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Neuronal inhibitory mechanisms in overweight/obesity and food/eating addiction: The role of frontal brain coherence, dysregulated eating, and depressive symptoms

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BRIEF REPORT

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ABSTRACT

Background and aims: Obesity and food/eating addiction are associated with deficits in inhibitory control mechanisms manifested in the lateral prefrontal cortex (LPFC) bilaterally. We explored abnormalities in the functional coherence between the hemispheres and within each of them. *Methods:* Resting-state electroencephalography (EEG) was utilized to examine intra- and inter-hemispheric frontal brain coherence in individuals with obesity and food/eating addiction (FAOB, $n = 27$), those with obesity without addiction (Non-FAOB, $n = 11$), and healthy controls (HC, $n = 15$). Participants were included in this study following assessment of their BMI and Yale Food Addiction Scale (YFAS) scores. In addition, depressive symptoms were measured with the Beck Depression Inventory (BDI). *Results:* Individuals with obesity (FAOB and Non-FAOB combined) exhibited higher interhemispheric frontal (IF) and right frontal (RF) coherence compared to healthy controls. Within the obesity group, Non-FAOB participants showed higher IF and RF coherence than the FAOB group. These results remained significant after controlling for BDI scores. Depressive symptoms in the FAOB group were higher than those of the HC group. *Discussion:* Elevated frontal coherence, both between the frontal hemispheres and within the right frontal hemisphere, may be associated with effortful cognitive control and possibly a compensatory neurocognitive correlate associated with differential susceptibility to developing addictive-like overeating. *Conclusions:* Frontal brain coherence patterns represent candidate neurophysiological correlates of differential vulnerability to addictive-like overeating in individuals with overweight/obesity and warrant longitudinal investigation to establish their prognostic value.

KEYWORDS

obesity, food/eating addiction, functional connectivity, frontal cortex, inhibitory control, EEG coherence

INTRODUCTION

Obesity and overweight are multifaceted conditions defined by a Body Mass Index (BMI) of ≥28. They are characterized by behavioral and neurometabolic adaptations that promote overeating, even in the absence of metabolic hunger (Ho & Verdejo-Garcia, 2021). During such episodes, individuals often struggle to inhibit their eating, especially when rewarding foods are readily available (Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016). This difficulty has been linked to deficits in inhibitory control systems. Consistently, the inhibitory control deficit theory (Devoto et al., 2018) posits that overeating in obesity reflects impaired higher-order control processes governed by the prefrontal cortex (PFC), particularly the lateral prefrontal cortex (LPFC), which nonetheless fails to suppress food intake despite satiety cues or awareness of adverse consequences (Lavagnino et al., 2016).

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Effective eating regulation depends on the balance between the left and right LPFCs (Lowe, Reichelt, & Hall, 2019). Although both PFC hemispheres contribute to appetite regulation, they fulfill distinct roles: neural activation in the left LPFC is associated with approach behavior and cognitive reappraisal, whereas the right LPFC is involved in impulse control and inhibition, core elements compromised in addiction (Volkow, Wang, Tomasi, & Baler, 2013). The "right hypothesis of obesity" suggests that diminished right LPFC engagement contributes to overeating and obesity (Alonso-Alonso & Pascual-Leone, 2007). However, this simplistic description of differential roles between the hemispheres has been challenged by various findings including improvement of eating-related outcomes following left LPFC stimulation, indicating both hemispheres may support inhibitory control in different ways (Alhindi, Khalifa, Al-Khyatt, & Idris, 2023). Efficient inter-hemispheric communication via the corpus callosum is also essential; its disruption in obesity is associated with impaired white matter integrity and reduced cognitive regulation of eating (Hinkley et al., 2012; Hu et al., 2024).

A subset of individuals with obesity exhibits food/eating addiction (FA; Gearhardt, Corbin, & Brownell, 2009), sharing neurobiological mechanisms with substance use disorders (Volkow et al., 2013). It remains unclear why some individuals with obesity develop FA while others do not, and what neurobehavioral distinctions differentiate these groups. Resting-state EEG coherence quantifies inter-regional oscillatory synchrony and has been linked with executive functions and behavioral inhibition (Park et al., 2017). Yet, only a few studies have examined this in individuals with obesity and FA (FAOB) (De Ridder, Manning, Leong, Ross, & Vanneste, 2016; Imperatori et al., 2015), and none have specifically focused on interhemispheric frontal coherence or group differences in this measure between individuals with and without FA among those with obesity. To fill this gap, the present study examined both intra- and inter-hemispheric resting-state frontal EEG coherence across three groups: FAOB, Non-FAOB, and healthy controls (HC).

