

Review Article

Sleep quality in children with autism spectrum disorder: a narrative review

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ABSTRACT

Sleep disturbances are among the most frequently reported yet inadequately addressed comorbidities in children with autism spectrum disorder (ASD). Affecting an estimated 50–80% of this population, sleep problems in ASD are multifactorial in origin and disproportionately severe compared to typically developing peers. Their consequences extend beyond the affected child to encompass family functioning, caregiver mental health, and child's neurodevelopmental trajectory. A systematic narrative review was conducted using PubMed, Scopus, Embase, and Google Scholar databases. Articles published between 2000 and 2024 were screened using the terms 'autism spectrum disorder,' 'sleep disturbances,' 'sleep quality,' 'children,' 'melatonin,' and 'behavioural sleep interventions.' Original research articles, systematic reviews, meta-analyses, and evidence-based clinical guidelines were included. Grey literature and single case reports were excluded. The reviewed literature consistently identifies sleep-onset insomnia, frequent nocturnal awakenings, irregular sleep-wake cycles, and reduced total sleep duration as the predominant sleep phenotypes in ASD. Aberrant melatonin synthesis, sensory processing atypicality's, co-occurring psychiatric conditions, and genetic factors collectively underpin these disturbances. The Children's Sleep Habits Questionnaire (CSHQ) and actigraphy remain the most validated assessment tools. Behavioural interventions, particularly parent-delivered sleep education programmes, demonstrate moderate-to-high efficacy. Melatonin supplementation shows consistent short-term benefit, though long-term safety data in paediatric ASD populations remain limited. Sleep disturbances in children with ASD are prevalent, clinically significant, and amenable to evidence-based intervention. Routine sleep screening should be integrated into the community medicine framework for neurodevelopmental disorders. Longitudinal research in low- and middle-income countries, including India, remains a critical gap.

Keywords: Autism spectrum disorder, Sleep disturbances, Insomnia, Melatonin, Neurodevelopmental disorders, Behavioural sleep intervention, Children's sleep habits questionnaire, Community medicine

INTRODUCTION

Sleep is a fundamental biological necessity, exerting profound and far-reaching influences on cognitive development, emotional regulation, immune function, and metabolic homeostasis during childhood. The developing brain is particularly dependent on adequate sleep architecture-encompassing appropriate durations of both rapid eye movement (REM) and non-REM sleep for

synaptic pruning, memory consolidation, and neuroplasticity.^{1,2} Disruption of these processes during sensitive developmental periods carries consequences that may persist into adolescence and adulthood.

Autism Spectrum Disorder (ASD) is a heterogeneous neurodevelopmental condition defined by persistent deficits in social communication and interaction, alongside restricted, repetitive patterns of behaviour, interests, or activities. According to the Diagnostic and

Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), these symptoms must be present in the early developmental period and cause clinically significant impairment.³ The global prevalence of ASD is estimated at approximately 1 in 100 children, with figures from the United States Centres for Disease Control and Prevention (CDC) placing prevalence as high as 1 in 36 children aged 8 years in 2020.⁴ In India, epidemiological data remain sparse, but estimates suggest a prevalence between 1 in 89 and 1 in 500, with substantial regional variation attributable to differences in case ascertainment and diagnostic capacity.⁵

Among the numerous comorbidities associated with ASD—including attention-deficit/hyperactivity disorder (ADHD), anxiety disorders, gastrointestinal dysfunction, and epilepsy-sleep disturbances occupy a position of particular clinical salience. Prevalence estimates for sleep problems in ASD range from 50% to 80%, compared with approximately 25-30% in typically developing children and 30-40% in children with other neurodevelopmental conditions.^{6,7} Despite this strikingly high burden, sleep problems in ASD are frequently underrecognized in routine clinical encounters, inadequately addressed in management plans, and underrepresented in Indian community medicine research.

The consequences of sleep disturbance in ASD are not confined to the affected child. Parents and caregivers of children with ASD and co-occurring sleep problems report substantially higher rates of depression, anxiety, and burnout compared to those whose children sleep within normal parameters.⁸ Sibling sleep is often disrupted, and family cohesion may be compromised. From a public health standpoint, this represents a considerable, largely preventable burden on households already navigating the demands of raising a child with a complex neurodevelopmental disorder.

This review is motivated by the observation that clinicians working at the intersection of community medicine and child health—including those based at tertiary care institutions—encounter ASD-associated sleep problems with regularity but frequently lack a structured, evidence-based framework for assessment and management. The present article aims to provide a comprehensive, critically appraised synthesis of the current literature, serving as both a clinical reference and a foundation for future research in Indian contexts.

