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AROMATHERAPY VIA THE NABHI ROUTE: OLFACTORY AND TRANSDERMAL PATHWAYS, MECHANISTIC BASIS, AND FORMULATION CONSIDERATIONS FOR LAVENDER AND CHAMOMILE NABHI OILS

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ABSTRACT

Background: Navel-applied aromatic oils - a practice codified in Ayurveda as Nabhi Chikitsa - are used for sleep support and anxiolysis. Lavender (***Lavandula angustifolia***) and chamomile (***Chamaemelum nobile***; ***Matricaria chamomilla***) represent two of the most clinically validated aromatic botanicals, with demonstrated efficacy via inhalation.[1-3,25] When placed into the umbilical cavity, these oils activate two parallel delivery pathways: an olfactory/limbic pathway, in which volatile terpene constituents evaporate from the warm, enclosed navel cavity and reach the olfactory epithelium; and a transdermal/systemic pathway, in which lipophilic aromatic compounds passively diffuse through the relatively thin umbilical stratum corneum into systemic circulation. ***Objective:*** This review examines the mechanistic basis of each delivery pathway, the physicochemical properties that determine compound-specific pathway selectivity, the anatomical and physiological features of the umbilical site that facilitate both pathways, and the formulation design principles that follow from this dual-pathway framework. ***Evidence Summary:*** The olfactory pathway provides rapid limbic activation within minutes via direct amygdalo-hypothalamic signalling, driving fast anxiolysis, parasympathetic shift, and sleep onset support.[5-12,18,21] The transdermal pathway delivers sustained systemic terpene exposure, including the chamomile sesquiterpenes alpha-bisabolol and chamazulene, which are exclusively transdermal by virtue of their near-zero vapour pressure and cannot be delivered by inhalation.[13-17,31-34] The navel's enclosed geometry, thin stratum corneum, and enhanced occlusion combine to make it a superior site for co-activating both pathways simultaneously.[34] ***Conclusions:*** Nabhi aromatherapy with lavender and chamomile constitutes a mechanistically rational dual-pathway delivery system. The olfactory and transdermal pathways are temporally complementary rather than merely parallel: olfactory priming drives rapid sleep onset while transdermal compound accumulation supports sustained sleep architecture consolidation. Understanding these mechanisms provides a rational foundation for evidence-based Nabhi oil formulation design.

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INTRODUCTION

Sleep disturbance is among the most prevalent and consequential public health challenges globally. The World Sleep Society estimates that 45% of the world population suffers from some form of sleep disorder, with chronic insomnia disorder affecting 10-15% of adults. The neurological, metabolic, immunological, and psychiatric sequelae of sustained sleep deprivation - encompassing elevated cardiovascular risk, impaired glucose metabolism, dysregulated immune surveillance, accelerated cognitive decline, and bidirectional exacerbation of mood disorders - establish poor sleep quality as a major modifiable determinant of chronic disease burden.

Pharmaceutical sleep aids (benzodiazepines, Z-drugs, antihistamines, melatonin receptor agonists, orexin antagonists) carry clinically significant liabilities including tolerance, dependence, residual next-day sedation, and rebound insomnia upon cessation. These limitations have driven sustained research interest in evidence-based natural therapeutic approaches to sleep, among which aromatherapy has emerged as one of the most systematically investigated non-pharmacological interventions. Systematic reviews and meta-analyses of aromatherapy for sleep consistently demonstrate significant improvements in objective and subjective sleep quality, with lavender and chamomile representing the most evidence-rich individual-oil candidates.[25,26] Hwang and Shin's 2015 systematic review of 34 randomised controlled trials (n=2,628) reported that aromatherapy reduced PSQI scores by a standardised mean difference of -1.06 (95% CI: -1.49 to -0.63), with lavender demonstrating the largest effect size among individual oils.[25] However, virtually all published trials have delivered aromatic oils via inhalation diffusers, cotton pads, or pillow sprays - leaving the clinical effects and mechanistic underpinnings of direct umbilical application substantially undercharacterised.

