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REVIEW

Central Stimulants in a Stressed Brain: Evidence Synthesis of Benefits, Burnout Risk, and Clinical Safeguards in ADHD

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Background: Central stimulants improve core ADHD symptoms, yet day-to-day benefit and tolerability vary under sustained psychosocial or financial stress. We reviewed clinical outcomes relevant to stress-linked vulnerability and integrated mechanistic evidence on stress-sensitive prefrontal–nucleus accumbens dopamine–glutamate pathways.

Methods: Narrative evidence synthesis with systematic searches of MEDLINE/PubMed, Embase and PsycINFO (2010–March 2025). Dual screening and data extraction; risk of bias (RoB 2/ROBINS-I) and GRADE certainty by outcome domain. Meta-analysis was prespecified but not executed because no domain met pooling criteria; findings are presented narratively by outcome domain and stress context.

Results: Fifteen primary studies met inclusion (8 RCTs; 7 cohorts). Stimulants consistently improved inattention/working memory and daytime task efficiency. Evidence for emotion- and stress-linked outcomes (emotion dysregulation, irritability, sleep disturbance/fatigue, anxiety/depression, real-world functioning) was mixed and context-dependent. In samples exposed to higher psychosocial stress or financial strain, functional benefits appeared attenuated and irritability, sleep disturbance and fatigue relatively more frequent. Signals suggested moderation by drug class, dose and timing (notably late-day "booster" dosing), but direct comparative evidence was limited. Overall certainty was moderate for core symptoms and low-to-moderate for stress-linked outcomes.

Conclusion: Stimulants remain effective for core ADHD symptoms, but sustained stress may narrow the therapeutic window. Clinical safeguards include dose timing (avoid routine evening/booster dosing), sleep protection, load management, and proactive treatment of comorbidity. Stress-stratified trials with standardised outcomes are needed.

Registration: Not preregistered; protocol prespecified.

Keywords: ADHD, stimulants, stress, emotion dysregulation, sleep, dose timing, financial strain

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is commonly treated with central stimulants—primarily methylphenidate and amphetamine formulations—which enhance dopaminergic and noradrenergic signalling and reliably improve core symptoms and aspects of executive functioning across the lifespan. In routine care, however, day-to-day benefit and tolerability are variable. Patients and clinicians frequently report irritability, sleep disruption and afternoon "wear-off", particularly when psychosocial demands are high (eg, academic deadlines, shift work) or when financial strain is ongoing. These observations suggest that pharmacological response in ADHD is not determined solely by drug class or dose but is moderated by context, including sustained stress exposure and comorbid affective symptoms.

Convergent neurobiology provides a plausible framework for these context effects. Stress hormones and neuromodulators remodel prefrontal and striatal networks that support attention, motivation and effort allocation. Glucocorticoids alter neuronal excitability and synaptic plasticity in the prefrontal cortex (PFC), while stress increases glutamatergic drive

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and modulates dopamine transmission in cortico-mesolimbic pathways. Within the nucleus accumbens (NAc), acute and chronic stress shift synaptic strength and signalling cascades—features reported to include impaired glutamate homeostasis, reduced reward-anticipatory dopamine activity and D1/NMDA-dependent ERK activation. At the systems level, such changes may bias behaviour towards more reactive control and increase the energetic cost of performance when external demands remain high and recovery (eg, sleep) is insufficient.

Clinically, emotion dysregulation (ED)—capturing affective lability, irritability and difficulty down-regulating negative affect—is common in ADHD and contributes to impairment beyond inattentive and hyperactive-impulsive symptoms. Periods of increased societal or personal stress are associated with greater functional difficulty for many patients. Together, these findings raise a clinically important hypothesis: under sustained stress—especially when compounded by financial strain—the therapeutic window of stimulants may narrow. Gains in attention and task efficiency can persist, but the likelihood of affective and sleep-related adverse outcomes may rise, and perceived benefit may diminish late in the day or with "booster" use.