METHODS

Participants

As part of a larger clinical trial on transcranial magnetic stimulation as a therapeutic aid for FAOB, we recruited 66 adults aged 18–65 years, of whom 7 were excluded due to technical EEG recording failures or excessive muscular artifact, and a further 12 were excluded due to incomplete questionnaire data or corrupted EEG segments, yielding a final analytic sample of $n = 53$ (FAOB: $n = 27$; Non-FAOB: $n = 11$; HC: $n = 15$; M/F: 34/19). Food/eating addiction status was established with the Yale Food Addiction Scale (YFAS) (Gearhardt et al., 2009).

The original YFAS was used, which has since been superseded by a version with improved diagnostic and psychometric properties (Schulte & Gearhardt, 2017); however, the study was conducted before the updated scale was widely adopted. Body height and weight were self-reported and confirmed with a Tanita scale (Tokyo, Japan).

Measures

Participants completed multiple questionnaires, including the Beck Depression Inventory (BDI; Beck, Steer, & Carbin, 1988), and their resting-state EEG was measured during a 5-min resting period, with eyes closed while remaining alert.

Procedure

Further recruitment and procedural details can be found in (Aviram-Friedman, Kafri, Baz, Alyagon, & Zangen, 2020).

EEG acquisition and processing. EEG was recorded using a 64-electrode Waveguard cap (ANT neuro) and a TMS-compatible EEG amplifier (TMSi), with impedance kept below 5 kΩ. Data were processed offline with the EEGlab 14.0.0 toolbox in Matlab (version R2013a; MathWorks Inc., Natick, MA) (Delorme & Makeig, 2004). First, 1–100 Hz bandpass and 48–52 Hz notch filters were used to remove DC offset, drift, and line/high-frequency noise. Then, the data were segmented into 1 s epochs, which were screened for ocular and muscular artifacts using an automatic amplitude-threshold algorithm, followed by manual inspection. To avoid any mixing of the signals, which might bias phase-dependent measures, the data were kept Cz-referenced, rather than re-referenced to the average.

Functional connectivity analysis. Coherence was calculated using the fieldtrip toolbox (Oostenveld, Fries, Maris, & Schoffelen, 2011). First, data were fast-Fourier transformed (1–45 Hz; 1 Hz resolution) to yield the Fourier coefficients for each channel and epoch. Magnitude-squared coherence was computed across all channel pairs ($n = 2016$), yielding values from 0 to 1. Coherence scores were then averaged into five bands: delta (1–4 Hz), theta (5–7 Hz), alpha (8–13 Hz), beta (14–20 Hz), and gamma (31–45 Hz). A *Fisher-Z* transformation was applied to normalize the coherence scores. One outlier deviated by > 3 SD above the overall mean coherence score and was excluded from the analysis.

Finally, data dimensionality was reduced by averaging the coherence scores into four regions (right-frontal, right-posterior, left-frontal, and left-posterior) and two coherence types (inter- and intra-hemispheric), resulting in eight region-type pairs (right-frontal, left-frontal, right-posterior, left-posterior, inter-hemispheric posterior, inter-hemispheric frontal, left frontal-posterior, and right frontal-posterior; Fig. 1).

Statistical analysis

Analysis was performed using non-parametric permutation tests for mixed effect models, as implemented in R (package 'permuco') (Frossard & Renaud, 2021), with group (three levels), frequency band (five levels), region type (eight levels) and BDI scores as fixed effects, and a random intercept. BDI

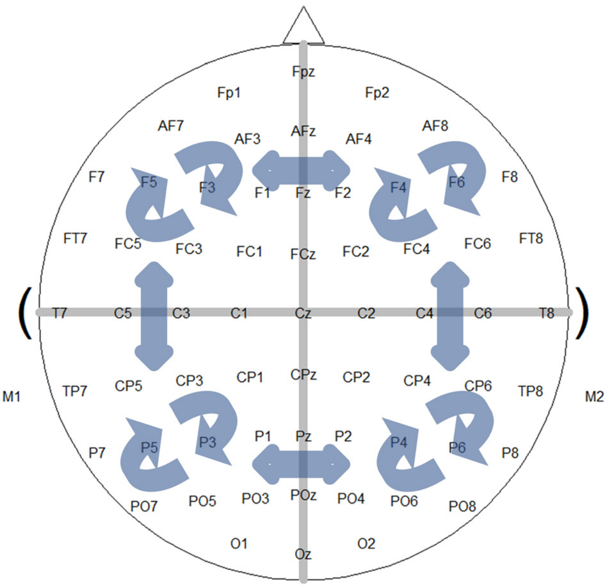

Fig. 1. Analytical approach. Functional connectivity was analyzed across eight region-type pairs