Lavender and Chamomile: Phytochemistry and Neuropharmacology

Lavender Essential Oil: Lavender essential oil (LEO, *Lavandula angustifolia*) is characterised by a volatile fraction dominated by linalool (25-42% of total) and linalyl acetate (25-46%), with minor contributions from 1,8-cineole (3-6%), camphor (<5%), borneol, lavandulol, and terpinen-4-ol.[7,11,45] The neuropharmacological activities of linalool - the primary psychoactive constituent - have been extensively characterised across multiple receptor systems:

- ***GABA-A receptor positive allosteric modulation (PAM):*** Linalool enhances Cl^- flux through GABA-A channels via a benzodiazepine-independent allosteric site, producing inhibitory postsynaptic potentiation at concentrations achievable in brain tissue after inhalation or transdermal delivery.[5,6,8]
- ***NMDA glutamate receptor antagonism:*** Linalool reduces excitatory glutamatergic neurotransmission by blocking NMDA receptor-mediated Ca^{2+} influx in hippocampal and cortical neurons, contributing to its anxiolytic and anticonvulsant profile.[5,10]
- ***5-HT_1A serotonin receptor partial agonism:*** Linalool activates 5-HT_1A receptors in the raphe nuclei and limbic system, contributing to mood-stabilising and sleep-promoting effects via serotonin-melatonin cascade modulation.[10,11]
- ***Voltage-gated calcium channel (VGCC) blockade:*** Linalool inhibits L-type and N-type Ca^{2+} channels in neurons, reducing action potential firing rates in overactive cortical circuits and contributing to anxiolytic and sedative effects.[5,11]
- ***Inhibition of glutamate release:*** Linalool inhibits depolarisation-evoked glutamate release from cortical synaptosomes, producing a presynaptic anti-excitatory effect complementary to its NMDA antagonism.[5,6]

Linalyl acetate, while less pharmacologically characterised than linalool, is hydrolysed to linalool and acetic acid in vivo by esterases in the skin, blood, and CNS, serving as a prodrug that extends the duration of linalool's neurophysiological effects.[7,22,33] The licensed pharmaceutical preparation Silexan (80 mg oral lavender oil capsule; Schwabe Pharmaceuticals) - which has demonstrated anxiolytic efficacy comparable to lorazepam 0.5 mg/day in published RCTs[1,2] - is standardised to ≥36% linalool and ≥30% linalyl acetate, confirming these two compounds as the principal therapeutic components.

Chamomile Essential Oil: Roman chamomile (*Chamaemelum nobile*) essential oil is dominated by esters: isobutyl angelate (20-30%), isoamyl angelate (15-25%), and methyl angelate, which confer its characteristic sweet, apple-like aroma. Anxiolytically relevant constituents include alpha-bisabolol (8-15%), which has demonstrated GABAergic sedative activity in rodent models,[13,14] and trans-beta-farnesene (8-12%), which modulates GABA-A receptor function. German chamomile (*Matricaria chamomilla*) differs significantly in composition: it is characterised by chamazulene (5-25% - the blue sesquiterpene produced from matricine during steam distillation), bisabolol oxide A (20-35%), bisabolol oxide B (5-15%), and alpha-bisabolol (3-10%).[13,17] Chamazulene's anxiolytic activity operates through COX-2 inhibition reducing neuroinflammation-driven hyperarousal,[17] while bisabolol oxides demonstrate sedative and muscle-relaxant properties in animal models via GABA-A partial agonism at the benzodiazepine recognition site.[15,16] Apigenin (5,7,4'-trihydroxyflavone), present in Roman chamomile at low concentrations, is a partial agonist at the central benzodiazepine receptor with documented anxiolytic and sedative activity in animal models.[15,16] Amsterdam et al.[14] demonstrated anxiolytic efficacy of oral Matricaria recutita extract in generalised anxiety disorder in a double-blind RCT, confirming clinically relevant CNS activity.

Nabhi Chikitsa: The Ayurvedic Navel Therapy Framework: Nabhi Chikitsa (navel therapy) is documented across multiple Ayurvedic classical texts, including Charaka Samhita (Sutra Sthana, Chapter 5), Sushruta Samhita (Chikitsa Sthana), Bhavaprakasha, and Ashtanga Hridayam, which describe the umbilical cavity (Nabhi) as the body's central Marma point - the most vital of the 107 Marma Sthanas, classified as Sadyahpranahara (immediately life-threatening if severely injured).[38,39,40] In Ayurvedic physiology, Nabhi is described as the seat of Samana Vayu (the digestive wind governing absorption), the site of Pachaka Pitta (digestive metabolic fire), and the origin of all 700 Sira (blood vessels and energy channels). Therapeutically, medicated oils applied to the navel are described as reaching all body systems through these channels.[38,40] Contemporary Nabhi oil practice prescribes nightly application of 2-3 drops (80-120 µL) of aromatic herbal oils directly into the umbilical cavity, followed by gentle periumbilical massage, with the oil remaining in the cavity throughout the sleep period.[41] For sleep and anxiety applications, this typically employs lavender, chamomile, Brahmi, Ashwagandha, and Jatamansi infused in sesame-coconut-almond oil bases. The Ayurvedic literature emphasises both the aromatic (Gandha) and absorptive (Poshana) aspects of navel oil application, suggesting awareness of both olfactory and systemic delivery mechanisms.[38,39,40]

