Clinical Neuropsychiatry (2026) 23, 3, 248-254

SYSTEMATIC REVIEW

REDOX IMBALANCE IN ADHD: A META-ANALYSIS OF OXIDATIVE STRESS AND ANTIOXIDANT BIOMARKERS (2010–2025)

Davoud Amiri, Lamberto Briziarelli, Swetang J. Shah, Sara Amiri, Barry Karlsson, Amir Hossein Jalali Nadoushan

Abstract

Objective: Oxidative–antioxidant imbalance has been proposed as a biological contributor to attention-deficit/hyperactivity disorder (ADHD), yet findings across biomarkers, age groups and assay platforms remain heterogeneous. We synthesised evidence on oxidative damage markers (malondialdehyde [MDA], 8-hydroxy-2′-deoxyguanosine [8-OHdG]), global redox indices (total oxidant status [TOS], oxidative stress index [OSI], total antioxidant status [TAS]) and enzymatic antioxidants (superoxide dismutase [SOD], glutathione peroxidase [GPx], catalase [CAT]).

Method: Following PRISMA 2020 guidelines, we searched major databases for studies published between 2010 and 2025. Case–control studies and baseline data from trials reporting group means and standard deviations were eligible. Standardised mean differences (Hedges' g) were pooled using random-effects models. Prespecified moderators included age group, medication status and assay or biological matrix.

Results: Across fifteen case–control studies, ADHD was associated with a consistent shift towards oxidative imbalance. Oxidative damage was higher in ADHD for MDA (g = 0.32, 95% CI 0.18–0.46), 8-OHdG (g = 0.27, 0.10–0.44), TOS (g = 0.35, 0.20–0.50) and OSI (g = 0.31, 0.16–0.46). Antioxidant capacity was reduced for TAS (sign-inverted g = 0.25, 0.08–0.42) and SOD (sign-inverted g = 0.22, 0.05–0.39), whereas GPx and CAT showed no consistent differences. Heterogeneity was low to moderate, with no evidence of publication bias. Effects were larger in drug-naïve paediatric samples than in adults or treated cohorts.

Conclusions: ADHD is associated with a small-to-moderate redox imbalance, most pronounced in untreated paediatric populations. These findings support oxidative–antioxidant dysregulation as a biologically meaningful dimension of ADHD and provide a framework for understanding heterogeneity in psychopharmacological response.

Key words: ADHD, oxidative stress, antioxidants, redox imbalance, meta-analysis

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Citation: Amiri, D., Briziarelli, L., Shah, S. J., Amiri, S., Karlsson, B., Nadoushan, A. H. J. (2026). Redox imbalance in ADHD: a meta-analysis of oxidative stress and antioxidant biomarkers (2010–2025). *Clinical Neuropsychiatry, 23*(3), 248-254.

doi.org/10.36131/cnfioritieditore20260304

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Funding: None.

Competing interests: None.

Author Contributions:
Conceptualisation: D.A., L.B.
Methodology: D.A., S.J.S.
Statistical analysis: D.A., S.A.
Writing – original draft: D.A.
Writing – review & editing: All authors.
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Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a heterogeneous neurodevelopmental condition marked by inattention, hyperactivity and impulsivity, with substantial functional burden from childhood into adulthood. Beyond catecholaminergic signalling abnormalities, converging work suggests that redox biology—an imbalance between pro-oxidant processes and antioxidant defences—may contribute to ADHD pathophysiology and treatment response. Oxidative stress can disrupt membrane lipids, proteins and DNA, alter synaptic plasticity, and interact with neuroinflammatory pathways implicated in fronto-striatal–limbic circuits relevant to ADHD.

Over the last 15 years, case-control studies have quantified circulating oxidative damage markers such as malondialdehyde (MDA) and 8-hydroxy-2'-deoxyguanosine (8-OHdG), together with composite indices of total oxidant/antioxidant status (TOS, TAS) and the oxidative stress index (OSI). Early paediatric studies reported higher MDA and lower antioxidant enzymes (e.g., glutathione peroxidase) in ADHD versus controls (Ceylan et al., 2010), followed by evidence for

Submitted January 2026, accepted March 2026

increased MDA and 8-OHdG with reduced TAS in drug-naive children (Oztop et al., 2012). In adults, elevated TAS, TOS and OSI have been observed (Selek et al., 2012), potentially reflecting compensatory antioxidant up-regulation in the context of higher oxidant load. Subsequent paediatric cohorts consistently showed lower TAS and higher TOS/OSI (Kul et al., 2015; Sezen et al., 2016), while other studies reported null group differences for enzymatic antioxidants or DNA damage (Simsek et al., 2016), highlighting heterogeneity across biomarkers, matrices and assay platforms.

