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Effect of Preadolescents' Obesity on Inhibitory Control During Stop-Signal Task: A Functional EEG Network Study

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ABSTRACT *Objectives*. Until now, limited knowledge remains regarding the association among childhood obesity, cognitive behavior, and brain networks. Utilizing a publicly available dataset, we aimed to investigate the relationships between childhood obesity and functional networks during the stop-signal task. *Results*. Given the huge conflict-monitoring and inhibitory control demands of the task, both enhanced network connectivity and properties were observed under the "No-go" compared to the "Go" condition for both obese and non-obese preadolescents. Obese preadolescents exhibited significantly increased frontal-parietal, frontal-occipital, and frontal-temporal linkages, as well as heightened network efficiency under both "Go" and "No-go" conditions compared to non-obese counterparts. Additionally, significant correlations were found between network connectivity and properties and preadolescents' body mass index (BMI), with their combination predicting BMI scores successfully. *Conclusions*. These findings support that childhood obesity is not simply a deviant habit with restricted physical health consequences, but rather associated with the atypical development of frontal-based networks involved in inhibitory control and cognitive performance.

INDEX TERMS Obese preadolescents, inhibitory control, functional network, stop-signal task.

IMPACT STATEMENT We found that childhood obesity is associated with atypical development of frontal-based networks involved in inhibitory control and cognitive performance, which may lead to specific, targeted obesity interventions.

I. INTRODUCTION

The epidemic of childhood obesity has emerged as one of the most severe public health concerns in the present century, increasing the risk for various ailments including cardiovascular disease, hypertension, diabetes, cancers, and psychiatric disorders [1]. Additionally, psychosocial consequences of obesity such as poorer educational attainment, higher rates of poverty, and lower household income [2]. Given the detrimental impact of obesity on health and cognitive function in adulthood, the question of whether these

relationships are evident in childhood has received increasing attention [3]. Recent studies suggest that childhood obesity adversely affects the brain development that occurs over childhood and adolescence, along with negative consequences for cognitive control development, such as attention and visuospatial performance, and academic achievement [4], [5], [6]. In this study, we specifically investigated whether childhood obesity is associated with alterations in the brain's functional network and cognitive control, particularly among obese preadolescents, aiming to provide new insights into the association between childhood obesity and brain cognition.

Cognitive control refers to "the ability to coordinate thought and action according to internal goals" and encompasses inhibition, working memory, and cognitive flexibility [7], [8]. It is thus important in preadolescents with obesity, facilitating the orchestration and maintenance of healthy goal-directed behaviors related to food intake and physical activity [9]. In support of this view, numerous experimental studies have indicated that childhood obesity correlates with deficits in cognitive inhibitory control, manifested by prolonged response ctimes and worse accuracy during the task [10], [11]. The stop-signal task, commonly employed to evaluate cognitive response abilities in preadolescents [12]. Specifically, the "Go" and "No-go" conditions instruct participants to engage in proactive and inhibitory responses towards a string of stimuli, primarily relying on the activation of frontoparietal and ventral networks, with a robust right-lateralized reactive mechanism [13], [14]. It has been proposed that the right prefrontal cortex plays a crucial role in cognitive control of feeding and obesity [15]. Given this right-lateralized mechanism for reactive inhibition, we hypothesize that this task will reveal greater deficits in obese preadolescents compared to their non-obese counterparts. When comparing obese and non-obese preadolescents, despite the insignificant behavioral difference, the event-related potentials (ERPs) derived from individual electroencephalogram (EEG) indicated that the obese preadolescents exhibited an electrophysiological pattern associated with variations in attentional allocation during the "Go" condition (less modulation of P300a latency) and conflict monitoring during the "No-go" condition (less modulation of N200 latency) [16].

Moreover, obesity is linked to abnormalities in specific brain regions and disruptions in inter-regional communication [17]. Notably, distributed regions such as the dorsal and pregenual anterior cingulate cortex, posterior cingulate, and inferior parietal area, etc. are implicated in "obesity neural activity" [18]. Recent investigations on childhood and adolescent obesity have also revealed anomalies across various brain regions and neurocognitive domains [19], [20]. Generally, connections across regions have been extensively studied as prominent features [21], [22], for instance, weaker connectivity between cortical and subcortical elements of the reward system has been associated with higher body mass index (BMI) in children with obesity [23]. However, most studies have primarily focused on resting-state activity rather than task characteristics in individuals with obesity, failing to reveal the network mechanisms underlying childhood obesity-induced decline in executive control. It is worth noticing that preadolescence is a key period characterized by structural and functional changes in brain network reorganization [24], which are relevant to the development of executive control functions. Recent research has shown significantly increased normalized clustering coefficient and small-world property of resting-state functional network in obese preadolescent children compared to healthy controls [25]. Nevertheless, to date, no study has assessed potential changes in the EEG functional network using the stop-signal task (reactive inhibition) in the preadolescent population. Herein, utilizing a publicly available dataset, our objective is to investigate the deficits in functional networks among obese preadolescents during the stop-signal task. Given previous studies that have demonstrated the promising validity of graph metrics and network connectivity as "biomarkers" for examining task-induced alterations [26], [27], [28], we believe our research will also deepen the network knowledge of childhood obesity and executive functions. Furthermore, identifying critical functional network nodes or connections associated with obesity in preadolescents could offer valuable insights for the development of neural interventions aimed at enhancing executive functions. Techniques such as transcranial magnetic stimulation or transcranial direct current stimulation hold potential as effective preventive or therapeutic measures for obesity."

