International Journal of Basic & Clinical Pharmacology
Banerjee PG et al. Int J Basic Clin Pharmacol. 2026 Jul;15(4):823-832
https://www.ijbcp.com
pISSN 2319-2003 | eISSN 2279-0780

DOI: https://dx.doi.org/10.18203/2319-2003.ijbcp20261977

Review Article

Liposomal vitamin B5 (pantothenic acid): enhanced delivery, mechanisms, and clinical applications

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Received: 28 April 2026
Revised: 02 May 2026
Accepted: 14 May 2026

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ABSTRACT

Vitamin B5 (pantothenic acid) is a critical precursor for the synthesis of coenzyme A (CoA), facilitating essential metabolic pathways including the Krebs cycle and fatty acid synthesis. Despite its biological importance, conventional oral supplementation faces significant pharmacokinetic challenges, primarily the saturation of sodium-dependent multivitamin transporters (SMVT) and susceptibility to gastric degradation, which limit its systemic bioavailability. Liposomal encapsulation offers a biomimetic "Trojan horse" delivery mechanism that protects vitamin B5 from the acidic gastric environment. By utilizing a phospholipid bilayer, this technology enables non-saturable absorption pathways such as passive diffusion, endocytosis, and lymphatic uptake, effectively bypassing the competitive limitations of SMVT and the hepatic first-pass effect. Reported literature suggests that enhanced delivery of vitamin B5 through advanced formulations holds significant potential in several clinical domains: dermatological health: supporting skin barrier repair and addressing conditions such as acne, wound healing: facilitating tissue regeneration and dermatological repair, metabolic management: aiding in the regulation of dyslipidaemia through its role in lipid metabolism and steroid hormone synthesis, and neuroprotection: enhancing the synthesis of neurotransmitters like acetylcholine to support neurological health. While liposomal technology significantly optimizes the bioavailability and therapeutic potential of pantothenic acid, further clinical evidence is required to fully map its long-term efficacy across diverse populations. Future research should focus on addressing existing evidence gaps to establish standardized protocols for its application in chronic metabolic and neurological therapy.

Keywords: Vitamin B5, Pantothenic acid, Coenzyme A, SMVT

INTRODUCTION

Pantothenic acid, commonly known as vitamin B5, is a vital water-soluble nutrient that serves as a cornerstone of cellular metabolism.[1] Its biological significance is primarily derived from its role as the fundamental precursor for the biosynthesis of coenzyme A (CoA) and acyl carrier proteins.[2] These cofactors are indispensable for the tricarboxylic acid (TCA) cycle, fatty acid synthesis, and the metabolism of carbohydrates and proteins. Beyond energy production, pantothenic acid is essential for the synthesis of cholesterol, steroid hormones, and the neurotransmitter acetylcholine, making it a critical component for maintaining physiological homeostasis across various organ systems.[3]

The challenges of achieving adequate vitamin B5 status are further compounded by substantial losses during food processing and preparation. Industrial and domestic processing can result in the degradation of up to 80% of the vitamin's activity due to its sensitivity to heat and acidic or alkaline conditions.[6] This fragility, coupled with the limited capacity of conventional transport mechanisms, necessitates the development of advanced delivery systems. Liposomal formulations represent a promising technological advancement, offering a means to bypass saturable transporters and protect the nutrient from

environmental degradation, thereby enhancing systemic bioavailability and therapeutic potential.[7,8]

To address the inherent pharmacokinetic hurdles of traditional supplementation, West Bengal Chemical Industries Ltd., Kolkata, India (WBCIL)—a WHO-GMP certified manufacturer with over six decades of expertise in API and fine chemicals—has engineered a high-performance liposomal vitamin B5. LipoedgeTM , i.e., WBCIL's liposomal encapsulation technology utilizes a high-purity phospholipid matrix, typically derived from sunflower-based phosphatidylcholine, to provide both gastric protection and biomimetic absorption.[9,10] By mimicking the structure of human cell membranes, these liposomes facilitate nutrient uptake via passive diffusion and endocytosis, effectively bypassing the saturated SMVT transporters and avoiding the hepatic first-pass effect.[11,12] This review article evaluates the current landscape of vitamin B5 therapy, focusing on its biochemistry and physiology, the principles and advantages of liposomal delivery, and clinical applications in dermatology (specifically acne and wound healing), dyslipidemia, and cardiovascular risk. Furthermore, it explores emerging evidence regarding its role in inflammation and neurodegeneration, alongside an assessment of safety, dosing, and tolerability protocols.

