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EMOTIONAL REACTIVITY AND REGULATION IN CHILDREN AND ADOLESCENTS WITH ADHD: A NARRATIVE REVIEW OF CLINICAL AND NEUROPSYCHOLOGICAL EVIDENCE

Rona Retsa Aurellia [1] and Yunias Setiawati [1, 2, 3, *]

[1] Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.
[2] Department of Psychiatry, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.
[3] Department of Psychiatry, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia.
* Corresponding Author

ORCID Details
Rona Retsa Aurellia: https://orcid.org/0009-0009-0126-4802
Yunias Setiawati: https://orcid.org/0000-0002-5920-3676

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ABSTRACT

Emotional difficulties are common and impairing in children and adolescents with attention-deficit/hyperactivity disorder (ADHD), yet emotional reactivity and emotion regulation are often treated as a single construct. This narrative review examines whether the distinction is supported by clinical, neuropsychological, psychophysiological and treatment evidence published from 2016 to October 2026, together with key theoretical work. Both domains are impaired, with larger meta-analytic effects for reactivity and lability than for regulation. Emotion dysregulation is associated with aggression, maladaptive regulation strategies with depressive symptoms, and irritability with both externalizing and internalizing outcomes. Working memory is consistently linked to emotion regulation, including experimentally, whereas irritability is unrelated to performance-based executive function. Autonomic and neuroimaging findings point to interacting affective and regulatory processes. In a recent randomized trial, methylphenidate and atomoxetine reduced emotional lability beyond ADHD symptom change without altering self-reported regulation strategies. These findings support assessing reactivity and regulation separately. A two-pathway account is a useful working hypothesis, but measurement overlap and largely observational designs prevent confirmation of independent mechanisms.

Keywords: ADHD; Emotional Reactivity; Emotion Regulation; Children; Adolescents; SDG 3 Good Health and Well-Being

1 INTRODUCTION

Attention deficit/hyperactivity disorder (ADHD) is one of the most common neurodevelopmental disorders in childhood, with a worldwide prevalence estimated at 8.0% [1]. Salari et al. (2023) estimated 7.6% of 96,907 children aged 3 to 12 years had ADHD (95% confidence interval: 6.1–9.4%), while 5.6% of teenagers aged 12 to 18 years have ADHD [2]. Its diagnosis rests on inattention and hyperactivity-impulsivity, whereas irritability, low frustrations

tolerance and mood lability are listed only as associated features [3]. Emotion dysregulation has nonetheless been estimated to affect approximately 25% of children with ADHD[4], although estimates depend strongly on the threshold applied.

The place of emotion in ADHD remains contested. Self-regulation of affect was included among the executive functions in early theory [5]; later work considered whether emotion dysregulation is a core feature, a correlated dimension or the marker of a subgroup [4]; and a practitioner review noted that no sufficiently specific definition has been agreed [6].

Part of the difficulty lies in the construct itself. Emotion science distinguishes the generation of an emotional response from its regulation [7], and a recent scoping review identified the same two themes in definitions of dysregulation in middle childhood [8]. A child may react intensely but recover quickly, or react modestly but fail to recover, yet both can receive the same global dysregulation score. The question parallels earlier work on ADHD heterogeneity, in which similar presentations were proposed to arise through separable executive and motivational routes [9,10].

Earlier syntheses documented emotional impairment in ADHD [11,12], an a recent meta-analysis contrasted lability with regulation [13], but subsequent clinical, cognitive, physiological and treatment findings have not been integrated around this distinction. This narrative review draws on literature identified through PubMed, theoretical and measurement work, with priority given to children and adolescents aged 6-18 years. The original search dates, complete Boolean strategies and screening counts were not prospectively documented. Selected citations and contested claims were checked on 29th October 2026 through exact-title/DOI searches of PubMed-indexed records and publisher pages; the substantive evidence window remains 2016–October 2026. This verification was not a rerun of an exhaustive PubMed search. Explicit reporting of aims, searching, references and evidence limitations was informed by SANRA [14–22]. It addresses four questions: how reactivity and regulation are defined and measured; how they relate to clinical outcomes; which neuropsychological and psychophysiological findings distinguish them; and whether they respond differently to treatment.