In this study, we investigated the distinctive functional network patterns in obese preadolescents, as well as probed potential disparities in brain activity between obese and non-obese preadolescents during the stop-signal task. This exploration aims to deepen our understanding of the relationship between weight status and cognitive control. Given previous studies have reported no discernible differences in behavioral performance among weight groups during the "Go" trials and "No-go" trials, our objective is to uncover cognitive deficits associated with childhood obesity at the level of brain networks and demonstrate that neural resource utilization during task engagement may precede observable behaviors (Table 1).

TABLE 1. The Definitions of the Acronyms

Acronyms	Definitions
BMI	Body Mass Index
Obese group	Preadolescents with obesity
Non-obese group	Healthy preadolescents
ERP	Event-Related Potential
EEG	Electroencephalogram
FDR	False Discovery Rate
CC	Clustering Coefficient
CPL	Characteristic Path Length
GE	Global Efficiency
LE	Local Efficiency
RMSE	Root Mean Square Error
NRMSE	Normalized Root Mean Square Error
CN_{Go}	The sum of correlated edges under "Go" condition
$CN_{No\text{-}go}$	The sum of correlated edges under "No-go" condition

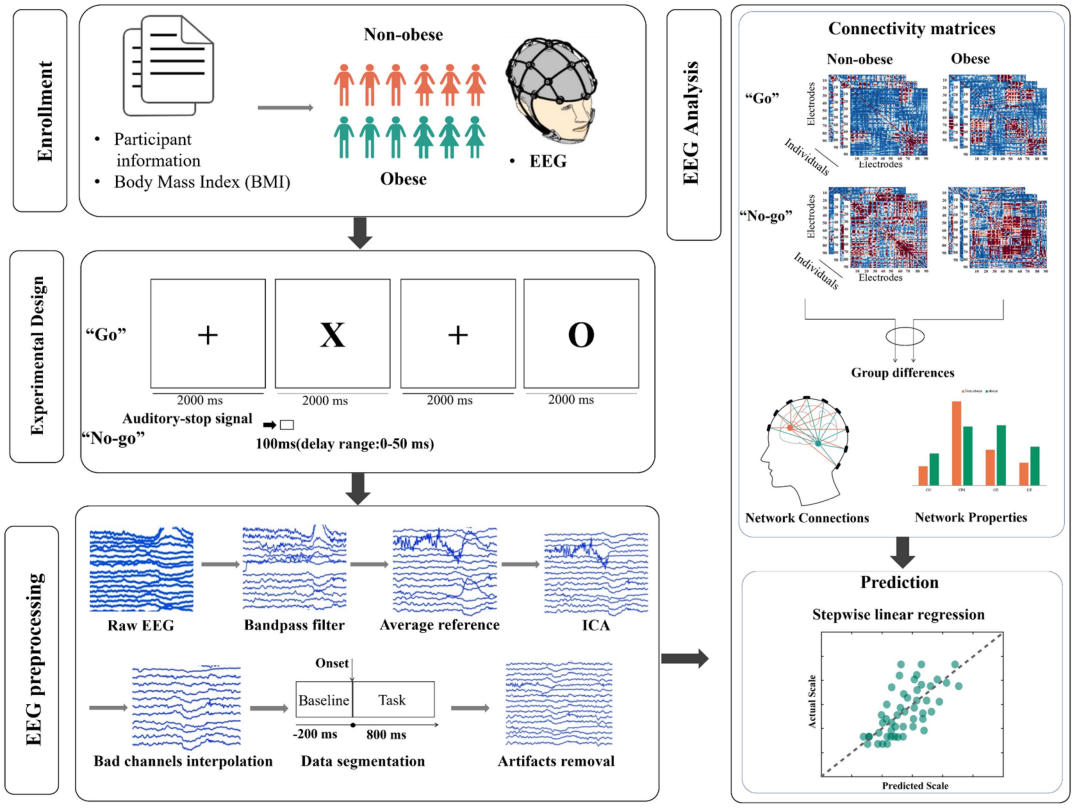

FIGURE 1. The workflow of data analysis. The procedure consists of four parts: participant enrollment, experiment design (stop-signal task: "Go", and "No-go" conditions are represented in a timeline), EEG preprocessing (ICA: Independent component analysis. The ICA was used to remove residual artifacts by removing artifact components), and EEG analysis. Specifically, the EEG analysis including the network construction using the weighted phase lag index, calculation of network properties, BMI scores prediction based on network features using the stepwise linear regression). Herein, the "Non-obese" represents the healthy preadolescents and the "Obese" represents the preadolescents with obesity.

