



A functional neuroimaging review of obesity, appetitive hormones and ingestive behavior



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HIGHLIGHTS

- Obese and lean individuals show differences in neural response to food stimuli.
- Neuroendocrine functioning & ingestive behavior also relate to weight status.
- A better understanding of these factors will provide greater insight to weight gain.
- Impulsivity, food macronutrient content & reward learning impact ingestive behavior.

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ABSTRACT

Adequate energy intake is vital for the survival of humans and is regulated by complex homeostatic and hedonic mechanisms. Supported by functional MRI (fMRI) studies that consistently demonstrate differences in brain response as a function of weight status during exposure to appetizing food stimuli, it has been posited that hedonically driven food intake contributes to weight gain and obesity maintenance. These food reward theories of obesity are reliant on the notion that the aberrant brain response to food stimuli relates directly to ingestive behavior, specifically, excess food intake. Importantly, functioning of homeostatic neuroendocrine regulators of food intake, such as leptin and ghrelin, are impacted by weight status. Thus, data from studies that evaluate the effect on weight status on brain response to food may be a result of differences in neuroendocrine functioning and/or behavior. In the present review, we examine the influence of weight and weight change, exogenous administration of appetitive hormones, and ingestive behavior on BOLD response to food stimuli.

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1. Introduction

Nearly 30% of American adults are able to maintain a healthy weight (defined as body mass index (BMI) < 25.1) despite living in an environment that relentlessly presents large portions of energy-dense, highly palatable foods [1]. The excess adiposity tissue of the remaining 70% of Americans, who are overweight or obese, places them at higher risk for diseases such as atherosclerotic cerebrovascular disease, coronary heart disease, cancer, hyperlipidemia, hypertension, and diabetes mellitus, which result in as many as 300,000 annual deaths [2]. The dramatic rise and maintenance of the prevalence of obesity in the majority, but not the entirety of the population indicate that individual differences in the determinants of ingestive behavior play a role in weight regulation.

The determinants of ingestive behavior are frequently thought to involve parallel systems that interact with the external food environment to influence food intake [3,4]. First, homeostatic mechanisms, via appetitive hormones (e.g., leptin, ghrelin), act on neural circuitry converging through hypothalamus to stimulate or inhibit feeding in an effort to maintain energy balance and a healthy weight. Evidenced by the prevalence of overweight and obesity, this system is susceptible to failure. As a result, more research attention has focused on the impact of hedonic aspects of food and food cues on the brain's reward, attentional, and behavioral control circuitries. Theorists posit that neuropsychological constructs such as aberrant reward-related responses to food intake and/or cues override homeostatic processes, resulting in excess adipose tissue and weight gain (e.g., [3]).

Advances in neuroimaging techniques, particularly functional magnetic resonance imaging (fMRI) allow for the study of whole brain blood-oxygen level dependent (BOLD) response, a proxy used to indicate activity, during exposure various food stimuli in humans without use of contrast agents. This evolving technique has provided valuable insight into the neural correlates of ingestive behavior and weight

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regulation. Yet, some of the most prominent theories of aberrant neural responses to food reward and obesity appear to be in conflict. For example, obesity has been described as both a problem characterized by hyper- and hypo-responsivity of the reward circuitry [5–7]. Similarly, it has been suggested that these seemingly incompatible theories can operate concurrently and the valence of the neural response is contingent on the specific type of stimulus (i.e., the response to predictive cue versus actual receipt of a palatable tastant [8]). Independent of the exact hypothesis, all food reward based theories of obesity rely on the notion that the observed aberrant neural response to food stimuli relates to ingestive behavior. For example, it has been posited hypersensitivity to food cues places individuals at greater risk for overeating [7], presumably because these individuals are more susceptible to environmental food cues and eat more frequently (i.e., decreased satiety; Fig. 1.). Likewise, it has been suggested that those with a deficient neural response to food in reward-related regions consume excess food to compensate for a lack of reward or attenuated reinforcement [5], which could likely result in greater intake during a single eating occasion (i.e., delayed satiety; Fig. 1.). To date, most studies use weight status or weight change when examining BOLD response to food stimuli and have yet to directly examine the neural underpinnings of ingestive behavior, i.e., the neurobehavioral aspects that contribute to body weight.

