Pantothenic acid (vitamin B5) supplementation in rheumatological diseases: A systematic reviews

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Abstract. Pantothenic acid (PA), the dietary precursor of coenzyme A, plays a central role in mitochondrial metabolism, lipid regulation, and the synthesis of steroid hormones and neurotransmitters. Although PA has been traditionally applied in areas, such as wound healing and immunomodulation, its potential therapeutic relevance in rheumatology has not been well characterized. The aim of the present systematic review was to provide an updated overview of the clinical effectiveness, safety and research gaps related to PA supplementation in rheumatic diseases. A structured search was performed across the PubMed/MEDLINE, Web of Science, SciELO and LILACS databases for human studies published between 1966 and July 2024. Eligible articles investigated PA supplementation in patients with rheumatic diseases and reported clinical outcomes. Key data relating to population characteristics, dosage, treatment duration, outcomes and adverse effects were extracted. A total of seven studies involving 183 participants were included: Two focused on osteoarthritis (OA), one on fibromyalgia (FM) and four addressing systemic lupus erythematosus (SLE). PA was administered using heterogeneous regimens, generally in combination with other micronutrients, at doses ranging from 12.5 mg to 12 g/day and over variable follow-up durations. Clinical improvement was reported in the majority of studies, particularly in cutaneous lupus, in which the substantial resolution of lesions was frequently observed. Benefits in fatigue in SLE and pain reduction in OA and FM were also noted. Adverse events were rare and predominantly mild. On the whole, available clinical evidence suggests that PA supplementation may provide symptomatic benefit in selected rheumatic diseases, with a favorable safety profile. However, current data remain limited by small sample sizes, the lack of standardized protocols and frequent co-supplementation. Well-designed randomized clinical trials, particularly in SLE and OA, are required to determine therapeutic efficacy, optimal dosing and the mechanistic pathways involved.

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Introduction

Pantothenic acid (PA), traditionally referred to as vitamin B5, is an essential water-soluble micronutrient required for the biosynthesis of coenzyme A (CoA), a central cofactor involved in mitochondrial energy production, fatty-acid β-oxidation, acetylation reactions, lipid metabolism, steroidogenesis and neurotransmitter synthesis. Its metabolic relevance extends to immune regulation, epithelial repair and redox balance, processes that are critically involved in the pathophysiology of rheumatologic diseases (1,2). Historically, PA has been used in dermatology and wound healing; however, its therapeutic implications for systemic inflammation and autoimmunity have received limited attention (1).

Given its influence on mitochondrial bioenergetics, fatty-acid metabolism, acetyl-CoA generation and redox pathways, PA may exert anti-inflammatory and immunomodulatory effects relevant to fatigue, pain, immune dysfunction and epithelial integrity, features commonly observed in rheumatic diseases. Clinical symptoms associated with PA insufficiency or suboptimal levels, such as fatigue, musculoskeletal discomfort, mood disturbances, sensory symptoms and impaired immune responses, overlap with several rheumatologic conditions (1).

A growing body of research has focused on vanin-family pantetheinases, particularly vanin-1, an enzyme regulating PA homeostasis through pantetheine cleavage and cysteamine generation. Vanin-1 activity affects oxidative stress responses, leukocyte migration and inflammatory cascades, and its expression is increased in tissues relevant to rheumatologic pathophysiology, such as cartilage and renal epithelium (3). Notably, urinary vanin-1 has emerged as a potential biomarker of active lupus nephritis, reinforcing the biological plausibility of interventions affecting PA metabolism (4).

Early clinical studies from the mid-20th century documented substantial improvement in cutaneous lupus with high-dose PA or panthenol, often combined with vitamin E,

demonstrating reductions in erythema, scaling and lesion size (5-7). Later investigations have explored PA supplementation in osteoarthritis (OA), fibromyalgia (FM) and systemic lupus erythematosus (SLE), reporting benefits in pain, stiffness, fatigue, depression and cutaneous manifestations, although findings remain limited by small sample sizes, heterogeneous dosing, and confounding by co-supplementation (8-11).