2 CONCEPTUALIZATION AND MEASUREMENT OF EMOTIONAL REACTIVITY AND REGULATION

2.1 Conceptual Distinction

Emotional reactivity describes how readily, quickly and intensely an individual responds to an emotionally relevant event. Emotional lability refers more specifically to rapid changes in emotional state, and negative affect to the tendency to experience distress. Emotion regulation comprises processes that monitor and modify an emotional response in line with situational goals, such as redirecting attention, reappraisal or controlling expression; it does not simply mean suppression [7]. The ADHD terms emotional impulsivity and deficient emotional self-regulation are related but do not map perfectly onto these constructs [6]. Reactivity itself is heterogeneous. In a community cohort of 452 children followed from age 6, tonic irritability (irritable mood) and phasic irritability (temper outbursts) were distinguishable and had different outcomes [23]. Although not specific to ADHD, the finding indicates that reactivity should not be treated as uniform.

2.2 Measurement Approaches

Instrument content must guide construct classification. Shields and Cicchetti's 1997 paper concerns development and validation of an Emotion Regulation Q-Sort scale [24]; the Q-sort should not be conflated with the questionnaire-based Emotion Regulation Checklist (ERC). The ERC subscale descriptions used here are supported by the explicit instrument description in Marques et al [25]. Lability/negativity includes mood lability, anger dysregulation and inflexibility, whereas Emotion Regulation includes awareness, expression, and empathy. Neither subscale directly isolates a pure generative or regulatory process. Composite Child Behavior Checklist (CBCL) dysregulation profiles combine Anxious/Depressed, Attention Problems, and Aggressive Behavior; inclusion of attention items introduces construct overlap with ADHD [14]. Conners emotional lability scores quantify observable lability rather than strategy use [15]. Repeated observations, behavioral coding, and physiological recording provide complementary and context-dependent information [16–18]. Associations between parent-rated emotional and executive scales may also reflect shared-rater and method variance rather than a specific cognitive mechanism.

The ERC Lability/Negativity subscale captures mood lability and negative affect, whereas the Emotion Regulation subscale assesses adaptive emotional functioning, including emotional awareness and empathy. The BRIEF Emotional Control scale assesses how children modulate their emotions in daily life, and the Conners lability subscale captures observable mood changes. The CBCL dysregulation profile combines emotional, attentional, and behavioral symptoms. Self-report questionnaires assess reported strategy use, facial coding captures observed emotional expressions, and respiratory sinus arrhythmia (RSA) and electrodermal activity (EDA) index autonomic responses during specific tasks.

Because these measures capture different aspects of emotional functioning, they may yield different results. A null finding on one task does not rule out the underlying mechanism [14–22].

2.3 Magnitude of Impairment

Across 77 studies (32,044 participants), children with ADHD differed most from controls in reactivity, negativity and lability (d = 0.95) and somewhat less in emotion regulation (d = 0.80) [11]. Across 75 studies of externalizing disorders (63 on ADHD), emotional lability correlated more strongly with symptom severity (r = 0.47) than emotion regulation did (r = 0.24) [13]. Both domains are therefore substantially affected, but the two meta-analyses used different metrics and populations, and their estimates should not be compared directly.

3 CLINICAL CORRELATES OF EMOTIONAL REACTIVITY AND REGULATION

3.1 Prevalence and Functional Impairment

In 696 children with ADHD. 91% showed at least one irritable symptom and 31% met a three-month definition of disruptive mood dysregulation disorder. Higher irritability was associated with anxiety, depressive symptoms and family history of depression [19]. These figures reflect broad thresholds in a referred sample, but they confirm that emotional symptoms are a major source of impairment that not captured by core criteria [6].

3.2 Externalizing and Internalizing Correlates

Marques et al. examined 38 children with ADHD and 34 controls aged 6–10 years [25]. Within the ADHD group, their cross-sectional model identified a statistical indirect association between inhibitory-control difficulty and aggression through an emotional dysregulation composite. The final composite combined BRIEF Emotional Control and ERC Lability/Negativity after the adaptive ERC regulation subscale was excluded for weak loading. It therefore represents mixed emotional-control/lability content, not isolated strategy use. Neither temporal ordering nor causal mediation can be inferred, and shared parent reporting may contribute to covariance. Wu et al. [20] reported more emotional-control problems, aggression and anxious/depressed mood among children with ADHD and ODD (n = 161) than among those with ADHD alone (n = 354).