BIOCHEMISTRY AND PHYSIOLOGY OF VITAMIN B5

Pantothenic acid exists in several biologically active forms, including D-pantothenic acid, its alcohol analog dexpanthenol, and common supplemental forms such as calcium pantothenate and pantethine.[13] Structurally, it is composed of pantoic acid joined to the amino acid β-alanine via an amide bond. Within the body, the most critical physiological function of this vitamin is its role as the fundamental structural component of CoA and acyl carrier proteins (ACP). These cofactors are indispensable for a vast array of enzymatic reactions, with CoA serving as a carrier for acyl groups in over 4% of all known cellular chemical processes.[14]

The physiological importance of vitamin B5 is centered on its contribution to acetyl-CoA metabolism, which serves as a crossroads for energy production and macromolecule synthesis. As part of CoA, it facilitates the Krebs cycle by enabling the conversion of carbohydrates, fats, and proteins into adenosine triphosphate (ATP).[15] Furthermore, it is a primary driver of fatty acid synthesis and degradation, as well as the biosynthesis of cholesterol, phospholipids, and steroid hormones like cortisol and progesterone. In the nervous system, it acts as a precursor for the neurotransmitter acetylcholine, making it vital for cognitive function and peripheral nerve signaling (Figure 1).[16]

The absorption of vitamin B5 primarily occurs in the small intestine through an active, energy-dependent process mediated by the SMVT. This transporter is also responsible for the uptake of biotin and lipoic acid, creating a competitive environment that can limit absorption efficiency.[17] Because this mechanism is saturable, fractional absorption decreases significantly as the dose increases; studies indicate that high-dose intake can cause the absorption rate to drop to approximately 10%.[18] Following absorption, the vitamin is transported to tissues for CoA synthesis, while excess amounts are not stored and are instead excreted in the urine, making urinary levels a primary indicator of dietary status.[18]

To maintain these critical metabolic functions, the National Institutes of Health (NIH) and other health authorities have established adequate intake (AI) levels based on age and life stage. For healthy adults, the AI is set at 5 mg per day.[19] Requirements increase during specific physiological states to support foetal development and milk production, with the AI rising to 6 mg per day for pregnant women and 7 mg per day during lactation.[19] Despite its wide availability in food, the inherent saturation limits of the SMVT and the vitamin's sensitivity to processing losses emphasize the need for delivery systems that can provide reliable physiological concentrations.[20]

LIPOSOMAL DELIVERY: PRINCIPLES AND ADVANTAGES

Liposomes are spherical, self-assembling vesicles characterized by one or more lipid bilayers surrounding an internal aqueous core. The primary structural components are phospholipids—amphiphilic molecules with a hydrophilic head and two hydrophobic tails.[21] In an aqueous environment, these lipids arrange themselves into a bilayer to shield their hydrophobic tails from water, creating a protected internal compartment.[21] Liposomes are generally classified by their structure and size: unilamellar vesicles (ULVs) consist of a single phospholipid bilayer and are ideal for rapid nutrient release, while multilamellar vesicles (MLVs) feature an "onion-like" structure of multiple concentric bilayers, offering a more sustained release profile and greater structural stability.[22]

The encapsulation of water-soluble vitamins, such as vitamin B5, within the aqueous core of a liposome provides a significant mechanistic advantage over conventional delivery. In traditional oral supplementation, vitamin B5 is highly susceptible to degradation in the acidic environment of the stomach and relies on an SMVT in the small intestine for absorption.[23] Liposomal encapsulation acts as a "bio-shield," protecting the nutrient from gastric acid and enzymatic breakdown. Furthermore, liposomes can be absorbed via multiple non-saturable pathways, including endocytosis, direct fusion with the intestinal mucosal cell membranes, and uptake through the lymphatic system (M-cells of Peyer's patches).[21,22] This multi-pathway absorption effectively bypasses the bottleneck of traditional transporters, leading to significantly enhanced systemic bioavailability.[22]