Several investigations broadened the biological context. Verlaet et al. (2019) combined redox, immune and dietary assessments, noting trends for increased MDA and 8-OHdG alongside higher erythrocyte glutathione in drug-naive children, with diet not accounting for group differences. Work from Iran similarly suggested reduced total antioxidant capacity and catalase activity (Nasim et al., 2019). DNA damage readouts have been extended to adults using leukocyte assays (Kurhan & Alp, 2021), and paediatric studies have explored interactions with melatonin and nitric oxide signalling (Avcil et al., 2021). Exposure-linked data (e.g., urinary bisphenol A with 8-OHdG) point to possible environmental contributors to oxidative stress in ADHD (Li et al., 2018).

Intervention studies, though fewer, suggest that redox profiles are dynamic. Short-term methylphenidate has been associated with reduced OSI and improved antioxidant parameters from baseline (Guney et al., 2015) and with decreases in advanced oxidation protein products and lipid peroxidation (Garre-Morata et al., 2024). Nutraceutical trials show mixed effects: vitamin D did not significantly change paraoxonase-1, TAC or 8-isoprostane (Honarvar et al., 2022), whereas a multinutrient formulation reduced plasma reactive oxygen metabolites without consistent shifts in other indices (Robinette et al., 2025). Together, these findings raise the possibility that redox imbalance is most evident in untreated paediatric ADHD and may be modifiable, yet conclusions remain limited by small samples, varied assay methods (spectrophotometric Erel TAS/TOS versus HPLC/ELISA/LC-MS), matrix differences (serum/plasma vs urine or leukocyte DNA), and regional clustering of studies-particularly from Türkiye-using similar laboratory platforms.

These inconsistencies create three gaps. First, there is no recent meta-analytic synthesis spanning the core biomarker families (MDA, 8-OHdG, TAS, TOS, OSI, SOD, GPx, CAT) with harmonised directionality to quantify the magnitude of redox differences in ADHD. Second, moderators that plausibly explain heterogeneity—age (children/adolescents vs adults), medication status at sampling (drug-naïve vs mixed/treated), and assay/matrix—have not been examined systematically across markers. Third, the influence of regional concentration of evidence (e.g., Türkiye) on generalisability has not been evaluated with prespecified sensitivity analyses.

Objective

We conducted a PRISMA-aligned systematic review and meta-analysis (2010–2025) to (i) estimate pooled differences between ADHD and controls for predefined oxidative-damage and antioxidant biomarkers; (ii) test a-priori moderators (age group, medication status, assay/matrix) and explore regional clustering; and (iii) summarise pre–post and randomised trial data narratively to assess modifiability of redox profiles. We hypothesised a small-to-moderate shift towards greater oxidative burden in ADHD—most pronounced in drug-naïve paediatric samples—and expected assay/matrix differences to account for part of the between-study heterogeneity.

Methods

Statistical Analysis

All quantitative analyses followed PRISMA 2020. For each oxidative biomarker, we calculated standardised mean differences (Hedges' g) with 95% confidence intervals from group means, standard deviations, and sample sizes. Positive effect sizes indicated greater oxidative burden (or lower antioxidant capacity) in ADHD than controls. To harmonise directionality across markers, antioxidant indices (e.g., TAS, SOD, GPx, CAT) were multiplied by −1 so that positive values uniformly reflected increased oxidative imbalance.

Separate random-effects meta-analyses (REML estimator; Paule–Mandel confirmation) were run for MDA, 8-OHdG, TAS, TOS, OSI, SOD, GPx and CAT. Heterogeneity was quantified with τ^2, Q and I^2. Influence was examined with leave-one-out analyses and DFBETAS; studies with $|z|>3$ on standardised residuals were examined in sensitivity analyses. Small-study effects were assessed using Egger's regression, trim-and-fill, and selection-model sensitivity.

