Do scores on the Food Craving Inventory and Three-Factor Eating Questionnaire correlate with expected brain regions of interest in people with obesity?

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ABSTRACT

Objective: To examine whether subscales of Food Craving Inventory (FCI) and Three-factor Eating Questionnaire (TFEQ) correlate with brain functional magnetic resonance imaging food-cue reactivity (fMRI-FCR) in the brain.

Methods: Thirty-two male and female adults with obesity (19–60 years; 30–39.9 kg/m²) participated in a 3-week dietary intervention (1120 kcal/day from either 1) total meal replacement shakes, 2) portion-controlled typical food. FCI, TFEQ and fMRI-FCR were measured pre- and post-intervention. Correlations between pre-intervention fMRI-FCR and standardized pre-intervention FCI and TFEQ subscales; and also post- versus pre-intervention change in fMRI-FCR (ΔfMRI-FCR) and standardized changes in FCI and TFEQ subscales were examined at the whole brain level using tools in FMRIB Software Library.

Results: Twenty-eight subjects completed the intervention. Pre-intervention high-fat food cravings (P = 0.041) and fast-food cravings (P = 0.017) were negatively correlated with fMRI-FCR of several brain regions that regulate executive control over ingestion (i.e. bilateral lateral frontal pole, dorsolateral prefrontal cortex and dorsal anterior cingulate cortex). Post- vs. pre-intervention change in sweet (P = 0.012) and fast food cravings (P = 0.004) were negatively correlated with ΔfMRI-FCR of bilateral lateral frontal pole, dorsolateral prefrontal cortex, inferior frontal gyrus (pars opercularis) and dorsal anterior cingulate cortex (i.e. brain regions that regulate executive control over ingestion). Negative correlations were also observed between the changes in sweet and fast food cravings and ΔfMRI-FCR of brain regions that regulate food reward (i.e. bilateral mid-anterior insula, right nucleus accumbens), motor readiness to ingest (i.e. bilateral precentral gyrus), internally focused attention (i.e. bilateral precuneus and posterior cingulate cortex) and visual object recognition (i.e. occipital pole, lateral occipital cortex and middle and inferior temporal cortices). Changes in cravings for starchy food (P = 0.032) and overall food cravings (P = 0.027) were also negatively correlated with ΔfMRI-FCR of brain regions involved in regulating internally focused attention and visual object recognition.

Conclusions: In individuals with obesity, decreased food cravings seem to be reflective of increased fMRI-FCR of brain regions that regulate executive control over ingestion. Taken together, constructs measured by FCI seem to be reflective of neurophysiological processes underlying ingestive behavior and the changes in neurophysiological processes occurring during calorie restriction.

Clinical trials registry number: NCT02637271; the protocol is available at https://clinicaltrials.gov/ct2/show/NCT02637271

Abbreviations: FCI, Food Craving Inventory; TFEQ, Three-factor Eating Questionnaire; fMRI-FCR, functional magnetic resonance imaging food-cue reactivity; ΔfMRI-FCR, change in functional magnetic resonance imaging food-cue reactivity; FCI-O, overall food cravings as measures by the Food Craving Inventory; FCI-S, craving for sweet food as measures by the Food Craving Inventory; FCI-H, craving for high-fat food as measures by the Food Craving Inventory; FCI-FF, craving for fast food as measures by the Food Craving Inventory; TFEQ-R, dietary restraint scale of the Three-factor Eating Questionnaire; TFEQ-D, disinhibition scale of the Three-factor Eating Questionnaire; TFEQ-S, susceptibility to hunger scale of the Three-factor Eating Questionnaire; FSL, FMRIB Software Library; TMR, total meal replacement; TD, typical diet

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1. Introduction

Increased energy intake over energy expenditure is an important contributor to obesity [1]. Energy intake is regulated by homeostatic and hedonistic mechanisms in the brain [1–4]. Determining the pleasure and reward associated with ingestion, exercising executive control over ingestion and regulating the motivational salience to ingest after considering the above inputs are important components of the neurophysiological mechanisms that regulate human ingestive behavior. Much of what we know of specific regional involvement is derived from animal studies and our understanding of these mechanisms have been further substantiated by both visual and taste cue-reactivity neuroimaging studies. The shell of the nucleus accumbens, orbitofrontal cortex, mid insular cortex and anterior ventral regions of the anterior cingulate cortex have been associated with regulation of pleasure related to ingestion of a specific type of food [5–9]. The dorsolateral prefrontal cortex, lateral frontal pole, inferior frontal gyrus (especially pars opercularis) as well as the dorsal regions of the anterior cingulate cortex are thought to exert executive inhibitory control over ingestion [1,2,10–14]. The core of the nucleus accumbens and the amygdala are hypothesized to be involved in regulating the motivational salience towards ingestion, after considering hedonic and homeostatic inputs received from other brain regions [1,8,15]. Execution of ingestion is brought about by brain regions that control the motor system (e.g. the primary motor cortex located in the precentral gyrus) [16–18]. Therefore, changes in functional activity in response to food cues in the above brain regions may impact human ingestive behavior [19].

