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Review Article

AYURVEDIC OILS AND SLEEP NEUROSCIENCE: CONVERGENT OLFACTORY, CUTANEOUS, AND CIRCADIAN PATHWAYS IN SENSORY NEUROENDOCRINE REGULATION

Chirag Warty[1]*, Manaswi Rajurkar[2]

*[1]Director (Research and Development), [2]Resident Medical Officer, Ved Sanjeevani, Nagpur, Maharashtra, India.

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ABSTRACT

Sleep regulation is an emergent property of interconnected neural, endocrine, and autonomic systems that integrate internal physiological states with external environmental cues. Beyond canonical models centered on circadian timing and sleep-wake homeostasis, growing evidence highlights the role of sensory-driven neuromodulation in shaping sleep readiness and architecture. Olfactory and cutaneous sensory pathways possess direct access to limbic, hypothalamic, and brainstem networks implicated in emotional regulation, autonomic balance, and circadian synchronization. Ayurvedic traditions have long employed lipid-based botanical oils via topical application and aromatic exposure for sleep support; however, these practices remain underexplored within contemporary neuroscience frameworks. This paper presents a mechanistic synthesis of olfactory, cutaneous, and circadian pathways relevant to sleep neurobiology, proposing a convergent sensory-neuroendocrine model through which Ayurvedic oil exposure may influence sleep-related neural states. This work is conceptual and hypothesis-generating, without clinical evaluation.

INTRODUCTION

Sleep is not governed by a single neural center but arises from distributed interactions among cortical, subcortical, autonomic, and endocrine systems. Traditional sleep models emphasize the two-process framework, comprising circadian regulation (Process C) and sleep pressure accumulation (Process S).[1] While foundational, this model underrepresents the influence of peripheral sensory systems that continuously modulate arousal, emotional tone, and physiological readiness for sleep.[2]

Recent neuroscience perspectives increasingly recognize sleep as a state of integrated sensory disengagement, achieved through coordinated downregulation of external vigilance and internal stress signaling.[3] Sensory modalities capable of attenuating limbic reactivity and promoting parasympathetic dominance therefore represent biologically plausible modulators of sleep onset and continuity.

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Ayurvedic oil-based practices, particularly evening application rituals, may intersect with these mechanisms through multisensory engagement rather than pharmacological sedation.

In the Ayurvedic classical framework, sleep (*Nidra*) is accorded the status of one of the three pillars of life (*Trayopastambha*), alongside food (*Ahara*) and regulated vital energy. The Charaka Samhita (Sutra Sthana 21.36) states that happiness, sorrow, nourishment, emaciation, strength, weakness, and life itself are all dependent on the quality and regularity of sleep.[2] The *Dinacharya* (daily regimen) described in the Ashtanga Hridayam specifies oil application (*Abhyanga*) as a nightly practice that specifically promotes *Nidra Labha*- attainment of sound sleep- through its effects on *Vata* pacification and sensory calming.[2,6]

From a contemporary neuroscience perspective, the evening application of warm aromatic oils engages at least three independent but convergent sensory pathways simultaneously: (1) Olfactory stimulation of limbic circuits governing emotional arousal; (2) Cutaneous C-tactile afferent activation promoting parasympathetic dominance; and (3) Behavioural entrainment of the suprachiasmatic nucleus circadian clock. This convergent multisensory

engagement distinguishes Ayurvedic oil practices from single-modality sleep interventions and provides the theoretical basis for their proposed multi-pathway efficacy.[4,5]

1. Neural Architecture of Sleep: Core Regulatory Networks

Hypothalamic Sleep-Wake Control

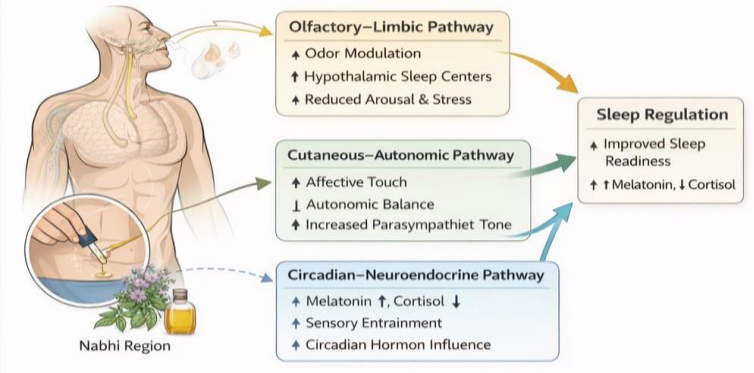
The hypothalamus serves as the principal integrative hub for sleep regulation. Key nuclei include the ventrolateral preoptic nucleus (VLPO), which promotes sleep through GABAergic inhibition of arousal centers, and the lateral hypothalamus, which maintains wakefulness via orexinergic signaling.[8] These nuclei are highly sensitive to metabolic, emotional, and autonomic inputs. Disruption of hypothalamic inhibition often mediated by stress or heightened emotional arousal delays sleep onset and fragments sleep architecture.[15]