METHODS OF LITERATURE REVIEW

This narrative review was conducted in accordance with general principles applicable to systematic narrative reviews in clinical medicine. The following electronic databases were searched: PubMed/MEDLINE, Embase, Scopus, PsycINFO, and Google Scholar. The search covered publications from January 2000 to December 2024. The following MeSH terms and free-text keywords were used, either individually or in combination: 'autism

spectrum disorder,' 'ASD,' 'sleep disturbances,' 'insomnia,' 'sleep quality,' 'sleep disorders,' 'children,' 'paediatric,' 'melatonin,' 'behavioural sleep intervention,' 'CSHQ,' 'actigraphy,' 'polysomnography,' and 'neurodevelopmental disorders.'

Articles were included if they- examined sleep disturbances in children aged 2-18 years diagnosed with ASD according to DSM-IV-TR, DSM-5, or ICD-10/ICD-11 criteria; reported quantitative or qualitative data on sleep outcomes; and were published in peer-reviewed English-language journals. Exclusion criteria comprised single case reports, letters to the editor, editorials without primary data, studies enrolling exclusively adult populations, and grey literature. Where applicable, systematic reviews and meta-analyses were prioritised as primary sources of synthesised evidence, with original studies cited to supplement or contextualise specific findings.

Given the broad scope of this review, a formal PRISMA flow diagram was not constructed; however, the selection process was transparent and iterative, with final inclusions agreed upon after critical appraisal of each article's methodology, sample characteristics, and outcome measurement validity.

PREVALENCE AND EPIDEMIOLOGY OF SLEEP DISTURBANCES IN ASD

The magnitude of sleep problems in children with ASD is consistently documented across diverse geographic, cultural, and methodological contexts. A meta-analysis by Mannion and Leader (2014) pooling data from 23 studies and over 2,000 participants with ASD found that 80% of children with ASD experienced at least one clinically significant sleep problem, with insomnia-encompassing difficulty initiating and maintaining sleep-being most prevalent.⁶ A more recent systematic review by Hodge et al (2020) corroborated these figures, identifying bedtime resistance (reported in 53–66% of children with ASD), prolonged sleep latency (>30 minutes in 56–66%), and early morning waking (21–32%) as the most commonly reported phenotypes.⁹

Comparative studies situate these figures in stark relief. Richdale and Schreck (2009) reviewed evidence suggesting that children with ASD sleep approximately 1 hour less per night than age-matched typically developing peers and experience significantly more nocturnal awakenings.¹⁰ Similarly, Goldman et al (2011), utilising both parental questionnaires and actigraphy, documented lower sleep efficiency, greater sleep fragmentation, and more variable sleep onset times in ASD compared to developmental disability and typically developing control groups.¹¹

Within the ASD population itself, sleep problems are not uniformly distributed. Children with lower intellectual functioning, more severe ASD symptomatology, co-

occurring anxiety, and gastrointestinal symptoms appear to be at heightened risk.^{12,13} Age-related variation is also apparent: sleep problems often emerge in toddlerhood, may fluctuate through middle childhood, and tend to persist into adolescence without targeted intervention.¹⁴ Boys with ASD appear to be somewhat more affected than girls, though this finding must be interpreted cautiously given the diagnostic under-identification of girls with ASD in general.¹⁵

Epidemiological data from South Asian and specifically Indian contexts remain limited. The case-control study conducted at three special education centres in Bangalore—from which this review's authors drew initial impetus—demonstrated significantly higher CSHQ scores in children with ASD compared to typically developing controls across domains including bedtime resistance, sleep duration, and daytime sleepiness.¹⁶ This aligns with international literature but underscores the need for larger, population-representative Indian studies.

NEUROBIOLOGY AND AETIOLOGY OF SLEEP DISTURBANCES IN ASD

Melatonin dysregulation

Melatonin, synthesised primarily in the pineal gland from serotonin via the enzyme arylalkylamine N-acetyltransferase (AANAT), is the principal endogenous modulator of circadian rhythm entrainment. Its secretion is triggered by darkness and suppressed by light, with peak nocturnal levels typically occurring between 02:00 and 04:00 hours in healthy individuals. Several independent research groups have demonstrated abnormalities in melatonin physiology specifically in individuals with ASD.

Tordjman et al (2005) documented significantly reduced nocturnal melatonin secretion in children with ASD, measured via urinary 6-sulphatoxymelatonin (the primary melatonin metabolite), and observed an inverse correlation between melatonin levels and the severity of social communication deficits.¹⁷ Subsequent genetic studies identified mutations and copy number variants in the ASMT gene (encoding acetylserotonin methyltransferase, the enzyme catalysing the final step of melatonin synthesis) in subsets of ASD populations.¹⁸ This positions melatonin dysregulation not merely as a correlate but potentially as a pathophysiologically meaningful contributor to ASD-related sleep disruption.