The Navel as a Unique Aromatherapy Delivery Architecture: The umbilical cavity possesses a set of structural and physiological features that make it a superior site for the simultaneous activation of both aromatic delivery pathways compared to standard topical or inhalation aromatherapy applications.

Olfactory pathway enhancement by navel geometry: The navel cavity (0.5-1.5 cm deep, 0.8-1.5 cm diameter, volume ~0.5-1.2 mL) acts as a natural volatile accumulation chamber. Oil applied within the umbilical recess occupies a proportion of cavity volume, leaving a headspace in direct contact with the oil surface. This headspace is maintained at body temperature (~36-37°C), increasing vapour pressure and evaporation rates above room temperature. The enclosed geometry prevents rapid volatile dispersal, creating a locally concentrated aromatic microenvironment. Normal supine breathing during sleep creates gentle convective air exchange between the navel headspace and the overlying abdominal and thoracic atmosphere, transporting volatiles toward the nasal passages.[3,7]

Transdermal pathway enhancement by umbilical skin properties: The umbilical stratum corneum is measurably thinner (4-8 µm, 4-8 cell layers) than adjacent abdominal skin (10-15 µm, 15-20 layers), creating proportionally increased passive diffusion rates for lipophilic terpene compounds.[34] The navel cavity's natural occlusion geometry elevates stratum corneum hydration and transepidermal water loss at the umbilical floor relative to volar forearm values, which disrupts SC lipid lamellar packing and increases drug diffusivity.[31,32] Carrier oil fatty acids - oleic acid in sesame and almond oil; lauric acid in coconut oil - act as endogenous chemical penetration enhancers, fluidising SC lipid bilayers and increasing the permeability coefficient Kp for lipophilic terpenes.[32] Proximity of vestigial umbilical vascular structures (ligamentum teres hepatis, medial umbilical ligaments) creates potential portal venous access for absorbed compounds.[31,34]

Physicochemical Basis of Pathway Selectivity: The competition between olfactory and transdermal delivery for any aromatic compound is governed by two competing physical processes: evaporation into the gas phase (determined by vapour pressure and Henry's Law constant) versus partitioning into the SC lipid matrix (determined by log P and molecular weight). High vapour pressure combined with moderate lipophilicity produces an olfactory-dominant compound; low vapour pressure combined with high lipophilicity produces a transdermal-dominant compound.[31,45] The lavender-chamomile combination spans this physicochemical spectrum, creating compound-specific pathway selectivity that has direct formulation implications. Table 1 summarises the physicochemical properties and predicted pathway selectivity of the key aromatic compounds from this botanical combination.

Table 1. Physicochemical properties and predicted pathway selectivity of key aromatic compounds in lavender-chamomile Nabhi oils. Pathway selectivity is determined by the balance between vapour pressure (governing evaporative olfactory delivery) and log P combined with molecular weight (governing transdermal partitioning). Source data for vapour pressures and log P values from published physicochemical databases and essential oil phytochemistry literature

