provided in Table 2.

Table 2 Other rheumatic and inflammatory conditions with observational data on red-meat intake.

Disease	Author, Year [Ref]	Study type	Main findings
Osteoarthritis	Wang Y et al., 2011 [16]	Prospective cohort	Moderate fresh-meat consumption is inversely associated with the risk of hip/knee arthroplasty (HR 0.94 per serving/week).
Chronic Inflammatory Diseases (cohort)	Rubin KH et al., 2020 [17]	Danish prospective cohort	High intake of red and processed meat is associated with increased risk of late-onset CID.
Gout in men	Choi HK et al., 2004 [13]	Prospective cohort (Nurses Health/HPFS)	Meat intake (>2 servings/day) ↑ risk of gout (RR 2.1 vs lowest quintile).
Autoimmunity/metabolism	Tilg H & Moschen AR, 2014 [18]	Narrative review	Gut microbiota and dietary fats modulate immune activation; meat is a pro-inflammatory factor.
Cardiometabolic risk/ rheumatic inflammation	Micha R et al., 2012 [19]	Review	Processed meat is associated with CVD and type 2 diabetes - conditions frequently coexisting with rheumatic diseases.
Meat composition and immune effects	Scollan ND et al., 2014 [20]	Review	Nutritional modification of beef lipids may reduce oxidative and inflammatory load in rheumatic patients.

CVD: cardiovascular disease, HR: hazard ratio, OA: osteoarthritis, OR: odds ratio, PsA: psoriatic arthritis, RR: relative risk, T2D: type 2 diabetes.

4. Discussion

This synthesis indicates that the relationship between red-meat intake and rheumatic diseases is disease-specific and context-dependent. For RA, large prospective cohorts and a recent meta-analysis converge on a neutral association, whereas smaller observational studies sometimes report associations with earlier onset or inflammatory polyarthritis [5-8, 14]. These discrepancies likely reflect differences in study design (cross-sectional vs. prospective), outcome definitions (incident RA vs. inflammatory polyarthritis or age at onset), exposure assessment (total vs. processed vs. unprocessed red meat), follow-up duration, and the extent of adjustment for key confounders such as smoking and adiposity. Controlled meal trials find no acute inflammatory worsening after red-meat meals compared with fish or soy meals [8, 9]. Importantly, these controlled postprandial studies address short-term biochemical inflammatory responses and cannot be directly extrapolated to long-term disease initiation, which may evolve over years or decades. These results collectively suggest that moderate intake of unprocessed red meat within balanced diets is unlikely to meaningfully elevate RA risk or short-term inflammatory activity, whereas potential risks—if present—may be more relevant to high intakes, processed meat exposure, and susceptible subgroups with higher baseline inflammatory or cardiometabolic burden.

The picture for gout and hyperuricemia is markedly different. Meta-analytic evidence consistently identifies higher red-meat consumption as a risk factor, reinforcing decades of clinical and epidemiological observations that purine-rich meats elevate serum urate and precipitate gout flares [10-12, 17]. The signal is reproducible across populations, clinically relevant in magnitude, and biologically plausible through purine load, xanthine oxidase activity, and potential effects of processed meat constituents on renal urate handling. Accordingly, dietary counseling for gout should explicitly prioritize restriction of purine-dense meats and processed meat products, particularly in patients with recurrent flares or persistently elevated serum urate.

For SLE, disease-specific evidence isolating red meat remains insufficient. Improvements observed with Mediterranean-type diets likely reflect synergistic nutrient effects (elevated omega-3 fatty acids, fiber, and antioxidants) rather than meat exclusion alone [12, 13, 19]. Therefore, statements regarding red meat and SLE should remain cautious and framed by the current absence of systematic reviews or clinical trials specifically addressing red-meat intake and disease activity in SLE. Nevertheless, given the elevated cardiometabolic risk in SLE, minimizing processed and saturated-fat-rich meats is reasonable within a pattern-first dietary approach, while maintaining adequate protein intake through healthier sources (e.g., fish, legumes, and lean unprocessed meats when appropriate).

