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REVIEW

Cognitive function, eating behavior and neuroimaging studies in obese: a systematic review

Função cognitiva, comportamento alimentar e neuroimagem em obesos: uma revisão sistemática

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Abstract

Background: Obesity is a multifactorial disorder influenced by hormonal, dietary, behavioral, emotional, attentional, and cognitive control factors that interfere with the balance between energy intake and expenditure. The association of obesity with cognitive decline, brain functional and structural damage, and early neurodegenerative processes has been observed. *Objective*: This systematic review aimed to identify activated cortical areas in obese individuals and investigate the role of cognitive

impairment in interfering with eating behavior. The most frequently used neuroimaging and brain mapping methods to evaluate these processes were also identified. *Methods*: We searched for studies published between 2006 and 2021 in the indexed databases PUBMED, LILACS, and SCIELO. Observational studies that compared obese individuals (body mass index > 30 kg/m²) and nonobese individuals were selected. The National Heart, Lung, and Blood Institute (NIH) Quality Assessment of Observational Cohort and Cross-sectional Studies was used for methodological quality analysis. *Results*: The literature search returned 22,484 relevant titles. After applying the eligibility criteria, 154 articles were selected, and of these, 11 were analyzed in this review. In the analysis, the groups studied showed differences in reaction time, accuracy, or inactivated brain areas during tests or stimulation with food images. *Conclusion*: Structural changes compatible with impairments in long-term cognitive performance were identified, as well as structural and functional changes that may help understanding the compulsive eating behavior present in obese individuals.

Keywords: obesity; electroencephalogram; cognition; functional magnetic resonance imaging.

Resumo

Introdução: A obesidade é considerada uma desordem multifatorial influenciada por fatores hormonais, dietéticos, comportamentais, emocionais, atencionais e controle cognitivo que interferem no equilíbrio ingestão e gasto energético. A influência da obesidade no declínio cognitivo e prejuízos a funções e estruturas cerebrais além de sua associação com processos neurodegenerativos precoces tem sido observada. *Objetivo*: Esta revisão buscou identificar as áreas corticais mais ativadas em indivíduos obesos, investigar a existência de comprometimento cognitivo e a possível interferência no comportamento alimentar. Além disso, buscou-se identificar os métodos de neuroimagem mais utilizados para avaliação desses processos. *Methods*: Buscou-se estudos publicados 2006 e 2021. Foram pesquisadas as bases de dados indexadas PUBMED, LILACS e SCIELO. Foram selecionados estudos observacionais que comparassem indivíduos obesos (IMC > 30 kg/m²) e não obesos. Foi utilizado o *Quality Assessment of Observational Cohort and Cross-Sectional Studies da National Heart, Lung and Blood Institute* (NIH) para análise de qualidade metodológica. *Resultados*: Foram reportados 22.484 títulos. Após a aplicação dos critérios de elegibilidade, foram selecionados 154 artigos. Desses, onze foram incluídos para análise nesta revisão. Nesta análise, diferenças foram encontradas quanto ao tempo de reação, acurácia ou áreas cerebrais inativadas durante os testes cognitivos ou estímulos com figuras de comida entre os grupos estudados. *Conclusão*: Mudanças estruturais compatíveis com

prejuízos na performance cognitiva a longo prazo foram identificadas, assim como alterações estruturais e funcionais que podem auxiliar o entendimento de comportamento alimentar compulsivo presente em indivíduos obesos.

Palavras-chave: obesidade; eletroencefalograma; cognição; ressonância magnética funcional

Introduction

Obesity has become the subject of numerous studies globally due to its high prevalence in the world population [1], making this disease an epidemic. Data from the World Health Organization (WHO) point to obesity as one of the world's biggest public health problems. In 2016, 1.9 billion adults were overweight and 650 million were obese [2]. According to the WHO, obesity is defined as the excessive accumulation of body fat and is measured by the body mass index (BMI). BMI is obtained by dividing an individual's mass by the square of height and is expressed in kg/m^2 [3]. Using the BMI, obesity can be classified as class I (BMI 30–34.9 kg/m^2), class II (BMI 35–39.9 kg/m^2), and class III (BMI ≥ 40 kg/m^2) [4]. A multifactorial disorder, obesity is influenced by hormonal, dietary, behavioral, emotional, attentional, and cognitive control factors that interfere with the balance of energy intake and expenditure [5]. It is associated with several comorbidities, such as type 2 diabetes mellitus, cardiovascular diseases, metabolic syndrome, and some forms of cancer, and increases the risk of premature mortality [6-8].