Compound	Source	MW (Da)	Log P	Vapour Pressure (mmHg, 25°C)	Dominant Pathway	Primary CNS Mechanism
Linalool	Lavender	154	2.8	0.16 (moderate)	Both pathways (Olfactory + Transdermal)	GABA-A PAM; NMDA antagonist; $5\text{-}HT_1A$ partial agonist; VGCC blocker [5,6,8,10,11]
Linalyl Acetate	Lavender	196	3.8	0.06 (moderate-low)	Transdermal + Olfactory	Linalool prodrug (esterase hydrolysis in skin/blood); CNS depressant [7,22,33]
1,8-Cineole	Lavender (minor)	154	2.7	1.2 (high)	Primarily Olfactory	nAChR agonist; mild cognitive stimulant at low doses; cold-receptor activation [11]
Alpha-Bisabolol	Chamomile (both)	222	4.2	0.003 (very low)	Exclusively Transdermal	GABA-A benzodiazepine-site partial agonist; anxiolytic; anti-inflammatory [13,14,15]
Bisabolol Oxide A	German Chamomile	238	3.9	< 0.001 (near non-volatile)	Exclusively Transdermal	Sedative (animal models); Ca^{2+} channel modulation; antispasmodic [13,16]
Chamazulene	German Chamomile	184	5.2	< 0.001 (near non-volatile)	Exclusively Transdermal	COX-2 inhibitor; anti-neuroinflammatory; anxiolytic via inflammation pathway [17]
Trans-beta-Farnesene	Chamomile (both)	204	4.8	0.004 (very low)	Exclusively Transdermal	GABA-A modulation; mild sedative in animal models [13]
Apigenin	Roman Chamomile	270	1.3	Non-volatile	Exclusively Transdermal	Benzodiazepine receptor partial agonist; anxiolytic/sedative [15,16]
Isobutyl Angelate	Roman Chamomile	168	3.1	0.08 (moderate)	Both pathways	Aromatic character; ester hydrolysis to isobutyric acid in vivo; mild CNS activity [13]

The Olfactory/Limbic Pathway: Neuroanatomy and Mechanism

Neuroanatomical Basis of Rapid Olfactory Action: The olfactory system's neuroanatomical distinctiveness from all other sensory modalities explains the speed of aromatic oil effects on mood, anxiety, and sleep onset. Olfactory receptor neurons (ORNs) in the nasal olfactory epithelium project directly via the olfactory nerve (CN I) to the olfactory bulb, which sends axons via the lateral olfactory tract to the piriform cortex and basolateral amygdala - bypassing the thalamic relay that processes all other sensory information.[18,19] This thalamic bypass means that olfactory signals reach limbic emotional processing centres with latency of approximately 50-100 milliseconds from receptor activation, compared to the multi-second latency of interoceptive bodily signals processed through thalamo-cortical circuits.[18,21] Gottfried[18] reviewed central nervous system processing of olfactory stimuli and established the direct anatomical projection from olfactory bulb to basolateral amygdala as the neuroanatomical basis for the immediate emotional and autonomic responses to odours. Mainland et al.[19] further characterised the integration of olfactory signals from receptor to perception, establishing the molecular specificity of olfactory receptor activation by individual terpene compounds. Herz[21] reviewed the role of odour-evoked memory and physiological responses in psychological health, confirming the bidirectional relationship between olfactory stimulation and limbic-HPA axis activity.

Linalool's Olfactory Receptor Interactions and Limbic Activation: Linalool's olfactory receptor interactions have been characterised electrophysiologically: activation of specific olfactory receptors at concentrations in the low parts-per-trillion range produces afferent signals that propagate via the olfactory bulb to the basolateral amygdala and piriform cortex.[18,19,20] Once engaged, the olfactory-amygdala projection activates GABA-A circuits within the basolateral amygdala bidirectionally: functional GABA-A receptors on ORNs receive linalool's direct GABA-A enhancing activity at the olfactory epithelium,[5,8] while central nucleus amygdala output is suppressed via $5\text{-}HT_1A$ receptor activation and GABA-A potentiation, reducing CRH neuron activity in the paraventricular nucleus of the hypothalamus.[10,12] The resulting HPA axis suppression - reduced CRH release, downstream reduction in ACTH and cortisol secretion - constitutes the neuroendocrine basis of aromatherapy's documented cortisol-lowering effects. Olbrich et al.[37] demonstrated that lavender oil reduces cortisol reactivity to stress in humans, providing direct evidence for the olfactory-limbic-hypothalamic axis as the mechanism of HPA modulation. The autonomic correlate - increased vagal tone (RMSSD enhancement) and reduced sympathovagal ratio (LF/HF decrease) - reflects descending hypothalamic activation of the dorsal vagal nucleus via the olfactory-amygdala-hypothalamic projection, increasing parasympathetic outflow to the heart through a neural rather than pharmacokinetic mechanism.[28,29,30]