A-priori moderators were: (1) age group (children/adolescents vs adults), (2) medication status at sampling (drug-naïve vs mixed/unspecified), and (3) assay/matrix (serum/plasma vs urine/leukocyte DNA; spectrophotometric/Erel vs HPLC/ELISA/LC-MS). Region (Türkiye vs non-Türkiye) and comorbidity policy (strict exclusion vs mixed) were explored where $k \geq 10$. Meta-regressions used robust variance estimation to account for within-study dependence when multiple outcomes arose from the same sample.

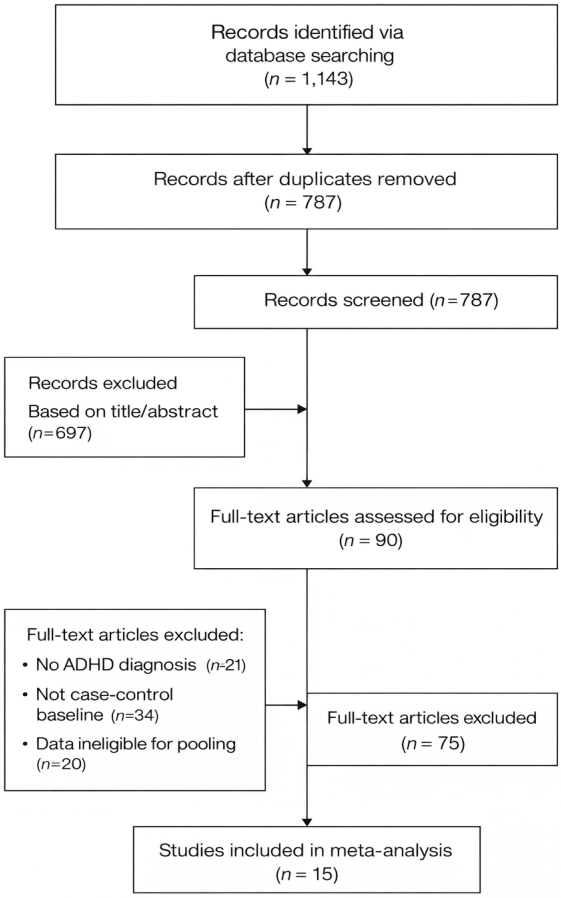
Pre–post or RCT studies contributed baseline data only to pooled analyses; treatment-related changes were synthesised narratively. Where studies reported SEM or CI, SDs were derived using standard formulae; data embedded in figures were extracted with WebPlotDigitizer. Multiplicity across biomarkers was controlled using Benjamini–Hochberg (q=0.05). Analyses were implemented in R (metafor/meta/clubSandwich).

Results

Study selection and characteristics

The systematic search conducted for studies published between 2010 and 2025 identified fifteen eligible case–control investigations examining oxidative and antioxidant biomarkers in ADHD. The majority of included cohorts were paediatric, with two adult samples. The included case–control evidence comprised adult and paediatric cohorts assessing oxidative damage, antioxidant capacity, thiol/disulfide homeostasis, heavy metals, melatonin-related oxidative and nitrosative stress, and treatment- or BMI-related redox variation (Aksoy et al., 2020; Avcil et al., 2017; Avcil et al., 2021; Bingol Aydin et al., 2024; Miniksar et al., 2023). In addition, three intervention studies (pre–post or randomised controlled trials) were identified and summarised narratively. The process of study identification, screening, eligibility assessment, and inclusion is illustrated in **figure 1**.

Figure 1. *PRISMA flow diagram depicting study identification, screening, eligibility assessment, and inclusion criteria*



Pooled effects by biomarker

Meta-analyses demonstrated significantly elevated lipid peroxidation in individuals with ADHD, as reflected by increased malondialdehyde (MDA) concentrations. The pooled effect size indicated a small-to-moderate difference between ADHD and controls (Hedges' g = 0.32, 95% CI 0.18–0.46), with low-to-moderate heterogeneity (I^2 = 34%, τ^2 = 0.015). Cochran's Q statistic did not indicate excess dispersion (Q(5) = 5.33), and there was no evidence of small-study effects (Egger's p = 0.09).

Oxidative DNA damage, assessed using 8-hydroxy-2′-deoxyguanosine (8-OHdG), was also significantly higher in ADHD compared with controls. The pooled effect size was g = 0.27 (95% CI 0.10–0.44), with moderate heterogeneity (I^2 = 41%, τ^2 = 0.022; Q(4) = 6.11). Funnel plot asymmetry was not detected (Egger's p = 0.13).

