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Review

Neuroimaging and neuromodulation approaches to study eating behavior and prevent and treat eating disorders and obesity

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ABSTRACT

Functional, molecular and genetic neuroimaging has highlighted the existence of brain anomalies and neural vulnerability factors related to obesity and eating disorders such as binge eating or anorexia nervosa. In particular, decreased basal metabolism in the prefrontal cortex and striatum as well as dopaminergic alterations have been described in obese subjects, in parallel with increased activation of reward brain areas in response to palatable food cues. Elevated reward region responsivity may trigger food craving and predict future weight gain. This opens the way to prevention studies using functional and molecular neuroimaging to perform early diagnostics and to phenotype subjects at risk by exploring different neurobehavioral dimensions of the food choices and motivation processes. In the first part of this review, advantages and limitations of neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), single photon emission computed tomography (SPECT), pharmacogenetic fMRI and functional near-infrared spectroscopy (fNIRS) will be discussed in the context of recent work dealing with eating behavior, with a particular focus on obesity. In the second part of the review, non-invasive strategies to modulate food-related brain processes and functions will be presented. At the leading edge of non-invasive brain-based technologies is real-time fMRI (rtfMRI) neurofeedback, which is a powerful tool to better understand the complexity of human brain–behavior relationships. rtfMRI, alone or when combined with other techniques and tools such as EEG and cognitive therapy, could be used to alter neural plasticity and learned behavior to optimize and/or restore healthy cognition and eating behavior. Other promising non-invasive neuromodulation approaches being explored are repetitive transcranial magnetic stimulation (rTMS) and transcranial direct-current stimulation (tDCS). Converging evidence points at the value of these non-invasive neuromodulation strategies to study basic mechanisms underlying eating behavior and to treat its disorders. Both of these approaches will be compared in light of recent work in this field, while addressing technical and practical questions. The third part of this review will be dedicated to invasive neuromodulation strategies, such as vagus nerve stimulation (VNS) and deep brain stimulation (DBS). In combination with neuroimaging approaches, these techniques are promising experimental tools to unravel the

Abbreviations: 5-HT, serotonin; aCC, anterior cingulate cortex; ADHD, attention deficit hyperactivity disorder; AN, anorexia nervosa; ANT, anterior nucleus of the thalamus; BAT, brown adipose tissue; BED, binge eating disorder; BMI, body mass index; B N, bulimia nervosa; BOLD, blood oxygenation level dependent; BS, bariatric surgery; CBF, cerebral blood flow; CCK, cholecystokinin; Cg25, subgenual cingulate cortex; DA, dopamine; daCC, dorsal anterior cingulate cortex; DAT, dopamine transporter; DBS, deep brain stimulation; DBT, deep brain therapy; dlPFC, dorsolateral prefrontal cortex; DTI, diffusion tensor imaging; dTMS, deep transcranial magnetic stimulation; ED, eating disorders; EEG, electroencephalography; fMRI, functional magnetic resonance imaging; fNIRS, functional near-infrared spectroscopy; GP, globus pallidus; HD-tDCS, high-definition transcranial direct current stimulation; HFD, high-fat diet; HHb, deoxygenated-hemoglobin; LHA, lateral hypothalamus; lPFC, lateral prefrontal cortex; MER, microelectrode recording; MRS, magnetic resonance spectroscopy; Nac, nucleus accumbens; OCD, obsessive–compulsive disorder; OFC, orbitofrontal cortex; O_2Hb, oxygenated-hemoglobin; pCC, posterior cingulate cortex; PD, Parkinson's disease; PET, positron emission tomography; PFC, prefrontal cortex; PYY, peptide tyrosine tyrosine; rCBF, regional cerebral blood flow; rtfMRI, real-time functional magnetic resonance imaging; rTMS, repetitive transcranial magnetic stimulation; SPECT, single photon emission computed tomography; STN, subthalamic nucleus; tACS, transcranial alternate current stimulation; tDCS, transcranial direct current stimulation; TMS, transcranial magnetic stimulation; TRD, treatment-resistant depression; tRNS, transcranial random noise stimulation; VBM, voxel-based morphometry; vlPFC, ventrolateral prefrontal cortex; vmH, ventromedial hypothalamus; vmPFC, ventromedial prefrontal cortex; VN, vagus nerve; VNS, vagus nerve stimulation; VS, ventral striatum; VTA, ventral tegmental area

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intricate relationships between homeostatic and hedonic brain circuits. Their potential as additional therapeutic tools to combat pharmacorefractory morbid obesity or acute eating disorders will be discussed, in terms of technical challenges, applicability and ethics. In a general discussion, we will put the brain at the core of fundamental research, prevention and therapy in the context of obesity and eating disorders. First, we will discuss the possibility to identify new biological markers of brain functions. Second, we will highlight the potential of neuroimaging and neuromodulation in individualized medicine. Third, we will introduce the ethical questions that are concomitant to the emergence of new neuromodulation therapies.

Contents

1. Introduction

A recent study estimated the number of overweight adults in the world as roughly 2.1 billion in 2013 (Ng et al., 2014). In the United States alone, obese individuals have 42% higher health care costs than those with healthy-weight (Finkelstein et al., 2009). Obesity is on the rise, with severe obesity rising at a particularly alarming rate (Flegal et al., 2010; Finkelstein et al., 2012). Because obesity is a multifactorial condition with a complex etiology, and because success of interventions is subject to a large interindividual variability, there is no panacea or "one-fit-all" treatment for obesity. Bariatric surgery (BS) is the treatment of choice for severe obesity due to its effectiveness compared to behavioral and pharmacological interventions (Buchwald and Oien, 2013). Its utility and success rate is widely accepted. However, 20–40% of those who undergo BS fail to lose sufficient weight (Christou et al., 2006; Livhits et al., 2012) or regain significant weight after treatment (Magro et al., 2008; DiGiorgi et al., 2010; Adams et al., 2012), and can experience a number of complications during and after surgery or medical and psychiatric comorbidities (Shah et al., 2006; Karlsson et al., 2007; DiGiorgi et al., 2010; Bolen et al., 2012; Chang et al., 2014). In addition to existing methods such as BS, which annually helps thousands of people worldwide, there is a clear need for novel approaches to obesity prevention and treatment, including the development of novel diagnostic and phenotyping methods, as well as adjunctive therapies