TABLE 2. The Demographic Information of the Participants

	Obese (n = 25)	Non-Obese (n = 29)	p value b
Age (Year)	9.59 (0.25)	9.64 (0.57)	0.35
Gender (M/F) [a]	10/15	11/18	0.55
BMI	25.18 (8.28)	17.53 (1.58)	0.00

Data was shown as Mean value (Standard Deviation). a. M for males and F for females. b. $\chi 2$ test for gender, independent sample *t*-tests for the other measures. BMI: body mass index. The "Non-obese" represents the healthy preadolescents and the "Obese" represents the preadolescents with obesity.

II. RESULTS

A. DEMOGRAPHIC RESULTS

In this study, we initially conducted statistical comparisons of demographic metrics between obese and non-obese preadolescents. Specifically, no disparities were observed in age as indicated by independent sample *t*-tests (t = -0.400, p = 0.346) or gender distribution as indicated by the Chi-square test ($\chi 2 = 0.024, p = 0.549$). However, significant differences in emerged in BMI scores (t = -12.970, $p < 0.001$), with the obese preadolescents exhibiting higher BMI scores (see Table 2).

B. DIFFERENCES IN NETWORK TOPOLOGIES AND NETWORK PROPERTIES

Initially, brain network analysis was conducted to investigate topological differences between obese and non-obese groups, as well as between "Go" and "No-go" conditions in the beta band. This aimed to explore the neural mechanism of obese preadolescents and validate the efficacy of the stop-signal task in this population. Fig. 2(a) revealed a significant main effect of the *Condition* by two-way repeated analysis of variance (ANOVA) ($p < 0.0001$, false discovery rate (FDR) corrected), where stronger connectivity was observed for the "No-go" condition compared to the "Go" condition (represented by the blue solid line). Subsequently, paired *t*-tests were conducted between both conditions per group. The results showed that the "No-go" condition exhibited enhanced connectivity spanning whole brain regions compared to the "Go" condition of the both obese ($p < 0.0001$, FDR corrected, Fig. 2(b)) and non-obese groups ($p < 0.0001$, FDR corrected, Fig. 2(c)). Additionally, the statistical analysis demonstrated a significant main effect of *Group* ($p < 0.01$, FDR corrected). As displayed in Fig. 2(d), prominently stronger edges were identified for the obese group than for the non-obese group. Specifically, under both "Go" (Fig. 2(e)) and "No-go" (Fig. 2(f)) conditions,

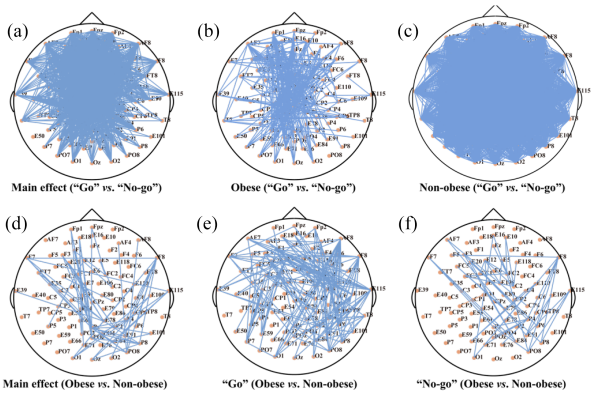

FIGURE 2. **Comparison of the network topology within the beta band between obese and non-obese groups, as well as between "Go" and "No-go" conditions. (a) Edges with significant main effect of the *Group* factor in network topology ($p < 0.0001$, false discovery rate (FDR) corrected). (b) Differences in network topologies between "Go" and "No-go" conditions for obese preadolescents ($p < 0.0001$, FDR corrected). (c) Differences in network topologies between "Go" and "No-go" conditions for non-obese preadolescents ($p < 0.0001$, FDR corrected). (d) Edges with significant main effect of the *Condition* factor in network topology ($p < 0.01$, FDR corrected). (e) Differences in network topologies between obese and non-obese groups under "Go" condition ($p < 0.01$, FDR corrected). (f) Differences in network topologies between obese and non-obese groups under "No-go" condition ($p < 0.01$, FDR corrected). In each subfigure, the blue lines denote the stronger edges of the obese group than that of the non-obese group, as well as denote the stronger edges of the "No-go" condition than that of the "Go" condition.**