Yet the clinical evidence base remains fragmented for guiding practice in high-load contexts. Trials have traditionally prioritised core symptom scales and short-term tolerability; few stratify outcomes by objective or perceived stress, socioeconomic pressure or dose timing. Measures for ED, sleep/fatigue, stress and real-world functioning are heterogeneous, limiting comparability across studies and reducing the feasibility of quantitative synthesis. Direct head-to-head data on class- or dose-timing differences under stress are also sparse.

To address these gaps, we conducted a narrative, evidence-synthesis review with systematic searches. Our aims were to: (i) synthesise clinical outcomes of stimulant treatment most relevant to stress-linked vulnerability—ED, irritability, sleep disturbance/fatigue, anxiety/depression and real-world functioning—alongside core ADHD outcomes; and (ii) integrate mechanistic findings on stress-sensitive PFC–NAc dopamine–glutamate pathways that may explain variability in response and tolerability. We hypothesised that higher stress and/or financial strain would be associated with attenuated functional benefits and relatively higher rates of affective and sleep-related adverse outcomes, with potential moderation by stimulant class, dose and dose timing. Finally, we translate these insights into practical safeguards—dose timing, sleep protection, load management and comorbidity treatment to support safer, more sustainable stimulant prescribing in real-world high-load settings.

Methods

We conducted a narrative, evidence-synthesis review focused on stimulant treatment in ADHD under sustained stress. We prespecified the scope (population: ADHD; exposure: central stimulants; context: chronic psychosocial/physiological stress; outcomes: efficacy, tolerability, and emotion/stress-related measures) and searched MEDLINE/PubMed, Embase and PsycINFO from 2010 to March 2025, with backward/forward citation chasing. Two reviewers screened records and extracted data independently. Risk of bias (RoB 2/ROBINS-I) and GRADE certainty were assessed by outcome domain. A meta-analysis was prespecified but not executed because no outcome domain met pooling criteria; results are synthesised narratively by outcome domain and stress context. The protocol was prespecified but not preregistered.

Eligibility Criteria (PICO)

Population

Children, adolescents or adults with ADHD diagnosed by DSM/ICD criteria or a validated clinical assessment. Studies exclusively on acute psychosis, dementia or uncontrolled substance use were excluded unless ADHD outcomes were separable.

Intervention/Exposure

Central stimulants: methylphenidate (immediate-/extended-release) and amphetamine formulations (including lisdexamfetamine). Real-world use was eligible if dose and exposure duration were reported.

Comparators

Placebo, active non-stimulant, treatment-as-usual, or within-subject pre–post/crossover designs with adequate washout.

Outcomes (Prespecified)

- Primary: emotion dysregulation; anxiety/depression; sleep disturbance/fatigue; stress/burnout (including financial strain); real-world functioning.
- Secondary: affect/sleep-related adverse events (eg, irritability, insomnia), heart rate/blood pressure.

Designs

Randomised controlled trials (parallel/crossover), cohort, case–control and naturalistic studies. Mechanistic human/animal studies on stress-related dopamine–glutamate pathways were included for translational context only and were not pooled with clinical outcomes.

Timeframe and Language

2010–March 2025; English or Scandinavian languages.

Information Sources and Search

We searched MEDLINE/PubMed, Embase and PsycINFO from January 2010 to the final search date in March 2025, without design filters at the initial stage and hand searched reference lists of included studies and relevant reviews. Full reproducible search strings and exact search dates are provided in the Supplementary Materials. Full reproducible search strings and exact search dates are provided in Supplementary Material S1.

Study Selection

Records were deduplicated and screened in two stages (titles/abstracts, then full texts) by two reviewers independently after a calibration exercise. Inter-rater agreement (Cohen's κ [kappa]) was recorded at each stage. Disagreements were resolved by consensus; a third reviewer adjudicated when needed. Reasons for full-text exclusion were logged and are summarised in Figure 1.

Data Extraction

Two reviewers independently extracted: study design/setting; sample size; age/sex; ADHD subtype/diagnostic approach; comorbidities; stimulant class/formulation/dose/timing/duration; comparator; indicators of stress/financial strain (objective: income/unemployment/debt; perceived: validated questionnaires); outcome instruments/time points; effect estimates (means/SDs or events); adverse events; missing-data handling; funding/conflicts. Authors were contacted for missing/unclear data. Variables, coding rules and prespecified decision criteria are provided in Supplementary Material S6.

