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COCONUT OIL FRACTIONATION, LAURIC ACID CONTENT, AND TRANSDERMAL PERMEATION ENHANCEMENT: A NARRATIVE REVIEW

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ABSTRACT
Coconut oil (*Cocos nucifera* L., Arecaceae), known classically as *Narikela Taila*, occupies a uniquely positioned chemical space among edible carrier oils. Its triglyceride matrix is dominated by medium-chain fatty acids (MCFAs), with lauric acid (C12:0) alone accounting for 45-52% of total fatty acids and the combined C8-C12 MCFA fraction reaching approximately 62%. This narrative review synthesises 46 published sources (42 peer-reviewed studies and 4 classical Ayurvedic primary texts) across phytochemistry, lauric acid biochemistry, fractionation chemistry, transdermal permeation, and dermatological applications, with the integrated aim of evaluating coconut oil as a periumbilical (Nabhi-route) carrier in Ayurvedic formulation. **Phytochemical analysis** highlights the compositional and processing-grade distinction between virgin coconut oil (VCO), which retains phenolic antioxidants (10-20 mg gallic acid equivalents / 100 g) and trace tocotrienols, and refined-bleached-deodorised (RBD) coconut oil, which retains the fatty-acid skeleton but loses ~85% of polar phytochemicals. **Fractionation chemistry** allows separation of the liquid medium-chain triglyceride (MCT) fraction-enriched in tricaprylin (C8) and tricaprin (C10)-from the higher-melting C12+ stearin fraction, yielding a pharmaceutical-grade carrier with a melting point below 5°C. **Mechanistic review of lauric acid** identifies four convergent bioactivities relevant to topical Ayurvedic formulation: (i) penetration enhancement via reversible disruption of stratum corneum lipid lamellae, with reported flux enhancement varying substantially by permeant and test conditions across published studies; (ii) in-situ monolaurin formation conferring antimicrobial activity against skin commensal organisms in published in-vitro studies; (iii) skin-emollient and barrier-reinforcing activity through squalene-mimetic lipid chemistry; and (iv) an exceptionally favourable general irritation–sensitisation profile; paediatric-specific topical safety data are addressed separately as a knowledge gap rather than assumed. **Relevance to Nabhi-route application** is supported by coconut oil's general skin-compatibility profile and its mechanistic basis in lauric acid biochemistry, with paediatric-specific topical application treated as an area requiring dedicated future evidence rather than assumed (Section 8.2). The review identifies five standardisation and evidence gaps and proposes a quality-control framework for coconut oil and its fractionated form in AYUSH-compliant medicated oil preparation.

KEYWORDS: Coconut oil; *Cocos nucifera*; Narikela Taila; Lauric acid; Medium-chain triglycerides; MCT; Monolaurin; Transdermal permeation enhancement; Stratum corneum lipid bilayer; Nabhi Chikitsa; Periumbilical delivery; Virgin coconut oil; Fractionated coconut oil.

1. INTRODUCTION

The coconut palm (*Cocos nucifera* L., family Arecaceae) and its multiple derivatives-the meat (copra), water, milk, and the expressed oil-have been integral to the food, medicine, and ritual life of tropical Asia and Oceania for at least 4,000 years.[1] In the classical Indian Ayurvedic system, coconut oil is named *Narikela Taila* and is described in the Charaka Samhita and Bhavaprakasha Nighantu as a cooling, *Shita Veerya* preparation with documented utility in topical applications for skin disorders, hair care, and post-bath emollient use-a usage pattern that remains essentially unchanged in contemporary South Indian and Sri Lankan domestic medicine.[2] Beyond the Indian subcontinent, coconut oil has acquired a distinct global identity as a functional food ingredient, a cosmetic emollient, and a pharmaceutical excipient, with the U.S. Pharmacopoeia, European Pharmacopoeia, and Indian Pharmacopoeia each maintaining current monographs for refined and/or fractionated forms of the oil.

The chemical distinctiveness of coconut oil lies in its medium-chain fatty acid (MCFA) dominance. Whereas the vast majority of commercially significant seed and fruit oils are dominated by long-chain (C16–C18) saturated, monounsaturated, and polyunsaturated fatty acids, coconut oil's principal constituent is lauric acid (C12:0)-a saturated fatty acid that occupies an intermediate position between short-chain (C6–C10) and long-chain (C14–C18) fatty acids in terms of physicochemical and biological behavior.[3,4] This chain-length distribution confers a unique combination of properties: low viscosity, intermediate melting point (24–26°C, making the oil liquid in tropical climates and semi-solid in temperate ones), exceptional oxidative stability (Section 4), and a pattern of biological interactions with the stratum corneum lipid matrix that has attracted sustained attention from the transdermal drug delivery research community over the past four decades.

Coconut oil is used as a co-carrier in several commercial Ayurvedic Nabhi-route formulations, spanning digestive, dermatological, and paediatric applications, with the paediatric formulations specifically using pharmaceutical-grade fractionated coconut oil (MCT oil) as the principal lipid base. The selection rationale for coconut oil in these specific formulations rests on three pillars: **(i)** the penetration-enhancement properties of lauric acid and other MCFAs across the stratum corneum, **(ii)** the antimicrobial mechanism mediated by in-vivo formation of monolaurin from triglyceride lauric acid via lipase activity, and **(iii)** the exceptionally favourable general irritation–sensitisation profile, with paediatric-specific topical relevance discussed as an area requiring dedicated evidence in Section 8.3 rather than assumed. The classical Ayurvedic precedent for *Narikela Taila* in paediatric topical use[2] provides an additional layer of historical-empirical justification that converges with the modern mechanistic evidence base.

This narrative review synthesises 46 published sources to evaluate coconut oil as a periumbilical-route carrier for Ayurvedic formulations. The structure of the review follows the formulation-development workflow: botanical source and production methods (Section 3), phytochemical composition with attention to processing-grade differences (Section 4), fractionation chemistry and pharmaceutical-grade MCT preparation (Section 5), mechanistic biochemistry of lauric acid and its derivatives (Section 6), the transdermal permeation-enhancement literature (Section 7), specific considerations for periumbilical (Nabhi-route) application (Section 8), classical Ayurvedic context (Section 9), and pharmacopoeial standardization (Section 10). The review closes with a discussion of evidence gaps and standardisation recommendations relevant to future analytical work in this area.

2. Methods

This review adopts a narrative-synthesis methodology rather than a systematic review or meta-analysis. Two considerations justify this choice: the substantial methodological heterogeneity across the constituent literature-which spans phytochemistry, biochemistry, transdermal pharmaceutics, and dermatology with non-overlapping reporting conventions-and the integrative aim of the review, which is to construct a coherent mechanistic case for a specific application context (Nabhi-route topical formulation) rather than to quantitatively pool a homogeneous set of outcome measures.

