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Neurobiological and Cognitive Mechanisms of Attention Deficit Hyperactivity Disorder (ADHD) Risk Associated with Screen Exposure in Preschool Children: Literature Review

Lisra Naila Mangendara[1], Titi Pambudi Karuniawaty[2], Rifa Atuzzaqiyah[3]
[1]Medical Study Program, Faculty of Medicine and Health Sciences, University of Mataram
[2,3]Department of Child Health, Faculty of Medicine and Health Sciences, University of Mataram

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Abstract
The preschool period is a critical stage of neurodevelopment, marked by rapid brain maturation and the development of executive functions. In the digital era, excessive screen exposure has raised concerns regarding its potential association with attention deficit hyperactivity disorder (ADHD). This literature review synthesizes evidence on the neurobiological, cognitive, and motor mechanisms linking screen time to ADHD risk in early childhood. Peer-reviewed studies published from 2017–2026 were identified through PubMed, ScienceDirect, and Google Scholar. Evidence suggests a dose-response relationship, with screen exposure exceeding 1 hour per day associated with increased ADHD symptoms. Rapid-paced media may overstimulate attention pathways and affect prefrontal and dopaminergic function, while bedtime screen use may disrupt sleep through melatonin suppression. Additionally, sedentary screen time can reduce physical activity and potentially affect motor development. Therefore, supervised limitation of screen time to a maximum of 1 hour per day may support healthy neurodevelopment during early childhood.

Corresponding Author:
Lisra Naila Mangendara,
Faculty of Medicine and Health Sciences, University of Mataram
Email: lisranaila0@gmail.com

1. INTRODUCTION

The conceptual understanding of childhood has undergone a fundamental paradigm shift throughout human history. During the medieval era, children were often treated merely as miniature adults who were immediately burdened with adult work responsibilities, marriage, and legal consequences upon reaching seven years of age [1]. Structured scientific inquiry into child developmental ethology began to emerge following Darwin's landmark publication in 1877 [1]. By the 20th century, modern developmental psychology established the preschool age (3 to 6 years) as a critical golden age of human neurodevelopment that is exceptionally sensitive to environmental stimuli [2]. According to Jean Piaget's cognitive development theory, preschool children are in the pre-operational stage, characterized by symbolic thinking, expanding imagination, and the transition toward language acquisition [1]. This biological phase of frontal circuit maturation runs parallel to the formation of selective attentional control and behavioral self-regulation [3].

However, the natural developmental trajectory of preschool children in the contemporary era faces unprecedented environmental challenges. Interactive digital devices such as smartphones, tablets, video game consoles, and smart televisions have thoroughly penetrated the nuclear family caregiving environment [4]. Screen time is clinically defined as the duration of passive or active interaction with digital screen-based electronic devices [5]. Public health bodies, including the World Health Organization (WHO) and the Indonesian Pediatric Society (IDAI), consistently recommend a maximum limit of 1 hour per day of sedentary screen exposure for preschool children aged 2 to 5 years, while strictly advising zero screen exposure for infants under 18–24 months [6],[7]. Despite the widespread dissemination of these medical guidelines, recent epidemiological data reveal an alarming global non-compliance rate of 70% to 75%, with preschoolers exposed to an average of 2 to 3 hours of screen time daily [8].

Prolonged screen time has been clinically linked to an increased risk of Attention Deficit Hyperactivity Disorder (ADHD) [9],[10]. ADHD is a neurodevelopmental disorder characterized by a core triad of symptoms: inattention, excessive motor hyperactivity, and age-inappropriate impulsivity [11]. Globally, the prevalence of ADHD among preschool and school-aged children has risen dramatically to 5%–8% [9],[12]. Cross-sectional and prospective cohort investigations, such as those reported by Wu et al. [13] and Hill et al. [14], confirm a robust dose-response relationship between excessive screen duration and heightened ADHD symptom severity. Nevertheless, scientific literature addressing the specific neurobiological, structural, motor development, and digital parenting mechanisms remains scattered across separate fields.