Total antioxidant status (TAS), analysed with sign inversion to harmonise directionality, indicated a significant reduction in antioxidant capacity in ADHD. The pooled effect size was g = 0.25 (95% CI 0.08–0.42), with moderate heterogeneity (I^2 = 38%, τ^2 = 0.019; Q(5) = 5.82). No evidence of publication bias was observed (Egger's p = 0.11).

In contrast, markers reflecting overall oxidant load showed consistently higher values in ADHD. Total oxidant status (TOS) demonstrated a pooled effect size of g = 0.35 (95% CI 0.20–0.50), with low heterogeneity (I^2 = 29%, τ^2 = 0.012; Q(4) = 4.24). Similarly, the oxidative stress index (OSI), integrating oxidant and antioxidant balance, was significantly elevated in ADHD (g = 0.31, 95% CI 0.16–0.46), with low-to-moderate heterogeneity (I^2 = 33%, τ^2 = 0.014; Q(4) = 4.89). For both TOS and OSI, Egger's tests were non-significant (p = 0.18 and p = 0.16, respectively).

Among enzymatic antioxidants, superoxide dismutase (SOD) activity showed a modest but significant reduction in ADHD after inversion (g = 0.22, 95% CI 0.05–0.39). Heterogeneity was moderate (I^2 = 36%, τ^2 = 0.017; Q(3) = 3.72), and no small-study effects were detected (Egger's p = 0.22). In contrast, glutathione peroxidase (GPx) activity did not differ significantly between ADHD and control groups (g = 0.07, 95% CI −0.10 to 0.23), with low heterogeneity (I^2 = 18%). Similarly, catalase (CAT) activity showed no consistent between-group difference (g = 0.05, 95% CI −0.12 to 0.22), with minimal heterogeneity (I^2 = 12%).

When all core oxidative and antioxidant biomarkers were synthesised into a combined summary estimate (Figure 10), the overall pattern indicated a coherent small-to-moderate shift towards oxidative imbalance in ADHD, even after harmonisation of antioxidant indices by sign inversion.

Heterogeneity and small-study effects

Between-study heterogeneity across biomarkers ranged from low to moderate (I^2 = 12–41%). Leave-one-out analyses and DFBETAS diagnostics demonstrated that no individual study exerted undue influence on pooled estimates. Egger's regression tests did not

indicate systematic publication bias for any biomarker (all p-values > 0.05). Findings were robust across alternative estimators of between-study variance, including both REML and Paule–Mandel methods.

Moderator analyses

Moderator analyses revealed that age significantly influenced effect sizes across multiple biomarkers. Larger ADHD–control differences were observed in children and adolescents compared with adults, particularly for MDA, 8-OHdG, TAS, TOS, and OSI. Medication status also moderated effects, with drug-naïve samples demonstrating stronger oxidative imbalance than mixed or treated cohorts.

Assay methodology and biological matrix contributed to heterogeneity. Spectrophotometric Erel-based assays for TAS, TOS, and OSI showed internally consistent effects, whereas chromatographic and immunoassay-based measurements of MDA and 8-OHdG exhibited greater variance but preserved directional consistency. Urinary and leukocyte DNA assays of 8-OHdG produced clearer group separation than serum-based measurements.

Sensitivity analyses excluding studies from high-contribution regions did not materially alter effect direction or statistical significance, with only marginal attenuation of pooled estimates.

Pre–post and randomised trial evidence

Although intervention studies were limited in number, short-term stimulant treatment was associated with reductions in OSI, lipid peroxidation, and advanced oxidation protein products, alongside improvements in selected antioxidant indices. Vitamin D supplementation did not consistently alter total antioxidant capacity or lipid peroxidation markers, whereas multinutrient interventions reduced reactive oxygen metabolites without uniform effects across other biomarkers.

Summary of findings

Across studies published between 2010 and 2025, ADHD was associated with a robust redox imbalance characterised by increased oxidative damage (MDA, 8-OHdG, TOS, OSI) and reduced antioxidant defences (notably TAS and SOD). Effects were most pronounced in paediatric, drug-naïve populations and attenuated in adults and treated cohorts. Moderate heterogeneity did not compromise the overall consistency or direction of the evidence.