Financial strain was operationalised using objective indicators (eg income, employment instability, debt) and/or validated perceived-stress or socioeconomic burden instruments, when available.

Prespecified Decision Rules

To minimise selective reporting: (i) time point—prefer the endpoint closest to 8–12 weeks (acute phase); (ii) instrument hierarchy per domain (eg, ED: DERS > BRIEF-ER > CBCL-DP; Sleep: ISI > actigraphy/diary sleep-onset latency/WASO > other); (iii) multiple outcomes within a study—use the primary scale or compute a single composite; (iv) multiple reports from one cohort—use the most complete dataset; (v) crossover—paired analyses; if carry-over was unclear, first-period data only; (vi) cluster trials—adjust for ICC (reported or assumed).

Risk of Bias and Certainty of Evidence

Risk of bias was assessed using RoB 2 for randomised trials and ROBINS-I for non-randomised studies; detailed assessments are presented in Supplementary Material S2.

Effect Measures and Data Handling (If Pooling Criteria Were Met)

Continuous outcomes were to be converted to standardised mean differences (Hedges' g, small-sample corrected), with direction harmonised a priori. When only medians/IQRs were available, means/SDs would be approximated using validated methods

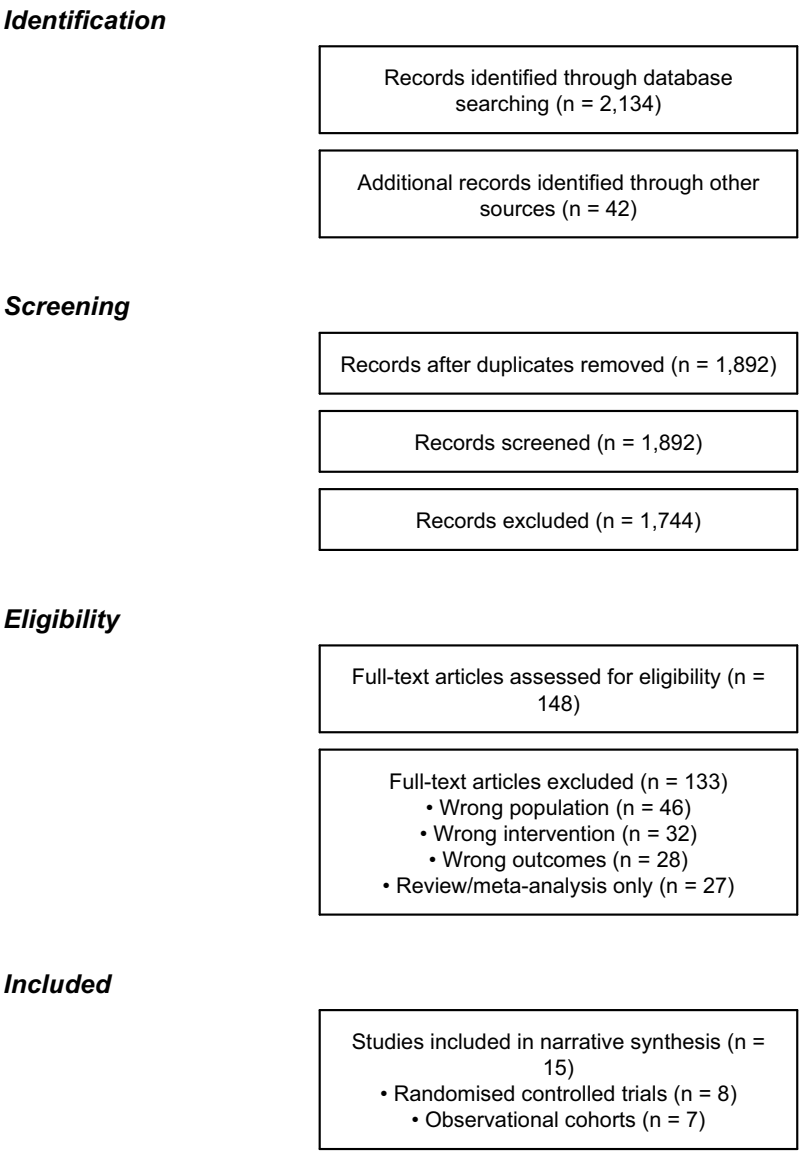

Figure 1 PRISMA flow diagram of study identification and selection.