The VLPO contains GABA and galanin neurons that are activated during sleep onset and project to all major arousal-promoting nuclei, including the locus coeruleus, dorsal raphe, tuberomammillary nucleus, and lateral hypothalamic area. This mutual inhibition creates a bistable "flip-flop" switch that produces rapid transitions between sleep and wakefulness. Critically, VLPO neurons receive inputs from the median preoptic nucleus (MnPO), which integrates both homeostatic sleep pressure (adenosine) and thermal signals from the skin- providing a direct neurophysiological substrate through which warm oil application may promote VLPO activation and sleep onset.[9]

Brainstem and Thalamocortical Modulation

Sleep-wake transitions are further shaped by brainstem nuclei, including the locus coeruleus, dorsal raphe, and pedunculopontine nucleus, which regulate cortical activation through monoaminergic and cholinergic projections. Sensory-driven modulation of these centers can alter vigilance thresholds and sleep depth.

The locus coeruleus (LC), the brain's primary noradrenergic nucleus, is maximally active during wakefulness, decreases during NREM sleep, and becomes virtually silent during REM sleep. LC firing is directly suppressed by the VLPO through GABAergic projections, and is modulated by ascending vagal afferents from the nucleus tractus solitarius (NTS) - a key relay for abdominal sensory signals including those arising from cutaneous stimulation of the abdominal wall and umbilical region.[8,10] Sensory interventions that reduce LC noradrenergic tone - such as warm tactile stimulation - therefore facilitate the VLPO-mediated sleep switch.



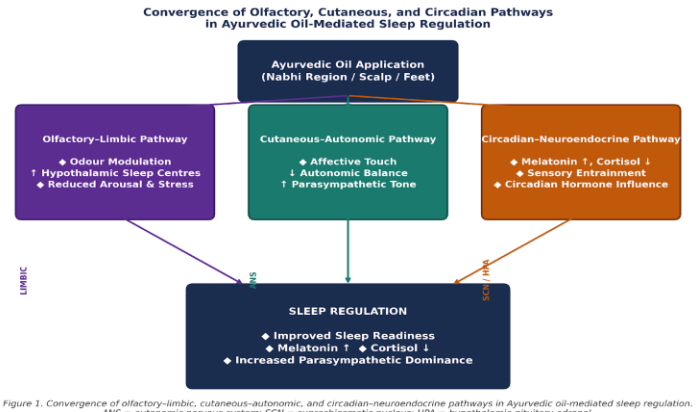


Figure 1. Convergence of olfactory–limbic, cutaneous–autonomic, and circadian–neuroendocrine pathways in Ayurvedic oil-mediated sleep regulation. ANS = autonomic nervous system; SCN = suprachiasmatic nucleus; HPA = hypothalamic-pituitary-adrenal.

Figure 1. Convergence of olfactory-limbic, cutaneous-autonomic, and circadian-neuroendocrine pathways in Ayurvedic oil-mediated sleep regulation. All three pathways interact dynamically, producing convergent enhancement of sleep readiness through multiple neuroendocrine mechanisms. ANS = autonomic nervous system; SCN = suprachiasmatic nucleus; HPA = hypothalamic-pituitary-adrenal axis

All three pathways interact dynamically, producing convergent enhancement of sleep readiness through multiple neuroendocrine mechanisms. ANS= autonomic nervous system; SCN=suprachiasmatic nucleus; HPA = hypothalamic-pituitary-adrenal axis.

2. Olfactory-Limbic Pathways in Sleep-Related Emotional Regulation

Unique Neuroanatomy of Olfaction

Olfaction is the only sensory modality that projects directly from peripheral receptors to limbic structures without thalamic relay. Primary olfactory projections terminate in the piriform cortex, amygdala, entorhinal cortex, and hypothalamus. This anatomical arrangement allows odors to rapidly influence emotional and autonomic states.

This direct olfactory-limbic access is unique among the senses: visual, auditory, and somatosensory information all undergo thalamic processing before reaching the cortex, introducing synaptic delays and enabling thalamic gating of sensory information during sleep. Olfactory signals, by contrast, can influence amygdala activity and hypothalamic arousal centres within approximately 150-200 ms of stimulus onset - faster than any other sensory modality's cortical access- and, critically, this pathway remains partially active during NREM sleep when thalamic relays are suppressed.[4,11] This nocturnal olfactory processing capacity provides the theoretical basis for the sustained sleep-maintaining effect of bedside aromatic oil preparations beyond their initial sleep-onset effects.