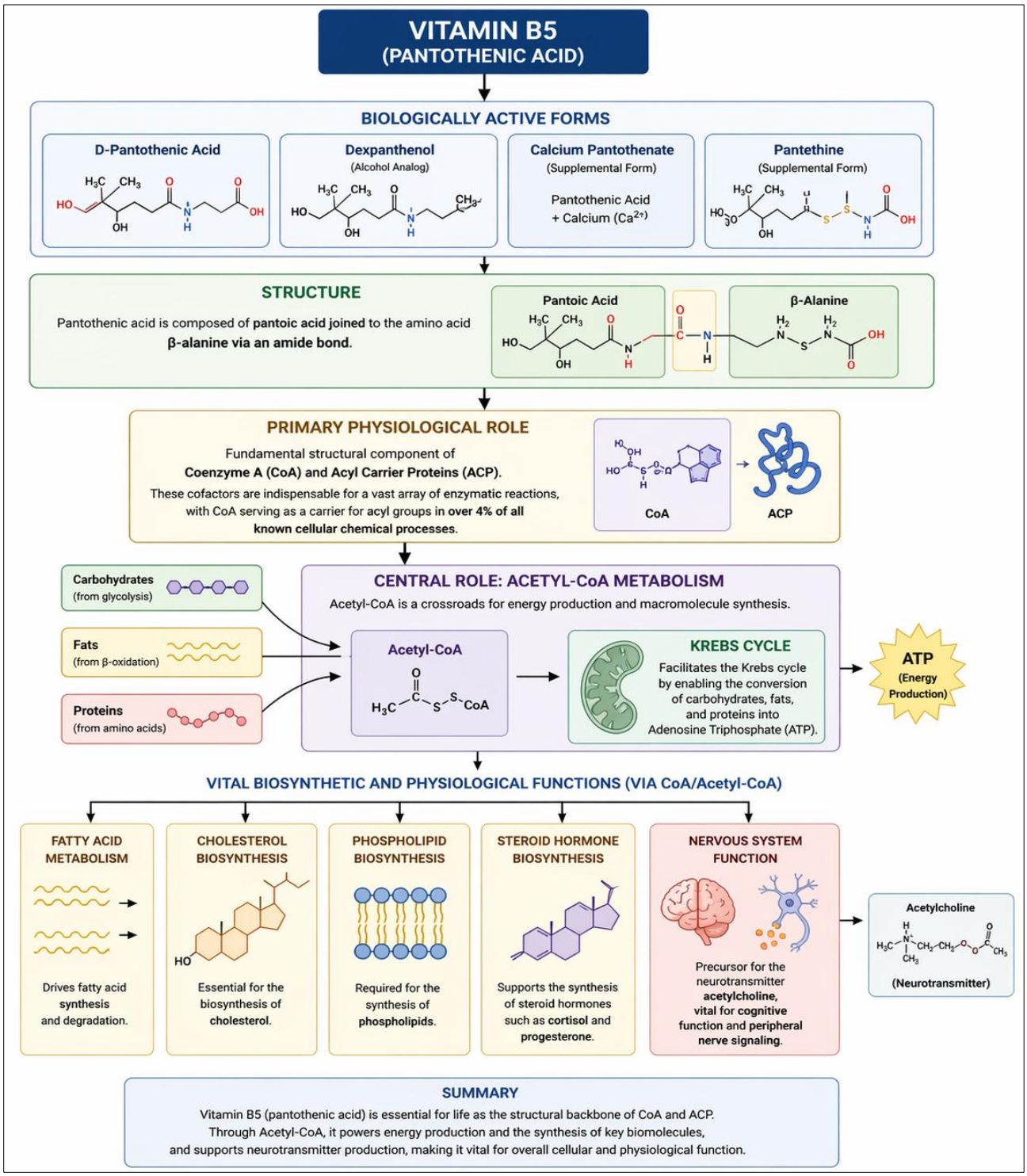

Figure 1: Infographic presentation of biochemistry and physiology of vitamin B5.

Manufacturing these sophisticated delivery systems requires precise control over particle size and stability. Common methods include thin-film hydration (the Bangham method), where a lipid film is hydrated to form MLVs, which are then typically processed via extrusion through polycarbonate membranes to achieve a uniform, nano-sized unilamellar distribution.[23] Emerging technologies like microfluidics allow for even greater precision by mixing lipid and aqueous phases in micro-channels, resulting in highly consistent liposomes with high encapsulation efficiency.[23]

The stability of the final product is often measured by its zeta potential—an indicator of the surface charge that prevents particles from aggregating—and its polydispersity index (PDI), which ensures a uniform size distribution necessary for predictable biological performance.[23]

From a regulatory and safety perspective, liposomal nutraceuticals occupy a unique space. While specific global regulations for liposomal supplements are still evolving, manufacturers often benchmark their processes against established pharmaceutical guidelines, such as those provided by the US-FDA and EMA.[24] High-quality formulations utilize ingredients that are generally recognized as safe (GRAS), such as sunflower-derived phosphatidylcholine. Although large-scale clinical randomized controlled trials (RCTs) specifically for liposomal vitamin B5 are still in development, the established mechanistic rationale—supported by decades of liposomal research in other water-soluble nutrients like vitamin C—provides a robust framework for its superior efficacy and safety profile in human nutrition.[25]