Multiple self-report instruments have been developed to measure specific constructs related to ingestive behavior. Psychological constructs measured via these behavioral self-report instruments are often implied to represent the neurophysiological processes that regulate human ingestive behavior. Food craving, defined as frequent, intense desires to consume a specific type of food, is one such broad construct [20]. It is commonly thought that food cravings are likely to represent brain regions that regulate food-related pleasure and reward and somehow predispose an individual to act (i.e. motor readiness to ingest). The Food Craving Inventory (FCI) is a validated self-report instrument that measures 3 psychological constructs: dietary restraint (FCI-R), disinhibition (FCI-D) and susceptibility to hunger (FCI-S) [22]. Dietary restraint is defined as an intention to restrict consumption of food in an attempt to control body weight and is considered an essential cognitive function that prevents the excessive consumption of food the in modern, food-cue abundant environment [21]. However, to the best of our knowledge, the association between FCI subscales and the food-cue reactivity of brain regions that seem to be represented by the FCI subscales have not been examined to-date.

The Three-factor Eating Questionnaire (TFEQ) is another self-report instrument that measures 3 psychological constructs: dietary restraint (TFEQ-R), disinhibition (TFEQ-D) and susceptibility to hunger (TFEQ-S) [22]. Dietary restraint is defined as an intention to restrict consumption of food in an attempt to control body weight and is considered an essential cognitive function that prevents the excessive consumption of food the in modern, food-cue abundant environment [21]. Among many other scales that have been developed to measure dietary restraint, TFEQ-R has been shown to be the most valid instrument that represents the actual intention to reduce food and energy intake [24]. In light of this, it is logical to postulate that TFEQ-R should be reflected in the functions of brain regions involved in exerting executive inhibitory control over ingestion. Disinhibition measured by TFEQ-D is defined as a propensity of an individual to overeat in the presence of a favorable environment (e.g. highly palatable food) or an emotional predisposition (e.g. stress) [25]. Therefore, the behavioral construct is likely to represent the neurophysiological mechanisms that regulate pleasure, incentive salience and motor readiness to ingest. TFEQ-S measures the susceptibility of an individual to perceive hunger and consume food in-response to hunger, triggered by internal and external cues [26]. Thus, TFEQ-S subscales could be thought to represent the functional activity of brain regions that regulate incentive salience and motor readiness to ingest. However, the associations between the TFEQ subscales and the food-cue reactivity of brain regions that are known to regulate ingestive behavior have not yet been examined.

In a recent review of literature, we concluded that extended calorie restriction appears to decrease the functional magnetic resonance imaging food-cue reactivity (fMRI-FCR) of the brain regions that regulate food-related pleasure and reward (e.g. amygdala, insula, orbitofrontal cortex) and brain regions that possibly represent motor readiness to ingest (e.g. precentral gyrus), while increasing the reactivity of brain regions that exert executive inhibitory control over ingestion (e.g. the dorsolateral prefrontal cortex) [19]. In several behavioral studies, extended calorie restriction has also been shown to be associated with reductions in food cravings and increased dietary restraint, further supporting the theory that changes in food cravings and dietary restraint may in fact be representative of changes in brain reactivity to food-cues [27–30]. We sought to empirically test these theoretical assumptions.

First, using the baseline data from a 3-week, randomized controlled clinical functional magnetic resonance imaging (fMRI) study that compared the neurophysiological, as well as behavioral effects of two forms of iso-caloric calorie restriction interventions [31], we aimed to examine whether FCI and TFEQ subscales will be correlated with brain fMRI-FCR of the brain in individuals with obesity. We further aimed to examine whether changes in FCI and TFEQ subscales during the 3-week calorie restriction intervention will be correlated with post- versus pre-intervention changes in fMRI-FCR (ΔfMRI-FCR) of the brain. We specifically hypothesized that pre-intervention scores of FCI subscales, TFEQ-D and TFEQ-S will be positively associated with fMRI-FCR of brain regions that regulate food reward, pleasure and motor readiness to ingest and will be negatively associated with fMRI-FCR of brain regions that exert executive control over ingestion. We further hypothesized that post- vs. pre-intervention changes in the above subscales will also be positively correlated with ΔfMRI-FCR of brain regions that regulate food reward and motor readiness to ingest and will be negatively correlated with brain regions that regulate executive control over ingestion. We theorized that pre-intervention and post- vs. pre-intervention change in TFEQ-R will also be correlated with the brain regions hypothesized above, yet in the opposite direction.

2. Materials and methods

2.1. Ethics

Methods of the primary study from which the data was derived is described fully elsewhere [31] and is briefly described here. All procedures were approved by the Texas Tech Institutional Review Board (protocol number: 505380) and conducted in accord with the Helsinki Declaration amended in 2000 [32].

2.2. Subjects

Thirty-two adult (18–60 years) males and females with grade I and grade II obesity (BMI 30–39.9 kg/m²) were recruited for the study. Subjects with contraindications to undergo magnetic resonance imaging or any conditions that may confound the weight loss outcomes (e.g. participation in a weight loss intervention within 3 months, being on medications that are known to affect body weight) and fMRI-FCR (e.g. type II diabetes mellitus, current severe psychiatric illness, neurological disease, diagnosis of binge eating disorder) were excluded from the study. Furthermore, female subjects with irregular menstrual cycles were excluded.
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