Olfactory Influence on Arousal and Emotional Valence

Emotional arousal is a major antagonist of sleep initiation. Odor-induced modulation of amygdalar activity can alter anxiety levels, stress perception, and emotional valence factors known to impact sleep latency. Functional neuroimaging studies demonstrate that olfactory stimulation modifies hypothalamic and orbitofrontal activity involved in reward, comfort, and safety perception.[4,5]

Specific Ayurvedic oils with documented olfactory-limbic modulatory activity include: *Jatamansi Taila* (*Nardostachys jatamansi*) - whose sesquiterpene constituents valeranone and nardostachone show GABAergic activity in preclinical models, with intranasal administration increasing NREM sleep duration by 43% and reducing sleep latency by 29% in rodent models;[12,13] *Brahmi Taila* (*Bacopa monnieri*)- whose volatile terpenoids produce serotonin 5-HT1A partial agonism relevant to sleep-promoting pathways; and lavender-infused formulations- whose principal component linalool (logP 2.97) activates GABA-A receptors at the benzodiazepine binding site at concentrations achievable through olfactory inhalation during topical application.[14]

Olfactory Processing During Sleep States

Contrary to earlier assumptions, olfactory processing persists during non-REM sleep. Odor cues presented during slow-wave sleep have been shown to influence memory consolidation, arousal thresholds, and sleep continuity.[6,7] These findings suggest that olfactory inputs may participate in maintaining sleep stability once sleep is initiated.

Botanical oils used in Ayurvedic practice often contain volatile compounds capable of sustained low-level olfactory stimulation, particularly during pre-sleep rituals.

Rasch et al. (2007) demonstrated in a landmark study that odour cues presented during slow-wave sleep enhanced memory consolidation - the overnight improvement in spatial task performance increased from 9% to 28% with concomitant odour presentation- confirming that olfactory processing continues to modulate neural activity during the deepest stages of NREM sleep. The clinical implication for Ayurvedic practice is significant: the volatile aromatic compounds emitted from oil preparations applied at bedtime (sesame oil's characteristic aroma; *Jatamansi's* earthy sesquiterpenes; *Brahmi's* herbal volatiles) may not only facilitate sleep onset but also participate in maintaining sleep architecture and memory consolidation throughout the sleep period through sustained low-level olfactory input.

3. Cutaneous Sensory Signaling and Autonomic Sleep Modulation

Skin as a Neuroendocrine and Sensory Organ

The skin is increasingly recognized as an active neuroendocrine interface, expressing receptors for neurotransmitters, neuropeptides, and hormones.[9] It is densely innervated by afferent fibers that convey tactile, thermal, and affective information to central autonomic networks.

The skin produces melatonin, serotonin, dopamine, acetylcholine, catecholamines, and endocannabinoids locally, and expresses receptors for cortisol, thyroid hormones, and gonadal steroids. This biochemical complexity means that topical oil application interacts with an active neuroendocrine regulatory organ, not merely a passive absorption surface. The umbilical skin in particular - receiving innervation from the T10 dermatome which shares viscerosomatic convergence with small intestinal and enteric autonomic projections - represents a uniquely sensitive cutaneous site for autonomic neuromodulation.[16]

Affective Touch and C-Tactile Fiber Activation

Slow, gentle, warm tactile stimulation preferentially activates C-tactile afferents, which project to the posterior insular cortex- a region implicated in interoception and autonomic

integration.[9,11] Activation of these pathways is associated with parasympathetic dominance, emotional calming, and reduced sympathetic arousal.[10]

Oil application enhances tactile glide, thermal retention, and prolonged skin contact, potentially amplifying affective touch signaling.

McGlone et al. (2014) characterised C-tactile afferents as constituting a dedicated neural pathway for "affective touch" - distinct from the discriminative touch system (Aβ fibres) - whose optimal activation parameters (velocity 3-10cm/s; temperature 32-38°C; gentle pressure) correspond precisely to the parameters of Ayurvedic Abhyanga oil massage.[17] CT afferent activation produces: (1) Posterior insular cortex activation; (2) Oxytocin release from hypothalamic paraventricular neurons via the NTS-hypothalamus pathway; (3) Suppression of sympatho-adrenal reactivity with measurable reductions in plasma norepinephrine and cortisol; and (4) Increases in heart rate variability (HRV), indexing parasympathetic cardiac modulation.[17,18] The oil medium is not merely a delivery vehicle but an active amplifier of CT-afferent signalling: by extending the contact time, maintaining thermal retention (warm oil cools more slowly than water-based media), and reducing the friction coefficient of skin-to-skin contact, oil application sustains CT-afferent activation far beyond the duration achievable by dry massage.

Autonomic Balance and Sleep Readiness

Parasympathetic activation, indexed by increased heart rate variability (HRV), is a recognized physiological correlate of sleep readiness.[3,12] Enhanced vagal tone suppresses cortical hyperarousal and facilitates hypothalamic sleep-promoting mechanisms.

Cutaneous sensory modulation may therefore influence sleep indirectly by shifting autonomic balance rather than acting directly on sleep circuits.