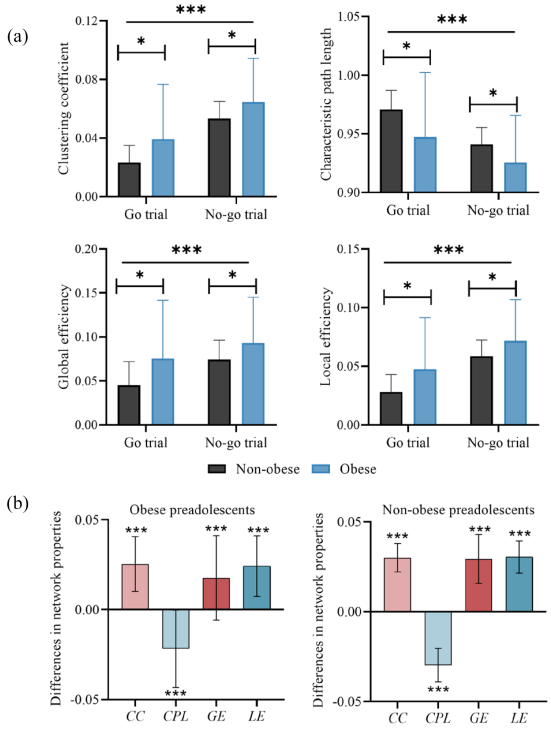

FIGURE 3. **Network properties of obese and non-obese groups under the "Go" and "No-go" conditions. (a) Comparison of the network properties between obese and non-obese groups under the "Go" and "No-go" conditions within the beta band. Values are the mean and standard deviation, ∗ represents $p < 0.05$, and ∗∗∗ represents $p < 0.001$. (b) Differences of the network properties between "Go" and "No-go" conditions for obese and non-obese preadolescents within the beta band. Values are the mean and standard deviation of differences, ∗∗∗ represents $p < 0.001$.**

independent sample *t*-tests indicated that frontal-parietal, frontal-occipital, and frontal-temporal connections exhibited significantly stronger strength in the obese than in the non-obese group ($p < 0.01$, FDR corrected). However, no significant interactive effect ($p < 0.01$, FDR corrected) of *Condition* × *Group* on network topologies was reported. Furthermore, results of the differential network topologies in other bands (i.e., delta, theta, alpha, and gamma) were summarized in Supplementary Materials (see Fig. S1) available online, further determining that brain network features in the beta band most strongly reflect abnormal neural mechanisms induced by childhood obesity.

We then focused on the quantified network properties, i.e., clustering coefficient (*CC*), characteristic path length (*CPL*), global efficiency (*GE*), and local efficiency (*LE*), to mine any potential difference in the aforementioned comparisons. The two-way repeated ANOVA revealed significant main effects of *Condition* on the network properties [*CC*, F (1,104) = 34.254, p <0.001, $\eta_p^2 = 0.021$; *GE*, F (1,104) = 7.543, $p = 0.007$, $\eta_p^2 = 0.015$, *LE*, F (1,104) = 23.712, p <0.001, $\eta_p^2 = 0.020$; *CPL*, F (1,104) = 14.868, p <0.001, $\eta_p^2 = 0.018$]. Specifically, paired *t*-tests indicated that compared to the "Go" condition, both non-obese and obese preadolescents exhibited higher *CC* (non-obese: $t = 20.425$, $p = 0.000$; obese: $t = 8.307$, $p = 8.049e\text{-}09$), *GE* (non-obese: $t = 11.605$, $p = 1.635e\text{-}12$; obese: $t = 3.744$, $p = 5.021e\text{-}04$), *LE* (non-obese: $t = 18.230$, $p = 0.000$; obese: $t = 7.164$, $p = 1.050e\text{-}07$), and lower *CPL* (non-obese: $t = -17.261$, $p = 1.110e\text{-}16$; obese: $t = -5.069$, $p = 1.745e\text{-}05$) under the "No-go" condition. Additionally, *Group* differences were observed for network properties [*CC*, F (1,104) = 8.428, $p = 0.005$, $\eta_p^2 = 0.005$; *CPL*, F (1,104) = 8.349, $p = 0.005$, $\eta_p^2 = 0.010$; *GE*, F (1,104) = 8.359, $p = 0.005$, $\eta_p^2 = 0.016$; *LE*, F (1,104) = 8.516, $p = 0.004$, $\eta_p^2 = 0.007$]. As shown in Fig. 3(a), independent sample *t*-tests revealed that under both experimental conditions, compared to the non-obese group, the obese group displayed decreased *CPL* ("Go": $t = -2.177$, $p = 0.017$; "No-go": $t = -1.914$, $p = 0.031$) and prolonged *CC* ("Go": $t = 2.194$, $p = 0.016$; "No-go": $t = 1.903$, $p = 0.031$), *GE* ("Go": $t = 2.281$, $p = 0.013$; "No-go": $t = 1.777$, $p = 0.041$), *LE* ("Go": $t = 2.238$, $p = 0.015$; "No-go": $t = 1.873$, $p = 0.033$). However, no significant interactive effect of *Condition* × *Group* was observed for these network properties [*CC*, F (1,104) = 0.241, $p = 0.624$; *CPL*, F (1,104) = 0.359, $p = 0.551$; *GE*, F (1,104) = 0.471, $p = 0.494$; *LE*, F (1,104) = 0.303, $p = 0.583$]. Moreover, considering that the mean values of network properties under the "Go" and "No-go" conditions are very close while their variances are large, we first performed a subtraction operation on these properties. Specifically, we subtracted the values in the "Go" condition from values in the "No-go" condition, followed by conducting a hypothesis test against zero. As depicted in Fig. 3(b), the disparities in *CC*, *GE*, and *LE* across all preadolescents were