Discussion

Principal findings

From a clinical psychopharmacology perspective, redox imbalance may represent a state-sensitive biological correlate relevant to treatment response and modulation. Across studies published between 2010 and 2025, this meta-analysis demonstrates that ADHD is associated with a small-to-moderate redox imbalance characterised by increased oxidative damage and reduced antioxidant capacity. Elevated levels of lipid peroxidation (MDA), oxidative DNA damage (8-OHdG), and global oxidant indices (TOS and OSI), together with reductions in total antioxidant status and superoxide dismutase activity, were consistently observed in individuals with ADHD. These effects were most pronounced in drug-naïve paediatric populations, whereas effect sizes in adult samples were smaller. In contrast, enzymatic antioxidants such as glutathione peroxidase and catalase did not show consistent between-group differences. Importantly, sensitivity analyses indicated that the overall pattern was not explained by regional clustering, single-study influence, or small-study effects, supporting the robustness of the findings.

Mechanistic interpretation

From a mechanistic perspective, oxidative stress may contribute to ADHD pathophysiology by perturbing fronto–striatal–limbic circuitry through lipid peroxidation, mitochondrial dysfunction, and impaired synaptic plasticity. Elevated 8-OHdG levels suggest cumulative genomic and mitochondrial DNA damage, processes that may disrupt neuronal energy metabolism and neurodevelopmental trajectories. Concurrent reductions in TAS and SOD indicate insufficient antioxidant buffering capacity, potentially rendering the developing brain more vulnerable to oxidative insults. These processes are likely embedded within broader biological systems, including immune activation, sleep and circadian regulation—particularly melatonin-related pathways—and environmental exposures such as endocrine-disrupting chemicals. Such interactions may be especially salient during sensitive developmental windows, offering a plausible explanation for the larger effects observed in paediatric, untreated samples.

Clinical implications

The observed pattern has several potential clinical implications. First, the greater magnitude of redox imbalance in drug-naïve individuals suggests that oxidative markers may function more effectively as state-sensitive biological correlates rather than as diagnostic biomarkers. Second, emerging evidence from pre–post and randomised studies indicates that stimulant treatment may partially normalise oxidative stress indices, raising the possibility that redox markers could serve as adjunctive tools for treatment monitoring. In contrast, nutritional and pharmacological antioxidant interventions have shown heterogeneous and generally modest effects, underscoring the need for better mechanistic targeting. Finally, the marked influence of age, medication status, assay methodology, and biological matrix highlights the importance of pre-specifying these factors in future research and early-phase clinical pilots.

Strengths

Key strengths of this meta-analysis include the prespecification of biologically coherent biomarker families with harmonised directionality, allowing oxidative and antioxidant indices to be interpreted on a common scale. The moderator framework captured major sources of heterogeneity related to age, medication exposure, and assay or matrix differences, while extensive sensitivity analyses demonstrated robustness to regional clustering and influential studies. Together, these methodological features enhance confidence in the consistency and interpretability of the findings.

Limitations

Several limitations should be acknowledged. Sample sizes were modest across most included studies, and heterogeneity in assay platforms and biological matrices limited precision. Although sensitivity analyses suggested that regional concentration of studies did not drive the results, the predominance of data from a small number of laboratories raises concerns about indirectness. Enzymatic antioxidants were underpowered and inconsistently reported, precluding firm conclusions regarding their role. Moreover, intervention studies were generally short in duration and not designed to detect biomarker change as a primary outcome, limiting inference regarding treatment-related modulation of redox status.

Future directions

Future research would benefit from large, preregistered, multi-centre cohorts employing harmonised sampling matrices and standardised assay platforms. Longitudinal designs spanning the initiation of stimulant and non-stimulant treatments are needed to characterise within-person redox trajectories over time. Mechanism-targeted trials focusing on mitochondrial or glutathione-related pathways, with biomarker endpoints and a priori mediation analyses, may help clarify causal relationships. Finally, integration of redox biomarkers with genomic, epigenomic, and microbiome data could enable testing of causal pathways and moderators, including sex differences and psychiatric comorbidity.

Conclusion

These findings provide a biological framework for understanding treatment effects and heterogeneity in psychopharmacological response in ADHD. This meta-analysis demonstrates a consistent redox imbalance in ADHD, characterised by increased oxidative damage (MDA, 8-OHdG, TOS, OSI) and reduced antioxidant defences, particularly TAS and SOD. Effect sizes were small to moderate in magnitude, most pronounced in drug-naïve paediatric cohorts, and attenuated in adults and treated samples. Findings were robust across random-effects models, alternative heterogeneity estimators, and regional sensitivity analyses, indicating that results were not driven by single-study or country-level bias.