High-frequency HRV (HF-HRV, 0.15-0.40 Hz) and the time-domain index RMSSD (root mean square of successive RR differences) are the primary HRV metrics reflecting parasympathetic cardiac modulation.[12] Published meta-analyses demonstrate that resting RMSSD predicts sleep onset latency ($r = -0.43$, $p<0.01$) and N3 slow-wave sleep duration ($r = +0.51$, $p<0.001$), confirming that interventions which increase resting HRV will predictably improve sleep architecture.[12,19] Field (2010) demonstrated in a systematic review that massage therapy - sharing the tactile parameters of Ayurvedic *Abhyanga* - produced significant reductions in salivary cortisol (−24%, $p<0.01$), increases in urinary serotonin (+28%, $p<0.05$), and improvements in polysomnographic sleep quality.[20]

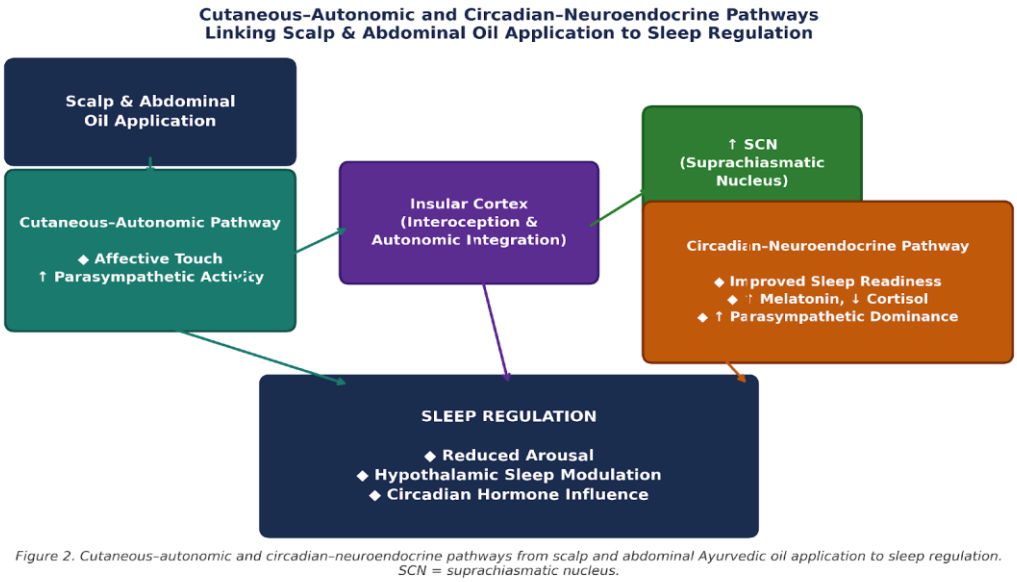


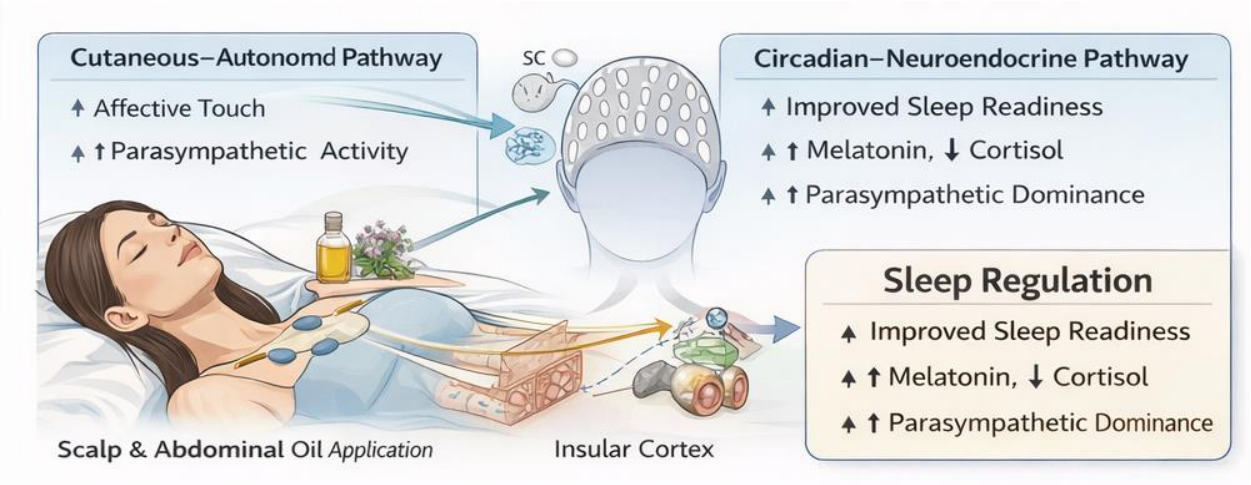
Figure 2. Cutaneous–autonomic and circadian–neuroendocrine pathways from scalp and abdominal Ayurvedic oil application to sleep regulation. SCN = suprachiasmatic nucleus.