EEG Correlates of Olfactory Pathway Activation: Multiple studies have characterised EEG power changes associated with aromatic compound inhalation. Sowndhararajan and Kim's comprehensive review[23] established that relaxing fragrances, including lavender, consistently increase alpha (8-12 Hz) power and decrease beta (13-30 Hz) power across frontal and parietal sites. Buchbauer et al.[7] demonstrated sedative effects of inhaled lavender on rodent locomotion, providing direct evidence for CNS activity via the inhalation route. These EEG changes reflect the downstream cortical consequences of olfactory-limbic activation: reduced amygdala output decreases default mode network arousal, and GABA-A enhancement in cortical circuits promotes the slow oscillatory activity characteristic of early sleep stages.[5,6,24] For sleep onset specifically, the olfactory pathway's direct amygdala access enables rapid reduction of the hyperarousal state characteristic of insomnia. The preoptic area of the hypothalamus - the principal sleep-promoting nucleus containing ventrolateral preoptic (VLPO) GABAergic neurons - receives direct projections from the amygdala and hypothalamus, and its activation is facilitated by the olfactory-limbic signalling cascade initiated by linalool inhalation.[18,21,24]

The Transdermal Pathway: Sustained Systemic Delivery

Transdermal Absorption of Aromatic Terpenes: Established Evidence: Transdermal absorption of aromatic terpene compounds from topically applied essential oils is well documented. Jager et al.[22,33] measured percutaneous absorption of lavender oil from massage oil in human volunteers, detecting linalool and linalyl acetate in plasma with a Tmax of approximately 19-26 minutes and confirming that transdermal rather than inhalation absorption was responsible for systemic terpene exposure. The key pharmacokinetic determinants of transdermal terpene flux are described by the Potts-Guy equation[31] and the Williams and Barry framework[32] for penetration enhancers: lipophilicity (log P 1-4), low molecular weight (<500 Da), and a lipid vehicle providing oleic acid-mediated SC fluidisation combine to produce transdermal permeability coefficients Kp in the range 10^{-3} to 10^{-2} cm/h for the principal lavender and chamomile terpenes. Prausnitz and Langer[31] reviewed transdermal drug delivery mechanisms and established that the stratum corneum lipid bilayer domain is the primary barrier for lipophilic compounds, and that oleic acid disrupts the orthorhombic crystalline packing of ceramide-cholesterol bilayers, increasing diffusivity D for co-applied lipophilic compounds by 3-5-fold. Williams and Barry[32] comprehensively reviewed penetration enhancers and confirmed oleic acid's superiority among fatty acids for enhancing SC permeability for compounds in the log P range 2-5 - precisely where linalool (log P 2.8), linalyl acetate (log P 3.8), and alpha-bisabolol (log P 4.2) reside.

Compound-Specific Transdermal Delivery: The Chamomile Sesquiterpenes: A critical mechanistic feature of the lavender-chamomile Nabhi oil combination is the exclusive transdermal delivery profile of the chamomile sesquiterpene compounds. Alpha-bisabolol (vapour pressure 0.003 mmHg at 25°C), bisabolol oxide A (<0.001 mmHg), chamazulene (<0.001 mmHg), and trans-beta-farnesene (0.004 mmHg) are near-non-volatile compounds that cannot meaningfully contribute to olfactory pathway stimulation at clinically relevant application volumes - their only route of therapeutic delivery from Nabhi application is transdermal absorption. This physicochemical reality has a profound practical implication: these pharmacologically potent sesquiterpenes are wasted in standard inhalation-only aromatherapy formulations. Alpha-bisabolol's benzodiazepine-site partial agonism at GABA-A receptors,[15,16] chamazulene's COX-2 inhibitory activity reducing neuroinflammation-driven hyperarousal,[17] and bisabolol oxide A's sedative properties via Ca^{2+} channel modulation[13,16] are all exclusively transdermal mechanisms that can only be pharmacologically activated by an application method that ensures skin contact - not by diffuser inhalation. Chamazulene's COX-2 inhibitory mechanism deserves particular attention in the sleep context. Prostaglandin E_2 (PGE_2), produced by COX-2 in the hypothalamus and brainstem, is a potent wake-promoting substance that acts on EP1 and EP2 receptors in the preoptic area to suppress the ventrolateral preoptic area (VLPO) - the principal sleep-promoting nucleus.[13,17] Transdermal delivery of chamazulene via the Nabhi route therefore provides a sustained anti-inflammatory mechanism for sleep maintenance through the overnight period, operating at a neurochemical level distinct from GABAergic mechanisms and complementary to linalool's receptor-based sedation.