Examining the neural responses to food stimuli that contribute to overeating behavior may prove critical, as changes in physiology, particularly neuroendocrine functioning, are associated with weight change. These alterations in neuroendocrine functioning may confound examinations of obese versus lean individuals that are aimed at understanding the etiology of obesity. Further, because habitual overeating resulting in a positive energy balance precedes initial weight gain, direct examination of the neural correlates of ingestive behavior provides insight into obesity risk factors; whereas assessing brain response to food stimuli in obese versus lean individuals or in obese individuals before and after weight change may elucidate obesity maintenance factors. Improved knowledge of neural risk factors for weight gain and differentiation of these risk factors from neural consequences of excess adipose tissue are needed, especially as data from food-related fMRI studies begin to serve as outcome measures of behavioral obesity prevention and treatment research. This review first examines human fMRI studies focused on brain-based correlates of obesity and weight change, briefly surveys studies examining the influence of select appetitive hormones on BOLD response to food stimuli, as well as studies focused on relation of acute and the habitual ingestive behavior to BOLD response to food stimuli. Lastly, we review emerging factors that are related to aspects of hedonically driven food intake.

2. BOLD response to food stimuli as a function of weight status

To date, fMRI studies assessing the relation between weight and BOLD response to food stimuli typically use one of three types of designs: 1) cross-sectional studies comparing overweight and/or obese versus lean individuals, 2) prospective ‘brain-as-predictor’ of weight change designs or 3) within-subject repeated scan designs. Each of

these designs provides unique insight into the neural underpinnings, consequences, and maintenance factors of obesity.

2.1. Cross-sectional evaluation of BOLD response in obese versus lean humans

One of the most replicated findings in the neuroimaging of obesity is the elevated neural response to images of palatable and energy-dense foods seen in obese versus lean humans. When compared to their lean counterparts, overweight and obese individuals show significantly more activity in the striatum, insula, orbitofrontal cortex (OFC), and amygdala [9–17], all regions thought to encode the reward value of stimuli and consequently influence future behavior [18]. When exposed to visual food images or cues predicting palatable food receipt, obese versus lean individuals also show greater activation in brain regions associated with visual processing and attention (visual and anterior cingulate cortices), the encoding of stimulus salience (precuneus), in the primary taste cortex (anterior insula, frontal operculum), and oral somatosensory regions (postcentral gyrus, rolandic operculum; [9–17]). In contrast, in several studies obese versus lean individuals showed less activity in the striatum in response to receipt of palatable food relative to a tasteless solution [17,19–22], though one study failed to replicate these findings [16].

These data indicate that overweight individuals show aberrant neural responses to food stimuli, specifically, an elevated striatal response to anticipatory cues (e.g., images, predictive cues) and decreased striatal response during consumption. As a result, these findings support both the hyper- and hypo-reward theories of obesity [5–7]. Critically, by nature of study design alone, cross-sectional investigations provide no ability to draw inferences regarding the temporal precedence of weight status and observed neural effects and thus equally support the notions that the BOLD response patterns are an underlying cause of weight gain or a consequence of habitual overeating and/or obesity. Further, these data and theories inherently presume a static neural response despite the high likelihood of neuroadaptations associated with repeated consumption of rewarding foods and recurring exposure to the associated food cues, as well as the known altered neuroendocrine functioning that is associated with obesity.

2.2. Prospective evaluation of BOLD response and weight change

Prospective studies provide insight into preexisting risk factors for weight gain relative to consequences of or maintenance factors associated with obesity. For example, neural activity can be used to predict subsequent behaviors in a ‘brain-as-predictor’ methodology wherein BOLD response to relevant stimuli is assessed at baseline and tested as a predictor of an outcome measure of interest assessed in the future [23]. Using a similar approach, individuals who showed greater activation in the OFC in response to a cue predicting palatable food gained more weight over one year [24]. Concordantly, ventral striatum and anterior cingulate activity in response to appetizing food images predicted weight gain at 6 month follow-up [25]. The authors concluded that individuals with elevated responses to food images in these reward and attention-related regions are at risk for future weight gain. Interestingly,

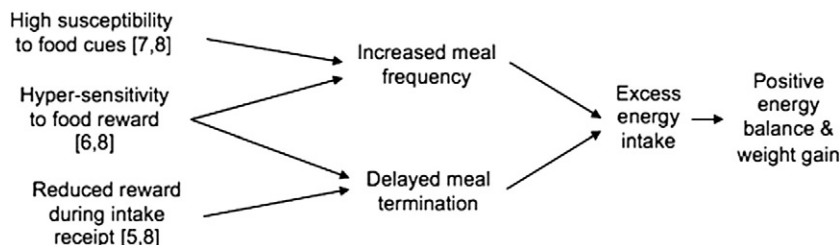


Fig. 1. Possible ingestive behavior mechanisms of reward-based theories of obesity.

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