Clinical Research

Food-induced brain activity in adult obesity: a quantitative electroencephalographic study

Kemas Abdurrohim[1,2], Pradana Soewondo[3], Fiastuti Witjaksono[4], Hasan Mihardja[2], Wresti Indriatmi[5], Heri Wibowo[6], Selfi Handayani[7], Nurhadi Ibrahim[8,9,10]

pISSN: 0853-1773 • eISSN: 2252-8083
https://doi.org/10.13181/mji.oa.236974
Med J Indones. 2023;32:98–104

Received: May 19, 2023
Accepted: July 21, 2023
Published online: October 03, 2023

Authors' affiliations:
[1]Doctoral Program in Medical Sciences, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia, [2]Department of Medical Acupuncture, Faculty of Medicine, Universitas Indonesia, Cipto Mangunkusumo Hospital, Jakarta, Indonesia, [3]Department of Internal Medicine, Faculty of Medicine, Universitas Indonesia, Cipto Mangunkusumo Hospital, Jakarta, Indonesia, [4]Department of Nutrition, Faculty of Medicine, Universitas Indonesia, Cipto Mangunkusumo Hospital, Jakarta, Indonesia, [5]Department of Dermatology & Venereology, Faculty of Medicine, Universitas Indonesia, Cipto Mangunkusumo Hospital, Jakarta, Indonesia, [6]Department of Parasitology, Faculty of Medicine, Universitas Indonesia, Cipto Mangunkusumo Hospital, Jakarta, Indonesia, [7]Department of Anatomy, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia, [8]Department of Medical Physiology and Biophysics, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia, [9]Neuroscience & Brain Development Cluster, Indonesian Medical Education and Research Institute (IMERI), Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia, [10]Medical Technology Cluster, Indonesian Medical Education and Research Institute (IMERI), Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

Corresponding author:
Nurhadi Ibrahim
Department of Medical Physiology and Biophysics, Faculty of Medicine, Universitas Indonesia, Jalan Salemba Raya No. 6, Kenari, Senen, Central Jakarta 10430, DKI Jakarta, Indonesia
Tel/Fax: +62-21-31930373/ +62-21-3912477
E-mail: ibmnurhadi@gmail.com

ABSTRACT

BACKGROUND Obesity may be associated with declined food consumption control through neurological and behavioral processes, as well as heightened responsiveness of the brain's reward systems. Performing neuroimaging and neurophysiological methods such as electroencephalography (EEG) can examine the connection between brain function and behavior. This study aimed to identify brain regulation of feeding behavior to food cues, which could be a potential neuromodulatory intervention target in adult obesity.

METHODS This cross-sectional study was conducted at Cipto Mangunkusumo Hospital, Jakarta, involving 40 adults with obesity. EEG analysis was performed to measure electrophysiological brain activity during eyes-open condition and during exposure to high-calorie food cues. Student's *t*-tests were performed to identify any significant differences between the groups ($p<0.05$).

RESULTS Beta waves in the frontal (channel F7) and gamma waves in the central (channels C3 and C4) and parietal (channels P3 and P4) regions were significantly increased during food cues compared to resting state/eyes-open condition without stimulation. Theta waves in the frontal (channels F7 and F8), central (channel C3), and parietal (channels P3 and P4) regions and alpha waves in the central (channels C3 and C4) and parietal (channels P3 and P4) regions were significantly decreased during food cues compared with resting state.

CONCLUSIONS In adults with obesity, increased beta activity in the frontal and gamma in the central and parietal regions suggested increased food-cue awareness and heightened attentional focus toward food stimuli. Additionally, decreased alpha and theta activities in frontal regions could underline deficits in executive functions and higher motivation.

KEYWORDS brain wave, food, electroencephalography, obesity

Obesity is a major public health challenge worldwide.[1] An increasing trend in obesity prevalence has been observed in Indonesia since 2007 and 2018, growing from 10.3–21.8%.[2] A recent study suggested that obesity might be associated with alterations in how the body and mind respond to food cues, particularly with increased reactivity to high-calorie foods. This reactivity can manifest as elevated neural activity, salivation, or physiological arousal and is known as high food-cue reactivity. According to the meta-analysis findings, significant medium-to-large effects were observed for food-related stimuli responsiveness, which was linked to overeating, weight gain, and becoming

overweight or obese in both children and adults, in cross-sectional and longitudinal studies.[3]