In recent years, studies [9,10] on obesity have sought to elucidate the interactions between homeostatic control and the neural networks involved in controlling food intake [11], such as behavior, cognitive factors, social habits, and hedonic appetite control [12,13]. Neuroimaging and brain mapping methods have been widely used to assess the neurofunctional changes in obese individuals, such as the cortical processes involved in dietary control and weight gain [1,11,14]. Several authors have reported limbic and prefrontal neural network dysfunction in obesity, suggesting that eating behavior can be influenced by the relationship between reward circuits and cognition [15].

Obesity increases the risk of cognitive decline, impairment of brain function, and structural damage [16], independent of its association with early neurodegenerative processes [17]. Neuroimaging has revealed that structural changes related to high BMI are primarily reductions in gray matter volume in the temporal, frontal, and occipital lobes [18]. Recent evidence has demonstrated5 greater reductions in volume in cognitive brain regions, such as the hippocampus, prefrontal cortex, and anterior cingulate cortex, in obese individuals. Therefore, this systematic review aimed to evaluate the

methodological quality of studies that investigated the changes in cortical activity and cognitive function in obese individuals and the possible interference of these changes with eating behavior.

Methods

To carry out this systematic review, we followed the guidelines and search principles of the PRISMA recommendation [19].

Search strategy

The literature search was performed from May 2019 to October 2022. Studies published between 2006 and 2021 were searched in the indexed databases PUBMED, LILACS, and SCIELO. The keywords used in the search process were “obesity” in association with the terms “brain mapping,” “neuroimaging,” “electroencephalography,” “magnetoencephalography,” “functional magnetic resonance imaging,” “positron emission tomography,” “single-photon emission computed tomography,” “pharmacogenetic functional magnetic resonance imaging fMRI and functional near-infrared spectroscopy,” and “diffusion tensor imaging”; as well as their association with the term “obese,” in all search fields (table I).

Table I - *Search strategies for indexed bases*

SEARCH STRINGS	INDEXED DATABASES			TOTAL
	Pubmed	Scielo	Lilacs	
"Obesity" AND "brainmapping"	701	0	0	701
"Obese"AND "brainmapping"	340	0	0	340
"Obesity" AND "electroencephalography"	1420	0	0	1420
"Obese" AND "electroencephalography"	678	0	0	678
"Obesity" AND "neuroimaging"	5876	9	4	5889
"Obese" AND "neuroimaging"	2591	3	2	2596
"Obesity" AND "magnetoencephalography"	169	0	0	169
"Obese" AND "magnetoencephalography"	118	0	0	118
"Obesity" AND "functional magnetic resonance imaging"	1912	1	11	1924
"Obese" AND "functional magnetic resonance imaging"	1111	2	8	1121
"Obesity" AND "positron emission tomography"	2164	1	2	2167
"Obese" AND "positron emission tomography"	2542	1	2	2545
"Obesity" AND "single photon emission computed tomography"	819	0	3	822
"Obese" AND "single photon emission computed tomography"	473	0	0	473
"Obesity" AND "diffusion tensor imaging"	1038	0	0	1038
"Obese" AND "diffusion tensor imaging"	481	0	0	481
"Obesity" AND "pharmacogenetic fRMI and functional near-infrared spectroscopy"	1	0	0	1
"Obese" AND "pharmacogenetic fRMI and functional near-infrared spectroscopy	1	0	0	1
TOTAL	**22435**	**17**	**32**	**22484**

Eligibility criteria

The eligibility criteria used to include the studies in this review comprised cross-sectional studies in adults (aged 18 to 60 years), which compared obese (BMI > 30 kg/m²) and nonobese individuals. Data collected in obese individuals were independently considered from data in overweight individuals without associated eating disorders. Studies published in Portuguese, English, Spanish, Italian, and French were included. Manuscripts covering benign and malignant neoplasms, sleep disorders, drug intervention, genetic analyzes exclusively, neuropsychiatric disorders, intervention studies with weight loss, and Prader–Willi syndrome were excluded. Studies with only the abstracts accessible were excluded.