scores were included as a covariate given established associations with both food/eating addiction and EEG coherence (Hardy, Fani, Jovanovic, & Michopoulos, 2018; Thibodeau, Jorgensen, & Kim, 2006). Permutation tests (10,000 iterations) resampled the data to evaluate the null distribution without assuming normality.

We concentrated on regional coherence difference between groups, and Bonferroni correction was used to account for multiple comparisons. Results were further investigated where a Bonferroni corrected test indicated significant differences, using two planned orthogonal contrasts: FAOB vs. Non-FAOB, and Obesity (FAOB and Non-FAOB, combined) vs. healthy controls (HC).

Ethics

All participants signed an informed consent form approved by the Helsinki Committee at the Soroka University Medical Center, Beer-Sheva, Israel.

RESULTS

The three groups (FAOB, Non-FAOB, and HC) did not differ significantly in either age ($F(2,50) = 0.72$, $p = 0.5$) or gender ($X^2(2) = 1.22$, $p = 0.54$), nor did the obesity groups differ in BMI ($t\ (39) = 0.46$, $p = 0.65$). However, the three groups differed in their mean BDI score ($F(2,50) = 7.9$, $p = 0.002$). Post-hoc comparisons revealed that the FAOB group exhibited higher BDI scores than those in the HC group (FAOB vs. HC ($Z = 3.28$, $p = 0.001$). No significant difference in BDI scores was observed between the Non-FAOB group and both the HC group ($Z = 1.65$, $p = 0.099$) and the FAOB group ($Z = 1.79$, $p = 0.074$).

Analysis of frontal coherence in the electrophysiological data revealed a significant interaction between group and region type ($F(14, 329) = 4.48$, $p = 0.0001$), but no significant interaction between group, region type, and frequency bands ($F(56,1316) = 1.13$, $p = 0.25$). Therefore, further analyses were conducted across frequency bands (1–45 Hz).

Significant differences among the three groups were identified in right-frontal (RF) coherence ($F(2,47) = 8.52$, $p = 0.008$; Fig. 2). Bonferroni-corrected post-hoc analyses revealed higher RF coherence in participants with obesity (FAOB and Non-FAOB combined) compared to the HC group ($F(1,49) = 5.94$, $p = 0.04$). Additionally, the Non-FAOB group exhibited higher RF coherence than their FAOB counterparts ($F(1,34) = 9.18$, $p = 0.01$).

The three groups also differed in interhemispheric frontal (IF) coherence ($F(2,47) = 7.83$, $p = 0.01$; Fig. 2). Bonferroni-corrected post-hoc analyses indicated greater IF coherence in participants with obesity when compared to the HC group ($F(1,49) = 6$, $p = 0.04$), and greater IF coherence in the Non-FAOB group when compared to the FAOB group ($F(1,34) = 8.07$, $p = 0.02$).

DISCUSSION

We found that both interhemispheric frontal (IF) and right frontal (RF) coherence are elevated in individuals with obesity compared to healthy controls, and particularly in participants with obesity that do not exhibit FA. These findings align with a prior EEG study of the same sample, which found heightened left-prefrontal alpha asymmetry (right prefrontal dominance) in the Non-FAOB group (Aviram-Friedman et al., 2020). These coherence differences remained significant after controlling for depressive symptoms, suggesting they reflect inhibitory control dysfunction rather than a secondary effect of comorbid depression.