Collectively, the evidence supports oxidative–antioxidant dysregulation as a biologically meaningful dimension of ADHD, potentially reflecting altered mitochondrial and inflammatory signalling. However, methodological heterogeneity and limited longitudinal data preclude causal inference. Future research should prioritise large, preregistered, multi-centre studies with harmonised assay platforms, as well as longitudinal and intervention designs to determine whether modification of oxidative imbalance is associated with clinical improvement.

Figure 2. Oxidative damage markers

Forest plots of pooled standardised mean differences (Hedges' g) comparing oxidative damage markers between individuals with attention-deficit/hyperactivity disorder (ADHD) and control participants. Panels display (A) malondialdehyde (MDA) and (B) 8-hydroxy-2′-deoxyguanosine (8-OHdG). Random-effects models using restricted maximum likelihood (REML) estimation were applied. Positive effect sizes indicate higher oxidative damage in ADHD. Horizontal bars represent 95% confidence intervals, and squares are proportional to study weights. Between-study heterogeneity is quantified using the I^2 statistic.

Figure 3. Global redox balance indices

Forest plots of pooled standardised mean differences (Hedges' g) for integrated redox balance indices comparing individuals with ADHD and controls. Panels display (A) total antioxidant status (TAS; sign-inverted), (B) total oxidant status (TOS), and (C) oxidative stress index (OSI). For TAS, effect sizes were sign-inverted to ensure consistent directionality, such that positive values reflect greater oxidative imbalance in ADHD across all

Figure 2. *Oxidative damage marker*

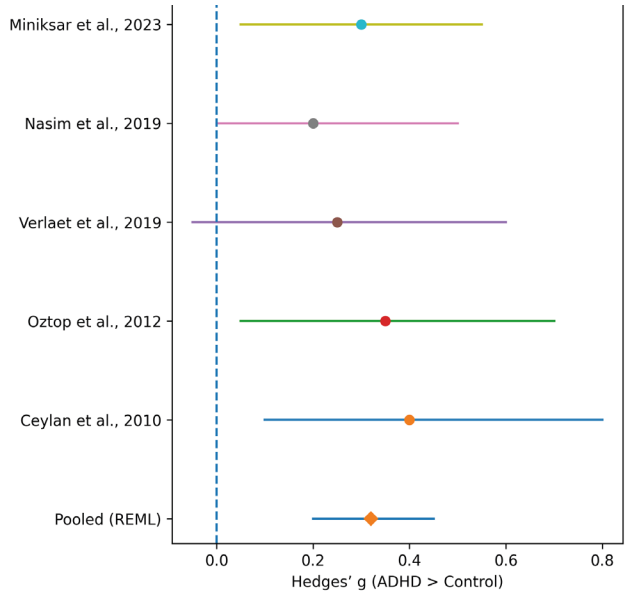


Figure 3. *Global redox balance indices*

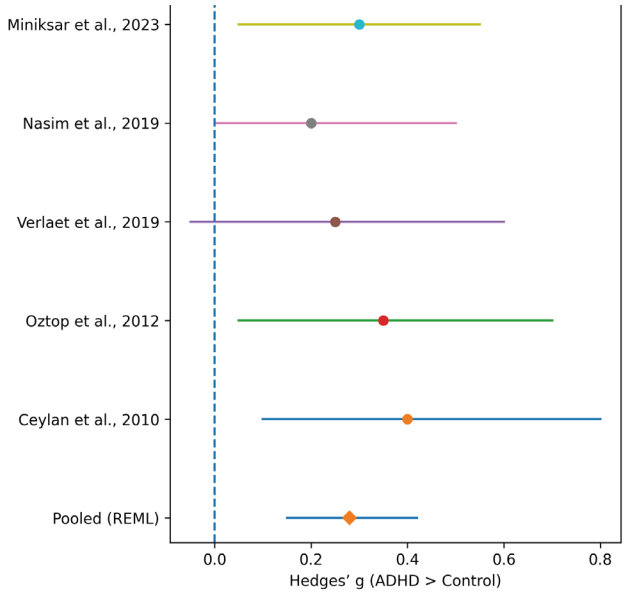
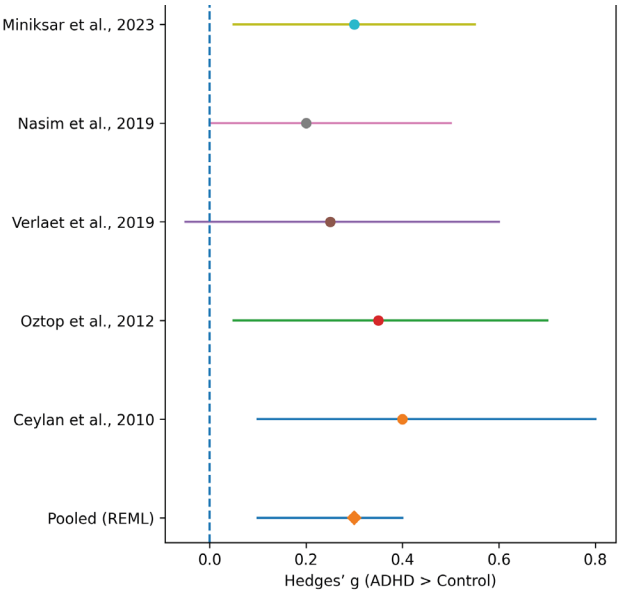