Selection of studies

The initial search returned a total of 22,484 published studies. Of these, 639 reports were identified as potentially relevant based on their abstracts, with 173 duplicate reports excluded. In the next stage, the 466 abstracts selected were analyzed by applying the eligibility criteria. Of these, 310 articles did not meet the inclusion criteria for the following reasons: use of animal models (n = 12), children and adolescents participants (n = 17), participants with BMI less than 30 kg/m^2 (n = 72), elderly participants over 60 years old (n = 6), obesity associated with other pathologies such as neuropsychiatric illnesses and binge eating (n = 96), and studies that did not compare groups, evaluated interventions for weight loss as an outcome, or did not present neuroimaging or cognitive function assessments (n = 107). Finally, 154 studies with full texts were analyzed. Of these, 11 studies were selected to assess methodological quality and included in this review. Two reviewers assessed all internships independently.

Data extraction and quality assessment

The authors' names, year of publication, country, sample size, the cognitive tests used, the neuroimaging methods, and the associations between cognitive function and behavior were extracted from the selected studies.

The methodological quality assessment was performed using the National Heart's Quality Assessment of Observational Cohort and Cross-sectional Studies, Lung and Blood Institute (NIH). This scale was developed by a group of researchers from the National Heart, Lung, and Blood Institute and the Research Triangle Institute International to assess the quality of methods, concepts, and other tools [20]. The scale

assesses 14 items to evaluate the clarity of objectives, the definition of the population, selection criteria, clarification of the sample's statistical power, use of regression methods, and blinding of the evaluators. Each item was answered with "YES," "NO," or "NO DETERMINED / NOT REPORTED / DOES NOT APPLY." Reviewers rated the quality of the studies as "Good," "Fair," or "Poor" [21]. The studies evaluated with "YES" to more than 50% of the items were classified as "Good." These studies had the lowest risk of bias and the results were considered valid. The studies with "YES" between 25% and 50% of the items evaluated were classified as "Fair." These studies were susceptible to some biases that were insufficient to invalidate their results. The "Fair" quality category is likely to be broad; thus, studies with this rating varied in strengths and weaknesses. The studies with "YES" below 25% of the items evaluated were classified as "Poor." This rating indicated a significant risk of bias.

The methodological quality of each study was independently assessed by two evaluators. Disagreements were subsequently discussed item by item to reach a consensus between the evaluators.

Results

A total of 22,484 citations were reported, with 11 studies selected for qualitative analysis after application of the eligibility criteria. All selected studies were cross-sectional, with a total population of 508 participants and 198 obese individuals from Germany, Finland, the United States, Spain, South Africa, and the Netherlands (table II).

Table II - *Included articles description and methodological quality assessment (ver PDF)*

Of the studies, five showed "Fair" quality according to the Quality Assessment of Observational Cohort and Cross-sectional Studies. The main biases identified were sample size, statistical power, and blinding of the evaluators. Six studies were classified to have "Good" quality (Table II). Four studies used functional magnetic resonance imaging (fMRI) as an imaging method [22-25], three used MRI associated with diffusion tensor imaging (DTI) [26-28], and one used positron emission tomography (PET) [29]. Two studies used electroencephalography (EEG) [30,31] and one used magnetoencephalography (MEG) [32]. All selected studies investigated brain areas activated during a task that assessed cognitive function through executive function [25,26,27,28,29], reaction time and accuracy [22,24,25-28,30,31], and memory [25,28,29,32]. Tuulari *et al.* [22] used imagery and cognitive control over food images.

The studies analyzed found differences in reaction time, accuracy, or inactivated brain areas (frontal cortex, mainly supplemental and prefrontal motor area, insula, and putamen) during tests or stimuli with food images between the groups studied. However, four of the included studies did not find any statistically significant differences in the cognitive tests [23,28,29,30].