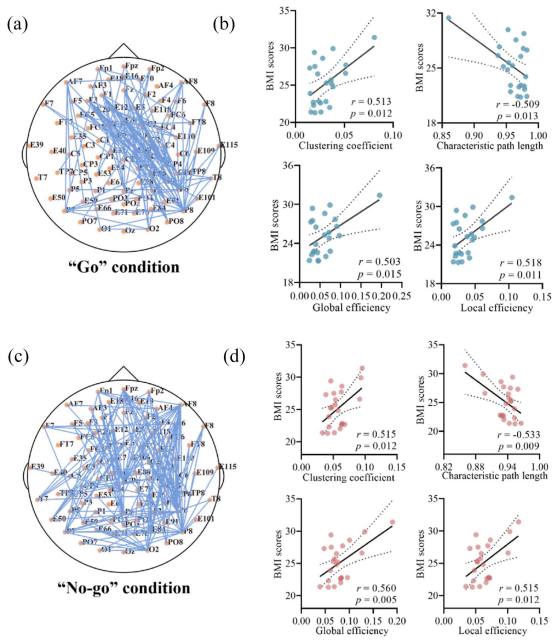

FIGURE 4. Relationships between body mass index (BMI) scores and network features. (a) Relationships between BMI scores and network connections under the "Go" condition. (b) Relationships between BMI scores and network properties under the "Go" condition, (c) relationships between BMI scores and network connections under the "No-go" condition. (d) Relationships between BMI scores and network properties under the "No-go" condition. In the network topologies, the blue lines indicate the significant positive correlations between the BMI scores and the network connections ($p < 0.05$, FDR corrected), and in each scatter correlogram, the black line is the fitted linear trend between two variables, the region of the black dashed line is the confidence interval, the circles are the included participants, *r* is the correlation coefficient, and *p* represents the statistical significance level.

found to be greater than 0, whereas the disparities in *CPL* were less than 0 within the beta band ($p < 0.001$). These observations align with the trends illustrated in Fig. 3(a).

C. BRAIN NETWORKS CORRELATED WITH BMI SCORES OF OBESE PREADOLESCENTS

Correlation analysis was further conducted to examine the relationship between brain networks and BMI scores of obese preadolescents in both experimental conditions. Fig. 4(a) and (c) illustrate network connections exhibiting a significant positive correlation with obese preadolescents' BMI scores ($p < 0.05$, FDR corrected), specifically highlighting frontal-parietal, and parietal-occipital connections. Meanwhile, participants with the 10% largest Malahanobis distances to the data center were considered as the outliers [29], and thus two obese preadolescents were accordingly excluded, to avoid obvious outliers that deviate from the data center would weaken the underlying relations. As depicted in Fig. 4(b) and 4D, *CPL* ("Go": $r = -0.509$, $p = 0.013$; "No-go": $r = -0.533$, $p = 0.009$) was significantly negatively correlated with the BMI scores, while *CC* ("Go": $r = 0.513$, $p = 0.012$; "No-go": $r = 0.515$, $p = 0.012$), *GE* ("Go", $r = 0.503$, $p = 0.015$; "No-go", $r = 0.560$, $p = 0.005$), and *LE* ("Go", $r = 0.518$, $p = 0.011$, "No-go", $r = 0.515$, $p = 0.012$) were positively

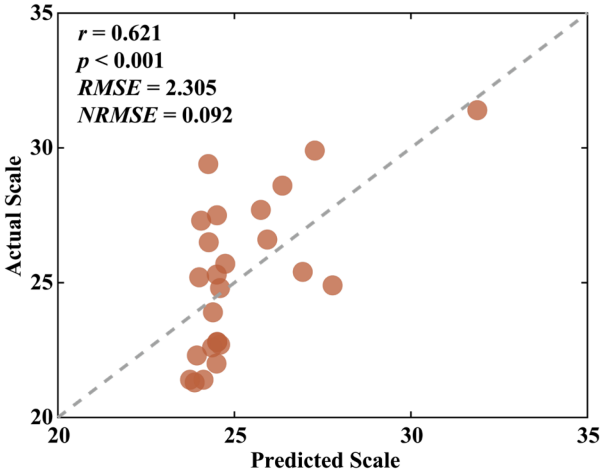

FIGURE 5. The correlation between predicted and actual BMI scores of preadolescents. The *p* represents statistical significance, *r* represents the correlation coefficient, *RMSE* is the root mean square error and *NRMSE* is the normalized root mean square error of predicting the model. The orange-filled circles are the enrolled participants.

correlated with the BMI scores under both experimental conditions. Given the importance of evaluating physical status for normal-weight preadolescents, we further explored the potential relationship between network features of all preadolescents and BMI scales, summarizing the findings in the Supplementary Materials (see Fig. S2) available online.