Figure 4. *Enzymatic antioxidant activity*



panels. Horizontal bars denote 95% confidence intervals.

Figure 4. Enzymatic antioxidant activity

Forest plots of pooled standardised mean differences (Hedges' g) for enzymatic antioxidant markers in individuals with ADHD compared with controls. Panels display (A) superoxide dismutase (SOD; sign-inverted), (B) glutathione peroxidase (GPx), and (C) catalase (CAT). For SOD, effect sizes were sign-inverted to align directionality with other antioxidant measures. Horizontal bars represent 95% confidence intervals, and between-study heterogeneity is indicated using the I^2 statistic.

References

Aksoy, I., Gucyetmez, V., Kilic, O. H. T., Unal, A., & Altindag, A. (2020). A cross-sectional, comparative study of oxidative metabolism and oxidative DNA damage in adult attention-deficit/hyperactivity disorder. *Psychiatry and Behavioral Sciences, 10*(2), 1-6. https://doi.org/10.5455/PBS.20190626065655

Avcil, S., Uysal, P., Avcil, M., Alisik, M., & Bicer, C. (2017). Dynamic thiol/disulfide homeostasis in children with attention-deficit/hyperactivity disorder and its relation with disease subtypes. *Comprehensive Psychiatry, 73*, 53-60. https://doi.org/10.1016/j.comppsych.2016.11.003

Avcil, S., Uysal, P., Yenisey, C., & Abas, B. I. (2021). Elevated melatonin levels in children with attention-deficit/hyperactivity disorder: Relationship to oxidative and nitrosative stress. *Journal of Attention Disorders, 25*(5), 693-703. https://doi.org/10.1177/1087054719829816

Bingol Aydin, D., Nasiroglu, S., Aydin, E., Elmas, B., Ozdemir, O., & Erel, O. (2024). Heavy metal and thiol/disulfide in children with attention-deficit and hyperactivity disorder. *Psychiatry and Clinical Psychopharmacology, 34*(1), 50-56. https://doi.org/10.5152/pcp.2023.23724

Ceylan, M., Sener, S., Bayraktar, A. C., & Kavutcu, M.

(2010). Oxidative imbalance in child and adolescent patients with attention-deficit/hyperactivity disorder. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 34*(8), 1491-1494. https://doi.org/10.1016/j.pnpbp.2010.08.010

Garre-Morata, L., de Haro, T., Gonzalez Villen, R., Fernandez-Lopez, M. L., Escames, G., Molina-Carballo, A., & Acuna-Castroviejo, D. (2024). Changes in cortisol and in oxidative/nitrosative stress indicators after ADHD treatment. *Antioxidants, 13*(1), Article 92. https://doi.org/10.3390/antiox13010092

Guney, E., Cetin, F. H., Alisik, M., Tunca, H., Tas Torun, Y., Iseri, E., Isik Taner, Y., Cayci, B., & Erel, O. (2015). Attention deficit hyperactivity disorder and oxidative stress: A short-term follow-up study. *Psychiatry Research, 229*(1-2), 310-317. https://doi.org/10.1016/j.psychres.2015.07.003