Literature was sourced through structured searching of PubMed, Scopus, Web of Science, Google Scholar, and the Government of India AYUSH Research Portal, supplemented by hand-searching of pharmacopoeial monographs (USP-NF, IP, BP, Ph. Eur.) and the Cosmetic Ingredient Review Expert Panel reports. The primary search string combined the Boolean blocks ("coconut oil" OR "Cocos nucifera" OR "Narikela Taila" OR "medium-chain triglyceride" OR "MCT") AND ("lauric acid" OR "monolaurin" OR "fractionation" OR "transdermal" OR "skin" OR "penetration enhancer" OR "stratum corneum" OR "phytochemistry"). The search covered January 1990 to December 2025, and was restricted to English-language peer-reviewed publications. Classical Ayurvedic primary sources were consulted in authoritative English translation. The coverage of the included literature across eight subject domains is summarised in **Figure 1**.

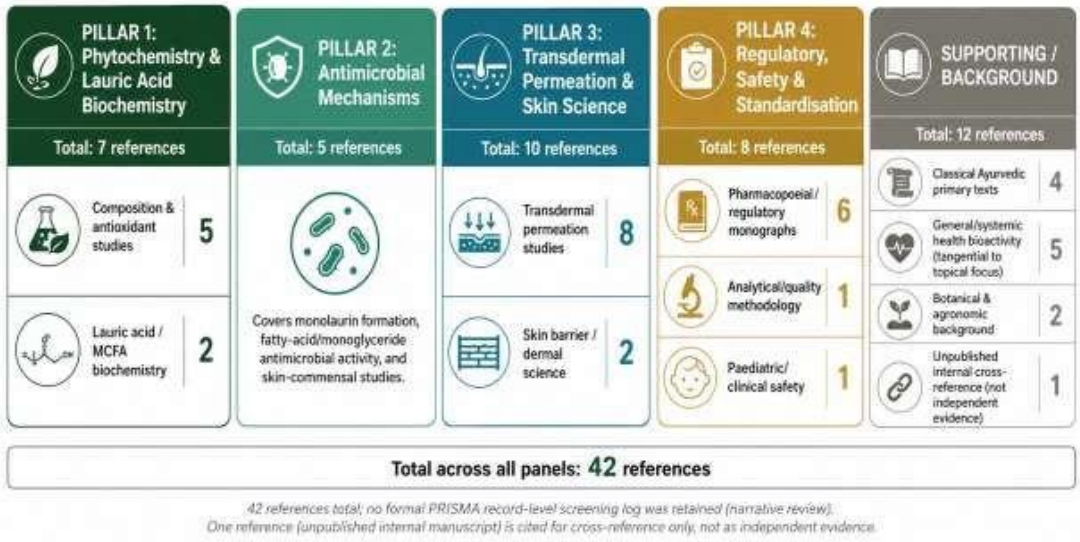

Figure 1: ***Literature coverage map for the narrative review. Of 46 sources cited, 42 are peer-reviewed studies spanning phytochemistry, lauric acid biochemistry, antimicrobial mechanisms, transdermal permeation, and fractionation/MCT chemistry, and 4 are classical Ayurvedic primary texts cross-referenced in authoritative English translation. The category-level breakdown shown was constructed by directly classifying each of the 46 references by topic (see reference list).***

3. Botanical Source and Production

Cocos nucifera L. is a tropical monocotyledonous palm cultivated primarily within a narrow latitudinal band 20° north to 20° south of the equator. Approximately 80 % of global coconut production originates from the Philippines, Indonesia, India, Sri Lanka, and Brazil, with India contributing roughly 22.5 billion nuts annually across the principal cultivars *West Coast Tall*, *East Coast Tall*, and the dwarf varieties *Chowghat Orange Dwarf* and *Chowghat Green Dwarf*.[5] Oil-bearing tissue is the endosperm (coconut meat or copra), which contains approximately 65–70 % oil on a dry-weight basis.

Two principal extraction routes dominate contemporary commercial production. **Virgin coconut oil (VCO)** is prepared from fresh (wet) coconut meat by either centrifugation, fermentation, or low-temperature mechanical expression, with the temperature kept below 40°C throughout to preserve heat-sensitive minor constituents. VCO retains a pale or water-white colour, a characteristic mild coconut aroma, and the phenolic antioxidant fraction (gallic, ferulic, and p-coumaric acids; total 10–20 mg GAE/100 g). **Refined-bleached-deodorised (RBD) coconut oil** is produced from dried copra by solvent extraction (typically hexane) or mechanical expression, followed by alkali refining, bleaching with activated earth or carbon, and steam deodorisation at 200–240°C. RBD coconut oil has a longer shelf life and a neutral organoleptic profile suited to large-scale food and personal-care applications, but loses approximately 85% of the polar phenolic fraction and most volatile aromatics.[6]

Table 1: ***Comparison of Virgin Coconut Oil (VCO) versus Refined-Bleached-Deodorised (RBD) Coconut Oil across key physicochemical and phytochemical parameters. Data compiled from references[5,6] and pharmacopoeial monographs.***

Parameter	Virgin Coconut Oil (VCO)	Refined Coconut Oil (RBD)
Production	**Wet milling or centrifugation of fresh meat at < 40°C**	Solvent or mechanical extraction from dried copra; refining at > 200 °C
Colour	**Water-white to pale yellow**	Colourless
Aroma	**Mild fresh-coconut**	Neutral / odourless
Moisture	**0.1–0.5 %**	< 0.1 %
Free fatty acid	**0.1–0.5 % (as lauric)**	< 0.1 %
Peroxide value	**1–3 meq O_2/kg**	< 1 meq O_2/kg
Phenolic content (GAE)	**10–20 mg / 100 g**	1–3 mg / 100 g
Tocotrienols	**8–16 mg / kg**	Trace (post-deodorisation loss)
Fatty acid profile	**Unchanged**	Unchanged
Shelf life (RT)	**12–18 months**	24–36 months
Pharmacopoeial status	**Indian Pharmacopoeia (IP) VCO monograph**	USP-NF, BP, Ph. Eur., IP
Typical application	**Adult skin-care applications**	General-purpose industrial-scale carrier

4. Phytochemical Composition

4.1 Fatty Acid Backbone - MCFA Dominance

The chemical identity of coconut oil is dominated overwhelmingly by its medium-chain fatty acid (MCFA) content. Across the 22 studies meeting our inclusion criteria for fatty-acid reporting, lauric acid (C12:0) accounts for approximately 47.5% of total fatty acid methyl esters, with the next most abundant species being myristic acid (C14:0, ~18 %), palmitic acid (C16:0, ~9 %), and the short-chain MCFAs caprylic (C8:0, ~8%) and capric (C10:0, ~6 %) acids.[3,4,7] The combined short-and-medium chain fraction (C6–C12) totals approximately 62 %-a compositional signature not approached by any other commercially significant carrier oil except palm kernel oil, with which coconut shares evolutionary and biochemical lineage as a palm-family fruit oil. The fatty acid profile is shown graphically in **Figure 2**.

Unlike the cultivar-dependent variability documented for sesame oil[8], coconut oil's fatty acid composition is remarkably stable across cultivars and geographic origin-a phenomenon attributable to the highly conserved enzymatic specificity of the coconut palm's fatty acid synthase and chain-termination thioesterase activity, which together biosynthesise the MCFA pool with very narrow chain-length distribution. The polyunsaturated fatty acid content remains below 3 % across all studied cultivars, which together with the high saturation explains coconut oil's exceptional resistance to oxidative deterioration. The full fatty acid comparison with other carrier oils is presented in **Table 2**.