Studies have revealed a significant increase in attentional processing triggered by environmental food cues, which have been linked to weight gain and obesity. This neurophysiological response can be measured using electroencephalography (EEG) event-related potentials.[4,5] Neuroimaging research has identified various important brain regions and networks that respond differently to visual food cues depending on the context, including fasting, weight loss, overfeeding, exercise, hormones, and cognitive control. These areas include those involved in visual processing and networks related to motivation and food cravings, particularly high-energy and palatable foods, located in the frontal cortex, striatum, and amygdala. This network, referred to as the "salience network," shows greater activation in obese individuals than in lean individuals.[1] Neuroimaging studies have shown that specific brain parts are associated with food-cue reactivity. Greater orbitofrontal cortex activity was observed in children with obesity than in those of normal weight when passively viewing high-calorie food images, which is related to the reward and motivation systems.[6,7] However, evidence regarding the relationship between the dorsolateral prefrontal cortex (PFC) and cognitive control is mixed.[4,7] This study aimed to identify the brain's regulation of feeding behavior in response to food cues, which could be a potential neuromodulatory intervention target in adults with obesity.

METHODS

This was a cross-sectional study with no control group that examined the effects of brain activity on adult obesity using EEG. The study was conducted at Cipto Mangunkusumo Hospital from October 2022 to May 2023 on participants who fulfilled the study criteria. The study protocol was approved by the Ethics Committee of the Faculty of Medicine, Universitas Indonesia, Cipto Mangunkusumo Hospital, Jakarta (No: KET-1236/UN2.F1/ETIK/PPM.00.02/2022).

Sample size

This was a large pre-intervention study employing a sample size formula designed for unpaired analytical research. Based on the research by Yeo et al,[8] the analysis of weight loss in patients with obesity had a mean difference of –2.05 (experimental mean –4.25 and control mean –2.2), standard deviation (SD) 2.03 in the experimental group, the SD was 1.9 in the control group ($p<0.05$), and the ratio of sample sizes between groups was 0.20. Calculations were performed using the MedCalc® statistical software version 20.111 (MedCalc Software Ltd., Belgium), and a minimum sample size obtained was 40, with an anticipation of 20% dropout.

Participants selection

Participants were recruited through advertisements on social media and banners placed at Cipto Mangunkusumo Hospital. Telephone screening was performed to evaluate the inclusion and exclusion criteria before scheduling the direct interviews and examinations. The inclusion criteria were females or males aged 19–35 years, body mass index (BMI) of ≥25.0 kg/m^2, waist circumference of ≥80 cm (woman) or ≥90 cm (man), agreed on signing informed consent. Participants were measured using calibrated scales and defined as obese according to the Asia-Pacific guidelines. Exclusion criteria for both groups were a history of epilepsy, diabetes mellitus, thyroid disease, medication intake affecting body weight, history of hormonal contraception, and pregnancy. Clinical data were obtained from direct observation and medical records. Figure 1 shows a flow diagram of the participant recruitment process and the number of eligible participants.

Procedure

Eligible participants (n = 40) were selected based on their medical history and physical examination screening. The screening and anthropometric examinations were conducted by a trained medical doctor using standardized procedures. Body weight was measured to the nearest 0.1 kg (in light clothes and without shoes) using a body composition analyzer (TANITA, BC-541, Tanita Corporation, Japan). The participants were instructed to stand at the center of the scale and look straight ahead with both arms in the resting position.[9] Height was measured to the nearest 0.1 cm without shoes on a stadiometer (Charder version ADE M320600-01, ADE Germany GmbH, Germany). Participants were instructed to stand barefoot on the stadiometer, and body height was measured as the distance between the soles of the feet and crown of the head. The back of the head, shoulder blades, buttocks,