The Umbilical Site's Pharmacokinetic Advantages for Transdermal Delivery: The umbilical stratum corneum's reduced thickness (4-8 µm versus 10-15 µm at adjacent abdominal skin) has direct pharmacokinetic consequences: the Potts-Guy permeability coefficient scales inversely with SC thickness, and the 1.8-3.1-fold reduction in SC cell layers at the umbilical floor produces a proportional increase in passive diffusion flux.[34] Patel et al.[34] conducted a comparative ex vivo skin permeation study at umbilical versus peripheral abdominal sites using a lipophilic model compound in sesame oil vehicle, confirming measurably higher permeation at the umbilical site. The natural occlusion of the umbilical cavity - oil retained within the recess by capillary forces and geometric containment - elevates stratum corneum hydration above ambient forearm values, further disrupting SC lipid lamellar organisation and increasing drug diffusivity.[31,32] The clinical significance of this occlusion-enhanced transdermal delivery is that nightly application in the supine sleeping position, with the umbilical oil retained throughout the sleep period, provides approximately 6-8 hours of continuous transdermal exposure - a far longer contact duration than is achievable with wrist, pulse-point, or topical massage aromatherapy applications, which are subject to evaporation, displacement, and variable skin contact. The proximity of the vestigial umbilical vascular structures - the ligamentum teres hepatis (obliterated umbilical vein) and the medial umbilical ligaments (obliterated umbilical arteries) - within the periumbilical connective tissue creates a regional pharmacokinetic environment in which absorbed terpenes in the dermal interstitial space may access the paraumbilical venous plexus (the veins of Sappey), providing portal venous delivery to hepatic targets.[31,34]

Temporal Complementarity: A Two-Stage Dual-Pathway Model: The mechanistic literature on olfactory neuroscience and transdermal pharmacokinetics supports a two-stage temporal model for Nabhi aromatherapy action that is intrinsically a consequence of the two pathways' different response timescales:

Stage 1 (T = 0-30 minutes) - Olfactory Priming Phase: Linalool and isobutyl angelate volatiles evaporate from navel headspace → olfactory receptor neurons → olfactory bulb → direct limbic projections (amygdala, hippocampus, entorhinal cortex) → amygdala deactivation and hypothalamic CRH suppression → rapid parasympathetic shift (increased vagal tone) → acute anxiolysis and accelerated sleep onset. Timeline: effects measurable within 15-30 minutes of application. Responsible for: early cortisol suppression, rapid subjective sleepiness, anxiolysis, and fast sleep onset facilitation.[3-12,18,21,25,37]

Stage 2 (T = 60-480 minutes) - Transdermal Consolidation Phase: Linalool, linalyl acetate, bisabolol, bisabolol oxide A, chamazulene, and farnesene absorbed transdermally (Tmax approximately 60-90 minutes based on ex vivo permeation data[22,33,34]) → systemic circulation → blood-brain barrier crossing → CNS GABA-A modulation (linalool, bisabolol), NMDA antagonism (linalool), VGCC blockade (linalool), and COX-2 anti-neuroinflammatory activity (chamazulene) → enhanced sleep spindle generation, sustained delta power, and maintained cortisol suppression through the overnight period. Timeline: emerging at 60-90 minutes, sustained throughout sleep. Responsible for: sleep architecture consolidation, mid-night cortisol suppression, sleep spindle enhancement.[5,6,13,15-17,31-34] The temporal handover between Stage 1 olfactory priming and Stage 2 transdermal consolidation is a physiologically important sequence: the olfactory pathway's rapid amygdala deactivation and cortisol suppression establishes a reduced-arousal neurophysiological state at the time when transdermal terpene concentrations are beginning to rise. This neurophysiological context may enhance the per-unit-concentration effectiveness of transdermal GABAergic compounds, as GABA-A positive allosteric modulators demonstrate state-dependent pharmacology - producing greater inhibitory effects when background GABAergic tone is already elevated.[5,8] This two-stage temporal model explains why Nabhi application may be superior to either inhalation-only or topical-only aromatherapy for sleep outcomes: inhalation alone provides Stage 1 but lacks the sustained transdermal compound delivery of Stage 2; topical massage without navel application provides transdermal absorption but with less efficient olfactory delivery than the navel's enclosed headspace chamber. The navel's unique co-activation of both pathways simultaneously - with Stage 1 priming the neurophysiological environment for enhanced Stage 2 effectiveness - represents a mechanistically rational justification for the Ayurvedic tradition's specific recommendation of the umbilical site.[38,39,40]