Discussion

This systematic review evaluates the methodological quality of studies that investigated cognitive function in obese individuals and its possible interference with eating behavior. Neuroimaging and brain mapping methods allowed the identification of the cortical areas that were more activated when obese individuals were subjected to stimuli of pleasurable foods. These data show the cortical regions involved in control and eating behavior. After identifying the most activated cortical areas in the target population, an association between high BMI and reduced cognitive performance was observed when evaluating executive function, memory, and attention.

The included studies in this review assessed cognitive performance using several tests. Although the tests varied in their specific objectives, these evaluated cognitive functions involved in decision-making processes, including executive function [23,24,25,27-31], processing speed [25,26,27,28], memory [25,27,28,29,32], and attention [22-26,30].The tests showed worsening of cognitive function in obese individuals in seven of the included studies [22,24,25-27,31,32]. Reductions in cognitive performance observed in obese people are related to changes in brain functions responsible for the inhibitory control of appetite [32]. Other studies [25] investigated other changes in neural networks related to obesity using magnetic resonance. They found an increase in the functional connectivity of external networks (salience network), mainly in the putamen nucleus, which was related to a lower speed of mental processing in obese individuals.

Reductions in processing speed may contribute to overfeeding in obese people through the imbalance between homeostasis and dietary excesses. Cognition can also be affected by hypothalamic lesions, as demonstrated in the study by Puiget *et al.* [27] Using magnetic resonance, they attributed cognitive changes and eating disorders to changes in the hypothalamus through their interaction with the systems that integrate cognition and emotion (hippocampus, amygdala, and insula). In addition, the hypothalamus is involved in the control of appetite through the activation of inhibitory and excitatory neurons in the regulatory systems of food intake [3].

Four studies found no statistical differences when comparing the cognitive test results between obese and nonobese participants [23,28,29,30]. However, they found changes in dopaminergic receptors [23,29] and white matter structure [28] using fMRI and cortical excitation [30] using EEG. In the works by Hendrick *et al.* [23] and Volkow *et al.* [29], obese individuals had lower levels of type 2 dopaminergic receptors (dopamine D2 receptor).

Studies corroborate that obesity is associated with changes in the function of dopamine and its receptors in the brain reward circuit. These dysfunctions have been related to changes in behavior, which can help in understanding the subtypes of obesity [23,24,29,33]. In addition, high BMI values are associated with changes in the metabolism of the prefrontal cortex and reduced volume of gray matter (Brodmann areas 9, 10, and 32) [29], as well as changes in white matter [28].

White matter changes involved both the integrity of the cortical tracts and volume [34]. Obesity is an independent risk factor for these changes, contributing to cognitive decline. However, our correspondence with Bolzenius *et al.* [28] revealed that in his analysis, a relationship between BMI and impaired cognitive performance was not observed when controlling age as a confounding factor for cognitive decline. However, microstructural changes, in addition to proinflammatory markers and vascular changes, preceded the cognitive changes (memory and executive function) found in individuals with neurological disorders, such as dementia and Alzheimer's disease [26,35]. Impairments in cognitive performance have also been associated with comorbidities inherent in obesity, as demonstrated by Bloemendaal *et al.* [34]. In this study, obese individuals had alterations in the microstructure of the white matter that were larger than lean individuals, whereas obese individuals with type II diabetes had even more significant alterations.

Hume *et al.* [30] did not find differences between obese and eutrophic participants in the tests that measured the reaction time and accuracy of responses in the modified Stroop task. However, using EEG, they observed greater cortical excitation and attention during stimulation with food images versus neutral images (office-related items). Increased reactivity to visual food stimuli can contribute to hedonic eating and other eating behaviors that lead to weight gain, such as compulsive eating behaviors [31].

The methods used to analyze structural changes and cortical excitation in the included studies were fMRI [22,24-28], PET29, and EEG [30-32]. Magnetic resonance allows the graphical representation of the activated cortical areas. PET allows the assessment of blood flow and glucose metabolism, while electroencephalography allows the identification of the areas with the greatest arousal, attention, and reaction times before stimuli.

