



Review Article

AYURVEDIC INTERVENTIONS FOR AUTISM SPECTRUM DISORDER IN CHILDREN: A CRITICAL NARRATIVE REVIEW OF CLINICAL EVIDENCE, MECHANISMS, SAFETY, AND RESEARCH PRIORITIES

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Article info

Article History:

Received: 23-07-2026

Accepted: 22-08-2026

Published: 10-09-2026

KEYWORDS:

Autism spectrum disorder, Ayurveda, Kaumarbhritya, Medhya Rasayana, Bacopa monnieri, Panchakarma, Gut-brain axis, Neuro-inflammation, safety.

ABSTRACT

Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental condition for which early developmental, behavioral, educational, communication, and family-centred interventions remain the foundation of care. **Objective:** To critically synthesize the Ayurvedic conceptual framework, clinical evidence, proposed mechanisms, and safety considerations for oral medicines and procedure-based interventions used in children with ASD. **Methods:** A structured narrative search of PubMed/MEDLINE, Google Scholar, AYUSH Research Portal, and relevant Ayurveda journals was considered through August 2026 using terms related to autism, Ayurveda, Panchakarma, Bacopa monnieri, Centella asiatica, Withania somnifera, Acorus calamus, Brahmi ghrita, Nasya, Basti, Abhyanga, and Shirodhara. Human evidence was prioritized; preclinical and indirect evidence was used only to discuss biological plausibility. **Results:** Direct clinical evidence for specifically Ayurvedic interventions in pediatric ASD is sparse and consists mainly of small uncontrolled studies, case reports, and protocols. Bacopa-related preparations have the most developed mechanistic rationale, involving antioxidant, anti-inflammatory, synaptic, and glutamatergic pathways, but ASD-specific human efficacy is unconfirmed. Evidence for other Medhya drugs, medicated ghee, and Panchakarma procedures is largely indirect. **Conclusion:** Individualized Ayurveda may be studied as an adjunct for co-occurring problems such as sleep disturbance, gastrointestinal symptoms, anxiety, and dysregulation, but it cannot presently be described as disease-modifying or a replacement for established developmental care. Future trials require standardized products, treatment manuals, validated core and functional outcomes, adverse-event monitoring, and long-term follow-up.

INTRODUCTION

Autism spectrum disorder is a lifelong, heterogeneous neurodevelopmental condition characterized by differences in social communication and interaction together with restricted or repetitive patterns of behaviour, interests, or sensory responses. Presentation, support needs, language, intellectual ability, adaptive function, and co-occurring conditions vary markedly between children.

Current estimates indicate that autism affects approximately 1 in 100 children globally, although identification varies by setting and surveillance method [1]. Autism should therefore not be approached as a single uniform disease entity or as a condition requiring normalization of neurodivergent traits.

Evidence-based care is individualized and may include caregiver-mediated intervention, speech-language and occupational therapy, educational supports, behavioural and developmental programmes, management of sleep, epilepsy, attention-deficit/hyperactivity disorder, anxiety, irritability, constipation, feeding problems, and support for family wellbeing [1,2]. Medicines used in conventional practice primarily target selected co-occurring symptoms rather than the defining features

Access this article online

Quick Response Code



<https://doi.org/10.47070/ayushdhara.v13i4.2949>

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of autism. This therapeutic gap, together with cultural accessibility, contributes to family interest in Ayurveda and other traditional systems.

The classical Ayurvedic compendia do not describe ASD as a one-to-one diagnostic entity. Retrospective identification of autism with *Unmada* is therefore an interpretive clinical model, not a literal classical diagnosis. Features relevant to an Ayurvedic formulation may be considered through impairment of *Manas*, *Buddhi*, *Smriti*, *Sheela*, *Cheshta* and *Achara*; *Vata*-predominant dysregulation; variable association of *Kapha* or *Pitta*; *Agni* and *Kostha* abnormalities; and the child's constitutional and developmental context. A responsible integrative formulation must remain symptom- and function-centred and must not force every autistic child into a single *Doshic* category [3-5].

The earlier version of this manuscript reported a PRISMA flow and pooled participant count without sufficient study-level documentation. Because systematic-review claims require a reproducible search, duplicate screening, protocol-level eligibility criteria, complete extraction, and formal risk-of-bias appraisal, the present revision is framed as a critical narrative review. Its purpose is to distinguish direct clinical evidence from indirect or preclinical plausibility and to develop testable mechanisms rather than therapeutic certainty.