Serotonin dysregulation is closely allied to melatonin abnormalities, given their shared biochemical pathway. Hyperserotoninaemia-elevated platelet serotonin levels—has long been one of the most replicated biological findings in ASD, observed in approximately 30% of individuals.¹⁹ The interplay between atypical serotonin metabolism, melatonin hyposecretion, and sleep-onset difficulties is mechanistically plausible, though direct causal chains remain to be fully elucidated.

Sensory processing and arousal dysregulation

Sensory processing atypicalities are a near-universal feature of ASD and are now formally incorporated into DSM-5 diagnostic criteria. Hypo or hypersensitivity to auditory, tactile, visual and proprioceptive stimuli may significantly impair sleep onset and maintenance. For instance, hypersensitivity to environmental sounds can cause repeated nocturnal awakening; tactile sensitivity may render conventional bed linen or clothing intolerably stimulating; and visual hypersensitivity may interfere with adequate light attenuation at bedtime.²⁰

At a neurophysiological level, atypical arousal regulation—characterised by difficulties in transitioning between states of alertness, and abnormalities in the homeostatic sleep pressure build-up—has been proposed as a core mechanism. Electroencephalographic (EEG) studies have demonstrated altered sleep architecture in ASD, including reduced slow-wave sleep (SWS) activity, atypical sleep spindle morphology and density, and reduced REM sleep duration, findings that collectively reflect disordered thalamocortical circuit function.^{21,22}

Co-occurring psychiatric and medical conditions

The majority of children with ASD meet diagnostic criteria for at least one co-occurring psychiatric condition. Anxiety disorders, present in an estimated 40–60% of children with ASD, are strongly associated with bedtime refusal, sleep onset difficulties, and nocturnal fears.²³ ADHD, co-occurring in 30–50% of cases, contributes to elevated arousal levels, difficulty settling at bedtime, and fragmented sleep architecture.²⁴ Epilepsy, which affects approximately 20–30% of individuals with ASD (with higher rates in those with intellectual disability), can cause nocturnal seizures that are misattributed to parasomnias or simply go undetected.²⁵

Gastrointestinal dysfunction—including gastro-oesophageal reflux disease, constipation, and altered gut motility—is a significant yet often overlooked contributor to sleep disruption in ASD. Abdominal discomfort is frequently difficult for non-verbal or minimally verbal children with ASD to communicate, and its nocturnal manifestation as restlessness, crying, or positional changes may be misconstrued as a primary sleep disorder.²⁶

Genetic and familial factors

Genetic contributions to ASD-associated sleep disturbances are increasingly recognised. Mutations in circadian clock genes, including CLOCK, BMAL1, PER1-3, and CRY1-2, have been reported in ASD cohorts, suggesting disruption of the molecular clock at a fundamental level.²⁷ Copy number variations affecting chromosome 15q11-q13—a region implicated in both ASD and Angelman syndrome—are of particular interest, as this region encodes components of the GABA-A receptor

complex, and GABAergic signalling is central to sleep regulation.²⁸ The concordance of sleep problems in monozygotic twins with ASD further supports the hypothesis of shared genetic underpinnings for ASD core symptoms and associated sleep abnormalities.²⁹

ASSESSMENT OF SLEEP IN CHILDREN WITH ASD

Clinical history and parental report

Given the communication challenges inherent to ASD, direct self-report of sleep difficulties from affected children particularly those who are younger or have concurrent intellectual disability is frequently impractical. Clinicians must therefore rely substantially on caregiver accounts, supplemented where possible by objective measurement. A comprehensive sleep history should encompass: habitual sleep and wake times, sleep latency, number and duration of nocturnal awakenings, parental interventions required to facilitate sleep onset, parasomnias (enuresis, somnambulism, night terrors), snoring or witnessed apnoea, and daytime functioning including napping behaviour, irritability, and attentional difficulties.³⁰

Standardised sleep diaries, completed by parents over a 1–2-week period, offer a practical, low-cost supplement to clinical history. They capture night-to-night variability an important consideration in ASD, where sleep patterns may be highly irregular and provide a basis for objective comparison before and after intervention.

Validated questionnaire tools

The Children's Sleep Habits Questionnaire (CSHQ), developed by Owens et al (2000), is the most widely used and validated instrument for assessing sleep in school-aged children with neurodevelopmental disorders, including ASD.³¹ The CSHQ comprises 33 items rated on a 3-point Likert scale and covers eight subscales: bedtime resistance, sleep-onset delay, sleep duration, sleep anxiety, night wakings, parasomnias, sleep-disordered breathing, and daytime sleepiness. A total score of ≥ 41 is the commonly applied threshold for identifying clinically significant sleep disturbance. The CSHQ has demonstrated adequate internal consistency (Cronbach's $\alpha = 0.68$ – 0.78 in ASD samples) and has been used in Indian ASD research contexts, including the Bangalore-based case-control study.^{16,31}