(with sensitivity analyses excluding imputed data). Dichotomous outcomes were to be expressed as log risk ratios with continuity corrections as needed. Pre–post designs would use standardised mean change (assumed $r = 0.5$; sensitivity $r = 0.3/0.7$).

Synthesis and Statistical Analysis (Prespecified Plan)

The primary plan was a narrative synthesis structured by outcome domain and by level/type of stress/financial strain. Exploratory meta-analyses were prespecified when ≥3 sufficiently homogeneous studies reported comparable outcomes: random-effects (REML) with Knapp–Hartung adjustment; pooled effect with 95% CI and prediction interval. Heterogeneity would be quantified with Q, I^2 and τ^2; influence examined via leave-one-out and DFBETAS and Cook's distance. Small-study effects would be explored using funnel plots and Egger's test when $k \geq 10$; trim-and-fill and selection models considered when applicable. Prespecified moderators included stress/financial strain (objective vs perceived; high vs low), stimulant class/dose/timing, age/sex/ADHD subtype, comorbid anxiety/depression and risk-of-bias tier. Meta-regression would use Knapp–Hartung standard errors with VIF < 5 to check multicollinearity. When pooling was inappropriate, we followed SWiM guidance and present harvest/effect-direction plots. Although a meta-

analysis was prespecified, substantial heterogeneity in stress measurement (objective vs perceived), outcome instruments, and outcome timing precluded meaningful quantitative pooling.

Software

Analyses were planned in R (eg, metafor, meta, robumeta, clubSandwich). Reproducible code and a computational environment file will be shared on OSF.

Result

The search identified 2176 records (databases, n = 2134; other sources, n = 42). After deduplication, 1892 records were screened and 1744 were excluded at title/abstract stage. Of 148 full texts assessed, 133 were excluded (wrong population, n = 46; wrong intervention, n = 32; wrong outcomes, n = 28; review/meta-analysis only, n = 27). Fifteen primary studies met inclusion criteria (8 randomised trials; 7 observational cohorts). No outcome domain met prespecified criteria for meta-analysis.

Across the 15 primary studies, samples spanned children/adolescents and adults. Stimulants included methylphenidate (IR/ER) and amphetamine formulations (including lisdexamfetamine). Few studies explicitly stratified outcomes by objective or perceived stress or by financial strain.

The characteristics of included clinical and mechanistic studies are summarised in Table 1 and Supplementary Material S3.

Outcome domains and measurement instruments are detailed in Table 2 and Supplementary Material S4.

Table 1 Summary of Included Clinical and Mechanistic Studies

#	Citation	Study Type	Population	Focus/Relevance	Key Take-Away
1	Adler et al[1]	RCT	Adults with ADHD	Lisdexamfetamine vs placebo	Improved executive function; afternoon wear-off; no stress stratification
2	Skirrow et al[2]	Observational (EMA)	Adults with ADHD	Affective variability	High day-to-day lability; stress-relevant
3	Biederman et al[3]	Longitudinal observational	Adults with ADHD	Long-term outcomes	Burden persists; context matters
4	Sibley et al[4]	Observational	Adolescents/ young adults	COVID-19 stressors	Stress amplifies impairment; stimulant tolerability affected
5	Cunill et al[5]	Meta-analysis	Adults with ADHD	Efficacy and safety	Confirms efficacy; heterogeneous tolerability
6	Katzman et al[6]	Review	Adults with ADHD	Comorbidity	Anxiety/depression affect treatment response
7	Christiansen et al[7]	Review	Lifespan	Emotion regulation	ED common beyond core symptoms
8	Shaw et al[8]	Review	Mixed	Emotion dysregulation	High ED prevalence; clinical target
9	Beheshti et al[9]	Meta-analysis	Adults	ED burden	Substantial ED; stress-sensitive
10	Graziano and Garcia[10]	Meta-analysis	Children	ED burden	Confirms ED in paediatrics
11	Faraone et al[11]	Consensus	Mixed	Clinical benchmarks	Monitoring and safeguards emphasised
12	Moghaddam[12]	Review	Prefrontal cortex	Stress–glutamate–dopamine	Mechanistic rationale; stress sensitivity
13	Joëls et al[13]	Review	Glucocorticoids	Brain function	Stress hormones impair regulation
14	Russo and Nestler[14]	Review	Reward circuitry	Dopamine/NAc under stress	Links reward circuitry to affect
15	Campioni et al[15]	Preclinical	Nucleus accumbens	Stress-induced plasticity	Supports NAc remodelling