METHODS

Review design and search approach

A structured narrative review was undertaken to map clinical and mechanistic evidence. PubMed/MEDLINE, Google Scholar, AYUSH Research Portal, and journal websites were searched from inception through August 2026. Reference lists of relevant articles were examined for additional sources.

Selection and interpretation

Human paediatric studies directly evaluating Ayurvedic medicines or procedures in diagnosed ASD were prioritized. Controlled trials were ranked above uncontrolled studies and case reports. Preclinical studies and human research in other cognitive, stress, sleep, or gastrointestinal conditions were included only to examine plausibility. Traditional East Asian, Persian, and Siddha interventions were not treated as proof for Ayurveda, although systematic reviews of herbal medicine were used to contextualize the broader evidence base [6-8]. No meta-analysis was attempted because interventions, diagnostic criteria, outcomes, and study designs were heterogeneous. Mechanistic statements were classified as: (i) clinically demonstrated in ASD; (ii) supported in preclinical ASD models; or (iii) extrapolated from non-ASD evidence.

Ayurvedic Clinical Framework

Dosha, *Manas* and developmental formulation

In an Ayurvedic clinical formulation, *Vata* provides a useful organizing principle for variability in movement, sensory reactivity, sleep, speech initiation, attention, bowel pattern, and emotional regulation. *Kapha*-associated features may include reduced initiation, heaviness, excessive sleep, or slower responsiveness, whereas *Pitta* features may include irritability, heat intolerance, or inflammatory complaints. *Raja* and *Tamas* describe qualities of mental activity and inertia but should not be used as moral labels. The clinically relevant task is to define the child's strengths, support needs, co-occurring symptoms, *Prakriti*, *Vikriti*, *Agni*, *Kostha*, sleep, diet, sensory profile, and family goals.

Therapeutic goals

Ayurvedic care should set measurable goals that matter to the child and family: improved sleep continuity, comfortable bowel function, reduced pain, safer feeding, better participation in therapy or school, reduced distress, and improved caregiver-reported quality of life. Change in eye contact should not be treated as a universal endpoint. Core autism severity scores alone are insufficient because they may not capture communication gains, adaptive function, or wellbeing.

Ayurvedic Medicines: Evidence and Proposed Mechanisms

The term "mode of action" must be used cautiously. Most proposed pathways below are pharmacological hypotheses derived from phytochemical or animal research, not confirmed mediators of clinical benefit in autistic children. Polyherbal formulations also cannot be reduced to the action of one constituent, and botanical identity, extraction method, dose, bioavailability, age, and co-medication influence effects.

Brahmi (*Bacopa monnieri*)

Bacopa monnieri contains bacosides and related saponins. Experimental literature supports antioxidant activity, modulation of inflammatory signalling, effects on cholinergic transmission, synaptic plasticity, dendritic architecture, and memory-related pathways^[9,10]. In a valproate-induced rat model relevant to selected ASD-like behaviours, a standardized extract improved behavioural and histological outcomes and modulated upregulated AMPA-receptor expression, oxidative stress, and neuroinflammation^[11]. A recent mechanism-focused review mapped *Bacopa* actions to oxidative stress, mitochondrial dysfunction, neuroinflammation, neurotransmitter imbalance, and altered cellular

signalling in ASD, while explicitly noting that efficacy has not been established in autistic people [12].

A small study of a *Brahmi*-based compound used with rehabilitation reported changes in CARS and Vineland scores, but the formulation, allocation, blinding, sample characterization, and analytic reporting limit causal inference^[13]. Accordingly, *Bacopa* is best described as a candidate adjunct requiring standardized randomized evaluation. Proposed benefits cannot be extrapolated from cognitive studies in non-autistic populations to core autism features. Gastrointestinal intolerance, sedation, thyroid-related interactions, and product quality require monitoring.

Mandukaparni (Centella asiatica)

Centella asiatica is sometimes referred to as *Brahmi* regionally, creating botanical ambiguity. Triterpenes such as asiaticoside and asiatic acid have shown antioxidant, anti-inflammatory, neuro-protective, and neurite-supporting effects in experimental systems. Proposed pathways include attenuation of NF-kappaB-mediated inflammatory signalling, protection against oxidative injury, and support of neurotrophic signalling [14]. These findings are not equivalent to evidence of efficacy in ASD. Any manuscript or trial must state the accepted botanical name, plant part, extraction ratio, chemical marker, and dose so that *Bacopa* and *Centella* are not conflated.