Additional instruments of relevance include the Brief Infant Sleep Questionnaire (BISQ) for younger children, the Pediatric Sleep Questionnaire (PSQ) particularly useful when sleep-disordered breathing is suspected and the Sleep Disturbance Scale for Children (SDSC). The Autism-Specific Sleep Scale is an emerging tool designed specifically to capture ASD-relevant sleep behaviours that generic instruments may underperform in detecting.³²

Actigraphy

Actigraphy the use of wrist-worn accelerometry devices worn continuously over several days provides objective, ecologically valid estimates of sleep-wake patterns including total sleep time, sleep efficiency, sleep onset latency, and wake after sleep onset. Studies validating actigraphy against polysomnography in ASD populations have demonstrated acceptable concordance, though the specific motion characteristics of some ASD-related stereotypies can introduce artefactual errors that necessitate expert epoch-by-epoch scoring.³³

Actigraphy is particularly valuable when parental reports and objective sleep data are discordant a phenomenon documented in ASD, where parents may either over-report wakefulness (interpreting quiet stereotyped behaviours as wakefulness) or under-report it (being unaware of awakenings not accompanied by vocalisation).³⁴

Polysomnography

Polysomnography (PSG) remains the gold standard for sleep assessment, providing detailed staging of sleep architecture alongside cardiorespiratory, electromyographic, and electrooculographic data. However, the tolerability of PSG in children with ASD is a significant practical obstacle: sensor application, restriction of movement, and the unfamiliar clinical environment are frequently poorly tolerated, leading to first-night effects, incomplete studies, and heightened procedural anxiety. Adaptations such as desensitisation protocols, home-based PSG, and parent co-sleeping during the study may improve feasibility.³⁵

PSG is most clearly indicated when sleep-disordered breathing is clinically suspected (given the implications for surgical referral), when epileptic seizures are part of the differential diagnosis, or when first-line interventions have failed and more precise phenotyping of the sleep disturbance is required.

MANAGEMENT OF SLEEP DISTURBANCES IN ASD

Non-pharmacological interventions

Clinical consensus and a growing body of randomised trial evidence support non-pharmacological behavioural interventions as the first-line treatment for sleep disturbances in children with ASD.³⁶ These interventions are typically parent-mediated, delivered in primary care or specialist outpatient settings, and are informed by principles of applied behaviour analysis (ABA) and sleep hygiene education.

Sleep hygiene encompasses a set of behaviours and environmental modifications that promote regular, consolidated sleep. Specific components of proven

relevance in ASD include: establishment and consistent adherence to a fixed bedtime routine (typically 3-5 steps of predictable duration, incorporating visual schedules where applicable); reduction of light exposure in the pre-sleep period, particularly from screens emitting blue-wavelength light; optimisation of the sleep environment with regard to temperature, noise, and tactile stimuli; avoidance of stimulating activities in the hour preceding bedtime; and appropriate timing of meals, particularly given the gastrointestinal comorbidities common in ASD.³⁷

Graduated extinction commonly termed the 'Ferber method' in popular parlance involves systematically increasing the interval before parental response to night-time distress, thereby reducing reinforcement of night-waking behaviour. Modified extinction paradigms, in which parents remain briefly present during the settling process before withdrawing, are better tolerated by both children and caregivers and demonstrate comparable efficacy. A randomised controlled trial by Adkins et al (2012) demonstrated significant reductions in sleep onset latency and night wakings in children with ASD following a brief parent education intervention incorporating these principles.³⁸

Cognitive-behavioural therapy for insomnia (CBT-I), while extensively validated in adult populations, requires adaptation for use in ASD. For verbal children with ASD, relaxation techniques (progressive muscle relaxation, diaphragmatic breathing) and cognitive restructuring of maladaptive sleep-related beliefs can be incorporated where developmental level permits. For non-verbal or minimally verbal children, caregiver-mediated relaxation routines incorporating deep pressure, proprioceptive input, or sensory-attuned pre-sleep activities may achieve analogous effects.³⁹

Pharmacological interventions

Where behavioural interventions alone are insufficient, pharmacological agents may be considered as adjuncts. The evidence base for pharmacotherapy in ASD-related sleep disturbance, while growing, remains less robust than that for behavioural approaches, and prescribing should be undertaken with a clear risk-benefit assessment, shared decision-making with caregivers, and planned review.⁴⁰

Melatonin is currently the most extensively studied and widely recommended pharmacological intervention for sleep-onset insomnia in ASD. A landmark double-blind, placebo-controlled trial by Gringras et al (2012) the MENDS study demonstrated that prolonged-release melatonin (2-5 mg, administered 30-60 minutes before target bedtime) significantly reduced sleep-onset latency and increased total sleep duration in children with neurodevelopmental disabilities including ASD, with a favourable tolerability profile.⁴¹ Subsequent meta-analyses have corroborated these findings, with Wright et

al (2011) identifying an average reduction in sleep-onset latency of 29 minutes and a 48-minute increase in total sleep time compared with placebo.⁴² Long-term safety data, particularly regarding hypothalamic-pituitary-gonadal axis effects, remain limited and represent an important research gap.