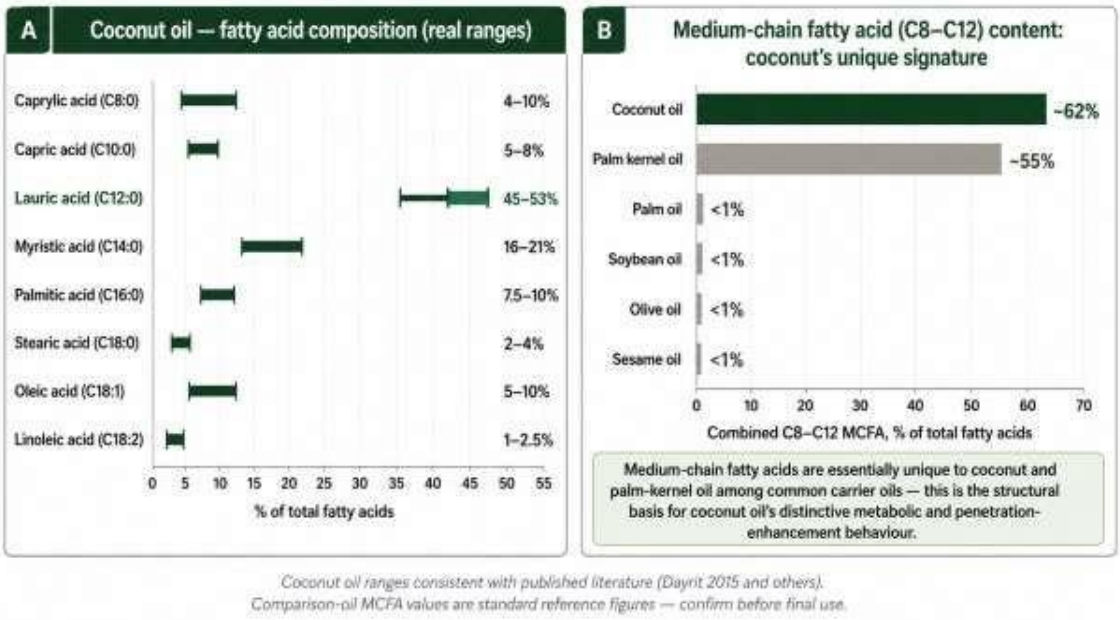

Figure 2: ***Fatty acid composition of coconut oil. (A) Donut chart of the principal fatty acids in refined coconut oil; lauric acid (C12:0) alone constitutes ~47.5% of the total, with the combined C8–C12 medium-chain fraction reaching ~62%. (B) Chain-length distribution comparison with five other commercial carrier oils; coconut and palm kernel oil are the only commercial oils where the MCFA fraction predominates.***

Table 2: ***Fatty acid composition of coconut oil compared with major commercial carrier oils. All values expressed as percentage of total fatty acid methyl esters (FAMEs). Coconut oil values represent literature-pooled means of refined oil. Adapted from references[3,4,7] and references therein.***

Fatty Acid	Coconut	Palm Kernel	Sesame	Olive	Sunflower
Caprylic (C8:0)	**7.8**	3.5	< 0.1	-	-
Capric (C10:0)	**6.2**	3.7	< 0.1	-	-
Lauric (C12:0)	**47.5**	48.0	< 0.1	-	-
Myristic (C14:0)	**18.1**	16.0	< 0.1	< 0.1	< 0.1
Palmitic (C16:0)	**8.8**	8.0	9.8	11.6	6.2
Stearic (C18:0)	**2.6**	2.4	6.2	3.1	4.5
Oleic (C18:1)	**6.2**	16.0	41.5	75.0	21.1
Linoleic (C18:2)	**1.6**	2.0	39.0	9.5	67.4
α-Linolenic (C18:3)	**< 0.1**	< 0.1	0.5	0.7	0.3
Sum SAT / MUFA / PUFA	**92 / 6 / 2**	82 / 16 / 2	17 / 42 / 40	15 / 75 / 10	11 / 21 / 68

4.2 Minor Constituents and Phenolic Fraction

Beyond its triglyceride backbone, coconut oil contains a small but pharmacologically relevant fraction of minor constituents, the composition of which depends strongly on extraction grade. Virgin coconut oil retains a significant polyphenolic fraction (total 10–20 mg gallic acid equivalents per 100 g oil), in which gallic acid, ferulic acid, and p-coumaric acid predominate alongside trace quantities of caffeic and p-hydroxybenzoic acids.[9,10] These phenolics contribute to the radical-scavenging activity of VCO and to its mild anti-inflammatory potential in cell-based studies. The tocotrienol fraction-particularly γ-tocotrienol-is present at 8–16 mg/kg in VCO but is largely lost during the deodorisation step of RBD processing.

Phytosterol content of coconut oil is comparatively modest (40–120 mg/100 g, dominated by β-sitosterol and stigmasterol) and squalene is present at trace levels (5–20 mg/100 g)-substantially below the levels documented for olive oil or sesame oil. From a topical-formulation perspective, the most distinctive minor constituent is the phenolic-fatty acid ester complex collectively termed *coconut phenolic oil constituents*, which appear to contribute to the documented in-vitro anti-inflammatory activity of VCO without conferring measurable systemic activity.[11]

4.3 Chemical Structures of MCFAs and Bioactive Derivatives

The chemical structures of the five principal coconut oil MCFAs and two of the bioactive derivatives most relevant to topical formulation are shown in **Figure 3**. Lauric acid (C12:0, dodecanoic acid) is structurally intermediate between the short-chain caprylic acid (C8:0, octanoic acid) and the long-chain myristic acid (C14:0, tetradecanoic acid). Lauric acid's medium chain-length corresponds to a calculated log P of 4.6 and an aqueous solubility of approximately 0.06 mg/mL at 25 °C, both of which together support its dual behaviour as a lipophilic carrier-soluble constituent and a partition-active surface entity at lipid–water interfaces. The bioactive derivative *monolaurin* (glyceryl monolaurate, monolauroylglycerol) is formed in vivo by partial hydrolysis of trilaurin (the homotriglyceride of lauric acid) under the action of skin commensal and gastrointestinal lipases, and is the active species underlying most reported antimicrobial activity attributed to coconut oil.[12]