Ashwagandha (Withania somnifera)

Withanolides and other constituents of *Withania somnifera* have been investigated for stress modulation, antioxidant activity, neuroprotection, GABAergic effects, and regulation of neuroinflammatory pathways^[15]. In theory, these actions could relate to anxiety, sleep disturbance, or stress reactivity in some autistic children, but ASD-specific pediatric clinical evidence is inadequate. Evidence from stressed adults or animal models should not be presented as proof of improvement in social communication. Sedation, gastrointestinal effects, thyroid effects, idiosyncratic liver injury, allergy, and interactions with sedatives or immunomodulatory drugs warrant attention.

Vacha (Acorus calamus)

Vacha is traditionally described as *Medhya* and *Vachya* and is used in formulations addressing speech and cognition. Experimental reports suggest acetylcholinesterase inhibition, antioxidant and anti-inflammatory effects, and modulation of monoaminergic or GABAergic systems. However, beta-asarone content varies by chemotype and is a major toxicological concern. *Calamus* and its derivatives are prohibited as food additives in the United States, illustrating the regulatory importance of species authentication and beta-asarone quantification^[16]. Pediatric oral use should not be recommended generically; only authenticated, quality-tested material under specialist supervision could be considered in research. Direct efficacy in childhood ASD is unproven.

Shankhapushpi, Jatamansi and other Medhya drugs

Shankhapushpi is a vernacular name applied to more than one species, commonly *Convolvulus pluricaulis* but also other regional substitutes. *Jatamansi* (*Nardostachys jatamansi*) and *Kushmanda* (*Benincasa hispida*) are similarly discussed for cognition, sleep, anxiety, or behavioural regulation. Antioxidant, anti-stress, sedative, or cholinergic mechanisms have been described experimentally, but product heterogeneity and absence of robust ASD trials prevent clinical claims. These drugs should be discussed as hypotheses for specific co-occurring symptoms, not as anti-autism medicines.

Brahmi ghrita and medicated ghee

Ghrita is traditionally considered a suitable vehicle for *Medhya* drugs. From a pharmaceutical perspective, a lipid matrix may solubilize lipophilic phytoconstituents, influence absorption, and alter release; however, enhanced delivery to the brain has not been demonstrated for *Brahmi ghrita* in autistic children. The formulation's effects cannot be attributed to ghee alone, and batch characterization is essential. Palatability, caloric load, cow-milk allergy, dyslipidaemia risk in predisposed children, rancidity, and concurrent dietary restrictions should be considered. Recent reviews describe theoretical relevance in pediatric neurodevelopment but do not substitute for controlled efficacy trials^[17].

Intervention	Ayurvedic rationale	Proposed biomedical pathway	Direct ASD evidence	Key safety/quality issue
<i>Bacopa monnieri</i>	<i>Medhya</i> , <i>Smriti</i> and <i>Buddhi</i> support	Antioxidant and anti-inflammatory effects; cholinergic/synaptic modulation; AMPA-receptor effects in an animal model	Very low: limited small human study; mechanistic and animal evidence predominate	GI effects, sedation, botanical and extract standardization

<i>Centella asiatica</i>	<i>Medhya/Rasayana</i>	Triterpene-mediated antioxidant, neurotrophic and inflammatory signalling effects	Insufficient; no persuasive pediatric ASD efficacy evidence	Do not conflate with Bacopa; define extract and markers
<i>Withania somnifera</i>	<i>Balya/Rasayana</i> ; stress and sleep support	Stress-axis, GABAergic, antioxidant and neuroimmune hypotheses	Insufficient for ASD; mostly indirect	Sedation, thyroid/liver concerns, interactions
<i>Acorus calamus</i>	<i>Medhya/Vachya</i>	Cholinergic, antioxidant and neurotransmitter hypotheses	No adequate ASD trials	Beta-asarone toxicity; chemotype authentication essential
<i>Brahmi ghrita/polyherbal Ghrita</i>	<i>Medhya</i> formulation and lipid vehicle	Possible solubilization of lipophilic constituents; unconfirmed CNS delivery	Very low; reviews/case-level evidence	Composition, stability, allergens, dose and calories
<i>Shankhapushpi / Jatamansi</i>	<i>Medhya, Nidra</i> and behavioural regulation	Anti-stress, sedative, antioxidant or cholinergic hypotheses	Insufficient	Species substitution, adulteration, sedation

