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Depressive symptoms and poorer performance on the Stroop Task are associated with weight gain

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ABSTRACT

Objective: Executive function impairments and depression are associated with obesity but whether they predict weight gain is unclear.

Methods: Forty-six individuals (35 m, 37 ± 10y) completed the Stroop Task, Iowa Gambling Task (IGT), Wisconsin Card Sorting Task (WCST), Inventory for Depressive Symptomatology (IDS-SR), Physical Anhedonia Scale (PAS), and Perceived Stress Scale (PSS). Body composition (DXA) and fasting glucose were also measured. Data from return visits were used to assess changes in weight.

Results: Poorer Stroop and WCST performance associated with higher BMI whereas poorer IGT and WCST performance associated with higher body fat (%; all p's ≤ 0.05). Stroop interference ($p = 0.04$; $p = 0.05$) and IDS-SR ($p = 0.06$; $p = 0.02$) associated with increased BMI and weight gain (%/yr). In a multivariate linear model Stroop interference ($\beta = 0.40$, $p < 0.01$; $\beta = 0.35$, $p < 0.01$) and IDS-SR ($\beta = 0.38$, $p < 0.01$; $\beta = 0.37$, $p < 0.01$) independently predicted increased BMI and weight gain (%/yr) even after controlling for baseline weight and glucose levels.

Conclusions: Poorer response inhibition and depressive symptoms, but not glucose levels, predicted weight gain. Evaluating neurocognitive and mood deficits could improve current treatment strategies for weight loss.

Clinical Trial Registration Numbers
NCT00523627, NCT00342732, NCT01224704. clinicaltrials.gov

1. Introduction

Obesity is associated with cardiovascular risk factors including impaired glucose regulation, and more recently, with increased risk of Alzheimer's disease and cognitive decline later in life [6]. The link between obesity and neurocognitive impairment has been most strongly inferred by adiposity associated alterations within the prefrontal cortex (PFC) [38]. Studies from our lab [25,34] and others have demonstrated that elevated BMI is associated with decreased activity [25,39] and lower gray matter volume [29] in the PFC.

A growing body of literature indicates a link between obesity and impairments in executive functions [20,36], higher order brain processes that regulate cognitive processes such as planning and reasoning, flexibility, inhibition, problem solving, and decision making [13]. Neuroimaging studies indicate that the PFC is activated during executive function related tasks [39]. Impairments in executive functions are associated with maladaptive eating behaviors, inability to resist impulses, impaired decision making and self-control, and generalized poor weight loss attempts ([20]; X [41].). The ability to make healthy choices in the face of biological drives relies heavily on executive functioning [5].

Executive functions represent several different neurocognitive domains and the literature is unclear as to specific areas of executive functioning that associate with obesity. For instance, compared to normal BMI adolescents those with higher BMI performed significantly worse on measures of inhibition, flexibility, and decision-making [37], but no differences were seen in measures of working memory, planning, or reasoning tasks. In contrast, one study found that overweight and obese individuals displayed significant impairments in working memory compared to lean individuals [9]. Furthermore, individuals with obesity, anorexia-nervosa, or bulimia nervosa displayed poorer decision making abilities on the IGT task [4] compared to controls. The lack of consensus as to which domains of executive function are impaired the most with increasing adiposity and therefore which measurements to use highlights a critical need for studies with longitudinal follow-up to fully uncover which measures are important.

Psychological variables are also related to impairments in executive functions, obesity and glucose regulation. A recent review suggests a

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neuropsychological model of obesity and obesity related behaviors [21] with interactions between psychological and cognitive factors [27]. Neuroimaging studies have reported that the altered brain areas seen in obesity and executive dysfunction are also associated with psychological factors such as stress and depression [22]. In healthy adults, decreased gray and white matter volumes within the dorsolateral and ventrolateral PFC were associated with greater perceived stress [30]. Compared to healthy controls, individuals with major depressive disorder (MDD) had decreased gray matter volume in the PFC [32]. However, independent of MDD, higher BMI across both groups was also associated with decreased gray matter volume in the PFC. Other studies looking at the impact of psychological factors on executive functioning found that nonsomatic (i.e. anhedonia and self-esteem) depression symptom clusters were predictive of executive functioning impairment [17] and our group recently showed that perceived stress, anhedonia and depression were associated with weight gain [19].

There is a known association between depression and impaired glucose regulation and many studies also link impaired glucose regulation [18] to lower levels of cognitive function on neuropsychological tests [23]. There is evidence that even mildly increased glucose concentrations in the non-diabetic range are associated with impaired cognition [18]. Moreover, a recent study from our lab found an association between impaired glucose regulation and poorer performance on the Stroop Task, a measure of selective attention [36].