Regulation difficulties are linked to internalizing symptoms. Individuals with ADHD reported more self-blame, rumination and catastrophizing, which were associated with depressive symptoms, although the sample was aged 14-34 years and included only nine adolescents [21]. Reactivity is not confined to externalizing outcomes either. Irritability carries depression liability [19], and tonic irritability predicted internalizing whereas phasic irritability predicted externalizing problems [23]. No strict reactivity-externalizing or regulation-internalizing mapping is therefore supported.

3.3 Prospective Associations

In 68 children aged 7–12 years (35 with ADHD), a parent-rated emotion measure labeled reactivity predicted ADHD severity six months later beyond baseline severity, whereas lability did not [16]; the measure, however, was derived from the regulation subscale of the Emotion Regulation Checklist, illustrating inconsistent terminology. Over three years, anger improved in 137 clinically referred youth with ADHD, whereas anxiety and depressive symptoms remained largely stable [22].

3.4 Heterogeneity within ADHD

An irritable temperament profile with high negative affect was replicated in 186 children and, in a pooled sample of 368, improved prediction of clinical outcomes beyond conventional indicators [26]. In 178 children, emotionally dysregulated personality profiles predicted benefit from starting medication over and above baseline ADHD severity [27]. Such profiles are research descriptions rather than diagnostic subtypes.

4 NEUROPSYCHOLOGICAL AND PSYCHOPHYSIOLOGICAL CORRELATES

4.1 Executive Function and Working Memory

Working memory has repeatedly been associated with emotion-related ratings. In 145 children aged 8-13 years, Groves et al. identified direct and indirect associations with emotional regulation through hyperactive/impulsive symptoms [28]. In 151 children, working memory retained a unique association when inhibition and set shifting were considered, while ADHD symptoms also contributed independently [29]. These individual studies do not establish superiority across executive-function domains. Letter-number sequencing was associated with parent-rated emotional control beyond ADHD and ODD symptoms, awithour a diagnosis-specific association [30]. Tarle et al. found disproportionately

increased self-criticism under experimentally increased working-memory demand in ADHD [17]. The manipulation links cognitive load to observed emotional behavior, but may affect frustration, affect generation or expression as well as regulation.

In Brænden et al.'s transdiagnostic referred sample of 208 children aged 6-12 years, irritability was associated with parent-rated emotional control and cognitive flexibility but not with performance-based executive measures [31]. Rating-task differences may reflect context, construct content, and common-method variance. They do not demonstrate a distinct irritability pathway without direct interaction or dissociation tests. Moreover, Bauer et al.'s cross-sectional model linked working memory to higher negative affect [32], and adjustment for cognitive functioning weakened some meta-analytic reactivity associations [11]. Cognitive and emotional response processes therefore cannot be assigned to separate pathways on these cross-study comparisons.

4.2 Motivational and Autonomic Processes

Hulsbosch et al. assessed task choice/response bias, response times after reward or punishment, and coded facial expressions in 53 children with ADHD and 46 controls; the video-analysis subset comprised 49 and 43 children, respectively [33]. Negative facial expressions increased across task blocks in ADHD, while punishment-related response bias did not differ between groups. This is a difference between observed affect and a specified choice metric, not evidence that learning, reinforcement sensitivity or response inhibition is intact or separate. In 259 children aged 5–13 years, including 160 with ADHD, Morris et al. found augmented respiratory sinus arrhythmia and electrodermal reactivity during emotion induction/suppression [18]. Physiological changes are context-dependent correlates, not selective markers of regulatory or reactive deficits.

4.3 Neural Circuitry

Children with ADHD and emotion dysregulation (n = 77) differed from those without (n = 53) and controls (n = 55) in dorsolateral prefrontal-orbitofrontal and amygdala-occipital connectivity, with greater prefrontal-orbitofrontal connectivity related to lower symptoms severity [14]; dysregulation was defined by a composite that includes attention items. Amygdala structural connectivity was associated with emotional problems in children with ADHD with and without ODD [20]. In a population cohort of 6,053 participants and a clinical sample of 672, dysregulation remained associated with ADHD symptoms after cognitive and motivational differences were considered, with mediation implicating right pars orbitalis surface area [34]. A later correction concerned cortical surface visualizations and did not alter the reported analyses or conclusions [35]. Gao et al. associated medication-related lability improvement with changes in amygdala–frontal and amygdala–cingulate connectivity [36]. These change correlations do not establish that connectivity changes mediated treatment effects. These findings support emotion-related neural correlates but do not yet separate reactivity from regulation. Table 1 summarizes the main studies.