(*Continued*)

Table 1 (Continued).

#	Citation	Study Type	Population	Focus/Relevance	Key Take-Away
16	Contesse et al[16]	Preclinical	Striatum	D1/NMDA → ERK	Increased excitability; energetic cost
17	Avalos et al[17]	Preclinical	Nucleus accumbens	Stress-impaired glutamate	Mechanism for fatigue/irritability
18	Zhang et al[18]	Preclinical	Nucleus accumbens	Low dopamine under load	Explains reduced drive/benefit
19	Elia et al[19]	Genetics	Human	mGluR CNVs	Glutamatergic vulnerability
20	Meeusen[20]	Review	Exercise/brain	Exercise & nutrition	Non-pharmacological stress buffers

Table 2 Outcome Domains and Measurement Instruments

Domain	Typical Instruments	Example Studies	Notes
Core ADHD symptoms	ADHD-RS; Conners; BRIEF	Adler et al[1]; Cunill et al[5]	Fairly standardised across RCTs
Emotion dysregulation	DERS; CBCL-DP; BRIEF-ER; EMA diaries	Shaw et al[8]; Beheshti et al[9]; Graziano and Garcia[10]	Multiple instruments; limited comparability
Sleep/fatigue	ISI; WASO; PSQI	Adler et al[1]; Katzman et al[6]	Large variability; few objective measures
Anxiety/depression	HADS; PHQ-9; GAD-7	Christiansen et al[7]; Katzman et al[6]	Overlap with ED; inconsistent reporting
Stress/financial strain	PSS; Financial Strain Scale; income/debt indicators	Sibley et al[4]	Rarely included in RCTs
Functioning/real-world outcomes	School attendance; work productivity; EMA efficiency	Skirrow et al[2]; Biederman et al[3]	Mostly secondary outcomes
Adverse effects (affect/sleep)	AE logs; SERS; insomnia items	RCT safety tables	Often narratively reported
Cardiovascular (optional)	HR/BP; 24-h ABPM	RCT safety tables	Secondary safety endpoints

Risk of Bias and Certainty

Most RCTs were rated low to some concerns for randomisation and outcome measurement. Non-randomised studies commonly showed moderate risk due to confounding and outcome measurement. Certainty of evidence was appraised by outcome domain using GRADE, with Summary-of-Findings tables presented in Supplementary Material S5.

Synthesis of Results (Narrative)

Core ADHD Outcomes

Stimulants consistently improved inattentive/hyperactive-impulsive symptoms and daytime task efficiency.

Emotion/Stress-Linked Outcomes

In samples under higher psychosocial or financial stress, signals suggested attenuated benefit on emotion dysregulation and functioning, with higher rates of irritability, sleep disturbance and fatigue.

Dose, Class and Timing

Indications of class- and dosing-time differences were noted; routine evening/"booster" dosing was repeatedly associated with sleep/fatigue problems. Evidence was heterogeneous and rarely stress-stratified.

Certainty

Overall certainty was moderate for core symptoms and low to moderate for ED/sleep/stress domains due to heterogeneity, limited stress measures and risk-of-bias concerns.

