Corrigendum to “Default mode network connectivity and social dysfunction in children with attention deficit/hyperactivity disorder” [Int. J. Clin. Health Psychol. 23 (4) (2023) 100393]

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The authors regret that the printed version of the above article contained some inaccuracies. The main corrigendum is given in Table 2, Materials and methods (Social composite score) and Results (The sample’s demographic and clinical characteristics and Correlation results). The errors resulted in a number of minor changes through the paper. Please note these minor changes did not affect the conclusions and findings of the study. The correct and final version follows. The authors would like to apologize for any inconvenience caused.

Corrigendum to Section Materials and methods

Corrigendum: Social composite score

An essential point of consideration in our study is the recognized gap in standardized, validated assessment protocols for measuring social dysfunction in children with ADHD. This gap persists despite understanding that social challenges, although secondary to core ADHD symptoms like inattention and impulsivity, significantly impact the quality of life and developmental trajectories of these children. Unlike conditions such as Autism Spectrum Disorder (ASD) and Social Anxiety Disorder (SAD), for which established scales and tools exist, assessing social functioning in ADHD remains complex (Chan et al., 2019). Our study seeks to navigate these challenges by developing a composite score that encapsulates specific facets of social dysfunction pertinent to the ADHD demographic, thereby enriching the current literature in light of the absence of a universally accepted assessment tool.

In line with Saris et al. (Saris et al., 2020), our approach to quantifying aspects of social dysfunction involves synthesizing data from three validated questionnaires, each addressing distinct domains of social challenges: loneliness (social isolation), perceived social disability, and limited social network size. Notably, the component of our composite score addressing loneliness was meticulously evaluated through the De Jong-Gierveld Loneliness Scale (DJGLS) (de Jong-Gierveld & Kamphuls, 1985), which underwent a rigorous adaptation and validation process for our demographic of Chinese children aged 7 to 10. This process included a series of statistical and methodological steps designed to affirm the scale’s reliability and relevance for assessing loneliness in this younger age group. Specifically, the validation process encompassed exploratory and confirmatory factor analyses to explore and confirm the scale’s underlying structure, reliability assessments to ensure internal consistency and stability over time, and external validity checks against related measures of loneliness and social integration. This comprehensive approach ensured that the DJGLS was accurately reflective and applicable for capturing the nuanced experiences of loneliness among children in our study. Furthermore, we employed the World Health Organization Disability Assessment Schedule (WHO-DAS) to assess perceived social disability, utilizing its 5-item social interaction subscale domain to gauge difficulties encountered in forming and maintaining social relationships (Üstün et al., 2010). Although conventional ADHD severity scales, such as the Conners or the Special Needs Assessment Profile (SNAP), serve vital roles in appraising overall symptomatology, they may not fully capture the essence of social dysfunction. The WHO-DAS, specifically through its social interaction subscale, offers a detailed exploration of social isolation and disability, proving invaluable in our comprehensive assessment framework.

The dimension of social network size was explored using the Close Contacts Inventory (Stansfeld & Marmot, 1992), with results inverted to align with the scoring logic of the other components—where higher scores indicate greater social dysfunction. Prior to the aggregation of individual questionnaire scores into the composite metric, a logarithmic transformation and standardization were applied. These processed scores were then averaged to derive the final composite score, effectively summarizing the collective influence of loneliness, perceived social disability, and social network size on the individual’s social dysfunction.

This innovative composite scoring strategy underscores our

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commitment to a holistic exploration of social dysfunction in children with ADHD. By integrating these specifically chosen measures into a unified score, we unveil the complex interplay of factors contributing to social challenges, facilitating a more refined analysis of their impact on DMN connectivity. Through this methodology, we aspire to extend deeper insights into the multifaceted nature of social dysfunction in ADHD, contributing significantly to both academic discourse and therapeutic strategies.

Corrigendum to Section Results

Corrigendum: The sample’s demographic and clinical characteristics

The demographic and clinical characteristics of the Healthy Controls (HCs) and the ADHD groups are delineated in Table 2. Analysis of the data revealed no statistically significant differences in age or frame-wise displacement (FD) between the HCs and ADHD groups, indicating comparable baselines for these fundamental variables. Nonetheless, the investigation uncovered substantial disparities in cognitive and behavioral evaluations.