Table 1: Neuropsychological and psychophysiological studies

Study	Sample	Design and measures	Emotional construct	Main finding and interpretive limit
Groves et al. 2020 [28]	145 children; 8–13 years; with and without ADHD	Cross-sectional; multiple working memory tasks; informant ratings	Regulation-related rating	Working memory associated with emotion regulation directly and indirectly through hyperactivity/impulsivity. Statistical paths do not establish causation.
Groves et al. 2022 [29]	151 children; 8–13 years; clinically diverse	Cross-sectional; working memory, inhibition, set shifting; parent emotion ratings	Regulation-related rating	Working memory retained a unique association; ADHD symptoms also contributed. No direct test of selective association with regulation versus reactivity.
Jensen et al. 2018 [30]	41 ADHD; 34 controls	Cross-sectional; letter–number sequencing, other memory tasks; BRIEF Emotional Control	Mixed control and lability	Complex verbal working memory associated with emotional control/lability beyond ADHD and ODD symptoms. Association not specific to ADHD.
Tarle et al. 2021 [17]	Children with ADHD and typically	Experimental variation of phonological working	Observed expression and regulation	High demand disproportionately increased self-criticism in ADHD. Cognitive load can affect both

	developing children	memory demand; behavioral coding		elicitation and regulation of emotion.
Bauer et al. 2018 [32]	623 children; 7–12 years; 388 ADHD, 235 controls	Cross-sectional mediation; working memory, inhibition, temperament negative affect	Negative affect	Working memory statistically mediated the ADHD–negative affect association. Challenges an exclusive cognitive link with regulation.
Brænden et al. 2024 [31]	208 referred children; 6–12 years; transdiagnostic	Executive tasks and parent ratings; dimensional irritability; diagnostic subsamples	Irritability and emotional control	Irritability related to everyday control/flexibility ratings, not performance-based executive function. Findings are not exclusively ADHD-specific.
Hulsbosch et al. 2024 [33]	53 ADHD, 46 controls; 6–12 years; video subset 49 and 43	Punishment task; choice behavior and coded facial expression	Reactive expression under demand	Negative expression increased over the task in ADHD; punishment response bias did not differ. Emotional and choice outcomes diverged.
Morris et al. 2020 [18]	259 children; 5–13 years; 160 ADHD	Emotion induction/suppression; RSA, EDA and PEP	Physiological reactivity and regulation	Augmented RSA and EDA reactivity in ADHD. Physiological indices require task-specific interpretation; sample includes preschool age.
Li et al. 2025 [14]	77 ADHD with ED; 53 ADHD without ED; 55 controls	Cross-sectional resting-state connectivity; CBCL dysregulation profile	Mixed CBCL phenotype	Prefrontal–orbitofrontal and amygdala–occipital differences. Greater prefrontal–orbitofrontal connectivity related to lower ED within ADHD+ED.

ADHD, attention-deficit/hyperactivity disorder; BRIEF, Behavior Rating Inventory of Executive Function; CBCL, Child Behavior Checklist; ED, emotional dysregulation; EDA, electrodermal activity; ODD, oppositional defiant disorder; PEP, pre-ejection period; RSA, respiratory sinus arrhythmia. Construct labels reflect operational definitions, not confirmed independent mechanisms. Sample denominators vary by analysis.

4.4 Effects of Treatment on Reactivity and Regulation

Gao et al. randomized 152 children to atomoxetine or methylphenidate (76 per group), followed by individualized titration and four weeks at a stable dose [36]. Emotional lability was the primary outcome and self-reported regulation strategies as secondary outcome. Mixed-effects linear models compared baseline, post-titration and endpoint assessments. Reported standardized changes were d = −1.46 with methylphenidate and d = −0.93 with atomoxetine; these are within-group changes, not placebo-adjusted or between drug effect sized. There was no significant between drug difference. Improvement remained significant after adjustment for ADHD symptom change, but self-reported strategies did not change significantly. This does not demonstrate selective effects on reactivity, equivalence between druges or an independent treatment mechanism. Delayed-release/extended-release methylphenidate improved lability relative to placebo in a post hoc trial analysis [15]; a separate randomised comparison reported greater dysregulation improvement with methylphenidate than neurofeedback [37].