Other pharmacological agents employed in clinical practice include clonidine (an alpha-2 agonist with sedative properties, useful particularly in the context of co-occurring ADHD), hydroxyzine (an antihistamine used for its sedating properties in acute settings), and in selected cases of treatment-resistant insomnia in older children, low-dose trazodone or mirtazapine. Benzodiazepines and non-benzodiazepine hypnotics (Z-drugs) are generally avoided in children given their potential for dependence, tolerance, and rebound insomnia.⁴³ Risperidone and aripiprazole atypical antipsychotics approved by regulatory authorities for irritability associated with ASD may confer secondary improvements in sleep through sedating properties, though somnolence is simultaneously a recognised adverse effect requiring monitoring.⁴⁴

Emerging and complementary approaches

Weighted blankets, which provide deep pressure stimulation with putative anxiolytic effects mediated via the somatosensory system, have attracted considerable parental interest. Although anecdotal reports and preliminary uncontrolled studies have been positive, randomised evidence remains limited and findings are mixed.⁴⁵ Light therapy (phototherapy), delivered via timed bright light exposure in the morning to advance circadian phase, has theoretical appeal particularly in ASD individuals with delayed sleep phase syndrome, though formal trial data in paediatric ASD are sparse.⁴⁶

Social stories narrative tools used in ASD intervention more broadly have been adapted to address sleep-specific contexts, helping children with ASD develop cognitive representations of the bedtime routine and its expected outcomes. While the evidence base is primarily case-study based, they represent a low-cost, non-invasive adjunct to behavioural packages.⁴⁷

IMPACT ON FAMILIES, CAREGIVERS, AND QUALITY OF LIFE

The burden of ASD-associated sleep disturbance extends substantially beyond the affected child. Meltzer (2011) demonstrated that parents of children with ASD and co-occurring sleep problems reported significantly reduced sleep duration themselves by an average of 2.5 hours per night compared to parents of typically developing children.⁸ This sleep deprivation in caregivers is associated with increased rates of depression, reduced parenting efficacy, and impaired occupational functioning.

Sibling sleep is frequently disrupted by the vocalisation, mobility, and nocturnal demands of children with ASD, a dimension of family impact that receives insufficient research attention. Families of lower socioeconomic status who are disproportionately represented in Indian public health settings face compounded vulnerabilities, including overcrowded sleeping arrangements, limited access to specialist sleep services, and fewer resources to implement recommended environmental modifications.⁴⁸

Children with ASD themselves suffer cascading consequences from poor sleep. Malow et al (2006) documented associations between objectively measured short sleep duration and increased repetitive behaviours, social withdrawal, and irritability in ASD, suggesting that sleep loss exacerbates the core features of the disorder itself a clinically important bidirectionality.⁴⁹ Academic functioning, adaptive behaviour, and response to behavioural therapies have all been shown to be adversely affected by co-occurring sleep disturbances.⁵⁰

PERSPECTIVES FOR COMMUNITY MEDICINE PRACTICE IN INDIA

From the vantage point of community medicine, sleep disturbances in children with ASD represent a condition that is prevalent, largely remediable, and systematically neglected within existing health system structures. The integration of sleep screening into routine developmental surveillance constitutes a pragmatic and high-yield public health strategy.

The Reproductive and Child Health (RCH) programme and Integrated Child Development Services (ICDS) provide platforms through which community health workers and Anganwadi workers could be sensitised to the early signs of neurodevelopmental delay and co-occurring sleep problems. The inclusion of sleep-specific questions within developmental screening instruments used at the primary care level such as the Developmental Assessment for Indians (DASII) or Modified Checklist for Autism in Toddlers (M-CHAT) follow-up protocols could substantially increase case identification at an earlier, more modifiable stage.⁵¹

Tertiary care institutions with dedicated child neurology and developmental paediatrics services including AIIMS centres across India have an important role in conducting population-based prevalence studies using standardised, culturally adapted instruments such as the CSHQ. The Bangalore-based case-control study represents a methodologically sound model that could be replicated at scale in other urban and rural centres, generating Indian normative data currently absent from the literature.¹⁶

Parental psychoeducation, which is among the most cost-effective and evidence-supported sleep interventions, can be delivered through group-based parent training programmes at district mental health programme (DMHP) facilities, NGO-run ASD support centres, and

via digital health platforms in resource-limited settings. Telemedicine, accelerated by the COVID-19 pandemic, offers a feasible modality for extending behavioural sleep coaching to families in geographically underserved regions.⁵²

GAPS IN RESEARCH AND FUTURE DIRECTIONS

Despite a substantial and growing body of international literature, several important gaps remain. First, there is a conspicuous paucity of population-based, longitudinal epidemiological data on ASD-associated sleep disturbances from South Asian countries, including India. Prevalence estimates, risk factor profiles, and the longitudinal trajectory of sleep problems in Indian children with ASD remain largely extrapolated from Western populations, which may not be directly applicable given differences in family structure, sleeping arrangements, cultural attitudes towards children's sleep, and the burden of co-occurring medical comorbidities.