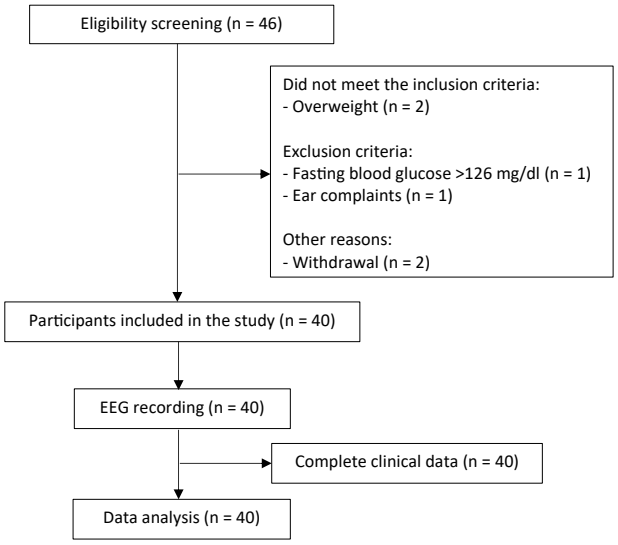

Figure 1. Flow diagram of the eligible participants. EEG= electroencephalography

Table 1. PSD during eyes-open condition *versus* food provocation

Scalp sites	Delta	Theta	Alpha	Beta	Gamma
FP1	0.4296	0.4326	0.7275	0.2261	0.8963
FP2	0.9744	0.8149	0.6874	0.6828	0.5987
F7	0.1303	**0.0327***	0.7535	**0.0407***	0.0799
F8	0.2377	**0.0229***	0.8483	0.1846	0.1685
F3	0.7709	0.2577	0.8409	0.4916	0.6872
F4	0.4068	0.6224	0.9839	0.8630	0.5503
C3	0.3897	**0.0464***	**0.0201***	0.6166	**0.0019***
C4	0.1023	0.0529	**0.0392***	0.4589	**0.0237***
P3	0.4034	**0.0252***	**0.0170***	0.9612	**0.0002***
P4	0.2332	**0.0081***	**0.0012***	0.8561	**0.0001***

C3=left central; C4=right central; FP1=left prefrontal; FP2=right prefrontal; F3=left frontal; F4=right frontal; F7=left frontal; F8=right frontal; PSD=power spectrum density; P3=left parietal; P4=right parietal
*Student's *t*-test, significant if $p<0.05$

and heels should be aligned with the stadiometer.[9] BMI was calculated by dividing the weight (kg) by the square of the height (m^2). Based on the Asia-Pacific guidelines, participants were classified as obese I (BMI of ≥25 kg/m^2) and II (BMI of ≥30 kg/m^2).[10] Waist circumference (cm) was measured at the midpoint between the lower margin of the least palpable rib and the top of the iliac crest at the umbilicus. The measurement was recorded at the end of a normal expiration.[11] All participants gave written informed consent before participating in the study, followed by EEG recordings.

EEG recordings

Participants fasted for 4–6 hours prior to EEG recordings. A 32-channel Easy III amplifier (Cadwell, USA) was used, and EEG signals were recorded over 19 scalp sites: prefrontal (channels FP1 and FP2); frontal (channels F3, F4, F7, F8, and Fz); central (channels C3, C4, and Cz); medial temporal (channels T3 and T4); posterior temporal (channels T5 and T6); parietal (channels P3, P4, and Pz); and occipital (channels O1 and O2) regions.[12] The gold cup electrodes were positioned according to the standard 10–20 international placement. The patient laid back in a semi-Fowler position in a silent room and was positioned in a quiet room for approximately 20 min. The recordings comprised specific segments, including 3 min of eyes-closed and 3 min of eyes-open condition without food cues. Following this, there was a 3-min period with high-calorie food cues, followed by another 3 min of eyes-closed and 3 min of eyes-open condition without food cues. Participants were presented with prepared instant noodles, a high-calorie food popular among Indonesians. The provocation of high-calorie food was provided 30 cm away from the participants for 3 min of eye-open condition. Artifact rejection (eye movements, blinks, muscular activations, or movement artifacts) was visually performed on the raw EEG, and the recordings were attended by trained technicians.

Pre-processing and analysis of EEG recordings

EEG was compared between participants during eyes-open condition *versus* provocation with food for each frequency band. A high-pass filter of 1 Hz, band-stop filter of 49–51 Hz, and low-pass filter of 100 Hz were used to filter the EEG signals. Following this, the EEG was filtered using a fourth-order Butterworth filter. Independent component analysis and visual rejection were applied to filter out artifacts and remaining impurities.

The extracted filtered EEG data included delta (1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta (13–30 Hz), and gamma (30.5–60 Hz). The EEG signals analyzed were 10 channels, and the extracted absolute power for each frequency band was grouped for frontal (FP1, FP2, F3, F4, F7, and F8); central (C3 and C4); and parietal (P3 and P4) and converted to a signal spectrum (microVolt^2/Hz).