A key historical point concerns the discrepancy in the nomenclature of B-complex vitamins, particularly between North American and continental European scientific traditions. PA was originally designated as vitamin B3 in the 1930s to 1940s, as the third nitrogen-containing B vitamin to be identified, while nicotinic acid/nicotinamide ('niacin') was designated as vitamin B5, based on chronological discovery (1,2). Although American literature gradually shifted after the 1990s, promoting the widespread use of the designations of niacin = B3 and pantothenic acid = B5, no formal IUPAC ruling has ever altered the classical nomenclature. Consequently, numerous textbooks and academic sources from continental Europe (e.g., Russia, Germany and France) continue to use PA = B3 and niacin = B5, occasionally causing confusion among international readers (12). This historical discrepancy is directly relevant to early dermatologic and rheumatologic studies on PA, which were authored during the period in which PA was universally referred to as vitamin B3, not B5 (5-7).

Recognizing this nomenclature divergence is essential, as it prevents misinterpretation of older literature and ensures accurate comparison of clinical interventions across historical and contemporary studies. The present systematic review aimed to synthesize the available human evidence on PA supplementation in rheumatic diseases, contextualize mechanistic pathways linking PA to inflammation and immune regulation, and identify gaps requiring rigorous modern investigation.

Data and methods

Search strategy. A comprehensive literature search was conducted across the PubMed/MEDLINE, Web of Science, SciELO and LILACS databases, encompassing the period from January, 1966 to July, 2024. To ensure methodological rigor, the present systematic review followed the principles of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA), although no protocol was prospectively registered. The search strategy employed combinations of the terms 'pantothenic acid', 'pantothenate', 'vitamin B5' and 'dexpanthenol' paired with disease-related descriptors, such as 'rheumatic', 'rheumatologic', 'osteoarthritis', 'fibromyalgia', 'systemic lupus erythematosus', 'rheumatoid arthritis', 'myositis', 'spondyloarthritis', 'Sjogren's syndrome', 'systemic sclerosis' and 'vasculitis'. Equivalent strategies were adapted for each database. No language restrictions were applied, and reference lists of included articles were manually inspected to identify additional studies. A single record retrieved during the search could not be accessed in full text or abstract despite multiple attempts; therefore, although acknowledged, it was not included in the analysis.

Eligibility criteria. Studies were considered eligible when they evaluated pantothenic acid, calcium pantothenate, panthenol or dexpanthenol, administered either alone or within multicomponent formulations, in human participants diagnosed with any rheumatic disease. Eligible studies were required to report clinical outcomes, including symptoms, disease activity, cutaneous manifestations, pain, fatigue, stiffness, flare frequency, or functional parameters. Accepted study designs comprised randomized or non-randomized trials, open-label or prospective studies, cohort studies and case series with at least 5 participants. Articles were excluded if they involved animal or *in vitro* models, narrative reviews, editorials, opinions, conference abstracts without original data, or case reports with <5 patients. Studies in which pantothenic acid could not be quantified or its contribution to clinical outcomes could not be determined were also excluded.

Study selection. The selection process adhered to the PRISMA structure. Following the identification of all records, duplicates were removed, and titles and abstracts were screened by two independent reviewers (JFDC and ATAM). Full-text versions of potentially eligible studies were obtained and assessed for inclusion. Disagreements were resolved through discussion until consensus was reached. Of the 127 records initially identified, 94 remained following the removal of duplicates, 15 were eligible for full-text evaluation, and seven studies met all criteria and were included in the final synthesis.

Data extraction. Data extraction was performed independently by the authors (JFDC and ATAM) using a structured approach. Extracted variables included authorship, publication year, country, study design, sample size and demographics, disease characteristics, pantothenic acid dosage and formulation, route and duration of administration, the presence of co-supplementation, and reported clinical outcomes. Additional information on adverse events, withdrawals and methodological limitations was collected when available. Due to extensive heterogeneity across studies, particularly as regards dosing, outcome measures and design, a meta-analysis was not feasible, and the findings were synthesized narratively.