Evidence for change in regulation is weaker. Regulation difficulties improved after six months of methylphenidate in an uncontrolled study of 82 adolescents [38], ever-medicated adolescents showed more favorable emotional patterns in an observational cohort [39], and two executive function training protocols produced similar improvements in dysregulation without a non-executive control condition [40]. Overall, treatment evidence is clearer for observable lability than for the acquisition of adaptive regulation strategies. Pozzi et al analyzed emotional adverse events across heterogeneous medication trials [41]. Irritability and emotional lability are different outcomes, and pooled class-level signals must not be interpreted as agent-specific efficacy or an inevitable response in every child. The available summary does not justify a blanket claim that all methylphenidate formulations reduce irritability or that all amphetamines worsen lability.

4.5 Toward a Two-Pathway Account

The selected evidence suggests difficulties across emotional domains and clinically relevant associations beyond core symptoms [11,13,29,36]. It does not establish independent cognitive mechanisms or differential pharmacological mechanisms. The provisional two-pathway account (Table 2; Figure 1) is an integrative hypothesis organizing affective responsivity, including salience, negative affect, arousal and frustration, alongside regulatory processes, involving goal maintenance, working memory and strategy use. The routes are expected to interact, so that greater demand can both increase distress and reduce the capacity to manage it. This is not an empirically identified dual-mechanism model or a validated classification of patients.

Several findings constrain the model: working memory also relates to negative affect [33], irritability signals internalizing as well as externalizing risk [19,23], and ADHD symptoms contribute to dysregulation after executive functions are considered [28,29]. The evidence therefore favors intersecting rather than independent pathways. A stronger test would measure both domains repeatedly in the same children and show that cognitive load preferentially affects recovery or strategy use, whereas motivational manipulation preferentially affects the initial response.

Table 2: Comparison of emotional response characteristics and regulation

Evidence domain	Reactivity and related response characteristics	Regulation and related processes	Boundary of interpretation
Magnitude of association	Large reactivity/negativity/lability estimate in ADHD synthesis; lability more strongly related to externalizing symptoms [11,13]	Large regulation estimate in ADHD synthesis; weaker symptom correlation in broader externalizing synthesis [11,13]	Different metrics and populations prevent direct pooling or individual-level ranking.
Clinical correlates	Outbursts and aggression are relevant; tonic irritability also relates to internalizing risk [19,23]	Maladaptive strategies relate to depressive symptoms; mixed-age evidence requires caution [21]	No exclusive reactivity–externalizing or regulation–internalizing mapping.
Executive function	Working memory also relates to negative affect and control/lability [30,32]	Working memory associations recur; cognitive demand alters emotional behavior [17,28,29]	A clean double dissociation is unsupported.
Motivation and autonomic response	Punishment-related expression and EDA suggest altered affective responding [18,33]	RSA modulation offers a candidate index of contextual regulation [18]	These are context-dependent indicators rather than diagnostic biomarkers.
Treatment	Randomized evidence supports lability improvement; one active-comparator trial found no significant strategy change [15,36]	Observational reports and cognitive-training trials suggest possible improvement [38–40]	Different informants, outcomes and designs prevent claims of selective treatment action.

This table summarizes patterns across heterogeneous designs. It does not establish independent pathways, diagnostic subtypes, or comparative treatment recommendations. EDA, electrodermal activity; RSA, respiratory sinus arrhythmia.