CLINICAL EVIDENCE: DERMATOLOGY AND ACNE

The use of vitamin B5 in dermatology is rooted in its role as a precursor to CoA, which is essential for lipid metabolism within the sebaceous glands. One of the most significant clinical demonstrations of this effect was a 12-week randomized controlled trial (RCT) by Yang et al (2014), involving 41 subjects with mild-to-moderate acne. Participants receiving a 2.2 g/day oral dose of vitamin B5 experienced a greater than 67% reduction in total facial lesion count compared to the placebo group ($p=0.02$).[26] This followed an earlier pilot feasibility study by Capodice et al (2012), which established the tolerability and safety of pantothenic acid-based dietary supplements in an 8-week open-label format, reporting significant improvements in skin quality and lesion reduction.[27]

Recent research has further validated the efficacy of B5 derivatives when used as adjunct therapy. Saki et al (2024–25) conducted an RCT with 59 patients comparing the use of adapalene alone versus a combination of adapalene and intramuscular dexpanthenol. The results demonstrated the superiority of the combined approach, suggesting that increasing systemic levels of B5 derivatives enhances the clinical response in acne management.[28]

These findings build upon the "Leung hypothesis" (1997), which posited that acne is essentially a manifestation of a localized CoA deficiency. In a 100-patient series, Leung observed that high-dose oral and topical administration led to rapid sebum reduction and the shrinking of pore size, effectively regulating sebaceous gland lipid synthesis and promoting keratinocyte differentiation.[29]

The therapeutic potential of these findings is amplified when considered through the lens of liposomal delivery. While conventional topicals often struggle to penetrate the lipid-rich environment of the sebaceous follicle, liposomal vitamin B5 and its derivatives (such as dexpanthenol) offer a distinct advantage.[30] Because liposomes are composed of phospholipids similar to skin surface lipids, they can more effectively deliver active nutrients directly to the target follicles. This liposomal hypothesis suggests that by bypassing the skin's barrier more efficiently than traditional creams, these formulations can achieve the necessary intracellular concentrations to regulate lipid metabolism at lower doses, potentially reducing the need for the extremely high-dose oral regimens previously explored in the literature (Table 1).[30]

Table 1: Clinical evidence of vitamin B5 in dermatology.

Study	Study type	Participants/dosage	Key findings	Mechanism
Yang et al (2014)	Randomized controlled trial (RCT)	n=41; 2.2 g/day oral B5 for 12 weeks	>67% reduction in total facial lesion count versus placebo (p=0.02)	Confirmed efficacy and safety of oral B5 for mild-to-moderate acne
Saki et al (2025)	Rct (adjuvant therapy)	n=59; IM Dexpanthenol + Adapalene versus Adapalene alone	Significant superiority of the combined approach in reducing acne severity	Systemic B5 derivatives enhance the clinical response of topical retinoids
Capodice et al (2012)	Pilot feasibility study	8-week open-label trial	High tolerability; significant improvement in overall skin quality and lesion reduction	Established the social and clinical significance of B5 supplementation
Leung (1997)	Case series/ clinical observation	100 patients; high-dose oral + topical B5	Rapid sebum reduction and shrinking of pore size observed in the majority of cases	Proposed that acne is a manifestation of localized CoA deficiency
Shields et al (2023)	Systematic literature review	Multiple clinical sources	Consistent evidence supporting B5 as a low-risk, effective alternative for acne	Validates the historical use of B5 through modern clinical standards

CLINICAL EVIDENCE: WOUND HEALING AND SKIN BARRIER

Dexpanthenol, the stable alcohol analogue of pantothenic acid (provitamin B5), has a long-standing clinical history in wound management and dermatological repair. Its efficacy is well-documented in post-operative recovery; for instance, randomized controlled trials have demonstrated that dexpanthenol significantly accelerates mucosal healing and reduces pain in patients following tonsillectomy, endoscopic sinus surgery, and post-

intubation sore throat.[8] In the context of inflammatory skin conditions, research by Udompataikul et al (2012) in a pilot study of paediatric atopic dermatitis indicated that a 5% dexpanthenol formulation could serve as a viable steroid-sparing agent, showing comparable efficacy to 1% hydrocortisone in improving skin barrier function and reducing transepidermal water loss (TEWL).[31]

The biological mechanism driving these clinical outcomes is centered on the stimulation of fibroblast activity. Vitamin B5 serves as a critical cofactor in the metabolic pathways that trigger fibroblast migration, proliferation, and protein synthesis. By increasing the availability of CoA, it fuels the high energy demands of tissue remodelling and enhances the production of collagen and glycans necessary for structural integrity.[32] Furthermore, dexpanthenol promotes rapid epithelial regeneration, acting as a humectant that maintains the hydration levels required for optimal enzymatic activity during the inflammatory and proliferative phases of healing.[32]