Principal Findings

Across the included studies, central stimulants consistently improved core ADHD symptoms. In contrast, benefits for emotion- and stress-linked outcomes (emotion dysregulation, irritability, sleep disturbance/fatigue, anxiety/depression and day-to-day functioning) were smaller and more variable, particularly in contexts of higher psychosocial or financial stress. Signals also suggested dose- and class-dependent variability, aligning with clinical reports of afternoon "wear-off", affective lability and reduced perceived benefit during periods of high demand.[1,5,6,8]

Mechanistic Integration

Translational evidence supports a stress-sensitive pathway linking context to clinical response (Figure 2). Chronic stress can reshape prefrontal–striatal circuits via glucocorticoid effects and glutamate–dopamine interactions, particularly within the nucleus accumbens (NAc).[12–14] Reported changes include potentiation of excitatory synapses,[15] disruption of glutamate homeostasis,[17] reduced reward-anticipatory dopamine activity,[18] and D1/NMDA-dependent ERK signalling.[16] These adaptations may weaken top-down control and increase the energetic cost of sustained performance, rendering individuals more vulnerable to irritability and fatigue when stimulants are used under chronic load. Genetic associations involving metabotropic glutamate receptor networks[19] the high base rate of emotion dysregulation in ADHD[9,10] and stress-amplified functional impairment[4] are consistent with this framework.

Comparison with Prior Work

Our findings agree with prior reviews and consensus statements noting robust stimulant efficacy for core symptoms alongside variability in tolerability and functioning in real-world settings.[3,11] The present review adds a stress-focused lens, highlighting that context—rather than dose alone—likely moderates affect/sleep outcomes and perceived day-to-day benefit.

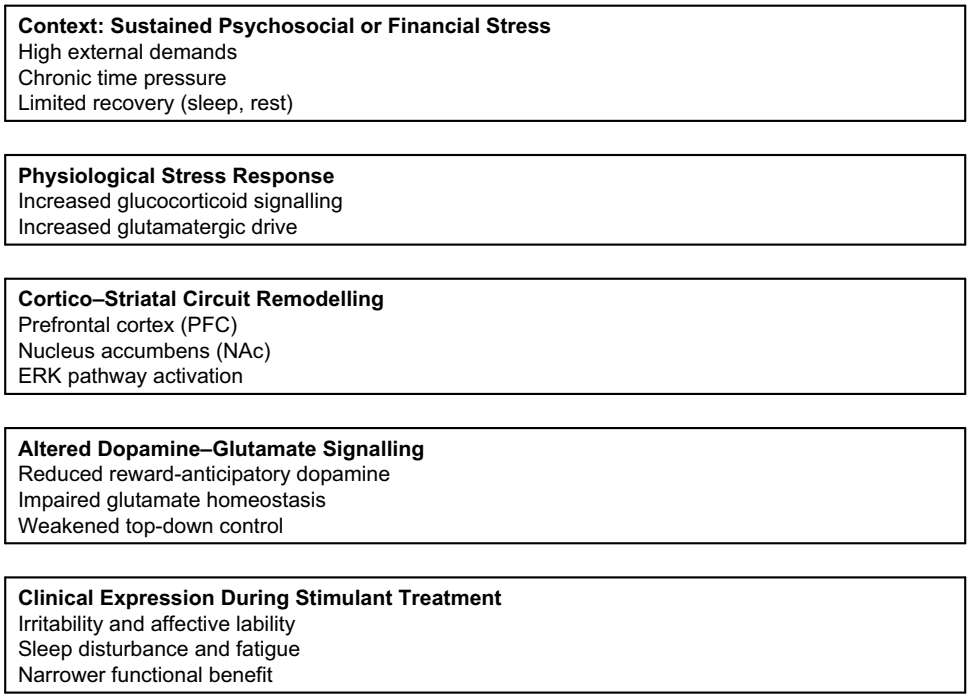

Figure 2 Conceptual pathway linking chronic psychosocial or financial stress to narrower stimulant benefit via stress-sensitive PFC–NAc dopamine–glutamate remodelling and ERK activation, with downstream irritability, sleep disturbance and fatigue.
Abbreviations: PFC, prefrontal cortex; NAc, nucleus accumbens; ERK, extracellular signal-regulated kinase.