Risk of bias considerations. Given the diverse nature of the included literature, the risk of bias was assessed narratively. Key aspects included the presence or absence of randomization, allocation concealment, blinding, sample size adequacy, co-supplementation and consistency in outcome reporting. A number of older studies lacked methodological elements required by contemporary research standards, such as validated outcome instruments or controlled designs, which is addressed in detail in the Discussion section below. For one historical publication without accessible text, author contact or archive retrieval was not possible; therefore, although cited historically, it was excluded from the qualitative synthesis.

Results

Overview of included studies. A total of seven clinical studies, comprising 183 participants, fulfilled all eligibility criteria and were included in the final synthesis (5-11) (Table I). These studies evaluated PA or related formulations in OA, FM and SLE, including discoid, subacute cutaneous and systemic forms. The studies were conducted in the USA, UK and Hong

Table I. Summary of clinical studies evaluating pantothenic acid (PA) in rheumatic diseases.

Author, year of publication	Country	Study design	Sample size and population	Disease	PA dose/ formulation	Duration	Main outcomes	Adverse effects	(Refs.)
Annand, 1962	UK	Open-label prospective	26 adults with OA	Osteoarthritis	12.5 mg/day PA + B-complex	Up to 18 months	77% improved pain and stiffness within 14 days; sustained benefit during follow-up	Mild asthenia (12%)	(9)
Haslock and Wright, 1971	Hong Kong	Double-blind RCT	40 patients with knee OA	Osteoarthritis	PA 50 mg + L-cysteine 30 mg, BID	12 weeks	No significant benefit vs. placebo	Headache, sleepiness, depression, memory issues, GI symptoms (15% withdrawal)	(8)
Ali *et al*, 2009	USA	Randomized, double-blind pilot trial	34 patients with FM	Fibromyalgia	IV 'Myers' Cocktail' including dexpanthenol	8 weeks	Improved pain, tender points, depression and quality of life vs. placebo	None reported	(10)
Leung, 2004	Hong Kong	Prospective open-label	12 female patients with SLE	SLE (systemic)	10 g/day PA	Up to 2 years	Improved fatigue in 4 weeks; fewer febrile episodes and flares; reduced medication use	None reported	(11)
Welsh, 1952	USA	Open clinical series	42 patients with SLE	Cutaneous/ Systemic Lupus	0.5-3 g/day initially, then 8-12 g/day PA + vitamin E	2-36 months	Marked improvement; 70-100% clearing of lesions	None reported	(5)
Goldman, 1950	USA	Open case series	46 patients	Discoid, subacute, acute lupus	0.5-10 g/day PA	NR	Improvement in 2/9 acute, 9/10 subacute, 17/27 discoid	One case worsening	(6)
Goldman, 1948	USA	Open-label	14 patients	Discoid lupus	200-400 mg/day PA	6 months	Marked responses, especially in subacute forms; 2 non-responders	One new lesion reported	(7)

BID, twice a day; FM, fibromyalgia; GI, gastrointestinal; IV, intravenous; OA, osteoarthritis; PA, pantothenic acid; RCT, randomized controlled trial; SLE, systemic lupus erythematosus.

Kong, over several decades (5-11). Sample sizes ranged from 12 to 46 participants, and demographic data were incompletely reported in some of the older studies. Across all trials, PA was rarely administered as monotherapy, and dosing regimens varied substantially, from 12.5 mg/day in combination with B-complex formulations to 10-12 g/day of high-dose therapy in SLE and cutaneous lupus (5-11). Of note, one additional study on rheumatoid arthritis was identified; however, this could not be retrieved in abstract or full text and is therefore mentioned, but not analyzed (12).

Studies on OA. Of note, two studies assessed PA in OA (8,9). An early open-label prospective study from the UK administered 12.5 mg/day PA plus vitamin B-complex to patients with OA and reported that 20 of the 26 participants (77%) experienced an improvement in stiffness and pain within 14 days, with benefits maintained during an 18-month follow-up; mild asthenia occurred in 3 of 26 patients (12%) (9). By contrast, a later double-blind, randomized, placebo-controlled trial evaluated 50 mg PA plus 30 mg L-cysteine twice daily vs. the placebo over a period of 12 weeks in knee OA, including 40 patients with long-standing disease (1-54 years of duration) (8). That trial found no significant differences between the active and placebo groups in global outcomes. Adverse events, such as headache, sleepiness, depression, memory loss, flatulence and abdominal pain, led to withdrawal in 6 of 40 participants (15%) (8). These contrasting results highlight the heterogeneity in study design, co-supplementation and outcome assessment, and make it difficult to isolate a specific therapeutic effect of PA in OA.