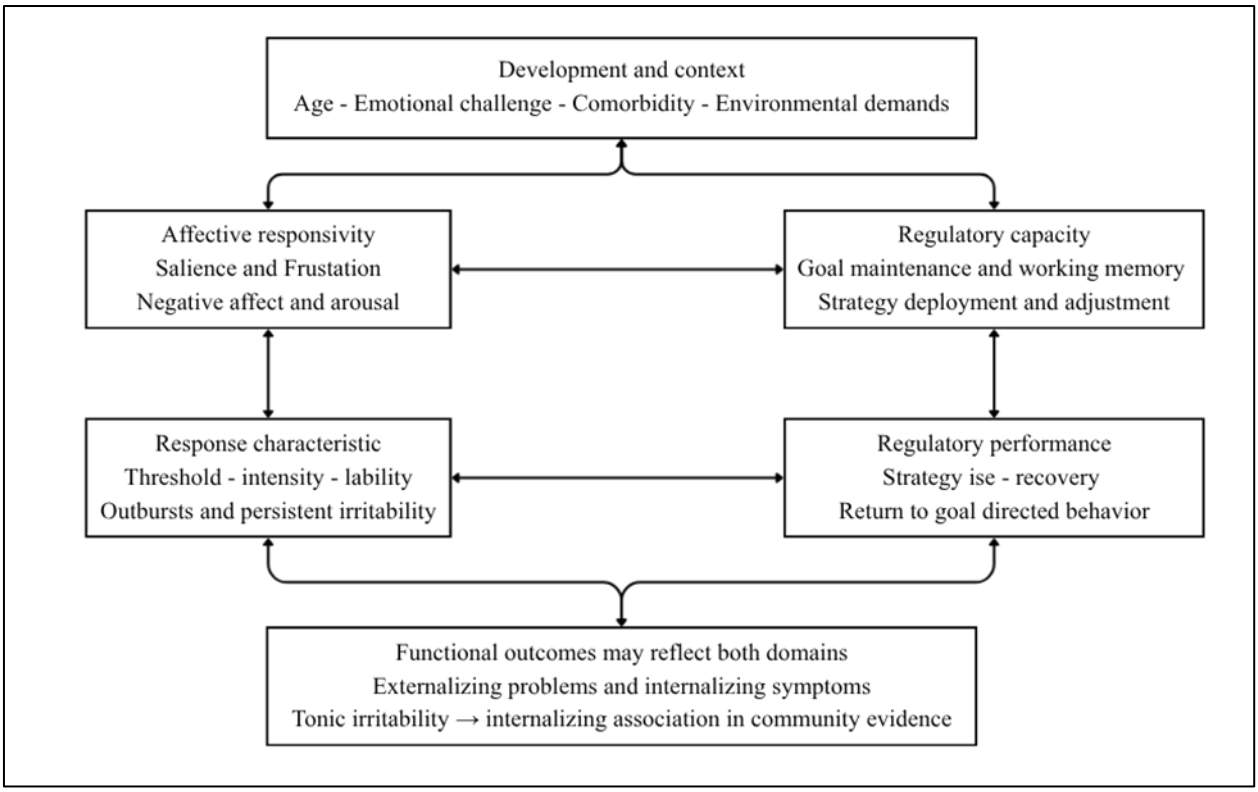


Figure 1: A provisional account of interacting emotional pathways. Dashed arrows represent hypothesized relationships rather than demonstrated causation; horizontal arrows indicate interaction and construct overlap. The tonic irritability–internalizing association comes from community evidence

Limitations and Future Directions

Construct inconsistency is the principal limitation: emotional domains are often measurd with overlapping items or composites that include ADHD symptoms [14,16,25]. Most studies are cross-sectional and small, rely on shared informants and handle ODD and medication inconsistently. Potential cohort overlap must be checked rather than assumed from institutional similarity [26,32]. Some evidence comes from mixed-age or community samples [21,23]. No Indonesian or Southeast Asian studies were identified among the studies discussed here; no reproducible regional search was conducted, so a regional evidence gap cannot be established.

This review also has methodological limitations. PubMed was the only database identified in the original account, whose exact search dates, strategies and selection record were incompletely documented. The revision used targeted source verification rather than exhaustive retrieval. Subjective narrative selection, lack of independent screening and absence of formal critical appraisal create risks of selective citation and uneven study weighting. Some claims rely on accessible abstracts rather than full methodological reports. Consequently, comparative statements about mechanisms and treatment response remain tentative.

Future research should develop measures that separate response intensity, lability, strategy use and recovery without including ADHD items; assess both domains and executive functions in the same children; use experimental and longitudinal designs; control for ODD and medication; and report reactivity and regulation as separate treatment outcomes. Clinically, assessment should describe triggers, intensity, duration, strategies and recovery rather than infer them from the ADHD diagnosis, and a reduction in outbursts should not be assumed to indicate improved regulation.

5 CONCLUSIONS

The available literature supports partially distinguishable measurement domains, but does not yet establish independent causal pathways or differential treatment mechanisms. Working-memory associations, irritability-related risks and treatment-related lability changes are informative but depend on operational definitions, design and informant. Separate assessment of response characteristics and regulation may improve characterization; the two-pathway account remains an integrative, testable hypothesis.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare no conflict of interest.

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