Panchakarma and External Procedures

Abhyanga and Shirodhara

Abhyanga supplies structured, predictable tactile and proprioceptive input. A plausible pathway is reduction of arousal through contextual safety, caregiver interaction, slow repetitive touch, and autonomic regulation. *Shirodhara* may similarly influence relaxation and sleep through repetitive sensory stimulation and expectancy. These mechanisms are plausible but nonspecific; direct evidence that either procedure changes the neurobiology of autism is lacking. Sensory aversion is common, and procedures must never be forced. Outcomes should include assent/tolerance, sleep, distress, and participation rather than only a global autism score.

Basti

Basti is often linked to *Vata* regulation and the colon. Contemporary discussion invokes the microbiota-gut-brain axis because gastrointestinal symptoms and microbiome differences are reported in subsets of autistic children^[18,19]. Yet an enema does not automatically or durably 'correct dysbiosis,' and no validated evidence shows that Ayurvedic *Basti* normalizes the microbiome or core autism features. Any proposed gut mechanism should be tested through predefined gastrointestinal outcomes, diet records, medication exposure, stool sampling, metabolomics, and clinically relevant neurobehavioural measures. Risks include discomfort, mucosal injury, electrolyte disturbance, infection, aspiration during restraint, and psychological distress; invasive procedures in children require strong justification and consent/assent safeguards.

Nasya

The nasal route can provide local, systemic, and- in selected pharmaceutical platforms- nose-to-brain delivery. This general pharmacological possibility does not establish that traditional oil-based *Nasya* reaches therapeutic brain concentrations or benefits ASD. Pediatric studies should characterize droplet size, volume, viscosity, administration posture, aspiration risk, excipients, and microbial quality. *Nasya* should be avoided in an uncooperative or distressed child and should not be described as direct brain delivery without pharmacokinetic evidence.

Swedana and intensive purification procedures

Mild warming after oil application may promote relaxation and comfort, but dehydration and heat intolerance are relevant in children with impaired interoception or communication. Therapeutic emesis, strong purgation, bloodletting, or prolonged fasting have an unfavourable risk-benefit profile in routine pediatric ASD care and should not be extrapolated from adult *Panchakarma* practice. The principle of *Mridu*, age-appropriate therapy and avoidance of distress is central.

Integrative Mechanistic Model

ASD arises from diverse genetic and developmental pathways; neuroimmune activity, oxidative stress, mitochondrial function, excitation-inhibition balance, autonomic regulation, sleep, and gut physiology may differ in subgroups rather than forming a single causal chain^[20-23]. An integrative model should therefore be modular and testable:

Neuroimmune/oxidative module: phytochemicals may alter NF-kappaB, Nrf2, cytokine, or redox

pathways in experimental models; clinical mediation in ASD remains unconfirmed.

Synaptic/neurotransmitter module: Bacopa-related effects on cholinergic signalling, plasticity, and AMPA receptors are preclinical hypotheses; target engagement must be measured before clinical claims are made.

Stress-autonomic module: Massage, predictable routines, sleep improvement, and caregiver co-regulation may reduce arousal and improve participation without changing core neurodevelopment.

Gut-symptom module: Dietary regularity and individualized management of constipation or abdominal pain may indirectly improve sleep and behaviour; microbiome modification by *Basti* is speculative.

Functional-learning module: Reduced pain, anxiety, or sleep loss may allow greater engagement with speech, occupational, educational, and caregiver-mediated interventions. This indirect pathway may be clinically important even when core autistic traits do not change.

This model avoids the unsupported proposition that 'detoxification' removes a causal toxin. Terms such as *Ama* or *Srotorodha* may retain Ayurvedic explanatory value, but biomedical translation should be framed as a hypothesis and linked to measurable variables rather than asserted equivalence.

DISCUSSION

Principal Interpretation

The central finding of this review is an asymmetry between extensive traditional rationale and limited direct clinical evidence. Ayurvedic literature provides an individualized framework for addressing digestion, sleep, behaviour, sensory regulation, and cognition, while contemporary laboratory research identifies pharmacologically interesting actions in several *Medhya* plants. Nevertheless, the chain from phytochemical activity to clinically meaningful improvement in autistic children has rarely been tested. Clinical benefit, if present, may arise through relief of co-occurring symptoms and better participation rather than modification of the underlying neurodevelopmental phenotype.