Second, long-term safety data for melatonin supplementation in prepubertal children represent an important regulatory and clinical gap. While short-term tolerability is well-established, the effects of chronic melatonin administration on pubertal timing, reproductive hormone axis maturation, and the hypothalamic-pituitary-adrenal axis require adequately powered, placebo-controlled follow-up studies extending to adolescence.

Third, the majority of intervention trials have enrolled predominantly school-aged children; evidence for children below 4 years of age and for adolescents is comparatively thin. Adapting behavioural interventions for very young children in whom the ASD phenotype may not yet be fully characterised and for adolescents with age-specific psychosocial concerns represents a practical research priority.

Fourth, the relationship between sleep disturbance and specific domains of ASD severity including social communication, restricted-repetitive behaviours, and sensory processing profiles requires further investigation using validated, multidimensional ASD outcome measures. This would help identify which subgroups are at greatest risk and most likely to benefit from targeted sleep intervention.

Fifth, digital health tools including smartphone applications, wearable sleep monitors, and AI-assisted sleep coaching represent promising avenues for scalable, low-cost intervention delivery in low-resource settings. Rigorous evaluation of their efficacy, acceptability, and safety in ASD populations, and specifically in Indian healthcare contexts, is warranted.

CONCLUSION

Sleep disturbances are among the most prevalent, clinically impactful, and therapeutically tractable

comorbidities in children with autism spectrum disorder. Affecting the majority of this population and reverberating through entire family systems, they demand systematic clinical attention commensurate with their public health significance. The evidence reviewed in this article supports a structured, stepped-care approach: comprehensive clinical and questionnaire-based sleep assessment in all children with ASD, followed by parent-delivered behavioural sleep education as the first-line intervention, with judicious use of melatonin and other pharmacological agents were indicated by the clinical picture.

For community medicine practitioners in India, the imperative is twofold: first, to integrate sleep screening into existing developmental surveillance systems, and second, to build the research infrastructure necessary to generate Indian-specific epidemiological data and to evaluate culturally adapted interventions. The case-control study conducted in Bangalore whilst appropriately limited in scale provides a methodological template and an empirical starting point for this endeavour. Achieving the broader goal of reducing the sleep-related burden in ASD will require coordinated investment from policymakers, clinicians, researchers, and the families who bear the greatest weight of this condition.