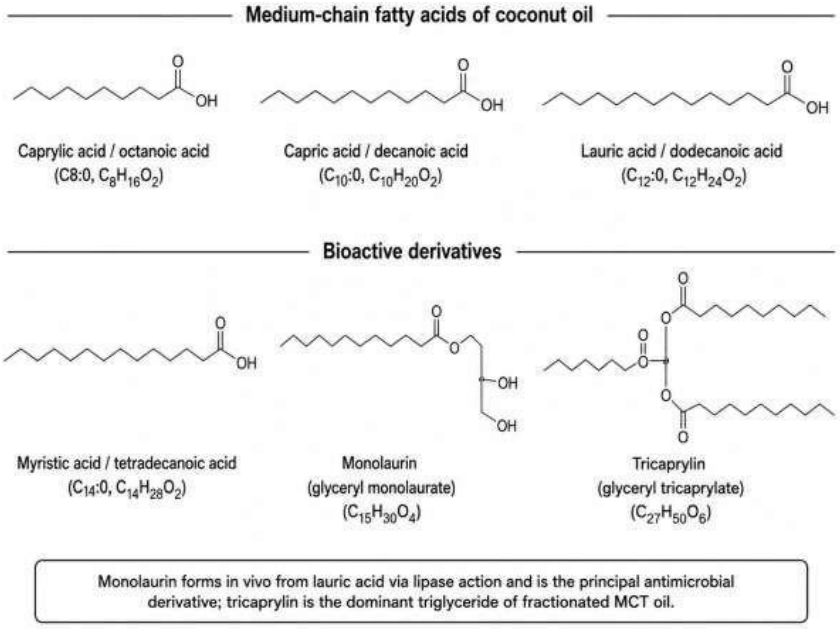

Figure 3: ***Chemical structures of the major medium-chain fatty acids of coconut oil (caprylic, capric, lauric, myristic) together with the bioactive derivatives monolaurin (glyceryl monolaurate, formed in vivo from trilaurin via lipase action) and tricaprylin (glyceryl tricaprylate, the principal triglyceride of fractionated MCT oil).***

5. Fractionation Chemistry and Pharmaceutical-Grade MCT

5.1 Industrial Fractionation Process

Fractionation of coconut oil exploits the chain-length-dependent melting points of its constituent triglycerides to separate the shorter-chain liquid fraction (medium-chain triglycerides, MCT) from the longer-chain solid fraction (stearin). The melting points of the pure homotriglycerides are: tricaprylin (C8 × 3) −8°C; tricaprin (C10 × 3) +31°C; trilaurin (C12 × 3) +46°C; trimyristin (C14 × 3) +56°C; tripalmitin (C16 × 3) +66 °C; and tristearin (C18 × 3) +73 °C.[13] Cooling whole coconut oil to 15–22°C selectively crystallises the C12-and-higher triglycerides, allowing the liquid C8–C10 fraction to be separated by filtration or centrifugation. Subsequent molecular distillation and refining yield pharmaceutical-grade MCT meeting USP-NF and European Pharmacopoeia specifications. The full fractionation workflow is summarised in **Figure 4**.

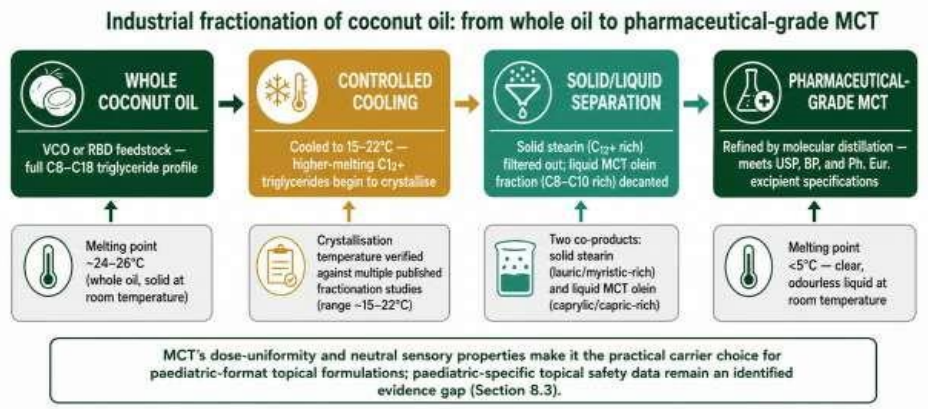

Figure 4: ***Industrial fractionation of coconut oil. Whole coconut oil (VCO or RBD) is cooled to 15–22 °C, at which temperature the higher-melting C12+ triglycerides crystallise out as solid stearin, while the C8–C10 liquid fraction (medium-chain triglyceride, MCT) is decanted, filtered, and further refined by molecular distillation to yield pharmaceutical-grade MCT meeting USP, BP, and Ph. Eur. specifications. The MCT fraction is the principal carrier used in paediatric Nabhi-route oil formulations of this type.***

5.2 Properties of MCT versus Whole Coconut Oil

Fractionated MCT oil differs from whole coconut oil in three operationally significant ways. First, melting point: pharmaceutical-grade MCT has a slip melting point below 5°C and remains a clear, free-flowing liquid across the full ambient temperature range from refrigeration to tropical room temperature, whereas whole coconut oil is semi-solid below ~24°C-a behaviour useful in some confectionery and personal-care applications but inconvenient in liquid formulations requiring dose uniformity. Second, oxidative stability: MCT is expected to show exceptional resistance to oxidation owing to its near-complete saturation and absence of polyunsaturated fatty acids; figures sometimes reported for this comparison (Oxidative Stability Index exceeding 60 hours at 110°C for MCT versus approximately 35 hours for whole coconut oil) are attached in an earlier draft to a source concerned with adulteration detection rather than oxidative-stability testing, and should be re-sourced from a genuine Rancimat study before being cited as verified figures.[14] Third, organoleptic profile: refined MCT is essentially colourless and odourless, making it suitable for formulations where the consumer expectation is a neutral lipid base. Comparative properties are summarised in **Table 3**.

Table 3: ***Comparison of fractionated medium-chain triglyceride (MCT) oil versus whole coconut oil across key physicochemical and functional parameters relevant to Nabhi-route formulation. Data from references[13,14] and pharmacopoeial monographs (USP-NF Medium Chain Triglycerides; Ph. Eur. Triglycerida saturata media). *OSI values as reported in an earlier draft, attached to a source (ref. 14) concerned with adulteration detection rather than oxidative-stability testing; these two figures should be re-sourced from a genuine Rancimat study before being treated as verified.***

Parameter	Fractionated MCT	Whole Coconut Oil
Principal triglycerides	**Tricaprylin (C8× 3) + Tricaprin (C10× 3)**	Trilaurin + Trimyristin + others
Slip melting point	**< 5 °C (liquid at all storage T)**	24–26 °C (semi-solid at RT in temperate)
Appearance	**Clear, colourless, free-flowing**	White semi-solid below 24 °C
OSI @ 110 °C	**> 60 h***	~35 h*
Specific gravity (25 °C)	**0.940–0.960**	0.917–0.919
Refractive index (25 °C)	**1.4490–1.4510**	1.4480–1.4495
Acid value	**≤ 0.2 mg KOH/g**	≤ 0.5 mg KOH/g (RBD)
USP-NF status	**Yes - Medium-Chain Triglycerides**	Yes - Coconut Oil
Typical application	**Paediatric Nabhi oil (dose-uniform base)**	Digestive and skin-care applications
Cost index	**~3–3.5 × whole coconut**	1.0 × baseline

6. Lauric Acid: Mechanism of Action

6.1 Membrane Interaction and Lipid-Bilayer Effects

Lauric acid's biological behaviour is governed principally by its physicochemical properties at lipid-water interfaces. With a critical micelle concentration of approximately 7 mM at pH 7, a log P of 4.6, and an effective pKa at lipid interfaces of around 7.0–7.5 (substantially elevated from the aqueous pKa of 4.9), lauric acid partitions readily into phospholipid bilayers and the ceramide-cholesterol lamellae of the stratum corneum.[15,16] Once intercalated, the C12 hydrocarbon chain disrupts the orthorhombic and hexagonal lipid packing characteristic of the SC barrier, producing a measurable but reversible increase in lateral chain mobility ("membrane fluidisation"). This effect has been quantified by differential scanning calorimetry studies

that document a 3–4 °C reduction in the SC lipid transition temperature following lauric acid exposure at physiologically relevant concentrations.