Data analysis

Power spectral density (PSD) was processed using the P-Welch method, and PSD normalization

was performed for comparison. The plot spectrum and topographic plot are visually presented. Shapiro–Wilk tests were conducted to examine data normality, and Levene's test was performed to examine the homogeneity of variance. Student's *t*-tests were performed to assess significant differences between groups and are presented as bar plots. The level of significance was set at a *p*-value of <0.05. All EEG analysis and statistics were performed by the Matlab® R2021a software (Mathworks Inc., Germany).

RESULTS

EEG recordings suitable for analysis were obtained from all patients. The total mean PSD and topographic PSD plots are shown in Figure 2, consisting of 10 subfigures representing the 10 selected channels. The x-axis of the figure shows the frequency (Hz), and the y-axis of the figure shows the normalized PSD (microVolt^2/Hz). The topographic plot was divided into five frequency ranges (delta, theta, alpha, beta, and gamma). The color bar shows the normalized PSD values based on the mean frequency ranges and the mean of all participants.

Bar plots PSD comparison between eyes-open and provocation of all participants are shown in Figure 3. Beta activity was significantly increased in the left frontal (F7) during the food-cue condition compared with the eyes-open condition. Additionally, gamma activity showed an overall increase, with significant increases in the left and right central (C3 and C4 [p<0.05]) and the left and right parietal (P3 and P4 [p<0.001]).

In contrast, theta activity decreased across all channels. However, the decrease was significant in specific areas, namely, the left and right frontal (F7 and F8), left central (C3), and left and right parietal (P3 [p<0.05] and P4 [p<0.01]). Finally, alpha activity exhibited a significant decrease in the left and right central (C3 and C4) and left and right parietal (P3 [p<0.05] and P4 [p<0.01]) (Tables 1 and 2).

DISCUSSION

Food consumption is influenced by both physiological hunger and the rewarding properties of food, indicating that human eating behavior results from a combination of needs (homeostasis) and desires (reward). The brain plays a crucial role in mediating the incentive value of food, attention allocation to food cues, and the motivation to obtain food rewards. It involves complex interactions within key brain regions, including the amygdala/hippocampus, insular cortex, orbitofrontal cortex, and striatum.[4,5,12] Pleasurable sensations and rewards are achieved by activating various neural circuits such as the dopaminergic, gamma-aminobutyric acid, opioid, and serotonergic pathways. These neural pathways, originating from the ventral tegmental area and substantia nigra pars compacta, regulate food consumption, and are implicated in addiction-related behaviors. The mesocortical and mesolimbic tracts of these circuits are particularly active during reward-seeking behaviors, including those related to pleasurable food intake. Interestingly, individuals with obesity and drug addicts show similar abnormalities in these neural pathways, such as increased anticipation of pleasure in response to rewards; however, a blunted pleasure response upon reward attainment.[5] Brain wave alterations in specific areas related to food regulation were identified in adult obesity when stimulated with food, suggesting an enhanced recognition of food cues, intensified concentration of food-related stimuli, and underlying impairments in executive functions.

This study showed greater beta activity in the left frontal lobe (F7) (p = 0.0407) during food cues than during the eyes-open condition. Consistent with the present study, Kösling et al[7] showed that overweight or obese children had increased beta activity when provoked by images of high-calorie food. The food stimulus was more effective than the landscape picture as it triggered higher levels of beta activity in the brain during the experiment. The human brain appears to be particularly responsive to food-related stimuli; studies have shown that displaying such stimuli can significantly increase brain metabolism in fasting, normal-weight individuals.[13] This result aligns with some studies showing a significant elevation in frontal beta activity, suggesting increased awareness of food cues and heightened attentional focus toward food stimuli.[13,14] Unlike the present study, Tammela et al[13] did not find differences in beta activity between the left and right frontal regions in their study. Greater increases in gamma activity were identified in the left and right central (C3 [p = 0.0019] and C4 [p = 0.0237]) and the left and right parietal (P3 [p = 0.0002] and P4 [p = 0.0001]) regions. Elevated gamma activity

