Review

Is the controversial role of alcohol in arthritis still a Hamletic dilemma?

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ABSTRACT

While alcohol consumption is widely considered detrimental in rheumatic diseases, literature suggests its effects might vary depending on dose, beverage type and specific disease context.

This narrative review examines current evidence studying the relationship between alcohol intake and disease activity in inflammatory arthropathies, such as rheumatoid arthritis (RA), gout and spondyloarthritis (SpA).

Specifically, we aim to evaluate the potential beneficial or harmful effects of alcohol on different inflammatory joint diseases, accounting for variations in beverage type and consumption volume. As a secondary objective, we investigate the impact of alcohol on intestinal homeostasis within these rheumatic conditions.

Observational studies in RA suggest that low-to-moderate alcohol consumption may be associated with a reduced risk of disease onset and lower disease activity; however, causality remains uncertain due to confounding factors. Conversely, alcohol intake in gout and SpA consistently shows dose-dependent detrimental effects that accelerate disease progression. Nevertheless, pivotal studies indicate that certain non-ethanol compounds found in red wine, such as polyphenols and resveratrol (RS), can reduce inflammation. While chronic alcohol consumption disrupts intestinal homeostasis by promoting gut dysbiosis and compromising barrier integrity, potentially fuelling systemic inflammation, RS may exert microbiota-modulating and anti-inflammatory effects in experimental models. Overall, current evidence does not support alcohol consumption as a therapeutic strategy in rheumatic diseases. Its effects must be interpreted with caution, balancing disease-specific risks against dose-dependent biological responses.

Introduction

In the past, alcohol was commonly considered an antagonist of rheumatic diseases, with known detriments in heavy drinking, recently defined by SAMHSA (1) as use as binge intake (five or more beverages in male, or four or more in female, in about two hours) on five or more days in the past month. Although several studies have correlated its consumption to the risk of developing arthritis (2), a moderate intake seemed to have differed and contrasting effects in terms of benefit/damage balance (3). We know from Teofrasto Paracelso (1493-1541) that "Everything that forms our nourishment corresponds to what we ourselves are: we eat therefore we eat ourselves. This is also true for medicine, with this only difference, that it adapts to the content of the illness".

While the detrimental effects of alcohol are well-established in gout, its role in other rheumatic diseases, such as rheumatoid arthritis (RA) or spondyloarthritis (SpA), remains more challenging to evaluate. The exact impact of various environmental factors, including lifestyle and diet on genetic predisposition (3, 4), is largely unidentified and limited by concomitant influence of multiple factors (5).

Furthermore , the emergence of the 'French paradox' concept (6), highlighting the antioxidant role of resveratrol (RS), a polyphenol found in red wine, and its bioactive compounds, challenged our previous assumptions by revealing its ability to modulate both inflammatory responses and intestinal permeability (7-9).

Fig. 1. Ampelographic images from Roberto Bandinelli's personal research data that extensively mapped Tuscan grape varieties and clones, particularly through regional initiatives like TOS.CO.VIT. and historical vineyard bio-diversity recovery projects.

As shown by Belli *et al*. (7), these effects appear to be dose-dependent and vary based on the RS and bioactive compound content across different clones (10-12) (Fig. 1).

Given the influence of RS on digestive processes, its potential impact on the gut microbiota (13-17) may be highly relevant to rheumatic diseases. Specifically, a disruption in the balance of *Streptococcus* or *Lactobacillus* relative to other commensal microbes has been shown to modulate inflammation (18).

The primary aim of this review is to evaluate the beneficial or harmful effects of alcohol on inflammatory processes in arthritis, based on current literature. We assessed its impact across various rheumatic diseases, accounting for variations related to alcohol type and intake levels. As a secondary objective, we investigated the potential impact of alcohol on intestinal homeostasis in rheumatic conditions.

Materials and methods

The literature included in this narrative review was identified through searches conducted in Google Scholar, Web of Science, Scopus and PubMed up to 30 December 2025, without temporal restrictions. The search strategy employed the following query: *[alcohol AND Rheumatoid Arthritis AND spondyloarthritis AND gout]*. The primary objective was the retrieval of original full-text studies investigating the potential benefits or risks of alcohol consumption in each of these diseases.