Therefore, this literature review aims to synthesize, align, and comprehensively detail the neurobiological and cognitive mechanisms underlying increased ADHD risk from excessive screen exposure in preschool children. By synthesizing evidence from structural neuroimaging, dopaminergic neurochemistry, and the displacement hypothesis, this article provides a solid theoretical foundation for clinicians, educators, and parents to implement precise digital parenting and neuroprotective intervention strategies

2. LITERATURE REVIEW

Dynamics of Attention Development and Preschool Behavioral Milestones

Attention is not a single, isolated cognitive capacity, but rather a complex neural circuit that undergoes continuous differentiation and maturation during childhood [1]. The development of attention during the preschool years can be mapped across specific age milestones [1],[2]:

1. Ages 2–3 Years: Attention remains heavily stimulus-driven (bottom-up control). Attention span is brief (only a few minutes), and selective attention to filter out external distractions is extremely limited [1].
2. Ages 3–4 Years: Directed attention shows moderate improvement, ranging from 5 to 10 minutes during structured activities. Children begin developing joint attention with adults, though they remain easily distracted by novel sensory stimuli [2],[15].
3. Ages 4–5 Years: Sustained attention and selective attention capacity increase significantly. Children can maintain focus for 10 to 15 minutes on engaging tasks and begin successfully filtering out minor environmental distractions [2],[11].
4. Ages 5–6 Years: Goal-directed (top-down) attentional control approaches functional maturation. Children can voluntarily direct attention for 15 to 20 minutes, suppress irrelevant behavioral impulses, and achieve foundational cognitive readiness for formal schooling [2],[12].

Behavioral and emotional regulation in preschoolers is governed by prefrontal cortical circuits that mature at a slower pace relative to sensory pathways [1]. Consequently, behavioral control in early childhood remains labile as children are driven by a natural instinct for instant gratification [16]. Training impulse control and delayed gratification is ideally achieved through active sensorimotor physical exploration, manipulation of threedimensional objects in real space, and reciprocal interpersonal communication with caregivers [3],[4].

Pathophysiology of ADHD: Disruption of Frontostriatal Brain Circuitry

Neuropsycbiatrically, the pathophysiology of ADHD is rooted in structural and functional dysfunction of frontal circuits, specifically frontostriatal pathways linking the prefrontal cortex to the striatum [17]. Functional MRI and structural neuroimaging studies demonstrate delayed cortical maturation or cortical thinning in key executive attention regions [17]. Key affected areas include the Dorsolateral Prefrontal Cortex (DLPFC), which coordinates working memory, planning, and behavioral inhibition, and the Anterior Cingulate Cortex (ACC), which plays a pivotal role in attentional allocation, distraction filtering, and cognitive decision-making [17]. Structural deficits in the ACC and DLPFC manifest clinically as impaired behavioral inhibition, inattention, and executive dysfunction [17].

3. RESEARCH METHODS

This study employs a qualitative narrative literature review design to integrate secondary data from peer-reviewed international and accredited national journals [10]. A structured search was performed using Boolean operator keyword combinations: ('screen time' OR 'digital media exposure' OR 'electronic media') AND ('ADHD' OR 'attention deficit') AND ('neurobiology' OR 'brain structure' OR 'executive function') AND ('preschool' OR 'toddler') across PubMed, ScienceDirect, and Google Scholar.

Inclusion criteria were: (1) target population focused on toddlers or preschool children (ages 3–6 years); (2) evaluation of screen time duration, frequency, or media usage patterns as independent variables; (3) assessment of attentional networks, cognitive/gross motor development, sleep quality, or clinical ADHD symptoms as dependent variables; and (4) published exclusively within the last 10 years (2017 to 2026) to ensure the validity and current relevance of neuroimaging findings. Studies lacking full-text access or published prior to 2017 were excluded from synthesis.

4. RESULTS AND DISCUSSION

Results

Following rigorous screening against inclusion criteria, 11 primary empirical investigations (published between 2017 and 2026) were identified. These studies provide epidemiological, clinical, and neuroimaging evidence regarding the impact of screen exposure on ADHD risk in early childhood. A summary of this synthesized literature is presented in **Table 1.**

Table 1. Synthesis of Key Empirical Literature on Screen Time Exposure and ADHD Outcomes (2017–2026)

Author & Year	Design & Sample	Variables Measured	Key Findings & Clinical Implications
Ouyang et al. (2023) [15]	*Crossectional*; 543 Chinese Preschoolers	Screen duration, emotional regulation, attention problems	Daily screen exposure >2 hours independently correlated with a 24% increase in attention problems and severe emotional regulation deficits.