Research on FM. FM was evaluated in a randomized, double-blind, placebo-controlled pilot trial using an intravenous micronutrient formulation known as \Myers' cocktail\, which included dexpanthenol and panthenol as PA derivatives among other vitamins and minerals (10). In that study on 34 patients, predominantly female patients, the active treatment group exhibited statistically and clinically significant improvements in tender point count, pain, depression scores and quality of life after 8 weeks, compared with the placebo (10). No adverse effects were reported in the intervention group, suggesting a favorable tolerability profile (10). However, as the infusion combined multiple micronutrients, including magnesium, B-complex vitamins and vitamin C, the specific contribution of PA cannot be determined, although the observed improvements are consistent with the hypothesis that correcting micronutrient deficits and supporting mitochondrial metabolism may alleviate the symptoms of FM (10).

Studies on SLE and cutaneous lupus. A total of four studies explored the role of PA in lupus, including the discoid, subacute cutaneous and systemic forms (5-7,11). In an open prospective trial from Hong Kong, 12 women with SLE received 10 g/day PA for up to 2 years and exhibited an improvement in fatigue within 4 weeks, followed by reductions in pyrexia and fewer major flares over a longer follow-up period (11). In a number of patients, background SLE medications could be tapered, and no adverse effects were reported (11).

Historical dermatologic series from the USA described high-dose calcium pantothenate or panthenol, often combined with vitamin E 400 mg/day, for the treatment of cutaneous lupus (5-7). In an open trial of 42 patients with longstanding SLE, high-dose PA (0.5-3 g/day initially, then 8-12 g/day) plus vitamin E resulted in an improvement of skin lesions in all patients, with a 70-100% clearing of cutaneous lesions in numerous cases and good long-term control over 2-36 months of follow-up (5). Another series of 46 patients with discoid, subacute and systemic lupus treated with PA doses ranging from 0.5 to 10 g/day reported clinical improvement in 2 of 9 patients with acute lupus, 9 of 10 with subacute lupus, and 17 of 27 with discoid lupus, with only 1 case of lesion aggravation (6). A smaller study on 14 patients with discoid lupus treated with 200-400 mg/day PA for 6 months documented marked responses particularly in subacute lupus, with 2 of 14 non-responders, 3 of 14 requiring phenol cauterization of residual lesions, and 1 of 14 developing a new lesion during follow-up (7). Collectively, these findings suggest a consistent therapeutic signal in cutaneous lupus, particularly in subacute and discoid forms, albeit in uncontrolled historical series.

Safety profile. Across all studies, the safety profile of PA appeared favorable. In the OA trials, adverse effects were generally mild and limited to a subset of participants, with symptoms such as headache, sleepiness, mood changes, flatulence and abdominal pain leading to withdrawal in a minority of patients (8,9). In the Myers' cocktail fibromyalgia trial, no adverse events were recorded in the active treatment arm (10). The lupus studies, particularly the high-dose regimens, with up to 10-12 g/day PA, reported no severe treatment-related toxicity, and historical reports emphasized good tolerability over prolonged administration (5-7,11). Nonetheless, the absence of systematic laboratory monitoring and standardized adverse event reporting in older studies warrants cautious interpretation and underscores the need for modern safety assessments.

General findings. Taken together, these seven studies suggest that PA supplementation may confer symptomatic benefits in selected rheumatic diseases, notably cutaneous lupus, with additional signals in SLE-related fatigue, fibromyalgia symptoms, and OA-related pain and stiffness (5-11). However, the evidence base is constrained by small sample sizes, heterogeneous dosing regimens, co-supplementation with other vitamins and amino acids, non-standardized outcome measures, and limited use of randomized, blinded designs. The additional, non-analyzed report in rheumatoid arthritis (12) illustrates that the potential spectrum of PA use in rheumatology may be broader than currently documented. Overall, these limitations highlight the urgent need for well-designed randomized controlled trials using standardized dosing, validated clinical endpoints, and systematic safety evaluation to clarify the therapeutic role of PA in rheumatologic practice.