Despite the identified associations between psychological factors, glucose regulation, executive function and obesity, few studies [6,21,32] have examined all 4 variables simultaneously and none with longitudinal data on weight change. We hypothesized that measures of executive functioning would be associated with adiposity measures at baseline and that impaired executive functioning and fasting glucose levels at baseline would predict increased body adiposity over time. Lastly, we hypothesized that the association between executive functioning and change in adiposity may be moderated by psychological variables (Depression, Anhedonia, Stress).

2. Methods

2.1. Participants

Forty-six individuals were recruited from the greater Phoenix area by advertisement to participate in one of three studies on our clinical unit (Clinical Trial Identifier: NCT00523627, NCT00342732, NCT01224704). All were observational studies of the effects of overconsumption and different diets on energy expenditure or food intake preference as risk factors for obesity. The studies did not include any medications or weight loss interventions. All measures were collected on the Clinical Research Unit of the National Institute of Diabetes and Digestive and Kidney Diseases – Phoenix (NIDDK). Inclusion criteria for all studies consisted of healthy adults, between the ages of 18–55, with no evidence of illness by history, physical or basic laboratory measures. No participants were taking medication. Exclusion criteria included substance abuse (positive urine test), nicotine use, or reported excess alcohol use (> 3 drinks/day). Prior to participation, all participants were informed of the nature, purpose and risks of the study and written informed consent was obtained. The decision to combine data from these studies to assess the impact of psychosocial measures on body weight was pre-planned as these studies were all relatively small. The experimental protocols were approved by the Institutional Review Board of the NIDDK.

2.2. Study design

The first four days of each study was identical. Upon admission to the Unit, participants were placed on a standard weight maintaining diet (WMEN) for three days (20% protein, 50% carbohydrate, and 30% fat), calculated for each individual based on weight [12]. Participants were instructed to consume the entirety of every weight maintaining meal. Within the first 2 days after admission, while participants were weight stable, percent body fat was measured using Dual-Energy X-Ray Absorptiometry (DPX-L; Lunar Radiation, Madison, WI). Further, all individuals completed the neuropsychological performance tests and questionnaires (described below) approximately 1-h after eating breakfast. After three days of the WMEN, a 75 g oral glucose tolerance test was done to exclude individuals with diabetes mellitus [1]. Plasma samples that were drawn at − 10 and 0 min were averaged as a measure of fasting glucose.

2.3. Participants completed 6 neuropsychological measures

1. Iowa Gambling Task [2]: Participants were presented with four decks of cards (A, B, C, D) from which they could choose 100 cards, one card at a time, resulting in either gain or loss of money. Two decks (A & B) were disadvantageous, giving large rewards ($100) in addition to large penalties that resulted in an overall net loss, while the remaining two decks (C & D) were advantageous, giving small rewards ($50) but also small penalties resulting in an overall net gain. Cards were presented in 5 blocks of 20 cards each. Instructed to win as much money as possible, participants had to resist the immediate payoff of disadvantageous decks to achieve the long-term rewards of the advantageous decks. The net IGT score is determined by the number of cards chosen from disadvantageous decks subtracted from the number of cards chosen from advantageous decks. A positive score indicates overall advantageous choices and a negative score indicates overall disadvantageous choices (e.g. poorer performance).
2. Wisconsin Card Sorting Task [28]: is a computer based test to measure cognitive flexibility or set shifting, the ability to shift cognitive strategies flexibly in the face of changing environmental contingencies. It is a measure of executive function and provides an assessment of prefrontal cortical function, specifically dorsolateral prefrontal function [16]. The task consists of four stimulus cards and 128 response cards which depict figures (crosses, circles, triangles, or stars), colors (red, blue, green, or yellow), and numbers (1, 2, 3, or 4). The participant is instructed to match response cards to one of the four stimulus cards according to the three possible dimensions (figures, colors, or numbers). The participant receives feedback as to if they were right or wrong. After achieving 10 consecutive correct matches, the matching principle is changed without warning. The test ends once the participant matches 6 categories (10 correct card matches per category) or all 128 trials have been attempted. One main outcome variable is perseverative errors, the inability to shift or modify the response to a stimulus despite negative reinforcement. Perseverative errors occur when the participant continues to apply the previous matching paradigm even though a new matching paradigm has been identified. Higher scores represent poorer performance and more perseverative errors.
3. Stroop Word Color Task [11]: is a computer-based test used to measure response inhibition, the ability to attend to certain environmental stimuli while inhibiting others. The task consists of three separate timed trails and participants are instructed to respond as quickly as possible. The stimulus appears in the center of the computer screen and participants are instructed to provide a verbal response prior to pressing the space bar, which displays the next stimulus. During the first trial the participant is instructed to identify the color-words on the screen in black ink (RED, GREEN, BLUE). In the second trial the participant is instructed to identify the color strings of “XXXX” which are written in red, blue, or green ink, and in the final trial participants are instructed to identify the color of the ink in which the color word is written. In this final condition, all the stimuli were incongruent (e.g., the word “BLUE” printed in red letters). The “Stroop effect” which is a measure of interference, is calculated based on the difference in speed and accuracy (e.g.,