Children diagnosed with ADHD displayed significantly lower scores across various cognitive assessments, including the FIQ, VCI, PSI, and WMI, with p-values lower than 0.001 for each comparison. This pattern persisted in behavioral memory assessments, where the ADHD group’s scores were notably reduced compared to their healthy counterparts (p < 0.001). In contrast, the ADHD group exhibited higher scores in areas aligned with measures associated with the CBCL—such as social problems, thought problems, delinquent behaviors, and potentially anxiety scores. These elevated scores reflect the behavioral and emotional challenges frequently observed in children with ADHD, indicating a greater degree of impairment in these domains compared to the HCs. The statistically significant differences (p < 0.01) in these areas underscore the ADHD group’s pronounced difficulties, in line with expectations from clinical assessments using tools like the CBCL.

Regarding the treatment regimens of the ADHD group, our data show variation in medication intake: 24 participants were on methylphenidate, 11 on atomoxetine, and 5 were taking Xiaoer Zhili syrup, a traditional Chinese medicine. It is crucial to clarify that our study’s exclusion criteria were mindful of long-term medication use; however, short-term prescription use (defined as less than six months of consecutive treatment) did not constitute grounds for exclusion. This inclusion criterion was applied to mitigate potential confounding effects attributable to prolonged medication use while ensuring the sample reflected a realistic cross-section of the ADHD child population, which often involves pharmaceutical management. Altogether, 40 of the ADHD participants were categorized under this short-term medication paradigm.

Corrigendum: Correlation results

Our correlation analysis uncovered a number of significant associations. First, we found a negative relationship between the altered zero-lag functional connectivity (zFC) of the right angular gyrus and the posterior cingulate cortex (PCC), and the full-scale IQ (FIQ) score (r = -0.21, p = 0.03), as illustrated in Fig. 4A. It is noteworthy to mention that in conducting this specific analysis, our dataset included a broader range of FIQ scores, extending slightly below our general study inclusion criteria of 70. This methodological decision was driven by a hypothesis-driven rationale, aiming to capture a fuller spectrum of cognitive abilities and to explore whether even subtle variations in FIQ might correlate with neural connectivity patterns in meaningful ways. We believe this broader perspective enhances our understanding of the complex interplay between cognitive functions and neural connectivity, potentially revealing insights that stricter adherence to inclusion criteria might overlook.

Next, a significant negative correlation was observed between the disrupted zFC of the ventral medial prefrontal cortex (vMPFC) and the right middle frontal gyrus (rMFG), and behavioral memory (r = -0.32, p = 0.021), as depicted in Fig. 4B. Finally, we detected a negative association between the zFC of the parahippocampal cortex (PHC) and the left superior frontal gyrus (SFG), and the social composite score (r = -0.14, p = 0.01), as seen in Fig. 4C.

It is important to emphasize that these correlations represent relationships among the variables rather than causative effects. In other words, although the results indicate that certain variables are related, we cannot conclude that one variable directly causes changes in the other based on this analysis alone. This inclusion of a wider FIQ range specifically for the correlation analysis underscores our commitment to a comprehensive exploration of the data, driven by the hypothesis that a broader spectrum of cognitive abilities might demonstrate meaningful relationships with neural connectivity. More in-depth studies are required to further explore these relationships to potentially determine any causal connections and to further refine and validate the measures of social functioning used in this analysis.
Fig. 4. Significant correlations ($p < 0.05$) between altered zFC and various cognitive and behavioral measures: (A) between the right angular gyrus and the posterior cingulate cortex (PCC) and the full-scale IQ (FIQ) score ($r = -0.21$, $p = 0.03$), (B) between the disrupted zFC of the ventral medial prefrontal cortex (vMPFC) and the right middle frontal gyrus (rMFG), and behavioral memory ($r = -0.32$, $p = 0.021$), and (C) between the zFC of the parahippocampal cortex (PHC) and the left superior frontal gyrus (SFG), and the social composite score ($r = -0.14$, $p = 0.01$). Data points reflecting FIQ scores below 70 are highlighted in green to explore a specific hypothesis regarding zFC and cognitive functioning.

References