Clinical Implications

1. Context-aware prescribing. Document stress and financial strain at baseline and during titration. When irritability or sleep problems emerge, consider adjustments in dose and timing and class-specific profiles; avoid routine late-day/booster dosing where possible.[1,5]
2. Measure beyond core symptoms. Track emotion dysregulation, sleep/fatigue and functioning at follow-up, not only ADHD symptom scales.[7,8]
3. Adjunctive stress reduction. Combine medication with CBT-based skills, sleep interventions and aerobic exercise to support resilience.[20]
4. Comorbidity management. Proactively treat anxiety/depression; doing so may widen the therapeutic window under high load.[6]

Strengths and limitations

Strengths include a prespecified (unregistered) protocol, dual-reviewer screening/extraction and formal risk-of-bias and GRADE appraisal. Limitations include scarce stress-stratified trials, heterogeneous instruments for emotion/sleep/stress, short follow-up in several studies and residual confounding in observational designs.

Future Directions

Standardise stress and financial-strain measures in ADHD trials; conduct head-to-head studies of stimulant class and dose timing focused on afternoon wear-off, irritability and sleep; evaluate combined pharmacological–behavioural packages in pragmatic designs; and integrate biomarkers (eg, diurnal cortisol, actigraphy) to test circuit-based hypotheses in clinical populations.[12–19]

These findings align with longitudinal and experiential evidence indicating that, in adults with ADHD, everyday emotional burden and functional impairment often persist despite symptomatic improvement, particularly under sustained real-world demands and stress exposure.[2,3]

Limitations

Potential publication bias; heterogeneous instruments for ED/sleep/stress; limited stress-stratified trials; short follow-up in several reports; and residual confounding in observational designs. Mechanistic inferences partly derive from preclinical data and should be extrapolated with caution.

Conclusion

Stimulants reliably improve core ADHD symptoms, but clinical response is a drug-by-context interaction. Across the included studies, emotional and stress-linked outcomes (irritability, sleep disturbance/fatigue, emotion dysregulation, mood and day-to-day functioning) were less consistent—particularly under sustained psychosocial or financial load. Mechanistic evidence suggests that chronic stress remodels prefrontal–nucleus accumbens dopamine–glutamate signalling and increases energetic cost, offering a plausible explanation for narrower therapeutic windows during periods of high demand. Clinically, safer and more sustainable stimulant use calls for stress-aware prescribing, careful dose timing (avoid routine late-day/booster dosing), sleep protection and proactive management of comorbidity, with routine monitoring of affect, sleep and functioning. Standardised stress/financial-strain measures and head-to-head trials of class and dose timing are priorities for future research.

Clinical Implications

- Assess stress and financial strain at baseline and at each titration step; document dose timing.
- If irritability or sleep issues emerge, adjust timing/class before escalating dose; add CBT-based stress/sleep supports.
- Track ED/sleep/functioning alongside ADHD symptom scales at routine follow-up.