AS and PsA literature does not reveal a consistent association with red meat. Systematic reviews emphasize evidence gaps, methodological heterogeneity, and small sample sizes [14, 15]. In OA, the inverse association between fresh red meat intake and hip arthroplasty risk is intriguing yet counterintuitive and requires replication [16]. If true, benefits might arise from higher protein intake, supporting muscle mass or influencing bone geometry, rather than from anti-inflammatory mechanisms. This finding also highlights the possibility that unprocessed red meat intake may act as a proxy for overall dietary adequacy, physical robustness, or socioeconomic factors—issues that are difficult to fully disentangle in observational research.

Mechanisms linking red meat to inflammation include purine-driven urate formation (relevant to gout), heme-iron-mediated oxidative stress, and AGEs-related innate immune activation [2, 3, 9].

Saturated fats in some red-meat cuts can engage Toll-like receptor signaling cascades [3]. Processed meats contribute nitrosamines and sodium, which may impair endothelial function and promote inflammation [19]. Moreover, high-temperature cooking methods (e.g., frying or grilling) may increase exposure to AGEs and other pro-inflammatory compounds, potentially modifying associations in studies that do not capture preparation practices. Conversely, lean unprocessed red meat provides essential amino acids and micronutrients important for musculoskeletal health, and husbandry or feeding practices can modulate lipid profiles of beef in ways that affect healthfulness [20]. The overall clinical impact thus depends upon meat type (unprocessed versus processed), portion size, preparation, and the broader diet (e.g., fish, fiber, whole grains). Taken together, processed meat consumption represents a more plausible target for risk reduction across rheumatic and cardiometabolic outcomes, whereas unprocessed lean red meat may be compatible with healthful dietary patterns when consumed in moderation.

Limitations of current evidence include reliance on self-reported dietary data (susceptible to recall and measurement error), inconsistent differentiation between fresh and processed meats, and residual confounding by smoking, adiposity, and socioeconomic factors [4]. Many studies are based on Western populations; generalizability to other regions remains uncertain. Interventional trials remain rare and often focus on short-term postprandial responses rather than long-term clinical endpoints. Publication bias and selective reporting cannot be excluded. Future research should prioritize prospective cohorts with repeated dietary measurements, explicit separation of processed versus unprocessed red meat, capture of cooking methods, and stratified analyses by smoking, BMI, and cardiometabolic risk. Long-term randomized dietary interventions in high-risk rheumatic populations may further clarify causality and inform evidence-based dietary guidance.

4.1 Clinical Implications

Dietary counseling in rheumatology should be pattern-oriented. For gout and hyperuricemia, reducing red and especially processed meat is strongly supported and aligns with urate-lowering strategies [10-12, 17]. For RA, SLE, PsA, and AS, moderate consumption of lean, unprocessed red meat within a Mediterranean-like dietary pattern focused on vegetables, whole grains, legumes, nuts, and fish appears compatible with disease control and comorbidity prevention. Weight management and cardiometabolic risk mitigation remain central across conditions. Clinicians should focus on limiting processed meats and saturated-fat-rich cuts while allowing moderate lean red-meat intake within balanced, anti-inflammatory dietary patterns.

5. Conclusion

Red meat consumption exhibits disease-specific associations in rheumatology. For gout and hyperuricemia, consistent epidemiologic and mechanistic evidence supports dietary restriction of red and processed meats. For RA, high-level syntheses and large cohorts indicate neutral associations, particularly for moderate intake of unprocessed red meat within balanced dietary patterns; however, processed meats and high-temperature preparation practices may represent more plausible sources of pro-inflammatory exposure and should be considered in clinical counseling. For other rheumatic diseases (including SLE, PsA, AS, and OA), evidence on red meat intake remains limited or heterogeneous, precluding definitive conclusions and reinforcing the need for better-designed prospective studies. Overall, dietary guidance should be prudent and stratified,

explicitly distinguishing processed versus unprocessed red meat, considering cooking methods, and accounting for individual susceptibility and cardiometabolic risk.

Author Contributions

J.F. Carvalho: conceptualization, literature search, formal analysis, writing, edition, submission.
R.P. Jesus: literature search, formal analysis, writing, and revision.

Competing Interests

The authors have declared that no competing interests exist.

Data Availability Statement

No additional data was generated to this study. All data is already included in the present format.

AI-Assisted Technologies Statement

AI tools were utilized exclusively to assist in language editing and improving the clarity of sentences in the manuscript. All ideas, data synthesis, and conclusions presented in this study are entirely the responsibility of the authors.

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