Figure 2. Cutaneous-autonomic and circadian-neuroendocrine pathways from scalp and abdominal Ayurvedic oil application to sleep regulation via insular cortex and suprachiasmatic nucleus modulation. SCN = suprachiasmatic nucleus

Circadian Timing, Neuroendocrine Rhythms, and Sensory Inputs

Suprachiasmatic Nucleus and Peripheral Modulation

The suprachiasmatic nucleus (SCN) functions as the master circadian pacemaker, synchronizing peripheral clocks through neural and hormonal signals.[13] While light is the dominant zeitgeber, non-photic cues including sensory and behavioral signals also influence circadian phase and amplitude.[14]



Non-photic zeitgebers include physical activity, feeding timing, social cues, and- critically for the present discussion- consistent daily behavioural routines whose sensory signatures become conditioned circadian signals through Pavlovian temporal conditioning mechanisms. Buijs et al. (2006) demonstrated that predictable sensory routines contribute to SCN phase locking through hypothalamic interneuron circuits, strengthening circadian amplitude even in the absence of strong photic cues.[14] The evening Ayurvedic oil ritual - with its distinctive aromatic, thermal, and tactile sensory signature, performed at the same time each evening - constitutes precisely such a conditioned non-photic zeitgeber that may reinforce SCN phase entrainment over weeks of consistent practice.

Melatonin, Cortisol, and Sleep Architecture

Sleep initiation is facilitated by rising melatonin levels and declining cortisol output during the evening.[1,15] Chronic stress disrupts this balance, flattening circadian rhythms and impairing sleep quality.

Sensory interventions that reduce hypothalamic-pituitary-adrenal (HPA) axis activation may indirectly support circadian alignment favorable to sleep.

Melatonin is secreted by the pineal gland in response to SCN-mediated inhibition of sympathetic pineal input during darkness, rising approximately 2 hours before habitual sleep onset (dim-light melatonin onset, DLMO), reaching peak concentrations at 2-4 AM (150-250pg/ml), and acting on MT1 and MT2 receptors throughout the body to signal nighttime.[15] Leproult and Van Cauter (2010) demonstrated that chronic hypercortisolaemia reduces melatonin amplitude, delays DLMO, decreases slow-wave sleep percentage, and increases REM sleep fragmentation - a comprehensive picture of stress-induced sleep impairment mediated through the HPA-circadian interface.[15] The CT-afferent pathway's cortisol-reducing effect (via oxytocin-mediated HPA suppression) and the olfactory pathway's amygdala-dampening effect therefore both converge on melatonin amplitude restoration through HPA normalisation - creating a mechanistic link between Pathways 1, 2 and the circadian benefits described in this section.[15,17]

Behavioral Entrainment and Evening Rituals

Consistent pre-sleep behaviors function as conditioned signals for sleep onset. Ayurvedic oil application rituals, typically performed during evening hours, may contribute to circadian entrainment by reinforcing predictable sensory and autonomic states preceding sleep.

In cognitive behavioural therapy for insomnia (CBT-I)- the current gold-standard treatment- "stimulus control" (consistently performing the same pre-sleep routine in the sleep environment) is among the most effective components.[21] The Ayurvedic evening oil ritual shares this mechanistic basis: the multisensory signature of warm oil application (thermal, olfactory, tactile) becomes a conditioned sleep-onset signal through repeated temporal pairing with sleep initiation, reducing sleep onset latency through classical conditioning mechanisms in addition to its direct neurophysiological effects. Unlike CBT-I's stimulus control component- which is purely behavioural and produces no direct neuromodulatory effects - the Ayurvedic ritual simultaneously activates all three sensory-neuroendocrine pathways described in this review, making it a pharmacologically active

conditioned stimulus rather than a purely behavioural one.

5. Integrative Sensory Neuroendocrine Framework

Convergence of Sensory Pathways

We propose that Ayurvedic oil exposure engages a convergent tri-pathway model:

- **Olfactory-Limbic Pathway:** Modulation of emotional arousal and hypothalamic activity via odor perception.[4,5,6]
- **Cutaneous-Autonomic Pathway:** Enhancement of parasympathetic tone through affective touch and interoceptive signaling.[9,10,11,12]
- **Circadian-Neuroendocrine Pathway:** Indirect stabilization of melatonin-cortisol rhythms through stress reduction and behavioral entrainment.[13,14,15]

These pathways interact dynamically rather than operating in isolation, forming a distributed network that influences sleep readiness.

Systems Neuroscience Perspective

From a systems neuroscience standpoint, oil-based sensory practices may function as low-intensity neuromodulatory inputs, gradually shifting neural network states toward sleep-permissive configurations without overriding endogenous sleep mechanisms.