Discussion

The present systematic review highlights both foundational and contemporary evidence on PA supplementation in rheumatic diseases, reframing these findings within the evolving field of immunometabolism. As the essential precursor to CoA, PA supports multiple biochemical processes relevant to inflammation and tissue repair, including mitochondrial

bioenergetics, fatty-acid oxidation, acetyl-CoA homeostasis and steroidogenesis. Clinically, insufficient PA may manifest as fatigue, musculoskeletal discomfort and impaired barrier integrity, features commonly observed in chronic inflammatory disorders and are thus supportive of supplementation strategies (1). In addition to these established mechanisms, PA also contributes to sphingolipid and phospholipid biosynthesis, epigenetic acetylation processes and antioxidant regulation, providing broader biochemical plausibility for its contribution to immune and tissue homeostasis. In comparison, although other B vitamins (such as B3 and B6) exert anti-inflammatory effects, PA is unique due to its obligatory incorporation into CoA-dependent metabolic pathways.

A key mechanistic connection lies in the vanin-1 pantetheinase pathway. Vanin-1 activity generates cysteamine, a modulator of oxidative stress responses and inflammatory signaling. Its expression in both leukocytes and renal tubular cells has prompted interest in its urinary secretion as a biomarker of lupus nephritis activity (3,4). A previous preclinical study also suggested that altered vanin-1 signaling may contribute to chondrogenesis dynamics and aberrant mineralization, linking this axis to degenerative joint conditions and soft-tissue calcification (3). Although a direct causal chain from PA supplementation to vanin-1 modulation remains hypothetical, this axis provides a compelling biological rationale for biomarker-driven interventional trials. Notably, the role of PA in regulating oxidative stress and immune activation may intersect with vanin-1-mediated redox pathways, strengthening the mechanistic framework for testing PA in SLE.

Among the available clinical data, cutaneous lupus exhibits the most consistent therapeutic signal. Small series and uncontrolled interventions report rapid and sustained improvements in dermatologic lesions with PA-based regimens (5-7). Despite considerable variability in dosing, administration route and co-supplementation, the recurrence of response patterns, alongside reductions in fatigue and flare frequency, suggests potential adjunctive utility in selected SLE phenotypes (5-7,11). Further research is required to incorporate validated dermatologic scoring systems, blinded outcome assessment and stratification according to cutaneous subtype (acute, subacute and discoid) to refine treatment expectations. The historical use of high-dose PA in these studies, without significant toxicity, further supports its safety profile and justifies renewed evaluation under controlled contemporary designs.

In FM, preliminary benefit was observed with intravenous multi-micronutrient therapy (10). Although attribution to PA alone is not possible, mechanistic plausibility is supported by its contributions to CoA-dependent cellular energy and acetylcholine biosynthesis, aligning with FM models of mitochondrial inefficiency and autonomic dysfunction. Comparative studies isolating PA or manipulating PA content within IV formulations, while incorporating objective endpoints, such as actigraphy, pressure pain thresholds and validated fatigue metrics, would clarify PA-specific effects. Additionally, classical descriptions of pantothenic acid deficiency leading to 'burning feet syndrome', a neuropathic condition responsive to PA supplementation, support the hypothesis that PA may influence sensory pathways relevant to FM and autoimmune neuropathies.

Evidence in OA remains inconclusive. A previous open-label study reported improvements in stiffness and pain with low-dose oral PA combined with B-complex vitamins (9), whereas another randomized controlled trial revealed no advantage of PA plus L-cysteine over the placebo (8). Differences in OA phenotype, baseline severity, intervention design and outcome measurement likely contributed to divergent results. Given the global burden of OA and the scarcity of disease-modifying options, larger rigorously designed trials incorporating standardized pain/function endpoints, imaging modalities and biomarkers of cartilage turnover are warranted. Potential mechanistic links between CoA-dependent metabolism, chondrocyte bioenergetics and osteoarthritic cartilage degeneration warrant further exploration.