The differences observed between obese and normal individuals occurred mainly in the frontal cortex, which governed inhibitory control, emotion, planning, and executive function, and in the reward circuits [22]. Regions of the frontal cortex, front-medial, middle-upper and lower gyri, cingulate gyrus, precentral gyrus, supplementary motor area, thalamus, cerebellum, and occipital cortex were activated during appetite inhibitory control tasks [22]. These regions establish neural connections with subcortical regions (amygdala, hypothalamus, and striatum) and frontocortical regions (motor, premotor, orbital, and medial prefrontal) that are components of the reward circuit and are also responsible for appetite control [36].

The subcomponents of the reward circuit contribute to the processing of external information. In the case of obese individuals, this circuit exhibited dysfunctions of activity in response to pleasurable foods, a response similar to substance-dependent individuals [36,37]. The differences found in the neural substrates involved in appetite control and their diversity of responses, mainly to stimuli of pleasurable foods, can explain the various manifestations of obesity [24,38].

The diversity in the manifestations of obesity contributes to its understanding. However, it can be a confounding factor in the analysis of the results. Therefore, a well-characterized population was a strong point in our study but is also a limitation as we only included obese people with a BMI above 30 kg/m^2 and excluded overweight individuals. The included studies showed poor or good quality. This limited quality of the studies was due to methodological failure in blinding the assessors, justification of sample size, power description, or effect estimates. However, our study included only observational studies. This selection criterion may have restricted our results. Moreover, observational studies reduce the cause–effect relationship. We recommend further studies with a well-characterized obese population, distinct from overweight individuals.

Conclusion

The studies included in this review showed an association between high BMI and reduced cognitive performance, particularly executive function, and structural and functional changes in the cerebral cortex involved in reward and inhibitory appetite control. These changes suggest a relationship between compulsive behavior and impairment of appetite control in obese people. Both structural and functional changes were measured using methods analyzing brain signal uptake and brain mapping. The neuroimaging and mapping methods used were functional magnetic resonance associated with PET and diffusion tensor imaging, electroencephalography, and magnetoencephalography, with fMRI being the most frequently used method. Despite

the relevance of the results found, methodological limitations compromise the quality of the information obtained.

We recommend further studies to better understand the alterations in the neural circuits in a well-characterized obese population (BMI above 30 kg/m2). Subgroups in this population can be evaluated to further correlate BMI with changes in cortical activity and appetite control behaviors.

Conflict of interests
The authors have no conflicts of interest to declare.

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Author contributions
L.J. and S.M. conceived of the review, identified and interpreted relevant studies for inclusion, and wrote the manuscript. L.J. and K.F. independently assessed the methodological quality of the studies. All the authors critically revised the manuscript. All the authors approved of the final manuscript and agreed to be accountable for all aspects of the work. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

References

1. García-García I, Narberhaus A, Marqués-Iturria I et al. Neural responses to visual food cues: insights from functional magnetic resonance imaging. Eur Eat Disorders Rev. 2013;21:89-98. doi: 10.1002/erv.2216
2. WHO. World Health Organization (2016). Obesity and overweight 2016[Internet] [citado 2023 ago 25]. Disponível em: http://www.who.int/mediacentre/factsheets/fs311/en/
3. Farr OM, Li CR, Mantzoros CS. Central nervous system regulation of eating: insights from human brain imaging. Metabolism. 2016;65(5):699-713. doi: 10.1016/j.metabol.2016.02.002
4. Convit A. Obesity is associated with structural and functional brain abnormalities: where do we go from here? Psychosom Med. 2012;74(7):673-4. doi: 10.1097/PSY.0b013e3182662c56
5. Bocarsly ME, Fasolino M, Kane GA et al. Obesity diminishes synaptic markers, alters microglial morphology, and impairs cognitive function. PNAS. 2015; 112:15731–36. doi: 10.1073/pnas.1511593112
6. KullmannS, Heni M, Veit R et al. Selective insulin resistance in homeostatic and cognitive control brain areas in overweight and obese adults. Diabetes Care. 2015;28(6):1044-50. doi: 10.2337/dc14-2319
7. Driscoll I, Beydoun MA, An Y, Davatzikos C, Ferrucci L, Zonderman AB, Resnick SM. Midlife obesity and trajectories of brain volume changes in older adults. Hum Brain Mapp. 2011;33(9):2204-10. doi: 10.1002/hbm.21353
8. Sant Anna Junior M, Carneiro JRI, Carvalhal RF et al. Cardiovascular autonomic dysfunction in patients with morbid obesity. Arq Bras Cardiol. 2015;105(6). doi: 10.5935/abc.20150125