Liposomal delivery systems provide a transformative advantage for these topical applications. Conventional creams often suffer from poor dermal penetration, as the skin's stratum corneum acts as a formidable barrier to water-soluble nutrients. Liposomes, being structurally similar to the lipid bilayers of human skin, facilitate "transdermal trafficking" of dexpanthenol deeper into the dermis.[33]

In vitro studies on human dermal fibroblasts have shown that liposomal encapsulation can lead to higher intracellular concentrations of B5 compared to free forms, resulting in a more robust stimulation of the genes responsible for extracellular matrix assembly. This enhanced penetration ensures that the therapeutic agent reaches the deeper layers of the wound bed where fibroblast activity is most critical (Figure 2).[33]

CLINICAL EVIDENCE: DYSLIPIDAEMIA AND CARDIOVASCULAR RISK

The therapeutic application of vitamin B5 in cardiovascular health primarily involves pantetheine, a double-bonded metabolite of pantothenic acid. Clinical research has consistently identified pantetheine as a potent lipid-modifying agent. In a landmark triple-blind, randomized controlled trial, Evans et al (2014) investigated the effects of 600–900 mg/day of pantetheine over 16 weeks in subjects with low-to-moderate cardiovascular disease (CVD) risk.[34]

The study reported significant reductions in total cholesterol, LDL cholesterol, and non-HDL cholesterol. These findings align with multiple earlier clinical trials where a standard regimen of 300 mg administered three times daily resulted in marked decreases in triglycerides and total cholesterol, often accompanied by a favourable increase in HDL cholesterol levels.[34]

The mechanism behind these lipid-lowering effects is multi-faceted. Pantetheine acts by inhibiting the activity of HMG-CoA reductase, the rate-limiting enzyme in cholesterol biosynthesis, and by modulating the activity of other enzymes involved in fatty acid metabolism.[35] Beyond direct cholesterol regulation, Vitamin B5 derivatives have been shown to reduce insulin resistance and activate lipolysis—the breakdown of fats. By increasing the available pool of CoA, pantetheine facilitates the efficient transport of fatty acids into the mitochondria for beta-oxidation, thereby preventing the accumulation of lipid precursors that contribute to dyslipidaemia.[8]

The introduction of liposomal pantetheine presents a significant clinical opportunity to optimize these outcomes. Conventional pantetheine supplementation often requires high, divided doses to maintain therapeutic serum levels, which can lead to gastrointestinal side effects in sensitive individuals.[36] Liposomal delivery offers a theoretical advantage by protecting the metabolite from premature breakdown and enhancing its absorption through non-saturable mucosal pathways. This improved bioavailability may allow for the achievement of lipid-modifying targets at lower total daily doses, potentially improving long-term patient compliance and reducing the metabolic burden on the gastrointestinal tract (Figure 3).[37]

EMERGING EVIDENCE: INFLAMMATION AND NEURODEGENERATION

Beyond its established roles in dermatology and lipid metabolism, pantothenic acid is increasingly recognized for its potential in modulating systemic inflammation and maintaining neurological health. Epidemiological data, such as the study by Jung et al (2015) involving Korean adults aged 40 and older, have revealed an inverse association between dietary vitamin B5 intake and serum levels of C-reactive protein (CRP), a hallmark biomarker of systemic inflammation.[38] This suggests that adequate B5 status may play a protective role in mitigating chronic low-grade inflammation, likely through its essential contribution to the synthesis of anti-inflammatory steroid hormones and the regulation of cellular energy metabolism.[38]

In the field of neurology, vitamin B5 is emerging as a nutrient of significant interest regarding neurodegenerative pathologies. Research by Xu et al (2020) identified a profound cerebral B5 deficiency in brain tissue from patients with Alzheimer's disease, proposing that this deficiency might be a reversible contributing factor to cognitive decline.[39] Similarly, Patassini et al (2019) documented localized B5 depletion in cases of Huntington's disease. These findings are particularly relevant given that pantothenate kinase-associated neurodegeneration (PKAN)—a rare but severe genetic disorder—is caused by mutations that impair the first step of CoA synthesis from vitamin B5, leading to iron accumulation in the brain and progressive motor dysfunction.[40]