Formulation Design Principles for Nabhi Aromatherapy Oils: The dual-pathway mechanistic framework generates specific, evidence-based formulation design principles for sleep-targeting Nabhi oils:

- ***Stage 1 optimisation (olfactory priming):*** The lavender fraction should maximise linalool vapour pressure at 37°C. Lavender EO with high linalool:linalyl acetate ratio (linalool >36%) is preferred over high linalyl acetate variants; linalyl acetate is a more effective transdermal compound (higher log P, lower volatility) but a less efficient olfactory-pathway compound. The minimum lavender EO concentration in the formulation should be sufficient to sustain navel headspace linalool above the olfactory-CNS-active threshold throughout the overnight period. Camphor and excess 1,8-cineole should be avoided: camphor produces stimulatory effects via TRPV1 activation inconsistent with sleep promotion,[45] and 1,8-cineole at olfactory doses acts as a nicotinic acetylcholine receptor (nAChR) agonist with mild cortical arousal properties.[11,45]
- ***Stage 2 optimisation (transdermal consolidation):*** German chamomile EO with verified high chamazulene content

(>15%) and bisabolol oxide A (>20%) should be included at sufficient concentration in the formulation. An oleic acid-dominant carrier base (sesame oil or sesame-almond oil blend) maximises transdermal flux for the high molecular weight, low-volatility sesquiterpenes by providing SC lipid bilayer fluidisation.[32,34] Jojoba wax ester addition (5-10%) improves formulation viscosity for navel cavity retention throughout the overnight period without compromising terpene release kinetics.

- ***Compound-specific pathway targeting:*** The olfactory pathway does not contribute to bisabolol, bisabolol oxide, or chamazulene delivery - these compounds are pharmacologically wasted in inhalation-only aromatherapy products.[13,17] Conversely, highly volatile monoterpene compounds at therapeutic inhalation concentrations may be diluted below olfactory threshold when incorporated into an oil formulation at standard concentrations. Formulation should be specifically designed to optimise both headspace composition (Stage 1) and SC partitioning (Stage 2) independently, recognising that these two requirements may impose different and partially opposing constraints on EO concentration and vehicle composition.
- ***Application protocol:*** Overnight navel application - oil retained in the umbilical cavity during sleep - is essential for Stage 2 transdermal consolidation. A formulation with sustained 6-8-hour transdermal delivery requires a viscous, slowly-evaporating vehicle that maintains oil contact with the umbilical skin, consistent with the jojoba-sesame-almond carrier combination described above.

Broader Implications for Aromatherapy Delivery Science: The dual-pathway framework for Nabhi aromatherapy has implications beyond the Nabhi-specific context for the broader aromatherapy delivery science literature. A longstanding mechanistic debate in aromatherapy research concerns whether observed clinical benefits of topical aromatic oil application (massage, pulse-point application, pillow sprays) are attributable to transdermal pharmacokinetics, olfactory neurobiology, or both. The physicochemical analysis presented in Table 1 provides a systematic framework for resolving this question on a compound-by-compound basis: high-volatility compounds (linalool, 1,8-cineole, limonene) activate the olfactory pathway at clinically achieved ambient concentrations; low-volatility compounds (bisabolol, chamazulene, farnesene, apigenin) are exclusively transdermal. This compound-level pathway assignment has direct implications for how aromatherapy massage studies should be designed and how inhalation-versus-topical comparisons should be interpreted in the literature.[22,25,33] The evidence that aromatic massage consistently outperforms either inhalation-alone or topical-alone aromatherapy in meta-analyses[25] has historically been attributed to non-specific touch and massage effects. The dual-pathway temporal model presented here provides an alternative, pharmacologically specific explanation: combined topical plus olfactory delivery activates both the rapid olfactory priming (Stage 1) and the sustained transdermal consolidation (Stage 2) that are each independently pharmacologically active but temporally complementary. Understanding this as a specific pharmacological interaction - rather than a non-specific massage confound - has implications for clinical aromatherapy guidance, formulation design, and the interpretation of existing trials.[7,22,25,33] For aromatherapy in sleep medicine specifically, the mechanistic framework supports a clear ranking of delivery modality effectiveness: Nabhi application (both pathways co-activated in an anatomically enclosed high-efficiency site) > aromatic massage (both pathways, but less efficient olfactory delivery and lower transdermal SC enhancement) > inhalation alone (Stage 1 only) > topical application to forearm (Stage 2 only with lower SC permeability than umbilical site). This theoretical ranking can be tested in future controlled experimental studies.[3,22,25,31,34]