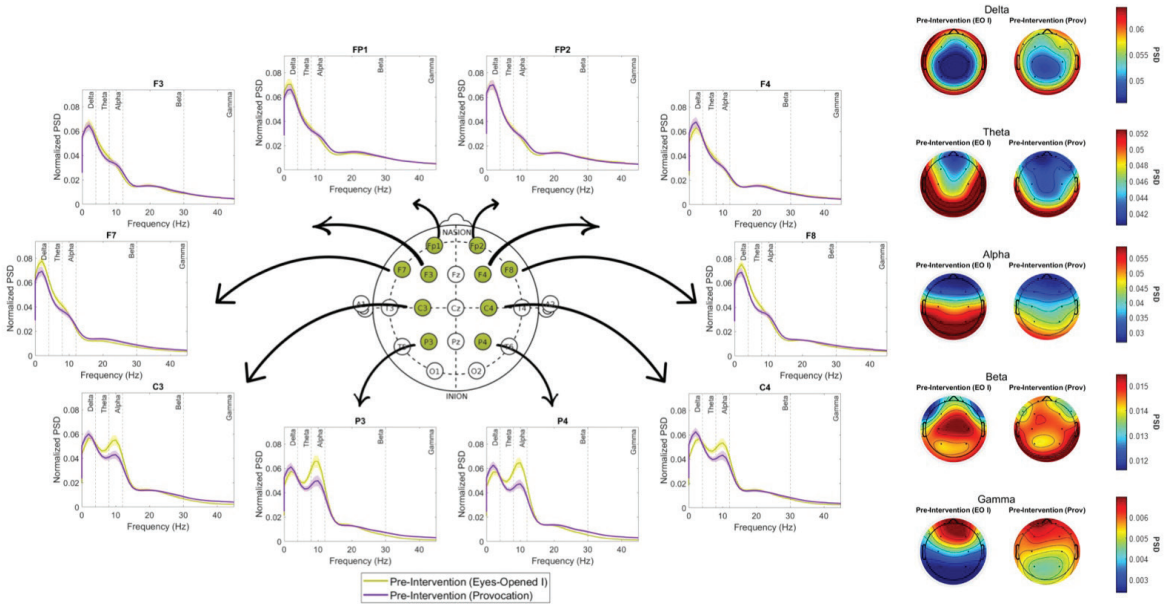

Figure 2. Total mean (grand mean) PSD along with the SEM of all participants (n = 40) during eyes-open condition and provocation (left). Yellow PSD and SEM represented eyes-open condition, while purple represented provocation. Topographic PSD plot of pre-intervention participants (n = 40) during eyes-open condition and provocation (right). Dark blue indicated low, and dark red indicated high. C3=left central; C4=right central; EO=eyes-open; FP1=left prefrontal; FP2=right prefrontal; F3=left frontal; F4=right frontal; F7=left frontal; F8=right frontal; O1, O2=occipital region; PSD=power spectrum density; P3=left parietal; P4=right parietal; SEM=standard error of the mean; T3, T4=medial temporal; T5, T6=posterior temporal