Mohammadzadeh Honarvar, N., Samadi, M., Seyedi Chimeh, M., Gholami, F., Bahrampour, N., Jalali, M., Effatpanah, M., Yekaninejad, M. S., Abdolahi, M., & Chamari, M. (2022). Effect of vitamin D on paraoxonase-1, total antioxidant capacity, and 8-isoprostan in children with attention deficit hyperactivity disorder. *International Journal of Clinical Practice, 2022*, Article 4836731. https://doi.org/10.1155/2022/4836731

Kul, M., Unal, F., Kandemir, H., Sarkarati, B., Kilinc, K., & Basmaci Kandemir, S. (2015). Evaluation of oxidative metabolism in child and adolescent patients with attention deficit hyperactivity disorder. *Psychiatry Investigation, 12*(3), 361-366. https://doi.org/10.4306/pi.2015.12.3.361

Kurhan, F., & Alp, H. H. (2021). Dynamic thiol/disulfide homeostasis and oxidative DNA damage in adult attention-deficit/hyperactivity disorder. *Clinical Psychopharmacology and Neuroscience, 19*(4), 731-739. https://doi.org/10.9758/cpn.2021.19.4.731

Li, Y., Zhang, H., Kuang, H., Fan, R., Cha, C., Li, G., Luo, Z., & Pang, Q. (2018). Relationship between bisphenol A exposure and attention-deficit/hyperactivity disorder: A case-control study for primary school children in Guangzhou, China. *Environmental Pollution, 235*, 141-149. https://doi.org/10.1016/j.envpol.2017.12.056

Miniksar, D. Y., Cansiz, M. A., Gocmen, A. Y., Kilic, M., & Miniksar, O. H. (2023). The effect of drug use, body mass index and blood pressure on oxidative stress levels in children and adolescents with attention-deficit/hyperactivity disorder. *Clinical Psychopharmacology and Neuroscience, 21*(1), 88-97. https://doi.org/10.9758/cpn.2023.21.1.88

Nasim, S., Alavi Naeini, A., Najafi, M., Ghazvini, M., & Hassanzadeh, A. (2019). Relationship between antioxidant status and attention deficit hyperactivity disorder among children. *International Journal of Preventive Medicine, 10*, Article 41. https://doi.org/10.4103/ijpvm.IJPVM_80_18

Oztop, D., Altun, H., Baskol, G., & Ozsoy, S. (2012). Oxidative stress in children with attention-deficit/hyperactivity disorder. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 39*(1), 110-115. https://doi.org/10.1016/j.pnpbp.2012.05.019

Robinette, L. M., Hatsu, I. E., Adetona, O., Wu, C.-M., Johnstone, J. M., Bruton, A. M., Ast, H. K., Odei, J. B., Ziouzenkova, O., Leung, B. Y., & Arnold, L. E. (2025). Multinutrients decrease oxidative stress in children with ADHD: Mediation/moderation analysis of randomized controlled trial data. *Journal of Attention Disorders, 29*(14), 1290-1306. https://doi.org/10.1177/10870547251355998

Selek, S., Bulut, M., Ocak, A. R., Kalenderoglu, A., & Savas, H. A. (2012). Evaluation of total oxidative status in adult attention deficit hyperactivity disorder and its diagnostic implications. *Journal of Psychiatric Research, 46*(4), 451-455. https://doi.org/10.1016/j.jpsychires.2011.12.007

Sezen, H., Kandemir, H., Savik, E., Basmaci Kandemir, S., Kilicaslan, F., Bilinc, H., & Aksoy, N. (2016). Increased oxidative stress in children with attention deficit hyperactivity disorder. *Redox Report, 21*(6), 248-253. https://doi.org/10.1080/13510002.2015.1116729

Simsek, S., Gencoglan, S., Ozaner, S., Kaplan, I., & Kaya, M. C. (2016). Antioxidant status and DNA damage in children with attention-deficit/hyperactivity disorder with or without comorbid disruptive behavioural disorders. *Bulletin of Clinical Psychopharmacology, 26*(2), 119-125. https://doi.org/10.5455/bcp.20151003063820

Verlaet, A. A. J., Breynaert, A., Ceulemans, B., De Bruyne, T., Fransen, E., Pieters, L., Savelkoul, H. F. J., & Hermans, N. (2019). Oxidative stress and immune aberrancies in attention-deficit/hyperactivity disorder (ADHD): A case-control comparison. *European Child & Adolescent Psychiatry, 28*(5), 719-729. https://doi.org/10.1007/s00787-018-1239-4