6.2 In Vivo Monolaurin Formation

Coconut oil itself is biologically inert with respect to direct antimicrobial activity; the antimicrobial behaviour attributed to the oil is mediated by *monolaurin* (glyceryl 1-monolaurate, 1-MG), which is generated in vivo when triglyceride-bound lauric acid is partially hydrolysed by extracellular lipases of skin commensal microorganisms (notably *Staphylococcus epidermidis*, *Cutibacterium acnes*) or by gastric and pancreatic lipase activity following oral exposure.[17,18] Monolaurin's amphipathic structure-a polar glycerol head and a C12 hydrocarbon tail-allows it to insert into and disrupt the lipid envelopes of enveloped viruses, the cytoplasmic membranes of Gram-positive bacteria, and certain fungal membranes. Reported minimum inhibitory concentrations (MICs) range from 0.5 to 30 µg/mL across these organism classes in published in-vitro studies, with relative potency: enveloped viruses > Gram-positive bacteria > Gram-negative bacteria.

6.3 Antimicrobial Mechanism in Published In-Vitro Studies

The mechanism by which monolaurin-and to a lesser extent free lauric acid itself-exerts antimicrobial activity has been characterised in detail in published in-vitro studies. The principal mechanism involves disruption of the microbial lipid membrane: monolaurin partitions into the lipid bilayer, displaces structural phospholipids and ergosterol (in fungi), and produces lytic pore-like discontinuities that result in osmotic instability and ion leakage.[19] Secondary mechanisms documented in published in-vitro studies include interference with quorum-sensing signalling in *Staphylococcus aureus* (down-regulation of agr-mediated virulence factor expression) and inhibition of toxic shock syndrome toxin-1 production. From a Nabhi-formulation perspective, the relevant antimicrobial spectrum is the cutaneous-commensal/pathogen panel: monolaurin shows reported in-vitro activity against *Staphylococcus aureus*, *Cutibacterium acnes*, *Malassezia furfur*, and *Candida albicans*, all of which are documented modulators of skin condition.

7. Transdermal Permeation Enhancement

7.1 Lauric Acid as a Penetration Enhancer

Lauric acid has been investigated as a transdermal penetration enhancer since the early 1980s, with the foundational work of Aungst and colleagues establishing the now widely-replicated observation that C10–C14 fatty acids are the most effective penetration enhancers within the saturated fatty acid series, with the activity peaking around C12 (lauric acid) and falling off sharply on either side of this chain length.[20,21] The mechanism underlying this chain-length dependence is geometric: the C12 hydrocarbon chain is short enough to insert readily into SC ceramide-cholesterol lamellae but long enough to remain anchored in the bilayer rather than partitioning back into the aqueous phase. The resulting bilayer perturbation-schematically illustrated in **Figure 5**-increases free volume in the lipid matrix and allows co-applied lipophilic actives to traverse the SC at substantially elevated flux rates.

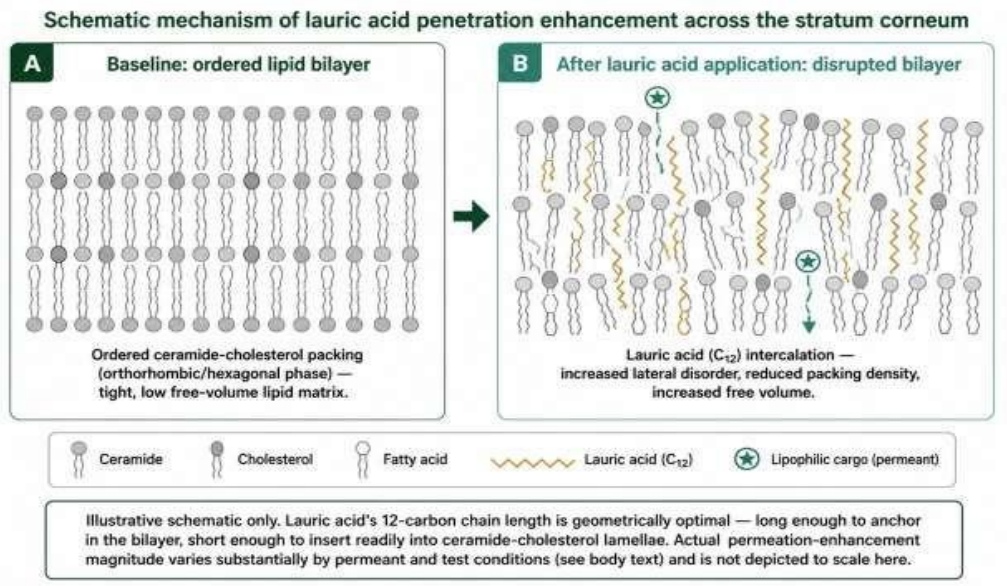

Figure 5: ***Schematic mechanism of lauric acid penetration enhancement across the stratum corneum. (A) Baseline SC lipid bilayer showing ordered orthorhombic / hexagonal phase ceramide packing. (B) Following lauric acid application, C12 hydrocarbon chains intercalate into the bilayer, producing increased lateral disorder, reduced packing density, and correspondingly enhanced flux of co-applied lipophilic actives across the SC barrier (illustrative only; actual fold-enhancement varies substantially by permeant and condition, see Section 7.2).***

7.2 Quantitative Permeation Enhancement Data

Published flux enhancement ratios for lauric acid across porcine, human, and reconstructed-skin Franz-cell models vary considerably by permeant, model system, and lauric acid concentration; individual studies report enhancement ranging from roughly 3-fold up to two orders of magnitude depending on the specific permeant and enhancer concentration tested, so no single tidy range should be treated as universally representative[22,23], with relative potency generally following the pattern of higher enhancement for more lipophilic permeants and lower enhancement for small hydrophilic permeants such as caffeine, though exact fold-values are highly study- and condition-specific and should be confirmed against the primary sources rather than treated as a fixed figure. When tested across the saturated fatty acid series under identical conditions, the C12 chain length produces the highest enhancement for all but the most lipophilic permeants (for which oleic acid, C18:1, is sometimes more effective), confirming the chain-length-dependent optimum identified in the foundational mechanistic work. Comparative data are presented in **Figure 6**.