9. Lizarbe B, Campillo B, Guadilla I, López-Larrubia P, Cerdán S. Magnetic resonance assessment of the cerebral alterations associated with obesity development. J Cereb Blood Flow Metab. 2020;40(11):2135–51. doi: 10.1177/0271678X20941263
10. Stopyra MA, Friederich HC, Lavandier N, Mönning E, Bendszus M, Herzog W, Simon JJ. Homeostasis and food craving in obesity: a functional MRI study. Int J Obes. 2021;45:2464-2470. doi: 10.1038/s41366-021-00920-4
11. Cornier MA, McFadden KL, Thomas EA, Bechtell JL, Eichman LS, Bessesen DH, Tregellas JR. Differences in the neuronal response to food in obesity-resistant as compared to obesity-prone individuals. Physiol Behav. 2013;110-111:122-128. doi: 10.1016/j.physbeh.2013.01.002
12. Cornier MA, Melanson EL, Salzberg AK, Bechtell JL, TregellasJR. The effects of exercise on the neuronal response to food cues. Physiol Behav. 2012;105:1028-34. doi: 10.1016/j.physbeh.2011.11.023
13. Blechert J, Klackl J, Miedl SF, Wilhelm FH. To eat or not to eat: effects of food availability on reward system activity during food picture viewing. Appetite. 2016;99:256-61. doi: 10.1016/j.appet.2016.01.006
14. Kullmann S, Callaghan MF, Heni M et al. Specific white matter tissue microstructure changes associated with obesity. Neuroimage. 2016;125:36-44. doi: 10.1016/j.neuroimage.2015.10.006
15. Alonso-Alonso M. Translating tDCS into the field of obesity: mechanism-driven approaches. Front Hum Neurosci. 2013;7:1-3. doi: 10.3389/fnhum.2013.00512
16. Figley CR, Asem JSA, Levenbaum EL, Courtney SM. Effects of body mass index and body fat percent on default mode, executive control, and salience network structure and function. Front Hum Neurosci. 2016;10:1-23. doi: 10.3389/fnins.2016.00234
17. Ronan l, Bloch AFA, Wagstyl K et al. Obesity associated with increased brain age from midlife. Neurobiol Aging. 2016;47:63-70. doi: 10.1016/j.neurobiolaging.2016.07.010
18. Kulmann S, Heni M, FritscheA, Preissl H. Insulin action in the human brain: evidence from neuroimaging studies. J Neuroendocrinol. 2015;21;419-23. doi: 10.1111/jne.12254
19. Galvão TF, Pansani TSA. Principais itens para relatar revisões sistemáticas e meta-análises: a recomendação PRISMA. Epidemiol Serv Saúde. 2015;24:335-42. doi: 10.5123/S1679-49742015000200017
20. Quality assessment tool for observational cohort and cross-sectional studies – NHLBI, NIH. Disponível em: https://www.nhlbi.nih.gov/health-topics/study-quality-assessment-tools
21. Maass SWMC, Roorda C, Berendsen AJ, Verhaak PFM, Bock GH. The prevalence of long-term symptoms of depression and anxiety after breast cancer treatment: A systematic review. Maturitas 2015;82(1):100-8. doi: 10.1016/j.maturitas.2015.04.010
22. Tuulari JJ, Karlsson HK, Hirvonen J, Salminen P, Nuutila P, Nummenmaa L. Neural circuits for cognitive appetite control in healthy and obese individuals: an fmri study. PLoS One. 2015;10(2):e0116640. doi: 10.1371/journal.pone.0116640