Broader reviews of herbal medicine in ASD have reported encouraging pooled signals when herbal formulas are added to conventional therapy, but the included interventions are mainly East Asian rather than Ayurvedic, with small samples, heterogeneous prescriptions, variable outcomes, and high or unclear risk of bias [6,7]. Such findings justify research but do not validate specific Ayurvedic formulations. A recent

Siddha study and a Persian-medicine trial similarly provide system-specific preliminary findings that should not be generalized across traditions [8,24].

Why previous positive reports may overestimate benefit

Uncontrolled autism studies are especially vulnerable to maturation, regression to the mean, expectancy, caregiver-report bias, concurrent therapies, and increased clinician attention. Developmental gains over weeks or months can be wrongly attributed to an intervention when speech therapy, schooling, or natural developmental change occurs simultaneously. Composite multimodal packages further prevent attribution to an individual medicine or procedure. Blinding is difficult for massage or *Shirodhara*, but blinded outcome assessors, objective sleep measures, treatment fidelity, and prespecified analysis remain feasible.

Statistical significance is not equivalent to clinical significance. Future studies should report absolute mean differences, confidence intervals, responder definitions, and minimal clinically important differences where available. CARS and ATEC are frequently used in Ayurveda reports; however, trials should also measure adaptive functioning, individualized goal attainment, participation, quality of life, sleep, gastrointestinal symptoms, caregiver strain, and adverse events. Outcomes should be selected with autistic people and caregivers.

Interpreting the mode of action

Mechanism claims should satisfy a hierarchy. First, the product must be chemically and microbiologically characterized. Second, exposure and target engagement should be demonstrated at the administered pediatric dose. Third, the proposed biomarker should change relative to a comparator. Fourth, biomarker change should mediate a functional clinical outcome. Most current Ayurveda-ASD publications begin at the last step, inferring neuroinflammation, gut-brain modulation, or neurotransmitter balance from symptom change alone. This is insufficient.

For Bacopa, animal evidence involving AMPA-receptor expression and oxidative/inflammatory markers provides a reasonable experimental starting point [11]. A pediatric trial could therefore combine a standardized bacoside profile with pharmacokinetics, redox and inflammatory markers, electroencephalographic or cognitive measures, and clinical outcomes. For *Basti*, stool microbiome and metabolite changes must be linked to gastrointestinal and behavioural outcomes, while controlling diet and antibiotics. For *Abhyanga* or *Shirodhara*, heart-rate variability, actigraphy, salivary cortisol, sleep diaries,

and tolerance measures could test autonomic and stress hypotheses. These designs turn traditional-clinical observations into falsifiable integrative science.

Individualization versus reproducibility

Ayurveda values person-specific treatment, whereas trials require reproducibility. These aims can be reconciled through a treatment manual containing a fixed core intervention plus prospectively defined decision rules based on *Prakriti*, *Agni*, *Kostha*, dominant co-occurring symptoms, and tolerance. Every component, dose change, and rationale should be recorded. Pragmatic trials can compare this protocolized individualization with enhanced usual care; explanatory trials can test a standardized formulation. Both designs should maintain stable developmental therapies across groups.

Safety, ethics and communication

Children with ASD may have restricted diets, pica, constipation, epilepsy, sleep problems, communication differences, and multiple medicines, making safety monitoring essential. Herbal products can be contaminated or adulterated, and some rasa preparations may contain intentionally processed metals. Only products manufactured under appropriate quality systems with identity testing, microbial limits, pesticides, aflatoxins, residual solvents, and elemental impurity testing should enter pediatric research. Batch certificates should be archived. *Vacha* requires particular attention to beta-asarone. Baseline and follow-up liver, renal, thyroid, or drug-level testing should be risk-based rather than assumed unnecessary because a product is traditional.

Ethically, clinicians must state that evidence is preliminary, avoid promises of cure, avoid blaming parents, and avoid discontinuing beneficial educational

or developmental services. The child's distress, sensory needs, and assent should guide external procedures. Restrictive diets require dietitian oversight because nutritional deficiency can worsen growth and function. Suspected adverse events should be documented and reported, including paradoxical agitation, sedation, diarrhoea, allergy, or procedure-related distress.