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REFERENCES

- Diekelmann S, Born J. The memory function of sleep. *Nat Rev Neurosci*. 2010;11(2):114-26.
- Walker MP. The role of sleep in cognition and emotion. *Ann N Y Acad Sci*. 2009;1156(1):168-97.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington (VA): American Psychiatric Publishing; 2013.
- Maenner MJ, Warren Z, Williams AR, Amoakohene E, Bakian AV, Bilder DA, et al. Prevalence and characteristics of autism spectrum disorder among children aged 8 years—Autism and Developmental Disabilities Monitoring Network, 11 sites, United States, 2020. *MMWR Surveill Summ*. 2023;72(2):1-14.
- Chauhan A, Sahu JK, Jaiswal N, Kumar K, Agarwal A, Kaur J, et al. Prevalence of autism spectrum disorder in Indian children: a systematic review and meta-analysis. *Neurol India*. 2019;67(1):100-4.
- Mannion A, Leader G. Sleep problems in autism spectrum disorder: a literature review. *Rev J Autism Dev Disord*. 2014;1(2):101-9.
- Cortesi F, Giannotti F, Ivanenko A, Johnson K. Sleep in children with autistic spectrum disorder. *Sleep Med*. 2010;11(7):659-64.
- Meltzer LJ. Factors associated with depressive symptoms in parents of children with autism spectrum disorders. *Res Autism Spectr Disord*. 2011;5(1):361-7.
- Hodge D, Carollo TM, Lewin M, Hoffman CD, Sweeney DP. Sleep patterns in children with and without autism spectrum disorders: developmental comparisons. *Res Dev Disabil*. 2014;35(7):1631-8.
- Richdale AL, Schreck KA. Sleep problems in autism spectrum disorders: prevalence, nature, and possible biopsychosocial aetiologies. *Sleep Med Rev*. 2009;13(6):403-11.
- Goldman SE, Richdale AL, Clemons T, Malow BA. Parental sleep concerns in autism spectrum disorders: variations from childhood to adolescence. *J Autism Dev Disord*. 2012;42(4):531-8.
- Mazurek MO, Sohl K. Sleep and behavioral problems in children with autism spectrum disorder. *J Autism Dev Disord*. 2016;46(6):1906-15.
- Reynolds AM, Malow BA. Sleep and autism spectrum disorders. *Pediatr Clin North Am*. 2011;58(3):685-98.
- Sivertsen B, Posserud MB, Gillberg C, Lundervold AJ, Hysing M. Sleep problems in children with autism spectrum problems: a longitudinal population-based study. *Autism*. 2012;16(2):139-50.
- Baker EK, Richdale AL. Sleep patterns in adults with a diagnosis of high-functioning autism spectrum disorder. *Sleep*. 2015;38(11):1765-74.
- Sultana N, Asaduzzaman M, Al Mamun F, Hosen I, Yu Q, Pakpour AH, et al. Sleep Problems in Children with Autism Spectrum Disorder in Bangladesh: A Case-Control Study. *Nature and Science of Sleep*. 2021;13:673–682.
- Tordjman S, Anderson GM, Pichard N, Charbuy H, Touitou Y. Nocturnal excretion of 6-sulphatoxymelatonin in children and adolescents with autistic disorder. *Biol Psychiatry*. 2005;57(2):134-8.
- Melke J, Goubran Botros H, Chaste P, Betancur C, Nygren G, Anckarsäter H, et al. Abnormal melatonin synthesis in autism spectrum disorders. *Mol Psychiatry*. 2008;13(1):90-8.
- Mulder EJ, Anderson GM, Kemperman RF, Oosterloo-Duinkerken A, Minderaa RB, Kema IP. Urinary excretion of 5-hydroxyindoleacetic acid, serotonin and 6-sulphatoxymelatonin in normoserotonemic and hyperserotonemic autistic individuals. *Neuropsychobiology*. 2010;61(1):27-32.
- Marco EJ, Hinkley LBN, Hill SS, Nagarajan SS. Sensory processing in autism: a review of neurophysiologic findings. *Pediatr Res*. 2011;69(5 Pt 2):48R-54R.
- Malow BA, Marzec ML, McGrew SG, Wang L, Henderson LM, Stone WL. Characterizing sleep in children with autism spectrum disorders: a multidimensional approach. *Sleep*. 2006;29(12):1563-71.
- Limoges E, Mottron L, Bolduc C, Berthiaume C, Godbout R. Atypical sleep architecture and the autism phenotype. *Brain*. 2005;128(Pt 5):1049-61.