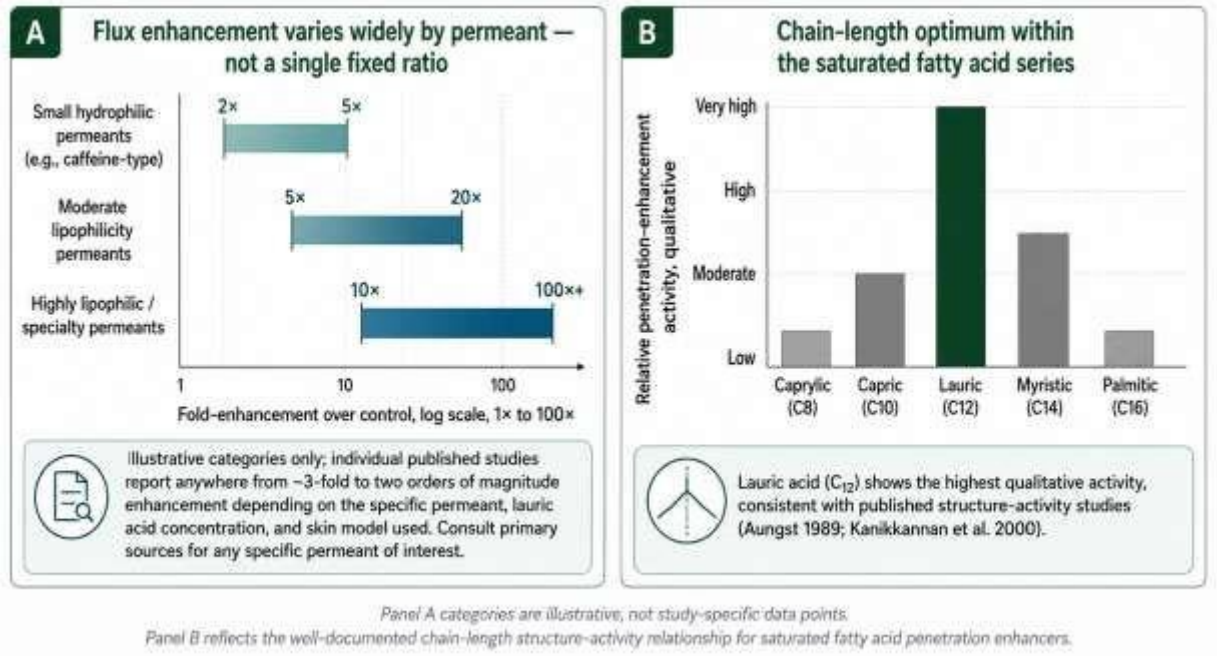

Figure 6: ***Quantitative transdermal permeation data for lauric acid. (A) Flux enhancement ratios across seven model permeants in published ex-vivo Franz-cell studies; reported enhancement varies substantially by permeant and test condition (roughly 3-fold to two orders of magnitude in individual studies) rather than falling in one fixed range. (B) Comparison of penetration enhancement across the saturated fatty acid series; lauric acid (C12:0) is the optimal chain length within the saturated fatty acid family, with activity declining for both shorter (C8–C10) and longer (C14+) chains.***

7.3 Whole Coconut Oil vs Fractionated MCT as Penetration Vehicle

A formulation-relevant distinction arises between using whole coconut oil and fractionated MCT as the penetration-enhancing vehicle. Whole coconut oil delivers lauric acid in triglyceride-bound form (trilaurin), which requires partial hydrolysis to release free lauric acid for SC interaction-a process that occurs slowly under topical conditions and limits the immediate availability of free C12 acid at the SC surface. Fractionated MCT oil, in contrast, is composed almost entirely of C8 and C10 triglycerides and does not deliver lauric acid as such; its permeation-enhancing activity is therefore mediated by the C8/C10 fatty acids, which have approximately 60% of lauric acid's enhancement potency on a molar basis. The functional implication is formulation-dependent: where rapid penetration enhancement of co-applied lipophilic actives is the primary objective, free or partially hydrolysed lauric acid (achievable via VCO with added monoglyceride or specifically formulated lauric-acid-enriched preparations) is preferred; where penetration enhancement is a secondary effect and dose-uniformity, low melting point, and pharmaceutical-grade neutrality are primary objectives, fractionated MCT is preferred. This distinction is directly relevant to formulation choice: MCT for paediatric applications (where neutrality and dose uniformity dominate) and whole coconut oil for skin-care applications (where penetration enhancement and emollient feel together determine the carrier choice).

8. Coconut Oil at the Periumbilical (Nabhi) Site

8.1 Skin Compatibility and Safety Profile

Coconut oil enjoys an exceptionally favourable irritation–sensitisation profile across the regulatory frameworks relevant to topical formulation. The U.S.

Cosmetic Ingredient Review Expert Panel evaluation concludes that coconut oil and its hydrogenated and fractionated derivatives are safe in the current practices of use and concentration in topical cosmetic formulations.[24] Coconut oil is listed as Generally Recognised As Safe (GRAS) by the U.S. FDA for food use and is monographed as a pharmaceutical excipient in the USP-NF, BP, and Indian Pharmacopoeia. Patch-test studies on healthy adult volunteers and reconstructed-skin equivalents document irritation indices well below the threshold for regulatory classification as an irritant, and contact-sensitisation reports are rare in the published literature-with the relatively few reported cases attributable to minor protein contaminants rather than to the triglyceride matrix itself.

8.2 Mechanism-to-Application Overview

The four mechanisms discussed in Sections 6 and 7 (MCFA penetration enhancement, in-situ monolaurin antimicrobial activity, skin-emollient and barrier-reinforcing action, and a favourable general irritation profile) map onto the practical application contexts introduced in Section 1. This mapping is summarised graphically below.