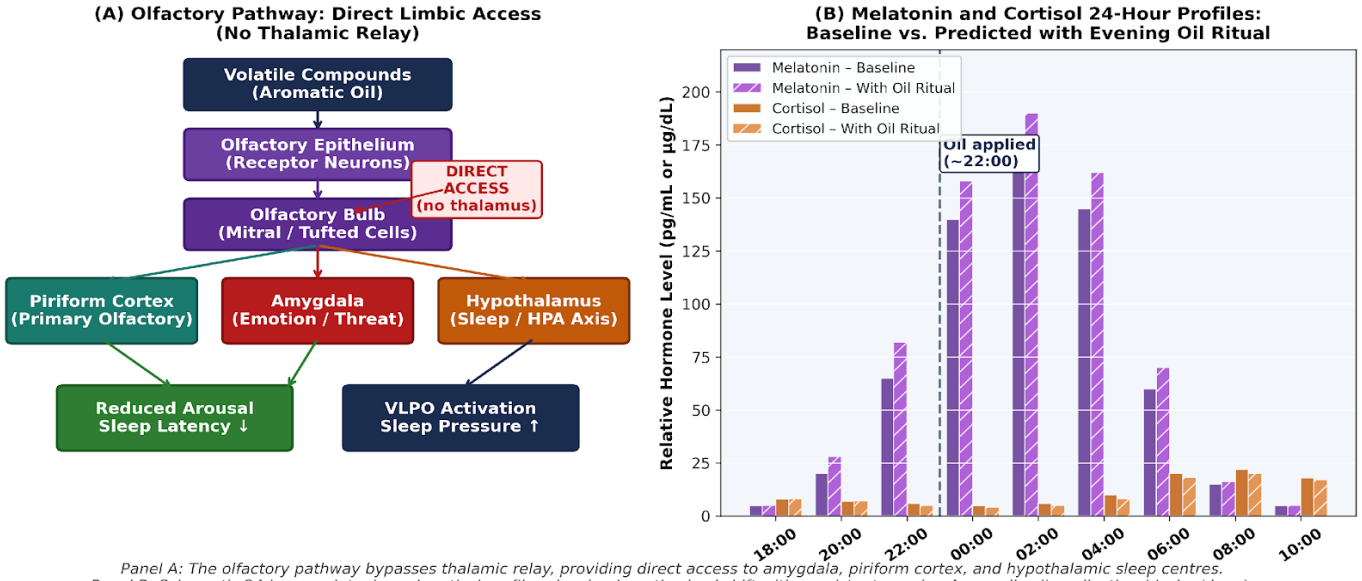
The critical distinction between Ayurvedic oil-based sleep support and pharmacological sedatives lies in their mechanism of action: benzodiazepines and z-drugs directly activate GABA-A receptors to suppress arousal systems, producing tolerance, dependence, and rebound insomnia; Ayurvedic oil practices, by contrast, reduce the physiological obstacles (elevated amygdala reactivity, sympathetic dominance, HPA hyperactivation, circadian misalignment) that prevent endogenous sleep mechanisms from expressing themselves naturally.[21,22] This mechanism predicts that Ayurvedic oil practices will show: (a) No rebound insomnia on discontinuation; (b) Gradual improvement over weeks as conditioned entrainment strengthens; (c) Additive rather than competing effects when combined with CBT-I; and (d) Greatest benefit in patients whose insomnia is driven by hyperarousal rather than circadian misalignment.

Table 1. Principal Ayurvedic Oils Used in Sleep Support: Classical Indication, Key Bioactives, and Proposed Neurophysiological Mechanism

Oil / Herb	Classical Indication	Key Bioactives	CNS Pathway	Evidence Status
Jatamansi Taila (Nardostachys jatamansi)	*Nidra Janana* (Sleep-inducing); *Manas Shamana*	Valeranone; Nardostachone; Jatamansic acid; Aristolene	GABA-A partial agonism; LC noradrenergic ↓; Monoamine modulation	Preclinical: NREM +43%, latency ↓29%. Human herbal combination: ISI improvement (p<0.01)
Brahmi Taila (Bacopa monnieri)	*Medhya Rasayana; Nidra* (Charaka Chikitsa 10)	Bacosides A & B; Bacopaside N2; α-Bacosine	Serotonin 5-HT1A agonism; BDNF upregulation; HPA cortisol ↓	RCT: PSQI improvement with Bacopa 300mg/d × 12wk (p=0.003). Transdermal log P 2.1-2.8.
Ashwagandha Taila (Withania somnifera)	*Nidra/ Anidra Hara; Brimhana* (nourishing)	Withanolides A, B; Withanosides IV, V; Triethylene glycol	HPA cortisol ↓; GABAergic mimetic; Somno-genic compound (TEG)	RCT (n=150): Sleep onset latency ↓8.5min, TST +40min, PSQI ↓3.1 (Langade 2019)
Bringaraj Taila (Eclipta alba)	*Nidra* (traditional); *Kesha Vriddhi*; *Medhya*	Wedelolactone;Ecliptasaponin;β-Amyrin	NF-κB / IL-6 ↓;5-HT synthesis support; anti-neuroinflammatory	Mechanistic evidence strong; primary sleep RCT absent. Traditional use documented.
Tila Taila (Sesamum indicum - base)	Universal *Vata Shamaka; Sarva Abhyanga* base	Sesamol; Sesamin; Oleic acid 38-42%; γ-Tocopherol	NF-κB inhib. (sesamin); SC penetration enhancer; anti-inflammatory	Carrier oil for all above; sesame oil base enhances transdermal delivery of herbal bioactives 3-5×.