The lateral prefrontal cortex plays a key role in balancing eating-related impulses, with an essential role for efficient interhemispheric communication and intra-hemispheric specialized functions (Lowe et al., 2019). In obesity, these mechanisms may be disrupted by neuroinflammatory processes and insulin resistance (Morys et al., 2025), potentially

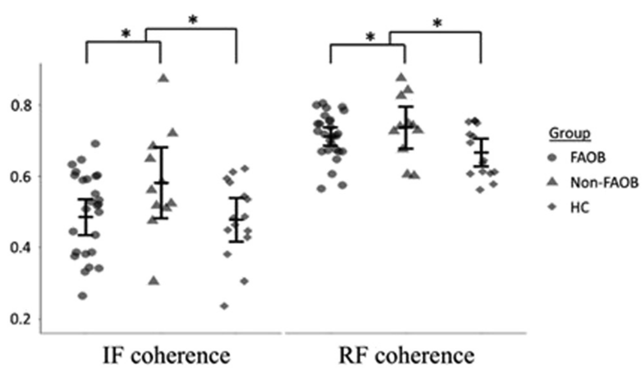

Fig. 2. Differences in interhemispheric frontal (IF) and right-frontal (RF) coherence between the groups. FAOB: Food/eating addiction in obesity. Non-FAOB: Obesity without food/eating addiction. HC: Healthy controls

impairing white and grey matter integrity and disrupting both inter- and intra-hemispheric communication (Kullmann et al., 2016).

Elevated frontal EEG coherence has been proposed as a compensatory mechanism in populations with executive function deficits, such as mild cognitive impairment (Jiang, 2005). In an obesogenic environment, where highly palatable food cues, such as food images or aromas, are ubiquitously available, individuals with obesity may need to exert greater cognitive efforts to inhibit their food cravings, and this may be reflected in elevated IF and RF coherence. Furthermore, the observed elevation in interhemispheric and right frontal coherence among Non-FAOB participants compared to FAOB participants suggests distinct coherence mechanisms within obesity subgroups. These findings suggest that higher frontal coherence may reflect a neurocognitive correlate associated with differential vulnerability to FA, a candidate marker for future longitudinal studies, rather than a confirmed protective mechanism.

These coherence differences are further contextualized by the neurobiological parallels between FA and alcohol use disorder, which are particularly pertinent to interhemispheric coherence. Intermittent excessive sugar intake produces addiction-like neurochemical and behavioral signatures, including dopamine sensitization, bingeing, and withdrawal, paralleling alcohol, itself derived from fermented sugars (Avena, Rada, & Hoebel, 2007). Converging translational evidence further consolidates the neurobiological continuum between sugar-driven eating and substance dependence (Avena, Gold, Kroll, & Gold, 2012; Gearhardt et al., 2011). Specifically, elevated interhemispheric EEG coherence was previously proposed as a biological marker of resilience against developing alcohol use disorders in those at risk for alcohol addiction (Michael, Mirza, Mukundan, & Channabasavanna, 1993). The present findings suggest a comparable neurophysiological signature in those vulnerable to FA. To our knowledge, this is the first study to demonstrate such patterns in the context of FA risk.

The current findings should be interpreted with caution. First, the original YFAS was used to classify FA status (Gearhardt et al., 2009). While this version established the foundational framework for assessing addictive-like eating, its psychometric limitations relative to YFAS 2.0 must be acknowledged: specifically, the original scale lacks dimensional severity grading (mild, moderate, severe) and may have lower diagnostic sensitivity in subthreshold cases (Schulte & Gearhardt, 2017). Second, the sample size was modest, particularly the Non-FAOB subgroup ($n = 11$), limiting statistical power and the generalizability of the findings to broader, more diverse populations. While nonparametric permutation testing mitigates normality assumptions, future studies with larger samples and preregistered power analyses are needed to replicate and extend these findings. Third, dedicated measures of binge eating or broader eating disorder psychopathology were not administered. FA shares core neurobehavioral features with binge eating disorder (BED) and bulimia nervosa (BN), including loss of control over eating, emotional dysregulation, and compulsive intake, reflecting overlapping deficits in inhibitory control and reward processing (Meule, 2024). Thus, the degree to which the frontal coherence patterns observed in this study are specific to FA, rather than shared with binge-spectrum disorders more broadly, cannot be fully determined without additional instruments such as the Binge Eating Scale. Fourth, psychotropic medications, such as antidepressants, may alter resting-state EEG functional connectivity (Zandstra et al., 2023) and should be documented in future studies, together with incorporating broader assessments of overeating disorders, larger and more diverse samples, pre-registered power analyses, and multimodal imaging (e.g., DTI) to examine structural-functional interactions.

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Conflict of interest: All authors declare no conflict of interest, had full access to the study data and complete freedom to direct the analysis and reporting without sponsor influence.

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