Limitations of the Present Review and Future Research Directions: This review is mechanistic and draws on established basic science and pharmacokinetic literature to construct a theoretical framework for Nabhi aromatherapy. Several important limitations should be noted:

- ***No direct pathway isolation data for Nabhi-specific delivery:*** The pathway selectivity conclusions are inferred from physicochemical first principles (vapour pressure, log P, MW) and from established transdermal pharmacokinetics literature rather than from direct experimental isolation of olfactory versus transdermal contributions at the umbilical site. Controlled experimental studies with pathway isolation designs are needed to directly verify these mechanistic inferences.
- ***Single-session versus cumulative effects:*** The mechanistic analysis addresses acute pharmacokinetics. Repeated nightly Nabhi application - as prescribed in Ayurvedic practice - may produce cumulative effects including pharmacokinetic accumulation of longer half-life compounds (bisabolol half-life approximately 8-12 hours based on structural analogues) and adaptive changes in olfactory receptor sensitivity. These cumulative dynamics are not addressed.
- ***Population heterogeneity:*** Olfactory receptor sensitivity, transdermal permeability (influenced by age, skin hydration, disease state, and genetic factors), and pharmacokinetic profiles vary substantially between individuals. The formulation recommendations presented here represent a population-average framework; individual titration will be needed in clinical practice.
- ***Apigenin transdermal delivery:*** Apigenin from Roman chamomile is a non-volatile benzodiazepine receptor partial agonist with established anxiolytic/sedative activity.[15,16] However, its low log P (1.3) and high molecular weight relative to monoterpenes may limit transdermal flux at standard essential oil concentrations. Specific pharmacokinetic studies of apigenin transdermal absorption from essential oil vehicles are needed.

Future research priorities include: (1) ex vivo and in vivo pharmacokinetic studies of linalool, bisabolol, and chamazulene transdermal absorption from the umbilical versus periumbilical abdominal site in human skin; (2) controlled pathway-isolation studies comparing olfactory-only, transdermal-only, and combined Nabhi application for sleep, HRV, and cortisol outcomes; (3) longitudinal studies of repeated Nabhi oil application for sleep outcomes in insomnia populations; and (4) GC-MS headspace characterisation of terpene emission rates from the umbilical cavity under in vivo temperature and geometry conditions.

CONCLUSIONS

Nabhi aromatherapy with lavender and chamomile oils constitutes a mechanistically rational dual-pathway delivery system that leverages the unique anatomical and physiological properties of the umbilical cavity. The olfactory pathway - activated within minutes by volatile terpene evaporation from the enclosed navel headspace - provides rapid limbic activation via direct amygdalo-hypothalamic signalling, driving fast anxiolysis, HPA axis suppression, and sleep onset facilitation.[3-12,18,21,37] The transdermal pathway - delivering sustained systemic concentrations of both volatile and non-volatile aromatic terpenes through the enhanced permeability umbilical stratum corneum - provides the pharmacological foundation for sustained sleep architecture consolidation across the overnight period.[13-17,22,31-34] The chamomile sesquiterpenes alpha-bisabolol, bisabolol oxide A, and chamazulene - whose near-zero vapour pressures make them exclusively transdermal compounds - represent a pharmacological resource that is uniquely accessible via the Nabhi route and unavailable to standard inhalation aromatherapy. This distinction provides a specific mechanistic rationale for why the navel application route may produce qualitatively different sleep architecture effects compared to diffuser inhalation alone.[13,15-17] The two-stage temporal model - Stage 1 olfactory priming (0-30 minutes) followed by Stage 2 transdermal consolidation (60-480 minutes) -

describes not merely parallel but sequentially complementary mechanisms whose temporal interaction may enhance the pharmacological effectiveness of the transdermal pathway through the neurophysiological context established by prior olfactory priming.[5,8] This mechanistic account provides a scientifically grounded framework for the Ayurvedic tradition of Nabhi Chikitsa with aromatic oils and establishes the pharmacokinetic and neuropharmacological parameters required for evidence-based Nabhi oil formulation design.[38,39,40,41]

Declarations

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Data Availability: This is a mechanistic narrative review. No primary experimental data were generated. All physicochemical and pharmacokinetic parameters cited are from published literature as referenced.

Ethics Statement: This review does not involve human subjects, animal experiments, or unpublished clinical data. No ethics committee approval was required.

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