Given the heterogeneity of study designs, methodologies, and outcome measures across the available literature, a non-systematic approach was adopted. Consequently, the review protocol was not registered in PROSPERO or other dedicated databases, and no PRISMA flow diagram is provided. In addition to database searches, reference lists of relevant publications were also manually screened to identify further eligible studies.

Table I. Main characteristics of the most diffuse fermented, brewed fermented and distilled alcoholic beverages, in terms of chemical composition and primary health benefits.

Alcoholic beverages	Average alcohol content	Key bioactive components	Beneficial effects
Fermented:			
Wine (especially red)	Around 13%	Polyphenols, resveratrol (34, 35)	Increase microbial diversity (35) and short chain fatty acid (SCFA) production (36); prevent reactive oxygen species (ROS) and protect cells from DNA damage (37-38); lower risk of cardiovascular disease with low to moderate consumption (21).
Brewed and fermented:			
Beer	Around 5%	Polyphenols, hop-derived compounds, vitamins, fibre, minerals (39)	Reduce mortality and morbidity of cardiovascular diseases (41, 42); melatonin, minerals and polyphenols (xanthohumol) contents promote cell protection (43) and prevent chronic diseases (44). Increase in bone mineral density and decrease diabetes risk (only in men) (45).
Distilled:			
Spirits	Around 40%	Minimal to none (fatty acids, volatile phenols) (46, 47)	Reduce risk of heart disease (48).

Given the paucity of literature on RS effects on rheumatic diseases (7), we explicitly define to include also cellular and animal models as well studies on real-world translational outcomes on human population. To mitigate bias, we clearly detail these distinct sources of evidence.

Beverages containing more than 0.5% alcohol by volume were categorized by alcohol content ranging from 5% to 40%, according to SAMHSA (1), and were classified into three principal categories: beer, wine, and spirits. Whenever available, information regarding grape clonal variation and the precise quantity of each alcoholic beverage evaluated was also reported.

Results

Type and quantity of alcohol can make a difference

Recent studies show that alcohol consumption has remained overall stable over the last 20 years (19), despite regional differences, while alcohol-related problems have increased. Alcohol is a leading risk factor for premature death among individuals aged 15-49, accounting for about 10% of deaths (20). The relationship between alcohol and mortality follows a J-shaped curve. Light to moderate drinking may be associated with slightly lower mortality risk compared to abstinence, whereas heavy drinking significantly increases the risk.

Harmful alcohol use, especially among young people, represents a major global public health, social and economic concern, influenced by consumption patterns, beverage type, and lifestyle factors (21-23). Although some studies suggest that moderate drinking may improve quality of life (24-31) and reduce disease activity or risk (*e.g.*, rheumatoid arthritis) (32), defining "moderate consumption" remains challenging. Alcohol content varies widely across beverages and countries, making it difficult to establish a standard definition of a "drink." Moreover, the concept of "moderate" is subjective, leading to inconsistencies in research findings. Therefore, a clear and standardised definition of moderate drinking is essential to better assess its potential risks and benefits (33).

Table I compares the main characteristics of the most diffuse fermented, brewed fermented and distilled alcoholic beverages, in terms of chemical composition and possible primary health benefits (34-47), highlighting differences among beverages, although it cannot provide sure clinical recommendations. Instead, these findings demonstrate that alcohol-related effects are beverage-specific and should not be generalised. Evidence regarding the potential health benefits of distilled spirits remains limited, as spirits like gin and vodka contain few non-alcoholic bioactive compounds. Because current literature offers little support for specific health benefits beyond the effects of ethanol itself, further studies are required to clarify their impact (49). This perspective transitions into the following section, which explores the clinical implications of alcohol consumption in rheumatic diseases.

Alcohol in arthritis: benefit or damage?

The effects of alcohol consumption in RA, Gout, and SpA are summarised in Table II.

- Rheumatoid arthritis

Alcohol plays a paradoxical role in RA, as shown in Table II. Cohort studies and meta-analyses show that light-to-moderate intake (up to ~10g/day) correlates with a reduced RA risk, particularly in women and seropositive subtypes (32, 50-52). This inverse association may stem from alcohol's immunomodulatory effects, such as suppressing T-cell responses and modulating cytokines (53-55).

Conversely, high or sustained intake increases RA risk (56-61). However, observational data faces reverse-causation bias, as patients with severe disease often quit or reduce drinking (24). While current drinkers with established RA report lower disease activity and better quality of life, this association weakens after adjusting for comorbidities, disability, and socioeconomic status (58).