Clinical Positioning

On present evidence, Ayurveda should be positioned as a carefully monitored adjunct, not a replacement for established care. A reasonable integrative pathway begins with multidisciplinary diagnosis and evaluation of hearing, language, cognition, sleep, feeding, growth, bowel function, epilepsy, attention, anxiety, and family priorities. Conventional and developmental supports continue. If an Ayurveda intervention is chosen, one or a small number of changes should be introduced with baseline measurement, a defined review period, stopping rules, and shared documentation. This approach protects children and also produces more interpretable clinical observations.

Limitations of This Review

This is a critical narrative review rather than a registered systematic review. Grey literature and regional journals may be incompletely indexed; publication bias is likely; and several Ayurveda reports provide insufficient detail for formal risk-of-bias assessment. Mechanistic sections necessarily use indirect and preclinical evidence and should not be interpreted as proof of clinical efficacy. Botanical nomenclature is inconsistent across publications. The field is evolving, and conclusions should be updated when adequately powered trials become available.

Research Priorities

Domain	Minimum recommendation
Design	Prospectively registered, adequately powered, multicentre randomized trial; assessor blinding; stable concomitant therapies
Participants	Standard diagnostic confirmation; age, sex, language, intellectual ability, support needs, comorbidities, diet and medications reported
Intervention	Botanical identity, plant part, extraction/manufacturing process, chemical markers, dose, adherence, and treatment-manual fidelity
Comparator	Enhanced usual care, credible attention control, or matched procedure where ethical and feasible
Outcomes	Adaptive function, communication, participation, individualized goals, sleep/GI outcomes, quality of life, caregiver burden, and adverse events
Mechanism	Predefined target-engagement biomarkers linked through mediation analysis; avoid post-hoc mechanism claims
Safety	Elemental impurities, beta-asarone where relevant, microbial/pesticide testing, laboratory monitoring, adverse-event grading and stopping rules
Duration	At least 12-24 weeks for clinical outcomes with longer follow-up for durability and development

CONCLUSION

Ayurveda offers a clinically rich, individualized framework for addressing the whole child, including sleep, digestion, stress regulation, sensory tolerance, behaviour, cognition, and family routines. This framework is conceptually relevant to the heterogeneity of autism, but conceptual relevance is not evidence of efficacy. Direct clinical evidence for Ayurvedic medicines and *Panchakarma* in autistic children remains very low in certainty.

Among oral interventions, *Bacopa monnieri* has the most coherent research rationale: bacosides may influence oxidative stress, inflammatory signalling, synaptic plasticity, cholinergic function, and AMPA-receptor regulation. Yet these mechanisms are supported mainly by laboratory and animal studies, and no robust pediatric trial has shown that *Bacopa* alters core autism characteristics. *Centella asiatica*, *Withania somnifera*, *Shankhapushpi*, *Jatamansi*, *Vacha*, and medicated *Ghrita* have plausible symptom-oriented or neuroprotective hypotheses but even less direct ASD evidence. *Vacha* additionally raises beta-asarone-related safety concerns. Proposed actions of *Abhyanga* and *Shirodhara* are more credibly framed as sensory, relational, sleep, and autonomic regulation; gut-brain claims for *Basti* and nose-to-brain claims for *Nasya* require direct pharmacological and clinical testing.

Therefore, no Ayurvedic intervention should currently be described as curing autism, reversing neurodevelopment, or replacing speech-language, occupational, educational, behavioural, medical, or family-centred care. When families choose Ayurveda, practice should be adjunctive, goal-based, time-limited, quality-assured, and monitored for benefit and harm. The strongest future contribution of Ayurveda will come from disciplined integrative research: standardized products, protocolized individualization, meaningful functional outcomes, transparent adverse-event reporting, and mechanistic studies that demonstrate target engagement rather than merely propose it. Such work may clarify whether selected interventions safely improve co-occurring symptoms and participation, while respecting neurodiversity and the priorities of autistic children and their families.

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Cite this article as:

Amandeep, Prem Prakash Vyas. Ayurvedic Interventions for Autism Spectrum Disorder in Children: A Critical Narrative Review of Clinical Evidence, Mechanisms, Safety, and Research Priorities. AYUSHDHARA, 2026;13(4):592-599.
<https://doi.org/10.47070/ayushdhara.v13i4.2949>

Source of support: Nil, Conflict of interest: None Declared

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