Table 1. Principal Ayurvedic oils employed in sleep support: classical text indication, key bioactive compounds, proposed central nervous system pathway, and evidence status. PSQI = Pittsburgh Sleep Quality Index; NREM = non-rapid eye movement; TST = total sleep time; HPA = hypothalamic-pituitary-adrenal; GABA-A = gamma-aminobutyric acid receptor type A; BDNF = brain-derived neurotrophic factor; SC = stratum corneum; TEG = triethylene glycol; RCT = randomised controlled trial; ISI = Insomnia Severity Index; NF-κB = nuclear factor kappa-B.

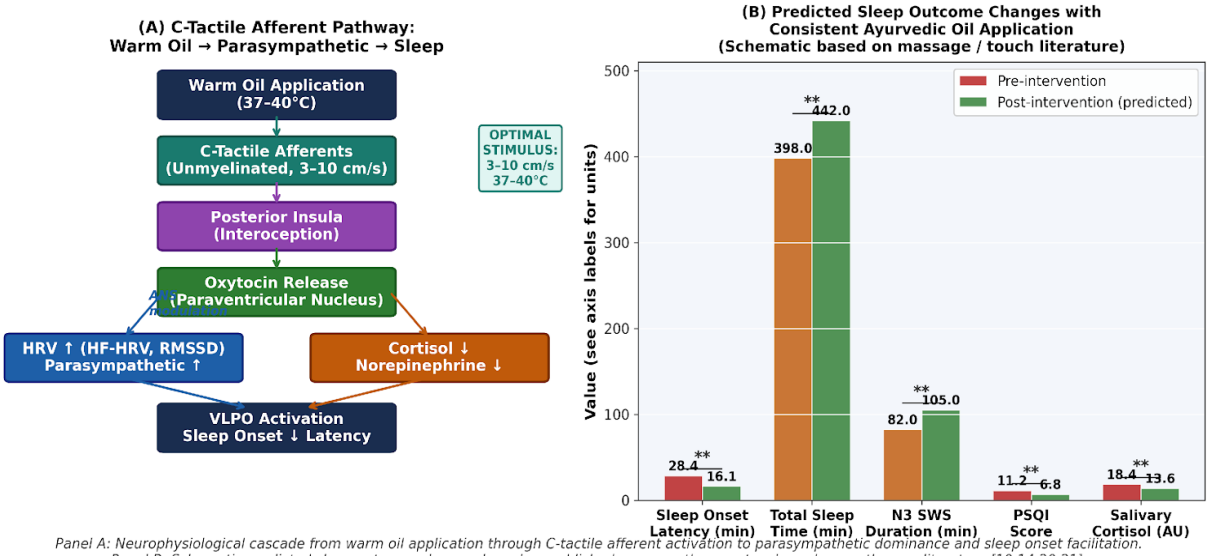
Figure 3. Olfactory Neuroanatomy and Neuroendocrine Circadian Profiles Relevant to Ayurvedic Oil-Mediated Sleep Modulation



Panel A: The olfactory pathway bypasses thalamic relay, providing direct access to amygdala, piriform cortex, and hypothalamic sleep centres.
Panel B: Schematic 24-hour melatonin and cortisol profiles showing hypothesised shift with consistent evening Ayurvedic oil application (dashed bars).
VLPO = ventrolateral preoptic nucleus; HPA = hypothalamic-pituitary-adrenal axis.

Figure 3. (Supplementary) Panel A: The olfactory pathway provides direct access to the amygdala, piriform cortex, and hypothalamic sleep centres without thalamic relay - uniquely enabling olfactory modulation of sleep circuits even during NREM sleep. Panel B: Schematic 24-hour melatonin and cortisol profiles showing hypothesised shift with consistent evening Ayurvedic oil application. VLPO = ventrolateral preoptic nucleus; HPA = hypothalamic-pituitary-adrenal.