Table II. Effects of alcohol intake on rheumatic diseases and gut microbiota.

Disease	Potential Beneficial Associations *(Low–Moderate Intake)*	Harmful Associations *(Dose-Dependent/High Intake)*	Microbiota
RA	Reduced risk of incident RA (particularly seropositive RA and in women) at ~≤10 g/day (32, 50-52); Lower disease activity and improved health-related quality of life in current drinkers (attenuated after adjustment) (58)	Increased RA risk with sustained or high intake in some cohorts (32, 50-52); Hepatotoxicity and interaction with methotrexate (55): Increased comorbidity burden (55); High dose/chronic use may worsen systemic inflammation (54-55, 59-61)	Potential increase in beneficial taxa and short-chain fatty acid production (62); High intake disrupts gut barrier integrity and promotes dysbiosis (154-55, 59-61)
Gout	No protective role	Strong, dose-dependent increase in serum urate, higher risk of incident gout and increased flare frequency (65-66, 69); Beer and spirits possibly higher risk than wine (all types implicated) (69)	High alcohol intake promotes gut dysbiosis (↑ pro-inflammatory taxa, ↓ beneficial bacteria) with increased gut permeability and systemic inflammation (70-72)
SpA	Observational association with lower reported disease activity (BASDAI, ASDAS) and less spinal pain (77-80)	Greater radiographic progression (mSASSS and syndesmophytes) (77, 81); Upregulation of pro-inflammatory cytokines (TNF, IL-17, IL-23) (83-84)	Alcohol-induced immune dysregulation (innate and adaptive) leading to gut microbiota alterations (55-85)

ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; HRQoL: health-related quality of life; mSASSS: modified Stoke Ankylosing Spondylitis Spine Score.

Consequently, no firm evidence supports recommending alcohol for disease modification. Clinicians must weigh potential benefits against risks like hepatotoxicity, comorbidities, and drug interactions, especially with methotrexate (55).

Alcohol modulates the gut microbiome in a dose-dependent manner. Low-to-moderate intake may increase beneficial microbial populations and anti-inflammatory metabolites like short-chain fatty acids (62). Conversely, high or chronic intake disrupts the gut barrier, causes dysbiosis, and fuels systemic inflammation, worsening disease activity in susceptible patients (54-55, 59-61).

Current data shows no significant risk differences between beer, red wine, or spirits (50). Moderate consumption of any type generally correlates with reduced RA risk and, occasionally, better functional status (2, 50-52, 55, 62).

While RS and other red wine polyphenols demonstrate significant anti-inflammatory and antioxidant properties in RA animal models (63-64), human clinical trials (9) remain limited and are often conflicting. Further studies are necessary to confirm clinical efficacy or support any specific dietary guidelines, because a major limitation in translating animal model successes to human treatment is poor bioavailability and rapid metabolism (7).

- Gout

Unlike in RA, alcohol consumption seems uniformly to worsen gout. Epidemiological and genetic studies confirm that alcohol intake increases serum urate levels, gout incidence, acute flare frequency, and tophi progression (65-69). This dose-dependent risk applies to all alcohol types, even in moderate amounts, though beer and spirits carry higher risks than wine (65-69). Consequently, the American College of Rheumatology explicitly recommends limiting or abstaining from alcohol to lower urate levels and prevent flares (65).

In fact, alcohol's impact on the gut microbiota represents a key difference between the two diseases.

While in RA, low-to-moderate intake may have neutral or modestly beneficial immunomodulatory effects on the microbiome (70-75), conversely in gout, alcohol consistently disrupts the gut microbiota. High intake exacerbates dysbiosis by reducing beneficial taxa and increasing pro-inflammatory bacteria, which triggers gut permeability, fuels systemic inflammation, and alters uric acid metabolism to aggravate flares (70-72).

Furthermore, certain patient subgroups are more susceptible to alcohol-induced dysbiosis and disease progression: patients with pre-existing low microbial diversity or established hyperuricemia (70, 72), old subjects with age-related declines in gut barrier integrity and immune regulation (60), individuals with the impaired acetaldehyde clearance due to *ALDH2*2* polymorphism (60), male more sensitive than females to alcohol-induced microbiota shifts (such as Gram-negative/positive bacterial changes) and TLR4-mediated inflammatory responses (76).