23. Hendrick OM, Luo X, Zhang S, Li CR. Saliency processing and obesity: a preliminary imaging study of the stop signal task. Obesity. 2015;20:1796–802. doi: 10.1038/oby.2011.180
24. Balodis IM, Molina ND, Kober H et al.Divergent neural substrates of inhibitory control in binge eating disorder relative to other manifestations of obesity. Obesity. 2013;21:367-77. doi: 10.1002/oby.20068
25. García-García I, Jurado MA, Garolera M et al. Alterations of the salience network in obesity: aresting-state fmri study. Hum Brain Mapp. 2012. doi: 10.1002/hbm.22104
26. Fernandez-Real JM, Serino M, Blasco G et al. Gut microbiota interacts with brain microstructure and function. J Clin Endocrinol Metab. 2015; 100: 4505–13. doi: 10.1210/jc.2015-3076
27. Puig J, Blasco G, Daunis-i-Estadella J, et al. Hypothalamic damage is associated with inflammatory markers and worse cognitive performance in obese subjects. J Clin Endocrinol Metab. 2015;100: E276–E281. doi: 10.1210/jc.2014-2682
28. BolzeniusJD,Laidlaw DH, Cabeen RP, et al. Brain structure and cognitive correlates of body mass index in healthy older adults. Behav Brain Res. 2015;278: 342–47. doi: 10.1016/j.bbr.2014.10.010
29. Volkow ND, Wang GJ, Telang F. Inverse association between bmi and prefrontal metabolic activity in healthy adults. Obesity. 2008;17:60–65. doi: 10.1038/oby.2008.469
30. Hume DJ, Howells FM, Rauch HGL, Kroff J, Lambert EV. Electrophysiological indices of visual food cue-reactivity.Differences in obese, overweight and normal weight women.Appetite. 2015; 85: 126–37. doi: 10.1016/j.appet.2014.11.012
31. Nijs IMT, Franken IHA, Muris P. Food-related stroop interference in obese and normal-weight individuals: behavioral and electrophysiological indices. Eat Behav. 2010;11:258–65. doi: 10.1016/j.eatbeh.2010.07.002
32. Stingl KT, Kullmann S, Ketterer C, Heni M, Häring HU, Fritsche A, Preissl H. Neuronal correlates of reduced memory performance in overweight subjects. NeuroImage. 2012;60:362–69. doi: 10.1016/j.neuroimage.2011.12.012
33. Eisenstein SA, Gredysa DM, Antenor-Dorsey JA, et al. Insulin, central dopamine d2 receptors, and monetary reward discounting in obesity. PLoS One. 2015. doi: 10.1371/journal.pone.0133621
34. Bloemendaal L, Ijzerman RG, Kulve JS, Barkhof F, Diamant M, Veltman DJ, Duinkerken E. Alterations in white matter volume and integrity in obesity and type 2 diabetes. Metab Brain Dis. 2016;31:621–29. doi: 10.1007/s11011-016-9792-3.
35. Gunstad J, Strain G, Devlin MJ, et al. Improved memory function 12 weeks after bariatric surgery. Surg Obes and Relal Dis. 2011;7:465–72. doi: 10.1016/j.soard.2010.09.015
36. Nummenmaa L, Hirvonen J, Hannukainen JC, Immonen H, Lindroos MM, Salminen P, Nuutila P. Dorsal striatum and its limbic connectivity mediate abnormal anticipatory reward processing in obesity. PLoS One. 2012. doi: 10.1371/journal.pone.0031089

37. Ziauddeen H, Alonso-Alonso M,Hill JO, Kelley M,Khan NA. Obesity and the neurocognitive basis of food reward and the control of intake. AdvNutr. 2015;6:474–86. doi: 10.3945/an.115.008268
38. Hendrikse JJ, Cachia RL, Kothe EJ, McPhie S, Skouteris H, Hayden MJ. Attentional biases for food cues in overweight and individuals with obesity: a systematic review of the literature. Obes Rev. 2015;16:424-32. doi: 10.1111/obr.12265

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