D. PREDICTION OF THE OBESE PREADOLESCENTS' BMI SCORES BY NETWORK FEATURES

Given the close correlations between network connections, and properties and obese preadolescents' BMI scores (Fig. 4), these network features may contribute significantly to predicting their BMI scores. To validate this, based on the correlated network parameters (i.e., CPL_{Go}, CC_{Go}, GE_{Go}, LE_{Go}, the sum of correlated edges under "Go" condition (CN_{Go}), $CPL_{No\text{-}go}$, $CC_{No\text{-}go}$, $GE_{No\text{-}go}$, $LE_{No\text{-}go}$, and the sum of correlated edges under "No-go" condition ($CN_{No\text{-}go}$)), a stepwise multiple linear regression model was constructed to predict the obese preadolescents' BMI scores. Fig. 5 depicts the correlations between predicted and original BMI scores, with the *X*- and *Y*-axes representing predicted and original BMI scores, respectively. The corresponding Pearson's correlation coefficient was $r = 0.621$ ($p < 0.001$), and the root mean square error (*RMSE*) was 2.305, with the normalized *RMSE* (*NRMSE*) of 0.092, indicating strong predictive power. Furthermore, the prediction results of BMI scores for all preadolescents were summarized in the Supplementary Materials (see Fig S3).

III. DISCUSSION

As previously explored, no significant differences in behavioral performance were observed between weight groups in the "Go" and "No-go" conditions based on this publicly available database. One possible explanation for this finding was proposed: both groups showed a similar response accuracy because the task required a higher level of inhibitory control

abilities than what preadolescents were able to compensate for using their previously acquired strategies or skills [16]. However, the study also found differences in N200 latency during the "No-go" condition and in P300 latency during the "Go" condition between weight groups, indicating that neural resources vary among weight groups even when behavioral performance remains unaffected [16]. The results of significant differences in functional networks between obese and non-obese preadolescents during the stop-signal task in the current study further support this finding and also enhance our understanding of executive control development in obese preadolescents.

Based on previous studies in adults using the stop-signal task, we would expect to observe enhanced functional connectivity during the "No-go" condition, reflecting increased conflict-monitoring and inhibitory control [30]. As mentioned above, stronger functional network connections are believed to indicate a compensatory function characterized by recruiting more neural resources and facilitating more efficient information exchanges to resolve the infrequent stimulus condition [31]. In line with these expectations, our findings demonstrate significant main effects of the experimental conditions on network topology. Specifically, Fig. 2(a)–(c) illustrates that the entire brain exhibits stronger functional connectivity during the "No-go" condition. In addition, increases in functional connectivity of the "No-go" condition were also quantified by the enhanced *CC*, *GE*, and *LE* as well as a shorter *CPL* (see Fig. 3). We suggest that the stronger network information transmission may be related to the need to compensate for the greater inhibitory control under the "No-go" condition. It is noteworthy that the non-obese group exhibited stronger connectivity differences between "No-go" and "Go" conditions compared to the obese group, which may be related to the fact that non-obese preadolescents recruited more neural resources to resolve infrequent stimuli ("No-go") [16]. Conversely, obese preadolescents showed smaller network differences between the two conditions, suggesting that the obese group might have lower neural efficiency due to executive function deficits under both conditions. This further highlights the association of childhood obesity with atypical development of functional networks involved in inhibitory control.

Next, we delved into the differences in functional networks between two weight groups. Prior investigations have indicated that childhood obesity correlates with compromised inhibitory control within the frontal-parietal network, diverging from the developmental trajectory of healthy-weight children [32], [33]. As anticipated, we observed a main effect of childhood obesity on the conflict-monitoring mechanism, with obese preadolescents displaying heightened frontal-parietal, frontal-temporal, and frontal-occipital connectivity within the beta band compared to their non-obese counterparts across both experimental conditions (Fig. 2(d)–(e)). As previously mentioned, the stop-signal task involves proactive control processes characterized by enhanced activation of the frontal-parietal network and lateral prefrontal cortex, pivotal components of the executive-control network whose dysfunction has been closely linked to obesity [34], [35]. Hence, the significantly distinct network patterns in obese and non-obese preadolescents within these regions are perhaps unsurprising. Furthermore, upon investigating the network properties of obese preadolescents, we identified the smaller *CPL* and longer *CC*, *GE*, and *LE*. As clarified in previous studies, increased *CC*, *GE*, *LE,* and decreased *CPL* consistently correspond to the promoted information transmission [36]. Consequently, it can be speculated that heightened information transmission, functional integration, and segregation observed in obese preadolescents relative to healthy-weight children reflect a top-down proactive control mechanism that may provide more effective strategies for responding to the demands of the stop signal task [21], [37], [38]. Moreover, we noted fewer connectivity differences between the obese and non-obese groups under the "No-go" condition, which we propose is attributable to the high degree of inhibitory control required for the task, not only for the obese group but also for the non-obese group. Despite the "Go" condition being the frequent stimulus, obese participants exhibit significantly different network patterns compared with their normal-weight peers, which may be related to the recruitment of additional neural resources for obese preadolescents due to the difficulty in processing the cognitive demands of the simple task [39]. Importantly, the differential network (see Fig. 2(e)) and related network (see Fig. S2A and Fig. S2C) in our results demonstrated a significant right-frontal lateralization, coinciding with the right brain hypothesis for obesity, whereby dysfunction of the right prefrontal cortex may represent a central event in the etiology of human obesity [15], [40]. Overall, these differences in neural resources of the brain may further reflect the atypical development of integrative and executive functions associated with childhood obesity, although not yet evident behaviorally.