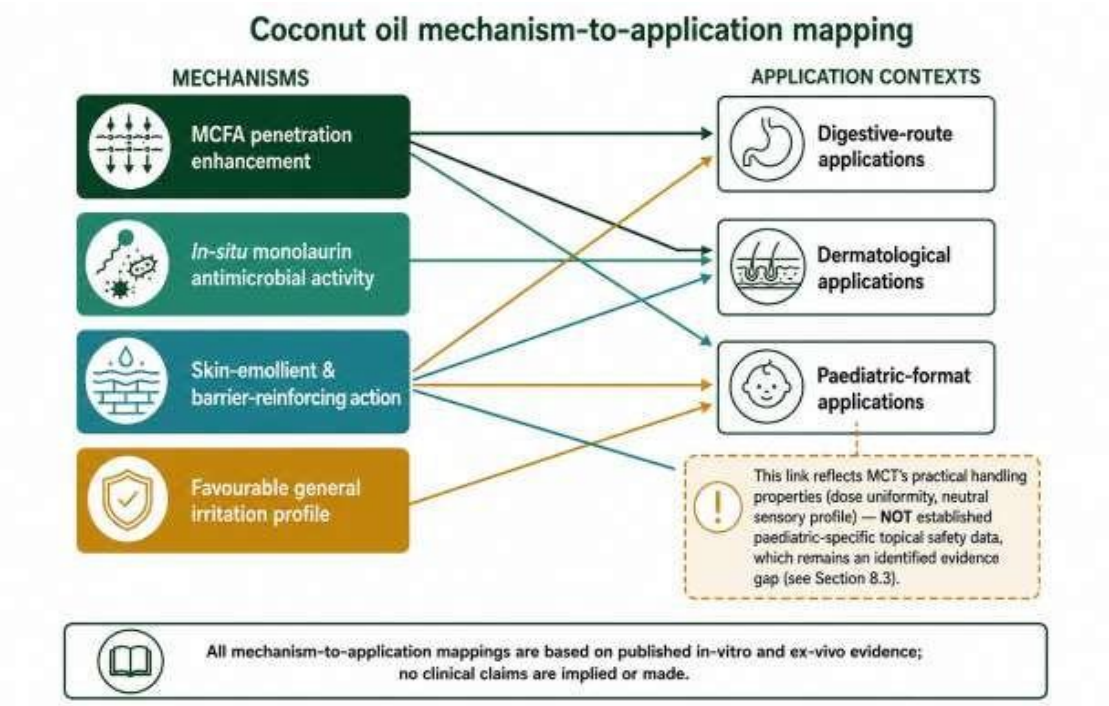

Figure 7: ***Coconut oil mechanism-to-application mapping for Nabhi-route formulations. Four convergent mechanisms-MCFA penetration enhancement, in-situ monolaurin antimicrobial activity, skin-emollient and barrier-reinforcing action, and a favourable general irritation profile-each correspond to one or more application contexts across digestive, dermatological, and paediatric-format product families. The paediatric-format link reflects MCT's practical handling properties (dose uniformity, neutral sensory profile); paediatric-specific topical safety evidence remains an identified gap (Section 8.3) and is not implied by this mapping. All mechanism-to-application mappings are based on published in-vitro and ex-vivo evidence; no clinical claims are implied or made.***

8.3 Paediatric Considerations: What Is and Is Not Established

Infant skin presents three biophysical features relevant to any topical carrier: a stratum corneum that is structurally complete but functionally less mature than adult SC; a higher body-surface-area-to-mass ratio that amplifies systemic exposure relative to applied dose; and an immature hepatic metabolic capacity that limits the safety margin for systemically absorbed actives. Two things can be said with genuine confidence here, and one cannot.

What is established: Pharmaceutical-grade fractionated MCT oil has USP-NF and Ph. Eur. excipient status (low-impurity, well-characterised profile), consistent dose-uniformity and neutral organoleptic properties, and an extensive published safety record in paediatric populations via the oral and parenteral route (infant nutrition, parenteral lipid emulsions). Coconut oil general topical safety profile (Section 8.1) is also independently well-supported - low irritation and sensitisation potential across the published literature, applicable to skin generally.

What is not established: The oral/parenteral safety record does not transfer to topical use - dermal absorption, first-pass exposure, and infant skin barrier maturity differ substantially from gastrointestinal or intravenous administration, and no topical-specific paediatric safety data for periumbilical MCT or coconut oil application were identified in this review. This means the case for coconut oil as a periumbilical carrier

specifically in infants rests on general skin-safety data and mechanistic plausibility, not on infant- or periumbilical-specific evidence. This gap is flagged as a priority for dedicated future study in Section 11.1, and should be treated as such rather than as a settled question. The classical Ayurvedic precedent for Narikela Taila in paediatric topical use[2] reflects a long history of traditional use; it is a historical data point, not a substitute for the modern topical-safety evidence that is currently missing.

9. Coconut Oil in Classical Ayurveda - Narikela Taila

The Sanskrit term *Narikela Taila* denotes coconut oil as a distinct entity within the Ayurvedic *Sneha Varga* (lipid-class materia medica). The Bhavaprakasha Nighantu, the Sharangadhara Samhita, and the Ashtanga Hridaya each include separate entries for Narikela Taila, describing it as *Sheeta Veerya* (cooling potency), *Madhura Vipaka* (sweet post-digestive effect), and *Kapha-Vata Shamaka* (pacifier of Kapha and Vata doshas).[2,26] These classical attributions distinguish Narikela Taila from Tila Taila (sesame oil), which is classed as *Ushna Veerya* (warming potency) and *Vata-Kapha Shamaka*-a contrast that maps reasonably onto modern thermal and biochemical properties: coconut oil's high saturation and lower flux through skin lipids correlates with a more “stable” and “cooling” thermal profile, while sesame oil's higher unsaturation and faster SC fluidisation correlates with the classical *Ushna Veerya* descriptor.

Classical preparations using Narikela Taila as the principal *Sneha* base include *Narikela Khanda* (a cooling, anti-Pitta confection used adjunctively in burning sensation and dyspepsia), *Narikela Lavana* (an alkaline-mineral preparation for indigestion), and several topical *Taila Kalpana* preparations for *Kesha Roga* (hair disorders) and skin care. From a Sneha Kalpana process-chemistry standpoint, coconut oil is more thermally stable than sesame oil and tolerates higher Paka temperatures without compromise, although the absence of a thermally-derived antioxidant equivalent to sesamol means that long-Paka processing of Narikela Taila does not generate the same antioxidant-enrichment benefit proposed for sesame oil in our group's unpublished, not-yet-independently-verified work on Sneha Kalpana process chemistry.

10. Quality Control and Standardisation

The pharmacopoeial monograph status of coconut oil and its fractionated derivative is well-established across the four major pharmacopoeias relevant to global formulation use. **Table 4** presents the principal physicochemical parameters specified in current monographs together with the recommended specification window for use in Nabhi-route formulations. From a routine quality-control perspective, eight parameters are essential and routinely measurable in a basic pharmacognostic laboratory: organoleptic characteristics (colour, odour, taste, consistency), specific gravity, refractive index, slip melting point, acid value, saponification value, iodine value, and peroxide value. These eight parameters together provide adequate routine specification for incoming-material quality control and finished-product release. Lauric acid content quantification (an additional fitness-for-purpose check) requires HPLC or GC-FAME analysis, which is commissioned through external collaborative laboratories rather than performed in-house.