- Spondyloarthritis

Alcohol presents a clinical paradox in axial SpA, as shown in Table II. Observational data and meta-analyses link consumption to lower reported disease activity (BASDAI, ASDAS) and less spinal pain, though sex and smoking may confound these modest findings (77-80). Conversely, longitudinal cohorts reveal that alcohol predicts greater radiographic spinal damage, accelerating m-SASSS changes and syndesmophytes formation (77, 81). No clinical guidelines recommend alcohol for SpA modification due to these structural risks and associated comorbidities (55, 83, 89).

Chronic alcohol use accelerates axial calcifications or heterotopic ossifications inside spinal ligament and annulus fibrosus, through interconnected pathways (81):

- Cellular stress triggers mitochondrial dysfunction and reactive oxygen spe-

Table III. Effects of resveratrol (RS) on main clinical trials (modified New Castle Ottawa quality scored) in animal and human models of arthritis.

RA, SpA, gout	Authors	Research on human or animal models	Main results of RS	m-NOS levels (0-9)
RA	Wahba *et al*. (2016)	RA induced rats received a 7-day treatment with either RS (10 mg/kg/day} or fenofibrate	Restored a smooth cartilage surface; enhanced modulatory (IL10 and GSH) and reduced inflammatory (rheumatoid factor, MMP-3, COMP, IgG, ANA, TNF-α, MPO, CRP, and MDA) biomarkers	7 (good-high)
RA	Yang *et al*. (2018)	Rat induced arthritis (*in vivo*) and IL-1 beta stimulated rat synovial cells were treated with trans-RV (200 or 400 mg/kg) for 8 weeks.	Markedly reduced synovial tissue inflammation and angiogenesis; lowered oxidative and inflammatory markers: reactive oxygen species (ROS), hypoxia-inducible factor-1α, p38 mitogen-activated protein kinase and c-Jun N-terminal kinase	7 (good-high)
RA and SpA	Lomholt *et al*. (2018)	Synovial fluid cells from 7 RA and 7 SpA patients analysed in a human case-control model at 48 hours and 21 days. Treatments consisted of resveratrol (RSV) monotherapy compared against combination therapies with MTX or adalimumab.	Significantly decreased monocyte chemo-attractant protein-1 (MCP-1) levels primarily at the 48-hour culture mark, influenced by patient profiles (low activity and high lymphocyte count) and treatment combinations (Methotrexate)	5 (fair)
RA	Wang *et al*. (2020)	RA induced rats were treated with intragastric 10 mg/Kg RS, for 24 days	Decreased synovial swelling and cartilage degradation; inhibited ROS and fibroblast-like synoviocytes proliferation by regulation of sirtuin 1 (SIRT1)/ nuclear-factor kappa-B (NF-κB)/miR-29a-3p/Keap1 and SIRT1/ NF-κB/miR-23a-3p/cul3 signalling pathways	7 (good-high)
RA	Fernández-Rodríguez *et al*. (2021)	Rats treated with 12,5 mg/Kg RS gavage, for 2 months, before inducing arthritis	Reduced chronic inflammatory (IL-1β, C-reactive protein, NFkb and PGE2) and angiogenesis (VEGF, Angiotensin 1) markers, after 8 weeks	7 (good-high)
Gout	Wang *et al*. (2023)	Gout induced rats *in vivo* and *in vitro* treated with RS in various concentrations	Reduced swelling and inflammatory synovial cell infiltration; reduce IL1beta secretion, inhibited NLRP3 inflammasome and Hypoxia-inducible factor 1-alpha (HIF-1) expression macrophages	7 (good-high)

cies production, causing tissue injury and aberrant bone remodelling (82).

- Immune dysregulation enhances pro-inflammatory cytokines (TNF, IL-17, IL-23), disrupts T-cell balance, amplifies neutrophil activation, and alters the gut microbiome, driving entheseal inflammation and osteo-proliferation (55, 83-85).
- Bone turnover stimulates osteoclast activity while simultaneously driving mesenchymal stem cell differentiation into osteoblasts, promoting ankylosis (83, 86).

Current evidence evaluates overall intake rather than beverage-specific effects, showing reduced peripheral manifestations but worse spinal progression regardless of whether patients drink beer, wine, or spirits (77-81). While general populations show distinct harm from binge drinking or specific beverage patterns, direct SpA data are lacking (78, 87).