Interestingly, we also observed a significant association between higher BMI scores in obese preadolescents and stronger frontal-parietal, and parietal-occipital connections (see Fig. 4(a) and (c)). An increased BMI also was accompanied by an increased *CC*, *GE*, and *LE* as well as a decreased *CPL* (see Fig. 4(b) and (d)). On the one hand, obesity is typically determined by BMI, with higher BMI scores indicating greater fat mass in individuals [41]; On the other hand, increased connectivity, *CC*, *GE*, and *LE*, as well as the decreased *CPL* reflect an enhancement in information processing efficiency within the brain [36]. Therefore, preadolescents with higher levels of obesity exhibit heightened network connectivity, potentially indicating a reduced ability to effectively evaluate proactive control in the stop-signal task. This may result in the expenditure of more neural resources than their normal-weight peers to compensate for it [37]. These findings further underscore the association between obesity and changes in brain functional networks, which may serve as potentially more sensitive markers of obesity-related decrements in cognitive control in children.

Furthermore, our attempts to use network features as predictors for obese preadolescents and construct a robust model for predicting BMI scores in children yielded valuable

insights. Specifically, we employed these EEG metrics (i.e., CPL_{Go}, CC_{Go}, GE_{Go}, LE_{Go}, the sum of correlated edges under "Go" condition (CN_{Go}), $CPL_{No\text{-}go}$, $CC_{No\text{-}go}$, $GE_{No\text{-}go}$, $LE_{No\text{-}go}$, and the sum of correlated edges under "No-go" condition ($CN_{No\text{-}go}$)) and conducted stepwise multiple linear regression analysis to develop predictive models for BMI scores of obese preadolescents. Fig. 5 presents scatterplots of the actual and predicted BMI scores for obese preadolescents, revealing a significantly positive relationship that validates the reliability of network connectivity. This finding further emphasizes that brain functional networks serve as a sensitive indicator for obese preadolescents.

However, the present study is limited by its relatively small sample size. Future investigations should aim to replicate these findings using larger groups. Furthermore, it remains unclear whether increased brain connectivity contributes to obesity or is a consequence of it. Longitudinal studies focusing on individuals at risk of developing obesity are needed to enhance the understanding of the relationship between brain function and obesity. Although this study is not without limitations, our preliminary findings are intriguing and provide initial evidence suggesting that childhood obesity is associated with the aberrant development of frontal-based networks involved in executive control function and cognitive performance. Research in this area is still in its early stages, and future studies will increase our understanding of the neural correlates of obesity, brain networks, and other health behaviors in children, which may lead to specific, targeted obesity interventions. For instance, targeting key nodes identified in the differential network from our findings, such as AF8 and F8, through interventions like transcranial magnetic stimulation could potentially enhance executive functioning in obese preadolescents. Better executive functioning might help optimize treatment results for obesity through enhancing the maintenance of a healthy and well-balanced diet.

IV. MATERIALS AND METHODS

A. EEG DATASETS

The dataset used in this study was collected by Alatorre-Cruz et al. at the Arkansas Children's Nutrition Center (ACNC) in Little Rock, Arkansas, with support from the University of Arkansas for Medical Sciences (UAMS) and the U. S. Department of Agriculture/Agricultural Research Service program 6026-51000-012-06S [16] (https://doi.org/10.18112/openneuro.ds004151.v1.0.0). Using E-Prime software (Version 2), EEG signals were recorded from 57 preschoolers during a stop-signal task. Following BMI scoring criteria, EEG data from 29 participants were categorized into the non-obese group, while data from 27 participants were categorized into the obese group after excluding one subject due to incorrect grouping. Comprehensive information regarding this dataset, including data collection procedures and demographic details of the enrolled children, can be accessed on the Openneuro website. Additional specifics regarding the experimental setup of this dataset are provided in the Supplementary Materials available online.