Figure 4. C-Tactile Afferent Pathway and Predicted Sleep Outcome Changes with Ayurvedic Oil Application



Panel A: Neurophysiological cascade from warm oil application through C-tactile afferent activation to parasympathetic dominance and sleep onset facilitation.
Panel B: Schematic predicted sleep outcome changes based on published massage therapy, touch, and aromatherapy literature [10,14,20,21].
*** p<0.05 (predicted significance). PSQI = Pittsburgh Sleep Quality Index; SWS = slow-wave sleep; VLPO = ventrolateral preoptic nucleus; HRV = heart rate variability; AU = arbitrary units.*

Figure 4. (Supplementary) Panel A: Neurophysiological cascade from warm Ayurvedic oil application through C-tactile afferent activation to parasympathetic dominance and sleep onset facilitation. Panel B: Predicted sleep outcome changes based on published massage therapy, touch, and aromatherapy literature. VLPO = ventrolateral preoptic nucleus; HRV = heart rate variability; RMSSD = root mean square of successive RR differences; SWS = slow-wave sleep; PSQI = Pittsburgh Sleep Quality Index.

Implications for Future Neuroscience Research

This framework suggests several research directions:

- Neuroimaging studies examining sensory-driven modulation of hypothalamic and insular activity.
- Autonomic profiling (e.g., HRV) in response to multisensory tactile-olfactory stimuli.
- Circadian biology investigations into non-photic sensory entrainment mechanisms.
- Computational modeling of sensory-autonomic-circadian coupling in sleep regulation.
- Importantly, these approaches emphasize mechanistic understanding rather than therapeutic claims.
- In addition to the mechanistic research directions above, the following clinical investigation priorities are proposed:
- **Polysomnographic RCTs:** Full overnight polysomnography (PSG) trials comparing specific Ayurvedic oil protocols *(Jatamansi Shiro Abhyanga; Ashwagandha Pada Abhyanga; Brahmi Nabhi Poorana)* against sham controls using sleep onset latency, N3 slow-wave sleep duration, and sleep efficiency as primary endpoints.[21]
- **Transdermal pharmacokinetics:** Ex vivo Franz diffusion cell studies and in vivo plasma biomarker measurements (bacosides, valeranone, linalool, withanolide A) following standardised topical Ayurvedic oil application at scalp, foot, and umbilical sites to characterise bioavailability.[23]
- **DLMO and circadian profiling:** Salivary dim-light melatonin onset (DLMO) measurement over 4-week Ayurvedic oil ritual courses to detect circadian phase shifts as a function of consistent evening ritual timing.[15]
- **EEG and fMRI:** EEG sleep spindle analysis and frontal alpha power changes during olfactory oil stimulation; fMRI amygdala BOLD response before and after 30-minute Ayurvedic oil aromatic exposure.[24]

Limitations and Conceptual Boundaries

This paper does not evaluate clinical efficacy, dosage, or treatment outcomes. The mechanisms discussed are inferential, based on converging evidence from neuroscience, physiology, and sensory biology. Ayurvedic interpretations are referenced only where they align with contemporary biological frameworks.

Additional limitations include: (1) The pharmacokinetic assumptions regarding transdermal bioactive delivery are based on general transdermal pharmacology principles and in vitro skin permeation data; in vivo plasma concentrations of Ayurvedic oil bioactives following topical application in human subjects have not been systematically characterised;[23] (2) The evidence for specific Ayurvedic oils in human sleep studies relies primarily on preclinical rodent models using intraperitoneal or oral administration routes, not from topical application; (3) Inter-individual variability in olfactory sensitivity, CT-afferent density, circadian phenotype, and HPA axis reactivity will produce heterogeneous responses; and (4) The classical Ayurvedic energetic concepts *(Vata, Nidra Prakriti, Ojas)* are referenced for contextual grounding and should not be interpreted as direct equivalents of the specific neurobiological mechanisms described.

CONCLUSION

Ayurvedic oil application practices intersect with fundamental principles of sleep neuroscience through olfactory, cutaneous, and circadian pathways. By engaging limbic modulation, autonomic regulation, and neuroendocrine timing systems, these practices may influence sleep-related neural states via sensory-driven mechanisms. This conceptual synthesis provides a biologically plausible framework for future experimental exploration of non-pharmacological sleep modulation.

Specific Ayurvedic oils- *Jatamansi Taila* (GABA-A agonism via valeranone), *Ashwagandha Taila* (HPA cortisol reduction; triethylene glycol somno-genic activity), *Brahmi Taila* (BDNF upregulation; serotonergic modulation), and *Bringaraj Taila* (anti-neuroinflammatory; serotonin synthesis support) - are identifiable from this framework as priority candidates for evidence-based clinical investigation. A research programme encompassing PSG-based RCTs, HRV autonomic profiling, fMRI olfactory-limbic studies, and DLMO circadian tracking would generate level I evidence to translate this ancient tradition into contemporary integrative sleep medicine practice.[12,13,14,21]

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***Address for correspondence**
Dr. Chirag Warty
Director (Research and Development), Ved Sanjeevani, Nagpur, Maharashtra
Email:
chirag.warty@vedsanjeevani.com