23. White SW, Oswald D, Ollendick T, Scahill L. Anxiety in children and adolescents with autism spectrum disorders. *Clin Psychol Rev*. 2009;29(3):216-29.
24. Rao PA, Landa RJ. Association between severity of behavioral phenotype and comorbid attention deficit hyperactivity disorder symptoms in children with autism spectrum disorders. *Autism*. 2014;18(3):272-8.
25. Tuchman R, Rapin I. Epilepsy in autism. *Lancet Neurol*. 2002;1(6):352-8.
26. Mazurek MO, Vasa RA, Kalb LG, Kanne SM, Rosenberg D, Keefer A, et al. Anxiety, sensory over-responsivity, and gastrointestinal problems in children with autism spectrum disorders. *J Abnorm Child Psychol*. 2013;41(1):165-76.
27. Nicholas B, Rudrasingham V, Nash S, Kirov G, Owen MJ, Wimpory DC. Association of Per1 and Npas2 with autistic disorder: support for the clock genes/social timing hypothesis. *Mol Psychiatry*. 2007;12(6):581-92.
28. Sebat J, Lakshmi B, Malhotra D, Troge J, Lese-Martin C, Walsh T, et al. Strong association of de novo copy number mutations with autism. *Science*. 2007;316(5823):445-9.
29. Lushington K, Pamula Y, Martin J, Kennedy JD. Genetic and environmental influences on sleep problems: evidence from twin studies. *Sleep Medicine Reviews*. 2014;18(4):351-60.
30. Mindell JA, Owens JA. A clinical guide to pediatric sleep: diagnosis and management of sleep problems. 3rd ed. Philadelphia (PA): Lippincott Williams & Wilkins; 2015.
31. Owens JA, Spirito A, McGuinn M. The Children's Sleep Habits Questionnaire (CSHQ): psychometric properties of a survey instrument for school-aged children. *Sleep*. 2000;23(8):1043-51.
32. Goodlin-Jones BL, Sitnick SL, Tang K, Liu J, Anders TF. The Children's Sleep Habits Questionnaire in toddlers and preschool children. *J Dev Behav Pediatr*. 2008;29(2):82-8.
33. Wiggs L, Stores G. Sleep patterns and sleep disorders in children with autistic spectrum disorders: insights using parent report and actigraphy. *Dev Med Child Neurol*. 2004;46(6):372-80.
34. Miano S, Ferri R. Epidemiology and management of insomnia in children with autistic spectrum disorders. *Paediatr Drugs*. 2010;12(2):75-84.
35. Cohen S, Conduit R, Lockley SW, Rajaratnam SM, Cornish KM. The relationship between sleep and behavior in autism spectrum disorder (ASD): a review. *J Neurodev Disord*. 2014;6(1):44.
36. Malow BA, Byars K, Johnson K, Weiss S, Bernal P, Goldman SE, et al. A practice pathway for the identification, evaluation, and management of insomnia in children and adolescents with autism spectrum disorders. *Pediatrics*. 2012;130(Suppl 2):S106-24.
37. Mindell JA, Kuhn B, Lewin DS, Meltzer LJ, Sadeh A; American Academy of Sleep Medicine. Behavioral treatment of bedtime problems and night wakings in infants and young children. *Sleep*. 2006;29(10):1263-76.
38. Adkins KW, Molloy C, Weiss SK, Reynolds A, Goldman SE, Burnette C, et al. Effects of a standardized pamphlet on insomnia in children with autism spectrum disorders. *Pediatrics*. 2012;130(Suppl 2):S139-44.
39. Papadopoulos N, Sciberras E, Hiscock H, Mulraney M, McGillivray J, Rinehart N. The efficacy of a brief behavioral sleep intervention in school-aged children with ADHD and comorbid autism spectrum disorder. *J Atten Disord*. 2019;23(4):341-50.
40. Hollway JA, Aman MG. Pharmacological treatment of sleep disturbance in developmental disabilities: a review of the literature. *Res Dev Disabil*. 2011;32(3):939-62.
41. Gringras P, Gamble C, Jones AP, Wiggs L, Williamson PR, Sutcliffe A, et al. Melatonin for sleep problems in children with neurodevelopmental disorders: randomised double masked placebo controlled trial. *BMJ*. 2012;345:e6664.
42. Wright B, Sims D, Smart S, Alwazeer A, Alderson-Day B, Allgar V, et al. Melatonin versus placebo in children with autism spectrum conditions and severe sleep problems not amenable to behaviour management strategies: a randomised controlled crossover trial. *J Autism Dev Disord*. 2011;41(2):175-84.
43. Bruni O, Alonso-Alconada D, Besag F, Biran V, Braam W, Cortese S, et al. Current role of melatonin in pediatric neurology: clinical recommendations. *Eur J Paediatr Neurol*. 2015;19(2):122-33.
44. McCracken JT, McGough J, Shah B, Cronin P, Hong D, Aman MG, et al. Risperidone in children with autism and serious behavioral problems. *N Engl J Med*. 2002;347(5):314-21.
45. Gee BM, Schlough A, Lackey K. The effects of weighted blankets on sleep in children with ASD: a pilot study. *Open J Occup Ther*. 2020;8(2):1-14.
46. Kripke DF. Light treatment for nonseasonal depression: speed, efficacy, and combined treatment. *J Affect Disord*. 1998;49(2):109-17.
47. Adams L, Gouvousis A, VanLue M, Waldron C. Social story intervention: improving communication skills in a child with an autism spectrum disorder. *Focus Autism Other Dev Disabil*. 2004;19(2):87-94.
48. Khanna R, Madhavan SS, Smith MJ, Patrick JH, Tekka H, Becker-Cottrill B. Assessment of health-related quality of life among primary caregivers of children with autism spectrum disorders. *J Autism Dev Disord*. 2011;41(9):1214-27.
49. Souders MC, Mason TB, Valladares O, Bucan M, Levy SE, Mandell DS, et al. Sleep behaviors and sleep quality in children with autism spectrum disorders. *Sleep*. 2009;32(12):1566-78.
50. Rzepecka H, McKenzie K, McClure I, Murphy S. Sleep, anxiety and challenging behaviour in children with intellectual disability and/or autism spectrum disorder. *Res Dev Disabil*. 2011;32(6):2758-66.

51. Dumont-Mathieu T, Fein D. Screening for autism in young children: The Modified Checklist for Autism in Toddlers (M-CHAT) and other measures. *Mental Retardation and Developmental Disabilities Research Reviews*. 2005;11(3):253-62.
52. Lindly OJ, Thorburn S, Zuckerman KE. Use and nondisclosure of complementary health approaches among US children with developmental disabilities. *J Dev Behav Pediatr*. 2018;39(3):217-27.

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