B. EEG ANALYSIS

In this study, our primary focus was on investigating EEG networks in preadolescents during the stop-signal task. Fig. 1illustrates the protocols employed to examine distinct network mechanisms between obese and non-obese preadolescents. These protocols encompassed participant enrollment, experimental design, EEG preprocessing, and EEG feature extraction. Below, we provide details of the corresponding sub-procedures.

C. EEG PREPROCESSING

In this study, data preprocessing was conducted offline using EEGLAB and custom MATLAB (v2014a; MathWorks, Inc., USA) scripts. To exclude lateral electrodes sensitive to eye, facial, and head movements [42], [43], 90 channels were selected from the original 128 channels. This channel subset included electrodes sparsely distributed on the scalp and covering the entire brain (e.g., Fpz/1/2, AF3/4/7/8, Fz/1/2/3/4/5/6/7/8, FC1/2/3/4/5/6, FT7/8/9/10, C1/2/3/4/5/6, T7/8, CPz/1/2/3/4/5/6, TP7/8/9/10, Pz/1/2/3/4/5/6/7/8, POz/3/4/7/8, Oz/1/2, etc). Thereafter, the average reference was applied. Independent component analysis was then utilized to remove residual artifacts by removing artifact components [44], followed by the replacement of noisy channels using spherical spline interpolation. Participants with more than 10 bad channels were excluded, resulting in the removal of two participants from the obese group. Given that related characteristics of obese preadolescents within the beta band have been proved to exist deviations during cognitive tasks [45], [46], [47], the data was bandpass filtered offline in the beta range (13 – 30 Hz). Finally, [−200, 800] ms segmentation (0 ms indicates the stimulus onset), [−200, 0] ms baseline correction, and artifact removal (± 120 μV as the threshold) were adopted to the filtered EEG signals.

D. POWER ANALYSIS

After EEG preprocessing, a total of 54 preadolescents were included in the subsequent analysis, comprising 25 obese preadolescents and 29 non-obese preadolescents. To evaluate the adequacy of our sample size for detecting significant differences or correlations, we conducted a power analysis using G Power 3 software [48]. With a large effect size ($f = 0.4$) [49], our experimental design (2 *Conditions* $\times$ 2 *Groups*) required a sample size of 52 subjects to achieve a statistical power of 0.8 at a significance level of 0.05. For correlation analysis, we employed 1-sided testing, with a significance level of 0.05 and a statistical power of 0.8. Given a null hypothesis Pearson correlation coefficient of 0 and an observed Pearson correlation coefficient of 0.34, the total sample size required was computed as 52. Therefore, the power analysis confirmed the reliability of the significant differences or correlations identified in our sample.

E. BRAIN NETWORK CONSTRUCTION

In our present study, to reduce the effect of volume conduction, brain networks were constructed using the weighted phase lag index (wPLI), which has previously been employed for characterizing connectivity in cognitive response [50]. Further details regarding wPLI can be found in the Supplementary Materials. The wPLI between 90 channels was computed for each "Go" trial and "No-go" trial in the beta frequency band using the FieldTrip toolbox in MATLAB software. The final weighted network for each subject, i.e., the 90×90 adjacency matrix, was obtained by averaging the matrices of all trials specific to the beta band.

Subsequently, to quantitatively measure the brain's efficiency in processing relevant information, four network properties, i.e., *CC*, *CPL*, *GE*, and *LE* were calculated based on the wPLI adjacency matrix. Further details on these network properties are reported in the Supplementary Materials available online.

F. PREDICTION OF THE BMI SCORES

In our present study, we established a stepwise multiple linear regression model based on these network metrics in the beta band to predict the BMI scores [51]. To ensure the generalizability of the predictive model, we employed the leave-one-out cross-validation (LOOCV) strategy [52]. Additionally, we calculated the *RMSE* and *NRMSE* [53] to quantify the prediction error (details provided in Supplementary Materials) available online.

SUPPLEMENTARY MATERIALS

Detailed information on the experimental design, brain network construction, network properties, prediction of the BMI scores, statistical analysis, and the differences in network topologies in other bands are provided in the Supplementary Materials available online.

CONFLICT OF INTEREST

The authors declare they have no conflicts of interest.

AUTHORS' CONTRIBUTIONS

Study conception and design: Yuqin Li, Qian Yang, Peng Xu, Fali Li, Yi Liang; data analysis and interpretation: Yuqin Li, Qian Yang, Yuxin Liu, Yutong Zheng, Jianfu Li; writing—original draft preparation: Yuqin Li, Chunli Chen, Baodan Chen, Dezhong Yao, Liang Yu, Fali Li, Yi Liang; writing—review and editing: Yuqin Li, Qian Yang, Yuxin Liu, Peng Xu, Fali Li, Yi Liang; funding acquisition: Peng Xu, Fali Li. All authors read and approved the final manuscript.

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