Animal models suggest non-ethanol compounds like resveratrol can attenuate SpA severity by suppressing TLR4/NF-κB/NLRP3 signalling and restoring the gut barrier (7). However, human data on beverage-specific components (*e.g.*, polyphenols, purines) remain insufficient (87-88). Ultimately, literature lacks interventional studies, randomised controlled trials on alcohol abstinence, and clear definitions of chronic use in SpA populations (77).

The role of alcohol on pathogenic microbiome signatures in arthritis and RS treatment perspectives

Intestinal dysbiosis is highly implicated in the onset and progression of RA, SpA, and gout (70-72, 96-104).

In RA, periodontal infection by *Porphyromonas gingivalis* (a Gram-negative anaerobe) acts as a potential trigger and driver of disease progression (102).

In SpA, driven by a "gut-synovial axis" pathogenesis, patients exhibit a distinct microbial signature that correlates with fecal calprotectin levels and subclinical inflammation (54, 59-61, 96, 97). Pathologically, *Enterococcus faecalis* increases, contrasting with beneficial *Lactobacillus* species (*plantarum, rhamnosus, acidophilus*) found in mild or remissive disease (7).

In Gout, patients exhibit elevated *Prevotella*, *Bacteroides*, and *Faecalibacterium*, alongside reduced *Enterobacteriaceae*, causing uric acid buildup (70-72). Vulnerability to this dysbiosis increases in older adults due to age-