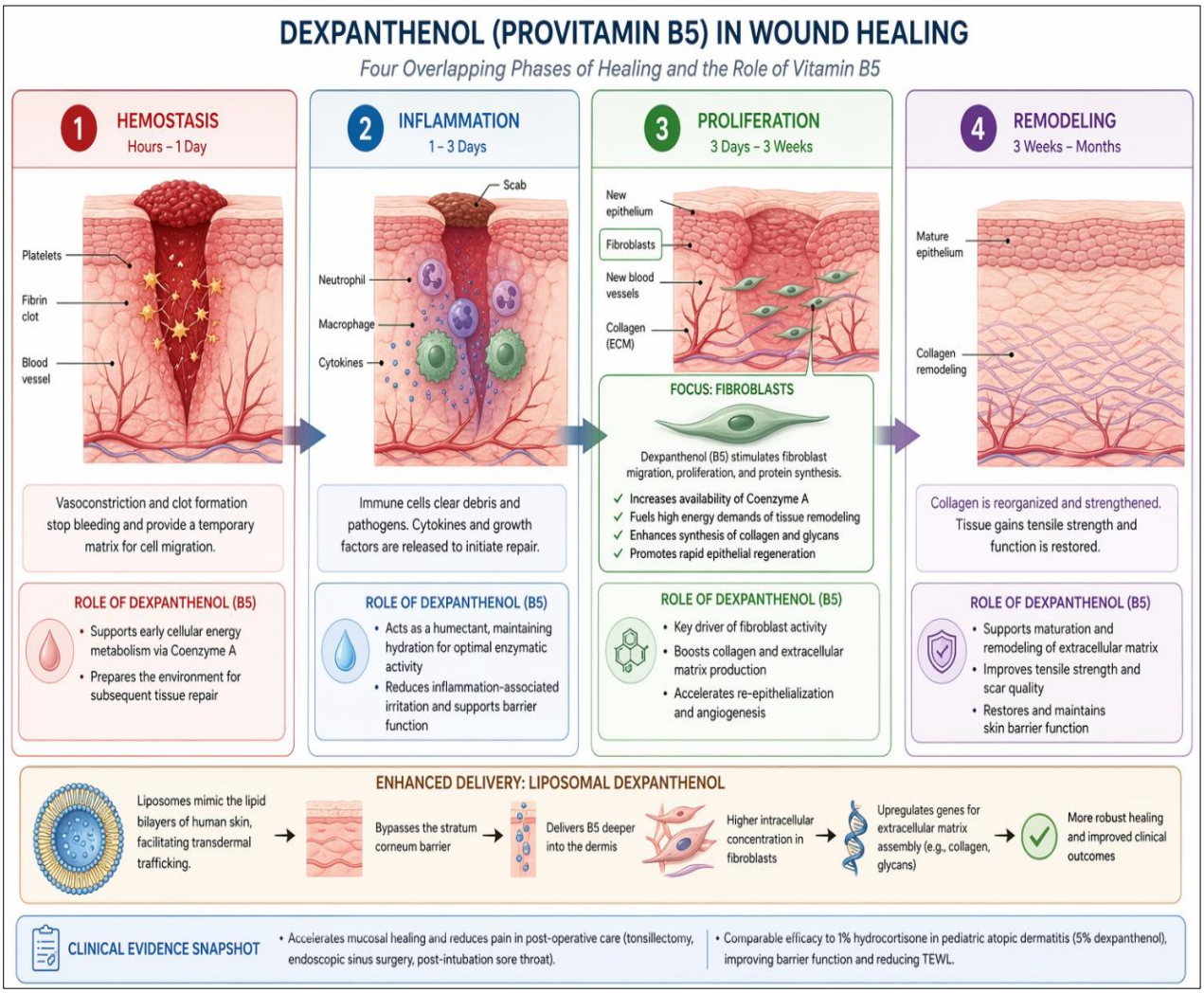

Figure 2: Infographic presentation of vitamin B5 in wound healing and skin barrier.

The application of liposomal technology to these emerging areas offers a compelling, albeit speculative, therapeutic frontier. One of the greatest challenges in treating CNS disorders is the penetration of the blood-brain barrier (BBB). Standard water-soluble vitamin B5 relies on specific transporters that may become compromised or saturated in disease states.[41] Liposomal B5, specifically if engineered with lipid compositions that favour BBB crossing or endocytic uptake by vascular endothelial cells, could potentially restore cerebral CoA levels more effectively than conventional supplements.[41] While this potential for targeted CNS delivery remains a hypothesis that requires rigorous clinical validation, the localized deficiencies observed in Alzheimer's and Huntington's disease provide a strong clinical rationale for exploring advanced delivery systems.[41]

SAFETY, DOSING, AND TOLERABILITY

Vitamin B5 is widely regarded as one of the safest water-soluble vitamins. Because it is not stored in significant quantities and is readily excreted through the kidneys, the risk of systemic toxicity is exceptionally low.[42] Consequently, neither the Food and Nutrition Board of the Institute of Medicine nor European regulatory bodies have established a tolerable upper intake level (UL) for pantothenic acid. Clinical data indicates that high oral doses of up to 10 grams per day are generally well-tolerated in healthy adults.[42] However, excessive intake exceeding 10,000 mg/day has been associated with mild gastrointestinal distress, primarily in the form of nausea and osmotic diarrhoea.[42]