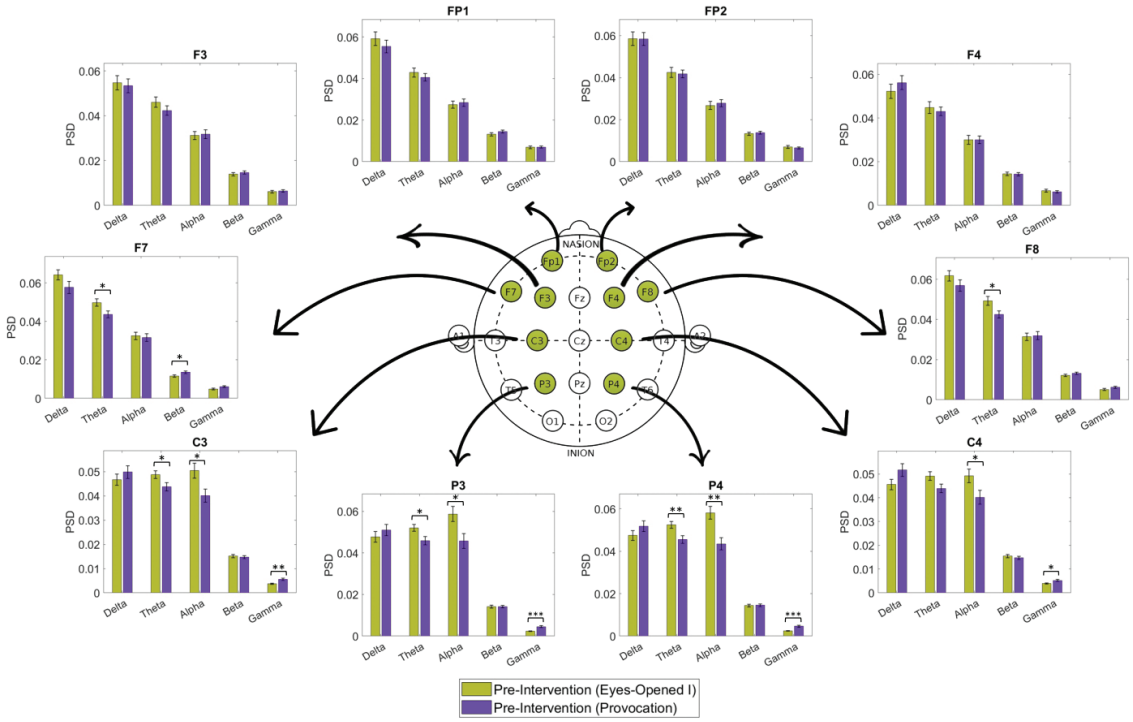

Figure 3. Bar plot PSD comparison between eyes-open and provocation of all participants. It consisted of 10 subfigures, representing the 10 selected channels/electrodes. The x-axis showed frequency ranges respectively (delta, theta, alpha, beta, and gamma), while the y-axis showed the normalized PSD (unit: microVolt^2/Hz) for each frequency range. The yellow bar showed the normalized grand mean PSD during eyes-open, the purple bar showed the normalized grand mean PSD during provocation. The line at the top of each bar represented the normalized mean (SEM) of PSD for each frequency range. *Significant if $p \leq 0.05$. C3=left central; C4=right central; EO=eyes-open; FP1=left prefrontal; FP2=right prefrontal; F3=left frontal; F4=right frontal; F7=left frontal; F8=right frontal; O1, O2=occipital region; PSD=power spectrum density; P3=left parietal; P4=right parietal; SEM=standard error of the mean; T3, T4=medial temporal; T5, T6=posterior temporal

Table 2. PSD during eyes-open condition *versus* food provocation

Scalp sites	Mean (SEM)									
	Delta		Theta		Alpha		Beta		Gamma	
	EO	Provocation	EO	Provocation	EO	Provocation	EO	Provocation	EO	Provocation
FP1	0.0592 (0.0033)	0.0554 (0.0031)	0.0429 (0.0022)	0.0405 (0.0018)	0.0274 (0.0017)	0.0284 (0.0018)	0.0131 (0.0007)	0.0145 (0.0007)	0.0069 (0.0007)	0.0070 (0.0005)
FP2	0.0585 (0.0032)	0.0584 (0.0031)	0.0425 (0.0024)	0.0417 (0.0018)	0.0267 (0.0019)	0.0278 (0.0017)	0.0133 (0.0007)	0.0137 (0.0007)	0.0070 (0.0007)	0.0065 (0.0005)
F7	0.0642 (0.0026)	0.0576 (0.0031)	0.0498 (0.0020)	0.0436 (0.0018)	0.0324 (0.0019)	0.0315 (0.0020)	0.0116 (0.0006)	0.0135 (0.0007)	0.0048 (0.0005)	0.0060 (0.0005)
F8	0.0617 (0.0026)	0.0569 (0.0028)	0.0493 (0.0022)	0.0425 (0.0018)	0.0314 (0.0018)	0.0319 (0.0020)	0.0121 (0.0006)	0.0133 (0.0006)	0.0051 (0.0005)	0.0062 (0.0005)
F3	0.0548 (0.0032)	0.0534 (0.0031)	0.0460 (0.0023)	0.0423 (0.0021)	0.0312 (0.0018)	0.0317 (0.0020)	0.0139 (0.0007)	0.0146 (0.0008)	0.0061 (0.0006)	0.0064 (0.0005)
F4	0.0522 (0.0032)	0.0562 (0.0032)	0.0448 (0.0027)	0.0431 (0.0020)	0.0300 (0.0020)	0.0301 (0.0017)	0.0144 (0.0008)	0.0142 (0.0008)	0.0067 (0.0007)	0.0062 (0.0005)
C3	0.0467 (0.0023)	0.0498 (0.0026)	0.0488 (0.0016)	0.0438 (0.0017)	0.0504 (0.0031)	0.0401 (0.0027)	0.0152 (0.0007)	0.0147 (0.0006)	0.0037 (0.0003)	0.0056 (0.0005)
C4	0.0456 (0.0022)	0.0517 (0.0027)	0.0491 (0.0018)	0.0439 (0.0018)	0.0492 (0.0029)	0.0402 (0.0029)	0.0155 (0.0007)	0.0147 (0.0007)	0.0039 (0.0003)	0.0052 (0.0005)
P3	0.0477 (0.0026)	0.0510 (0.0027)	0.0521 (0.0016)	0.0459 (0.0020)	0.0588 (0.0037)	0.0458 (0.0035)	0.0142 (0.0007)	0.0141 (0.0006)	0.0024 (0.0002)	0.0045 (0.0005)
P4	0.0474 (0.0023)	0.0518 (0.0025)	0.0524 (0.0016)	0.0455 (0.0018)	0.0581 (0.0030)	0.0435 (0.0029)	0.0144 (0.0007)	0.0145 (0.0006)	0.0025 (0.0002)	0.0047 (0.0005)