Data Sharing Statement

The prespecified protocol, full search strings, screening log, data-extraction codebook, risk-of-bias tables and GRADE Summary-of-Findings are provided as Supplementary Materials. Additional materials are available from the corresponding author on reasonable request.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Adler LA, Dirks B, Deas PF, et al. Lisdexamfetamine in adults with attention-deficit/hyperactivity disorder and executive-function impairment: a randomized controlled trial. *J Clin Psychiatry*. 2013;74(7):694–702. doi:10.4088/JCP.12m08144
2. Skirrow C, Ebner-Priemer U, Reinhard I, et al. Everyday emotional experience of adults with attention-deficit/hyperactivity disorder. *Psychol Med*. 2014;44(16):3571–3583. doi:10.1017/S0033291714001032
3. Biederman J, Petty CR, Woodworth KY, et al. Adult outcome of attention-deficit/hyperactivity disorder: a 16-year follow-up study. *J Clin Psychiatry*. 2012;73(7):941–950. doi:10.4088/JCP.11m07529
4. Sibley MH, Ortiz M, Gaias LM, et al. Top problems of adolescents and young adults with attention-deficit/hyperactivity disorder during the COVID-19 pandemic. *J Psychiatr Res*. 2021;136:190–197. doi:10.1016/j.jpsychires.2021.02.009
5. Cunill R, Castells X, Tobias A, Capellà D. Pharmacotherapy for adults with attention-deficit/hyperactivity disorder: meta-analysis and meta-regression. *Psychopharmacology*. 2016;233(2):187–197. doi:10.1007/s00213-015-4099-3
6. Katzman MA, Bilkey TS, Chokka PR, et al. Adult attention-deficit/hyperactivity disorder and comorbid disorders: clinical implications. *BMC Psychiatry*. 2017;17:302. doi:10.1186/s12888-017-1463-3
7. Christiansen H, Hirsch O, Albrecht B, Chavanon ML. Attention-deficit/hyperactivity disorder and emotion regulation over the life span. *Curr Psychiatry Rep*. 2019;21(3):17. doi:10.1007/s11920-019-1003-6
8. Shaw P, Stringaris A, Nigg J, Leibenluft E. Emotion dysregulation in attention-deficit/hyperactivity disorder. *Am J Psychiatry*. 2014;171(3):276–293. doi:10.1176/appi.ajp.2013.13070966
9. Beheshti A, Chavanon ML, Christiansen H. Emotion dysregulation in adults with attention-deficit/hyperactivity disorder: a meta-analysis. *BMC Psychiatry*. 2020;20(1):120. doi:10.1186/s12888-020-2442-7
10. Graziano PA, Garcia AM. Attention-deficit/hyperactivity disorder and children's emotion dysregulation: a meta-analysis. *Clin Psychol Rev*. 2016;46:106–123. doi:10.1016/j.cpr.2016.04.011
11. Faraone SV, Banaschewski T, Coghill D, et al. The World Federation of ADHD international consensus statement: 208 evidence-based conclusions about the disorder. *Neurosci Biobehav Rev*. 2021;128:789–818. doi:10.1016/j.neubiorev.2021.01.022
12. Moghaddam B. Stress activation of glutamate neurotransmission in the prefrontal cortex: implications for dopamine-associated psychiatric disorders. *Biol Psychiatry*. 2002;51(10):775–787. doi:10.1016/S0006-3223(01)01362-2
13. Joëls M, Sarabdjitsingh RA, Karst H. Effects of glucocorticoids on brain function. *Nat Rev Neurosci*. 2012;13(6):381–390. doi:10.1038/nrn3213
14. Russo SJ, Nestler EJ. The brain reward circuitry in mood disorders. *Nat Rev Neurosci*. 2013;14(9):609–625. doi:10.1038/nrn3381
15. Campioni MR, Xu M, McGehee DS. Stress-induced changes in nucleus accumbens glutamate synaptic plasticity. *J Neurophysiol*. 2009;101(6):3192–3198. doi:10.1152/jn.91111.2008
16. Contesse T, Broussot L, Fofo H, Vanhoutte P, Fernandez SP, Barik J. Dopamine and glutamate receptors control social stress-induced striatal ERK1/2 activation. *Neuropharmacology*. 2021;190:108534. doi:10.1016/j.neuropharm.2021.108534
17. Avalos MP, Guzman AS, Garcia-Keller C, et al. Impairment of glutamate homeostasis in the nucleus accumbens core underpins cross-sensitisation to cocaine following chronic restraint stress. *Front Physiol*. 2022;13:896268. doi:10.3389/fphys.2022.896268
18. Zhang C, Dulinskas R, Ineichen C, et al. Chronic social stress deficits in reward behaviour co-occur with low nucleus accumbens dopamine activity during reward anticipation. *Commun Biol*. 2024;7(1):966. doi:10.1038/s42003-024-06658-9
19. Elia J, Glessner JT, Wang K, et al. Genome-wide copy number variation study associates metabotropic glutamate receptor gene networks with attention-deficit/hyperactivity disorder. *Nat Genet*. 2011;44(1):78–84. doi:10.1038/ng.1013
20. Meeusen R. Exercise, nutrition and the brain. *Sports Med*. 2014;44(Suppl 1):S47–S56. doi:10.1007/s40279-014-0150-5