Safety findings across available studies appear reassuring, with predominantly mild and transient adverse events. Notably, lupus trials utilized supra-nutritional PA doses without major toxicity signals (5-7,11). Nonetheless, limited sample sizes, short follow-up intervals, and insufficient systematic reporting of laboratory trends and drug interactions restrict firm conclusions. As PA participates in acetylation reactions required for the metabolism of several pharmaceutical agents, its potential influence on drug pharmacokinetics, particularly in patients receiving corticosteroids, antimalarials, NSAIDs, or immunosuppressants, warrants further investigation in future studies.

The present systematic review provides a comprehensive appraisal of all human clinical studies in rheumatic settings to date, integrating mechanistic insight with clinical phenotype and immunometabolic context. However, the interpretability of available data is constrained by small cohorts, heterogeneous interventions, variable dosing, the absence of blinding in the majority of studies and reliance on historical controls in cutaneous lupus research. Additionally, the only OA randomized controlled trial and a pilot FM randomized controlled trial evaluated multi-ingredient preparations, obscuring the individual contribution of PA (8,10). Concomitant vitamin use (e.g., B-complex and vitamin E) and differing administration routes (oral vs. intravenous) further complicate the attribution of the effects. Furthermore, additional evidence from Eastern European literature, including a patented panthenol-containing formulation used to accelerate tendon healing in systemic sclerosis and a study demonstrating improved xerophthalmia with a panthenol-containing ophthalmic solution (Stillavit) in rheumatoid arthritis, suggests broader reparative and epithelial-trophic roles for PA that merit investigation.

Several priorities thus emerge for subsequent research: i) Clinical trials: Well-powered randomized studies of oral PA monotherapy in lupus and OA, including dose-ranging phases to determine minimal effective and maximal tolerated doses are warranted. ii) Mechanistic exploration: The quantification of CoA-related metabolites, acetyl-CoA and vanin-1 activity/expression is required to investigate PA-responsive biological pathways and evaluate vanin-1 as a predictive or pharmacodynamic biomarker in SLE (3,4,13). iii) Pragmatic FM trials: The assessment of PA as an adjunct to standard care using patient-centered outcomes (pain interference, sleep quality and fatigue) and metabolic profiling is warranted to identify responder subgroups with impaired CoA flux.

iv) Expansion to other rheumatic diseases: Systematic evaluation in rheumatoid arthritis, spondyloarthritis, Sjögren's syndrome, myositis, systemic sclerosis and vasculitis is required, given shared inflammatory and oxidative mechanisms (14-16).

In conclusion, across multiple decades of research, PA supplementation has exhibited potential therapeutic value in specific rheumatologic contexts, most notably in cutaneous forms of lupus, while producing preliminary yet promising findings in FM and more inconsistent outcomes in OA. Safety profiles reported to date are generally acceptable, with mostly mild and transient adverse effects. However, the evidence base remains limited by small sample sizes, heterogeneous dosing regimens, frequent co-supplementation and the absence of modern randomized designs, which collectively restrict firm conclusions regarding efficacy and optimal treatment strategies.

Given these limitations, PA should be considered an investigational adjunct rather than an established therapy, pending confirmation from contemporary randomized controlled trials. Future studies are required to incorporate standardized dosing protocols, validated dermatologic and musculoskeletal outcome measures, biomarker integration (particularly CoA-related metabolites and vanin-1), and systematic pharmacovigilance to evaluate potential interactions with immunomodulatory agents.

In the interim, PA supplementation may be cautiously explored as an adjunctive strategy in selected patient populations, provided that dosing, concomitant therapies and safety parameters are carefully monitored. Robust, well-designed clinical trials are urgently required to clarify its therapeutic potential and define its role within modern rheumatologic practice.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

JFDC performed the literature search, the analysis of data from the literature, and the writing and submission of the manuscript. ATAM performed the literature search, the analysis of data from the literature and was revised the manuscript. Both authors have read and approved the final manuscript. JFDC and ATAM confirm the authenticity of all the raw data.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Use of artificial intelligence tools

During the preparation of this work, AI tools were used to improve the readability and language of the manuscript or to generate images, and subsequently, the authors revised and edited the content produced by the AI tools as necessary, taking full responsibility for the ultimate content of the present manuscript.

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