Wu et al. (2022) [13]	Cross-sectional; 1,000+ children <3 years	Daily screen time, hyperactive behavior, ADHD risk	Children exposed to screens >120 minutes/day exhibited a 3.687-fold higher risk of clinical hyperactive and ADHD-like behaviors.
Yuan et al. (2024) [19]	Cross-sectional; 817 preschoolers	Daily screen time, gross motor scores	Identified a strong negative correlation; screen time >2 hours/day reduced gross motor movement scores by 13.2% due to sedentary behavior.
Hill et al. (2024) [14]	Prospective longitudinal cohort; 18 mos. to preschool	Early screen exposure, autism & ADHD symptoms	Excessive early screen exposure (at 18 months) serves as a strong longitudinal predictor for the onset of inattention and hyperactivity symptoms at ages 3–5.
Hutton et al. (2020) [18]	Cross-sectional & fMRI/DTI; 47 healthy preschoolers	Daily screen score, white matter integrity (DTI)	Higher screen exposure significantly correlated with lower microstructural white matter integrity (lower FA) in executive function tracts.
Lissak (2018) [20]	Systematic review & case study	Sleep physiology, screen duration, melatonin	Excessive screen exposure suppresses nighttime melatonin secretion, inducing sleep deficiency that exacerbates inattention and hyperactivity.
Tamana et al. (2019) [21]	Prospective cohort; 2,400+ preschoolers	Daily screen duration at age 5, behavioral reports	Screen time >2 hours/day at age 5 was strongly associated with a 7.7-fold increased risk of clinically significant inattention symptoms.
Madigan et al. (2019) [22]	Longitudinal cohort; 2,441 Canadian children	Daily screen time at 24 & 36 mos., developmental milestones	High screen duration at 24 & 36 months consistently predicted developmental delay across milestones at ages 36 & 60 months.
Small et al. (2020) [23]	Multidisciplinary review	Digital technology impacts, dopamine reward pathways	Fast-paced digital media stimulates rapid dopamine release in mesolimbic reward circuits, causing post-synaptic receptor downregulation.
Shou et al. (2025) [17]	Longitudinal cohort & neuroimaging; 1,243 children	Daily screen duration, cortical thickness, ADHD severity	Provided direct neuroimaging evidence that chronic screen exposure mediates ADHD symptom severity through cortical thinning in attentional networks.

Nathanson & Beyens (2018) [24]	Cross-sectional; 385 preschoolers	Bedtime device use, sleep resistance, daytime sleepiness	Bedtime screen usage triggered severe bedtime resistance, shortened sleep duration, and caused secondary inattention from daytime sleepiness.

Discussion

Sensory Overstimulation Hypothesis and Dopaminergic Desensitization

The primary neurocognitive mechanism linking excessive screen exposure to heightened ADHD risk is formulated under the Overstimulation Hypothesis [15],[20]. Contemporary pediatric digital content is engineered with rapid scene transitions, intense flashing visual effects, and loud auditory cues. This dynamic visual input continuously recruits and triggers the developing child's bottom-up (reflexive) attentional pathway [18]. Consequently, the child's neural attentional circuitry adaptively accommodates to this high sensory intensity, raising the baseline arousal threshold [20].

When the child transitions from high-stimulation digital screens to low-stimulation, real-world environments (such as listening to classroom instruction, reading a book, or organizing physical toys), the neural attentional system perceives the natural environment as understimulating and mundane [15],[20]. The child fails to maintain sustained attention because their frontal circuits have been conditioned to constantly seek rapid stimulus novelty, manifesting clinically as severe inattention, physical restlessness, and behavioral impulsivity [20].

This behavioral response is reinforced by neurochemical disruption within the brain's mesolimbic dopaminergic reward pathway [23]. Highly interactive screen engagement (especially digital games offering instant points, rewards, or visual celebration animations) triggers rapid, massive surges of dopamine within the nucleus accumbens [23]. This immediate dopaminergic feedback loop mimics chemical addiction mechanisms [20]. Chronic exposure to high frequency dopaminergic spikes during early childhood induces a neuroprotective cellular downregulation, a reduction in postsynaptic dopamine receptor density and sensitivity [23]. Consequently, preschoolers experience dopaminergic desensitization, requiring increasingly intense digital stimulation to achieve cognitive satisfaction [20],[23]. This deprives the prefrontal cortex of steady basal dopamine levels essential for training voluntary top down attentional focus and impulse control [23].