C3=left central; C4=right central; EO=eyes-open; FP1=left prefrontal; FP2=right prefrontal; F3=left frontal; F4=right frontal; F7=left frontal; F8 = right frontal; PSD=power spectrum density; P3=left parietal; P4=right parietal; SEM=standard error of the mean
Normalized PSD (per participant, per channel) was PSD per frequency point (per participant, per channel) divided by sum of the data (per participant, per channel)

suggests that a higher conflict situation needs to be resolved during food stimuli. However, studies on gamma radiation activity are limited.

Theta activity was significantly decreased in the left and right frontal (F7 [p = 0.0327] and F8 [p = 0.0229]), left central (C3) (p = 0.0464), left parietal (P3) (p = 0.0252), and right parietal (P4) (p = 0.0081) regions during food cues compared to when eyes were open. Hume et al[5] found that adults with obesity displayed lower theta activity than that in those without obesity. However, no significant effects were observed on theta activity during food cues. In contrast, Imperatori et al[15] observed increased frontal theta activity in adults who were overweight or obese and had symptoms of food addiction compared to weight-matched controls without food addiction after consuming a single milkshake. Theta activity may indicate cognitive control and is potentially relevant for food cue reactivity in obesity.[16]

The alpha activity was significantly decreased in the left and right central (C3 [p = 0.0201] and C4 [p = 0.0392]) and the left and right parietal (P3 [p<0.05] and P4 [p<0.01]) regions. This result contrasts with that of Kösling et al[7] who found no significant differences in alpha and theta activities between overweight/obese and normal-weight children. The specific areas mentioned above are similar to those in a functional magnetic resonance imaging study by Davids et al,[4] which showed increased activity in the dorsal PFC of individuals during food-related stimuli. This increased PFC activity is associated with various cognitive processes related to top-down control, particularly executive function, goal selection, planning, information manipulation, and response inhibition. As the level of emotional or cognitive conflict increases, the PFC activation also increases. In particular, food cues may induce a higher level of conflict as they exclusively result in heightened PFC activation. Intensified dorsal PFC activity may suppress appetitive reactions or approach behaviors triggered by food cues, leading to avoidance. Greater activation of the PFC indicates a greater need for inhibitory

control, suggesting that when faced with food-related cues, stronger regulation by the PFC is necessary to generate appropriate behavior. These specific areas of the PFC are often referred to as the "satiation domain" since they contribute to the termination of the feeding period by suppressing subcortical areas associated with hunger, such as the limbic/paralimbic areas, basal ganglia, thalamus, and hypothalamus. In individuals with obesity, the network responsible for promoting hunger (known as the orexigenic network) is consistently hyperactive, requiring increased effort from the PFC areas to suppress these hunger centers. Reduced neural activation in the putamen and amygdala results in difficulties activating the reward system in response to food cues. Furthermore, reduced hippocampal activation contributes to a decreased ability to regulate feeding behavior.

The results of this study can potentially serve as neuromodulatory intervention targets for adult obesity. However, this study was limited by the small number of EEG channel analyses (10); a greater number of electrodes would have allowed more detailed topographical analyses to assess other brain activity changes. Additional longitudinal studies are required to determine the association between feeding behavior and EEG changes.