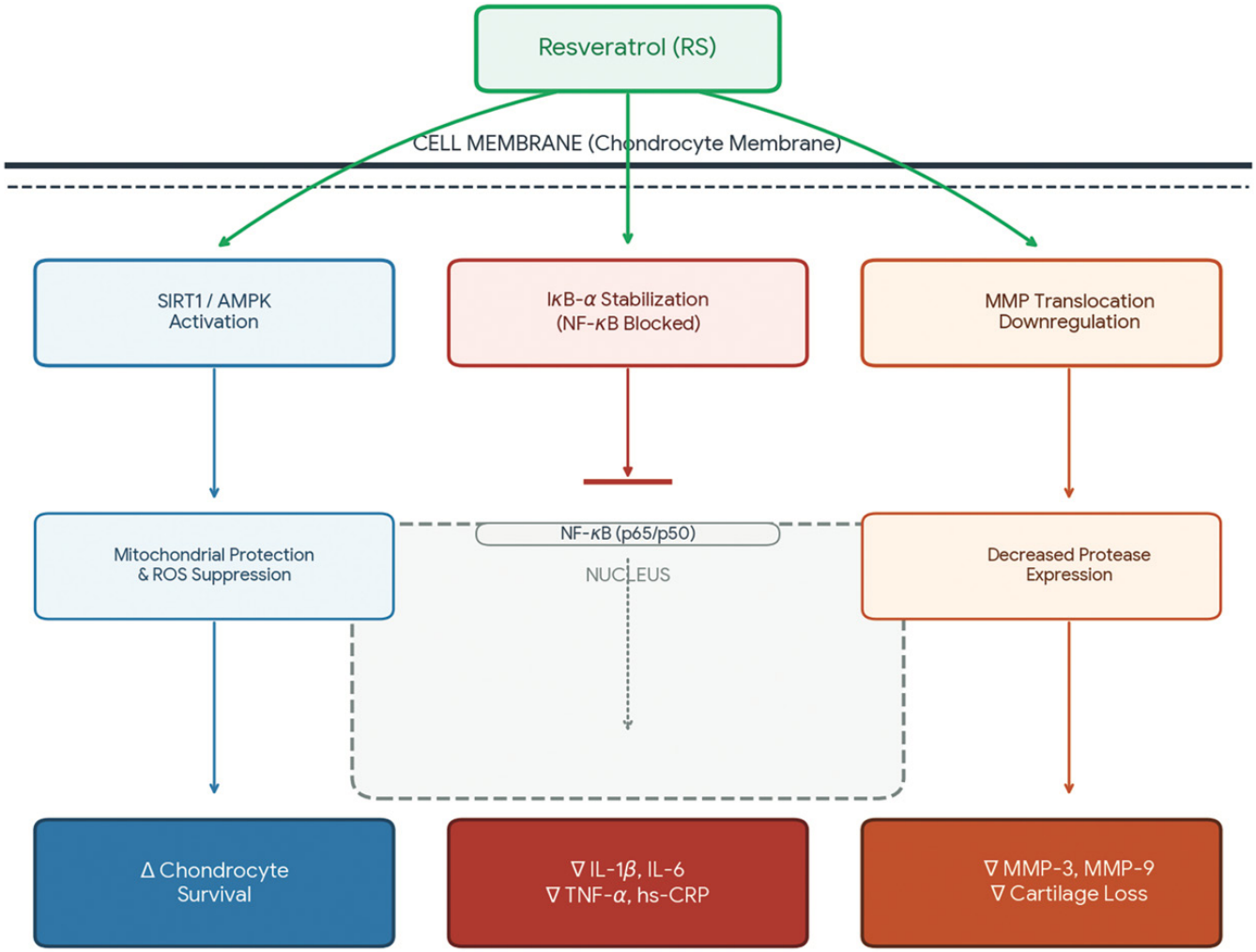

Fig. 2. Molecular effect of resveratrol in rheumatoid arthritis by sirtuin 1 (SIRT1), nuclear-factor kappa-B (NF-Kb) and metalloprotease (MMP) pathways on main inflammatory markers [interleukin 1 (IL-1) and 6 (IL-6), C reactive protein (CRP), tumour necrosis alpha (TNF-alpha)] and cartilage integrity (106).

related immune senescence and barrier decline (60, 76).

Regardless of beverage type, heavy ethanol disrupts gut barrier integrity, increases permeability, and drives dysbiosis, by decreasing beneficial taxa (*Lactobacillus, Bifidobacterium, Akkermansia*) and increasing Gram-negative bacteria (*Bacteroides, Prevotella*), fuelling systemic inflammation (60, 90-104).

On the other side, RS, found in red wine, enhances barrier function and reduces inflammation, though limited by low bioavailability and rapid clearance (7, 98-100). In fact, its biologically active RS metabolites stimulate *Lactobacillus reuteri* growth tenfold and upregulate tight junction proteins (7).

Preliminary evidence suggests RS may act against *P. gingivalis* in oral communities, though its specific role in RA remains uninvestigated (102-104). In murine SpA models, RS attenuates severity by boosting *Lactobacillus* and *Bifidobacterium* while suppressing *E. faecalis* and *E. coli* (98). In gout mice, RS reduces hyperuricemia and renal injury by cutting *Bacteroides* and increasing *Lactobacillus* (105).

RS-microbiota interactions are hypothesised to fight obesity, a shared rheumatic risk factor, though human metabolic syndrome trials show inconsistent results due to individual baseline microbiota variations (99-101). *In vitro* moderate beer intake increases short-chain fatty acids (SCFAs like butyrate) and supports *Bifidobacterium* (93-94). Traditional fermented rice liquors (*e.g.*, Makgeolli) contain live microbes that reverse alcohol-induced dysbiosis, boost SCFA, and lower inflammation in animal models (95).

Finally , the complex molecular anti-inflammatory effects of RS in RA, SpA and gout are summarised in Table III and shown in Figure 2 (63-64, 106-109). Even though the therapeutic perspectives of future RS clinical applications are promising, only few randomised controlled trials evaluated RS monotherapy or relied primarily on pain scores and most of studies explored preclinical and translational insights, as shown by current narrative (7) and systematic (106) literature reviews.

Conclusions

In contrast to gout patients, who are recommended to limit alcohol to prevent disease progression, moderate alcohol intake is linked to a lower risk of developing RA and milder disease activity.

Conversely, chronic alcohol use in SpA may accelerate radiographic spinal progression, though diverse study methods and analysis biases obscure a direct cause-and-effect relationship.

Furthermore, while red wine RS compounds show promising benefits for gut dysbiosis and arthritis severity in animal models of SpA, RA, and gout, human data remains insufficient to support their clinical or therapeutic use in rheumatic diseases.

Ultimately, any alcoholic beverage's net effect is dose-dependent-rather than to a specific beverage type- heavy intake universally causes barrier failure, whereas moderate, polyphenol- or microbe-rich options may partially mitigate ethanol-induced damage.

Overall, we conclude that the potential detrimental or beneficial effects of alcohol consumption should be interpreted with caution. Type- and dose-dependent investigation of different biological responses and further specific trials on rheumatic patients might help to better assess the balance in future.

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