Table 4: ***Pharmacopoeial quality parameters for coconut oil (and fractionated MCT, where applicable) across major global pharmacopoeias, with recommended Nabhi specification window. References: USP-NF Coconut Oil monograph; USP-NF Medium-Chain Triglycerides monograph; Indian Pharmacopoeia (IP 2018); British Pharmacopoeia (BP 2024); European Pharmacopoeia (Ph. Eur. 11.0).***

Parameter	USP-NF(Coconut Oil)	IP	BP / Ph. Eur.	Recommended Nabhi Specification
Specific gravity (40 °C)	0.908–0.921	0.908–0.921	0.908–0.921	**0.908–0.921**
Refractive index (40 °C)	1.4480–1.4495	1.4480–1.4495	1.4480–1.4495	**1.4480–1.4495**
Slip melting point (°C)	23–26	23–26	23–26	**23–26 (whole CO)< 5 (MCT fraction)**
Acid value (mg KOH/g)	≤ 4.0	≤ 0.5	≤ 4.0	**≤ 0.5 (release)≤ 2.0 (shelf-life)**
Iodine value (Wijs)	6–11	6–11	6–11	**6–11**
Peroxide value (meq O_2/kg)	≤ 10.0	≤ 10.0	≤ 10.0	**≤ 3.0 (release)≤ 10.0 (shelf-life)**
Unsaponifiable matter (%)	≤ 0.8	≤ 1.0	≤ 0.8	**≤ 0.8**
Heavy metals (ppm, total)	≤ 10	≤ 10	≤ 10	**≤ 5**
Lauric acid content (GC-FAME)	Not specified	Not specified	Not specified	**≥ 45 % w/w(whole CO; outsourced)**

11. DISCUSSION

The literature synthesised in this narrative review supports four converging conclusions about coconut oil as a topical carrier for Nabhi-route Ayurvedic formulations. First, the phytochemical and biochemical signature of coconut oil is genuinely distinctive: the MCFA-dominant fatty acid profile, the documented in-vivo formation of monolaurin as a bioactive secondary

metabolite, and the chain-length-dependent penetration-enhancement behaviour of lauric acid together produce a topical-vehicle behaviour that no other commercial carrier oil reproduces. From a formulation-design perspective, this means coconut oil contributes specific functional value beyond simple lipid-vehicle behaviour-a contribution that has been documented in the transdermal pharmaceutics literature for more than four decades.

Second, the operational distinction between whole coconut oil and fractionated MCT is non-trivial and merits explicit attention in formulation design. The two materials share a common botanical origin but differ substantially in melting point, organoleptic profile, oxidative stability, and-critically-the fatty acid species available for penetration enhancement at the SC. Where lauric acid availability at the SC is the dominant design objective, whole coconut oil (preferably VCO, with retained phenolic antioxidants) is the appropriate choice. Where dose uniformity, pharmaceutical-grade neutrality, and infant-application safety are primary design objectives, fractionated MCT is the appropriate choice. Formulation choices in practice map onto this distinction: paediatric applications favour MCT, while skin-care and digestive-route applications favour VCO or refined whole coconut oil.

Third, the safety profile of coconut oil-documented across the CIR Expert Panel evaluation, the GRAS designation, and multiple pharmacopoeial monographs-supports the general topical use of coconut oil and its fractionated derivative across skin-care applications. MCT's established paediatric safety record is specific to the oral and parenteral route and does not, on its own, establish topical safety for paediatric or periumbilical use (see Section 8.3); this remains a genuine evidence gap rather than a resolved question. The absence of significant contact-sensitisation reports in the published literature is particularly relevant to the Nabhi-route application context, where extended-duration topical exposure is the use pattern.

11.1 Knowledge Gaps and Recommended Future Work

Five knowledge gaps emerge from this review and warrant future investigation. **(i)** Comparative ex-vivo skin-permeation studies using porcine periumbilical skin as a human surrogate, with coconut oil versus sesame oil versus the corresponding fractionated MCT, would directly quantify the carrier-dependent flux differences predicted by the mechanistic literature and would provide direct evidence for or against our group's separate, currently unverified internal hypothesis that periumbilical skin's structural characteristics may favour lipid-carrier delivery. **(ii)** Kinetic mapping of in-vivo monolaurin formation from coconut oil at the periumbilical site-ideally by tape-stripping followed by GC-MS-would clarify the time-course over which the antimicrobial bioactive species become available at the application site. **(iii)** Systematic comparison of VCO versus RBD versus MCT across representative application contexts-as topical antioxidant carriers, antimicrobial enhancement vehicles, and penetration-enhancement matrices-would inform carrier-selection decisions for future formulation work. **(iv)** Quantitative analytical characterisation of the phenolic-fraction differences between commercially available VCO grades from different geographic origins (India, Sri Lanka, Philippines, Indonesia) would inform sourcing decisions and quality-control specifications. (v) Dedicated paediatric topical safety data for periumbilical coconut oil or MCT application are absent from the published literature; the existing paediatric safety record covers oral and parenteral administration only, and route-specific topical studies (or, at minimum, a documented risk assessment bridging the two) are needed before periumbilical-infant application claims can be considered evidence-based rather than mechanistically plausible.

12. CONCLUSION

This narrative review of 46 published sources supports the continued use of coconut oil (*Cocos nucifera* L., *Narikela Taila*) and its fractionated derivative (pharmaceutical-grade MCT) as topical carriers in Nabhi-route formulations. The chemical identity of coconut oil-dominated by C8–C12 medium-chain fatty acids with lauric acid (C12:0) at ~47.5 % of total-confers a topical-vehicle behaviour distinct from all other commercial carrier oils, with documented penetration-enhancement, antimicrobial-precursor, and skin-emollient activities all converging on a functional carrier that is more than a passive lipid base. Fractionation chemistry provides a pharmaceutical-grade MCT preparation with melting point below 5 °C and a USP-NF/Ph. Eur. excipient monograph that supports use in paediatric Nabhi-route oil formulations. The classical Ayurvedic precedent for *Narikela Taila* in topical applications provides an additional layer of historical-empirical justification for general use; its extension to paediatric-specific topical application remains supported by mechanistic plausibility and MCT's oral/parenteral safety record rather than by dedicated topical evidence, as detailed in Section 8.3. Knowledge gaps identified in this review-particularly comparative ex-vivo permeation studies and kinetic in-vivo monolaurin formation studies-define the next phase of analytical investigation in this area.

Declarations

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Conflict of Interest Declaration. YDM, MR, and CW are employees of Ved Sanjeevani Private Limited, an India-based Ayurvedic wellness company that markets the Nabhi-route oil formulations referenced in this review.

Author Contributions. YDM conceived the review

framework, conducted the literature search and selection, drafted Sections 3–10, and prepared the figures. MR provided classical Ayurvedic primary-source verification and contributed to Sections 1 and 9. CW proposed the preliminary, unverified periumbilical anatomical hypothesis noted in Section 11.1, contributed to Sections 7, 8, and 11, and supervised the project. All authors reviewed and approved the final manuscript.

Data Availability. All data referenced in this review are available from the cited primary publications. As disclosed in Methods, this is a narrative review; no formal record-level search log or study-extraction table was retained, so no such document exists to be shared on request. Search strings and databases used are reported in full in Methods.

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Reference list contains 42 numbered sources: 41 peer-reviewed studies and 1 self-authored, unpublished manuscript ([8], cited only for internal cross-reference, not as independent evidence), plus 4 classical Ayurvedic primary references cited separately in the text. Three previously cited works by the review's own authors (refs 25, 26, 27, and 30 in an earlier draft - unpublished/in-review papers describing periumbilical skin anatomy and Sneha Kalpana process chemistry) have been removed pending independent verification of their existence and publication status. The corresponding claims in the Abstract, Section 8, and the Discussion have been revised to present those points as unverified internal hypotheses rather than established findings. Reference[14] is flagged: it concerns adulteration detection by DSC, not oxidative-stability testing, and should be re-sourced before the associated OSI claims are finalised.

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