Despite its favourable safety profile, rare clinical complications have been documented in the literature. A notable case report by Debourdeau et al (2001) described a patient who developed eosinophilic pleuropericardial effusion following a high-dose regimen of 300 mg/day of biotin and 10 mg/day of pantothenic acid; however, this remains an isolated instance and is not representative of the broader clinical experience.[43] In supplemental forms like calcium pantothenate or dexpanthenol, the primary side effects are limited to the gastrointestinal tract, which are often the result of the physical presence of high-concentration powders rather than metabolic toxicity.[8]

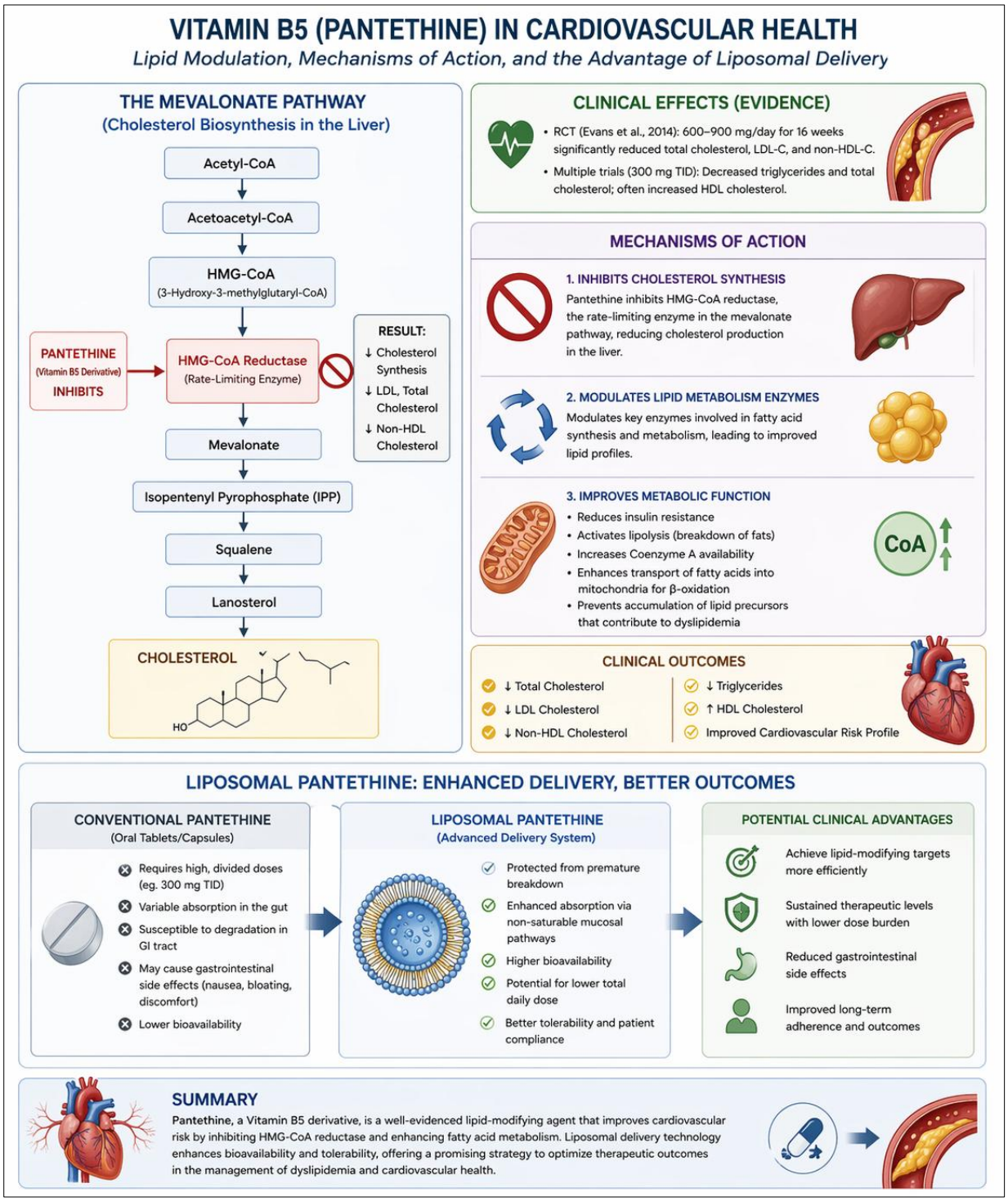

Figure 3: Infographic presentation of role of vitamin B5 in cardiovascular health.