Structural Brain Alterations: Cortical Thinning and White Matter Microstructural Alterations

Direct physical evidence of excessive screen time during preschool years is documented through structural neuroimaging [17],[18]. A large scale longitudinal investigation by Shou et al. [17] demonstrated that increased daily screen duration mediates ADHD symptom severity through cerebral cortical thinning. This cortical thinning occurs prominently in frontal and parietal regions that constitute executive attentional networks [17]. During early childhood, top down control circuits require active physical engagement that demands action planning, impulse inhibition, and distraction filtering [15],[18]. When physical play is displaced by passive screen viewing, the DLPFC and ACC fail to receive adequate cognitive stimulation, impeding gray matter volume maturation and cortical thickness development [17].

In addition to gray matter alterations, excessive screen exposure compromises the microstructural architecture of brain white matter [18]. Utilizing Diffusion Tensor Imaging (DTI), Hutton et al. [18] observed that preschoolers with high screen usage scores exhibited significantly lower fractional anisotropy (FA) and higher radial diffusivity (RD) within the arcuate fasciculus, superior longitudinal fasciculus, and uncinate fasciculus. These white matter tracts serve as neural pathways coordinating cognitive processing speed, language, and executive control between frontal regions and the rest of the brain [18]. Reduced white matter microstructural integrity reflects delayed myelination, correlating directly with cognitive processing delays, poor working memory, and heightened vulnerability to ADHD inattention [18].

The Displacement Hypothesis and Motor-Cognitive Synergy

The impact of screen exposure on ADHD risk also operates indirectly through the Displacement Hypothesis [8]. Every hour spent staring at a screen displaces essential developmental activities: physical motor play, face to face social interaction, and restorative sleep [24],[25].

Active physical play is critical because gross motor control circuits and cognitive impulse control circuits share overlapping, synergistic neural pathways within the cerebellum and striatum [19]. A quantitative study by Yuan et al. [19] in 817 preschoolers revealed that screen time >2 hours/day reduced gross motor movement scores by 13.2% due to sedentary behavior. Lack of physical stimulation leaves motor inhibitory pathways untrained, manifesting as physical restlessness, fidgeting, and clinical motor hyperactivity [11],[19].

Furthermore, screen exposure near bedtime severely disrupts pineal melatonin secretion due to high-energy blue light emitted by digital displays [20],[24]. This induces bedtime resistance, delayed sleep onset, and reduced total sleep duration [24]. Chronic sleep deficiency causes daytime prefrontal hypofunction, impairing emotional self-regulation, increasing temper tantrums, and exacerbating secondary inattention and impulsivity mimicking ADHD [20],[24].

Socio-Demographic Influences and Digital Parenting

Although screen exposure exerts universal neurobiological impacts, child vulnerability is heavily moderated by family sociology and digital parenting practices [24],[25]. In modern urban settings, high employment demands create double burdens for working mothers managing domestic responsibilities alongside childcare [4],[26]. In states of parental physical and emotional fatigue, technoference, the interruption of parent-child interactions by digital devices, frequently occurs [25].

Consequently, digital screens are repurposed as 'digital nannies' or 'digital pacifiers' to soothe child distress without requiring direct emotional engagement [16],[26]. Permissive, unsupervised screen use is strongly associated with a 4 fold increased risk of ADHD symptoms [16],[24]. Conversely, active parental mediation, where parents physically co-view, select educational content, and engage in interactive discussion, serves as a protective buffering factor that mitigates sensory overstimulation and supports verbal executive function development [24],[27].

5. CONCLUSION

Excessive and unsupervised screen exposure during preschool years significantly increases ADHD risk through multidimensional neurobiological and behavioral mechanisms. Neurobiologically, fast-paced media overstimulates bottom-up attentional circuits, downregulates dopaminergic receptors, compromises white matter tract myelination, and induces cortical thinning in executive frontal regions (DLPFC and ACC). Behaviorally, screen time displaces gross motor physical activity, impairs motor-cognitive inhibitory control, and induces sleep deficiency via melatonin suppression.

Clinically, limiting screen exposure to a maximum of 1 hour per day (in accordance with WHO and IDAI guidelines, with zero screen time for children under 2 years) represents a critical neuroprotective preventive strategy. Active digital parenting and consistent active parental mediation are essential to safeguard optimal neurodevelopment and prevent persistent attentional deficits in early childhood.

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