In conclusion, increased beta activity in the frontal and gamma in the central and parietal regions suggested increased awareness of food cues and heightened attentional focus toward food stimuli. Additionally, decreased alpha and theta activity in the frontal regions may underlie deficits in executive function and higher motivation.

Conflict of Interest

Pradana Soewondo is the editorial board member but was not involved in the review or decision making process of the article.

Acknowledgment

This work was supported by Universitas Indonesia and Cipto Mangunkusumo Hospital, Jakarta.

Funding sources

None.

REFERENCES

1. Gadde KM, Martin CK, Berthoud HR, Heymsfield SB. Obesity: pathophysiology and management. J Am Coll Cardiol. 2018;71(1):69–84.
2. Health Research and Development Agency (*Balitbangkes*) Ministry of Health Republic of Indonesia. Basic health research (*RISKESDAS* 2018) [Internet]. Health Research and Development Agency (*Balitbangkes*) Ministry of Health Republic of Indonesia; 2018 [cited 2021 Aug 3]. Available from: http://www.depkes.go.id/resources/down load/info- terkini/materi_rakorpop_2018/Hasil%20 Riskesdas%202018.pdf.
3. Boswell RG, Kober H. Food cue reactivity and craving predict eating and weight gain: a meta-analytic review. Obes Rev. 2016;17(2):159–77.
4. Davids S, Lauffer H, Thoms K, Jagdhuhn M, Hirschfeld H, Domin M, et al. Increased dorsolateral prefrontal cortex activation in obese children during observation of food stimuli. Int J Obes (Lond). 201;34(1):94–104.
5. Hume DJ, Howells FM, Rauch HG, Kroff J, Lambert EV. Electrophysiological indices of visual food cue-reactivity. Differences in obese, overweight and normal weight women. Appetite. 2015;85:126–37.
6. World Health Organization (WHO). Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8–11 December 2008. World Health Organization (WHO); 2011. p. 3.
7. Kösling C, Schäfer L, Hübner C, Sebert C, Hilbert A, Schmidt R. Food-induced brain activity in children with overweight or obesity versus normal weight: an electroencephalographic pilot study. Brain Sci. 2022;12(12):1653.
8. Samara A, Li X, Pivik RT, Badger TM, Ou X. Brain activation to high-calorie food images in healthy normal weight and obese children: a fMRI study. BMC Obes. 2018;5:31.
9. van Meer F, van der Laan LN, Charbonnier L, Viergever MA, Adan RA, Smeets PAet al,. Developmental differences in the brain response to unhealthy food cues: an fMRI study of children and adults. Am J Clin Nutr. 2016;104(6):1515–22.
10. Yeo S, Kim KS, Lim S. Randomised clinical trial of five ear acupuncture points for the treatment of overweight people. Acupunct Med. 2014;32(2):132–8.
11. Centers for Disease Control and Prevention (CDC). National Health and Nutrition Examination Survey (NHANES): anthropometry procedures manual. Centers for Disease Control and Prevention (CDC); 2007.
12. World Health Organization (WHO) Regional Office for the Western Pacific. The Asia-Pacific perspective: redefining obesity and its treatment. Sydney: Health Communications Australia; 2000.
13. Tammela LI, Pääkkönen A, Karhunen LJ, Karhu J, Uusitupa MI, Kuikka JT. Brain electrical activity during food presentation in obese binge-eating women. Clin Physiol Funct Imaging. 2010;30(2):135–40.
14. Hume DJ, Howells FM, Karpul D, Rauch HG, Kroff J, Lambert EV. Cognitive control over visual food cue saliency is greater in reduced-overweight/obese but not in weight relapsed women: An EEG study. Eat Behav. 2015;19:76–80.
15. Imperatori C, Fabbricatore M, Innamorati M, Farina B, Quintiliani MI, Lamis DA, et al. Modification of EEG functional connectivity and EEG power spectra in overweight and obese patients with food addiction: an eLORETA study. Brain Imaging Behav. 2015;9(4):703–16.
16. Blume M, Schmidt R, Hilbert A. Abnormalities in the EEG power spectrum in bulimia nervosa, binge-eating disorder, and obesity: a systematic review. Eur Eat Disord Rev. 2019;27(2):124–36.