Liposomal delivery systems are expected to significantly improve the tolerability of Vitamin B5 therapy. By encapsulating the nutrient within a phospholipid bilayer, the active compound is shielded from direct contact with the gastric mucosa, potentially eliminating the gastrointestinal upset associated with high-dose conventional tablets.[44] While standardized dosing for liposomal vitamin B5 has not yet been established by international health authorities, the enhanced bioavailability of these formulations suggests that therapeutic targets—particularly in acne and cardiovascular health—may be achieved at lower total daily dosages.[44]

Current protocols typically follow the manufacturers' guidance, often utilizing doses that align with or slightly exceed the AI to optimize cellular CoA status without exceeding the renal threshold.[44]

GAPS, LIMITATIONS, AND FUTURE RESEARCH DIRECTIONS

While the physiological rationale and preliminary clinical evidence for vitamin B5 are promising, a critical appraisal of the existing literature reveals several significant gaps that must be addressed to establish liposomal formulations as a standard of care. Most current research, such as the widely cited studies by Yang et al (2014) and Capodice et al (2012), is limited by small sample sizes and relatively short follow-up periods, which may not fully capture long-term safety or the sustainability of treatment effects.[26,27] Furthermore, several key trials have been industry-funded, highlighting a pressing need for independent, multi-centre replication to eliminate potential bias and validate the efficacy of vitamin B5 across broader, more diverse patient populations.[30]

A primary limitation in the current field of nutraceutical science is the total absence of head-to-head RCTs comparing liposomal vitamin B5 directly against conventional oral supplements. While the mechanistic advantages of liposomes—such as bypassing saturable transporters and protecting the nutrient from gastric degradation—are well-established in general pharmacology, direct pharmacokinetic data specifically for liposomal B5 is still required. Future research must prioritize human bioavailability studies to quantify the exact degree of absorption enhancement and determine whether liposomal delivery truly results in higher intracellular CoA levels compared to traditional forms.

Looking forward, research priorities should expand beyond dermatology into more complex systemic applications. There is a clear need for dose-ranging RCTs in the management of dyslipidaemia and cardiovascular risk to establish standardized liposomal protocols. Additionally, the emerging link between cerebral B5 deficiency and neurodegenerative diseases like Alzheimer's and Huntington's necessitates the development of CNS-targeted liposomal formulations. Research into liposomes engineered to cross the blood-brain barrier could open new therapeutic avenues for neuroprotection. Addressing these evidence gaps through rigorous, long-term safety studies and advanced clinical modelling will be essential for transitioning vitamin B5 from a supplemental aid to a precision-engineered metabolic therapy.

CONCLUSION

The clinical utility of vitamin B5 (pantothenic acid) and its metabolites is well-supported by an established evidentiary base, particularly in the management of acne vulgaris, wound healing, and dyslipidaemia. Randomized controlled trials have demonstrated that high-dose supplementation can significantly reduce inflammatory skin lesions and optimize lipid profiles by modulating CoA-mediated metabolic pathways. These findings underscore the vitamin's therapeutic potential as a low-risk, high-impact nutritional intervention for systemic and dermatological health.

However, the transition from clinical observation to standardized therapy is hampered by the known pharmacokinetic limitations of conventional oral vitamin B5. The reliance on saturable SMVT and the high rate of renal excretion often necessitate extremely high, divided doses to achieve therapeutic intracellular concentrations. Liposomal technology offers a sophisticated solution to these specific challenges. By encapsulating the vitamin in a biomimetic phospholipid bilayer, such as the sunflower-derived system engineered by WBCIL, the nutrient can bypass saturable pathways and resist gastric degradation, thereby significantly enhancing systemic bioavailability.

Given this robust mechanistic rationale, the adoption of liposomal vitamin B5 represents a justified and necessary progression in nutraceutical science. While current literature provides a strong foundation, there is an urgent call for dedicated clinical trials to directly compare the efficacy and safety of liposomal versus conventional formulations. For clinicians, liposomal B5 offers a promising alternative for patients who are non-responsive to traditional supplements or sensitive to high-dose gastrointestinal side effects. For researchers, it presents a new frontier in precision delivery, with the potential to address localized deficiencies in inflammatory and neurodegenerative conditions that were previously difficult to target.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Banerjee PG, Chakraborty A. Liposomal vitamin B5 (pantothenic acid): enhanced delivery, mechanisms, and clinical applications. Int J Basic Clin Pharmacol 2026;15:823-32.