



Academic stress and personality interact to increase the neural response to high-calorie food cues



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ABSTRACT

Psychosocial stress is associated with an increased intake of palatable foods and weight gain in stress-reactive individuals. Personality traits have been shown to predict stress-reactivity. However, it is not known if personality traits influence brain activity in regions implicated in appetite control during psychosocial stress. The current study assessed whether Gray's Behavioural Inhibition System (BIS) scale, a measure of stress-reactivity, was related to the activity of brain regions implicated in appetite control during a stressful period. Twenty-two undergraduate students participated in a functional magnetic resonance imaging (fMRI) experiment once during a non-exam period and once during final exams in a counter-balanced order. In the scanner, they viewed food and scenery pictures. In the exam compared with the non-exam condition, BIS scores related to increased perceived stress and correlated with increased blood-oxygen-level dependent (BOLD) response to high-calorie food images in regions implicated in food reward and subjective value, such as the ventromedial prefrontal cortex, (vmPFC) and the amygdala. BIS scores negatively related to the functional connectivity between the vmPFC and the dorsolateral prefrontal cortex. The results demonstrate that the BIS trait influences stress reactivity. This is observed both as an increased activity in brain regions implicated in computing the value of food cues and decreased connectivity of these regions to prefrontal regions implicated in self-control. This suggests that the effects of real life stress on appetitive brain function and self-control is modulated by a personality trait. This may help to explain why stressful periods can lead to overeating in vulnerable individuals.

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1. Introduction

Psychosocial stress has been linked to unhealthy eating and weight gain, but only for some individuals. Differences in stress vulnerability can explain this heterogeneity (Adam & Epel, 2007; Dallman, 2010; Groesz et al., 2012; Newman, O'Connor, & Conner, 2007; Tomiyama, Dallman, & Epel, 2011). The purpose of this study was to study the neural correlates of individual differences in stress reactivity on the brain circuits implicated in food intake.

Individual reactivity to psychological stress can be assessed via perceived stress questionnaires or by measuring levels of plasma or

salivary cortisol, a reflection of hypothalamo-pituitary-adrenocortical (HPA) axis activation (McEwen et al., 2015). Greater stress reactivity by either measure is associated with increased high-calorie food consumption outside of the laboratory (Groesz et al., 2012; Newman et al., 2007) and greater body mass index (BMI) (Tomiyama et al., 2011). The relationship between stress and weight gain in longitudinal studies has been more variable suggesting that there might be additional factors influencing this relationship (Wardle, Chida, Gibson, Whitaker, & Steptoe, 2011).

Personality traits have been proposed as a reflection of the vulnerability to stress. Stress reactivity has been related to scores on the Behavioural Inhibition and Activation System (BIS/BAS) scales of Carver and White (Carver & White, 1994a). The BIS measure of the BIS/BAS scale, as detailed in Gray's Reinforcement Sensitivity Theory, assesses an individual's sensitivity to

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punishments and aversive events (Gray, 1987), and correlates with the similar measure of Neuroticism from the Five Factor Model of personality (Keiser & Ross, 2011). For example, in one longitudinal study, BIS scores moderated the relationship between violence exposure and post-traumatic stress disorder symptoms (Gudiño, 2013). High scores in BIS are also related to increased perceived stress levels at work (van der Linden, Taris, Beckers, & Kindt, 2007) and eating disturbances in college students (Chang, Kahle, Yu, & Hirsch, 2014).

Heightened reactivity to stressors can increase the probability of self-control failure during food choice, especially for calorically-dense food items (Epel, Lapidus, McEwen, & Brownell, 2001; Maier, Makwana, & Hare, 2015; Tomiyama et al., 2011). Such impulsive behavior may be mediated by increased food-reward sensitivity and decreased executive function, features that have been reported consistently in the obesity literature (Vainik, Dagher, Dubé, & Fellows, 2013). In fMRI experiments, heightened reward sensitivity may be reflected as increased BOLD within the appetitive brain network while viewing high-calorie food pictures. This set of limbic and striatal brain regions is involved in coding the incentive salience of foods and engaging appropriate motor responses. It includes the hippocampus, the amygdala, the insula, the ventral and dorsal striatum, and the orbitofrontal cortex (OFC) and adjacent vmPFC (Dagher, 2012). For example, acute stress led to increased BOLD in the right amygdala upon ingestion of a high-calorie milkshake (Rudenga, Sinha, & Small, 2012), and increased connectivity of the vmPFC to the amygdala and ventral striatum when choosing a high-calorie over a healthy food item (Maier et al., 2015). These effects correlated with individuals' stress reactivity (i.e. changes in cortisol levels). On the other hand, decreased executive function, possibly reflecting poor self-control, has been associated to reduced activity of the dorsolateral prefrontal cortex (dlPFC) and the dorsal anterior cingulate cortex (dACC) (Verdejo-Garcia, 2014). Perceived stress during an acute laboratory stressor led to reduced functional connectivity between dlPFC and vmPFC during a food choice task that demanded self-control (Maier et al., 2015). This was thought to reflect a stress-induced impairment in self-regulation during food choice.

Effects of chronic stress on food intake may also work through a similar mechanism. For example, one cross-sectional study has shown that chronic stress load predicted greater BOLD signal in the appetitive regions and reduced activity in lateral PFC while viewing appetizing food pictures (Tryon, Carter, Decant, & Laugero, 2013). However, cross-sectional studies cannot directly address the cause and effect relationship between stress and brain reactivity, and whether personality traits might modulate this relationship.

We studied undergraduate university students on two occasions: during the final exam period and during the school year, to measure within-subject effects of subacute stress on the brain mechanisms of appetite control. We hypothesized that the BIS score would predict individual differences in stress reactivity and that this would be reflected in the fMRI response to high-calorie food stimuli in appetitive and executive brain regions.

2. Materials and methods

2.1. Participants

22 right-handed undergraduate students [13 females, BMI = 22.64 (SD = ±1.9); age = 20.5 (SD = ±2.9)] participated in the experiments. Participants were recruited via advertisements posted on campus and on online school classifieds at the beginning of each semester. Exclusion criteria included moderate or severe depression (score > 13 measured by the 13-item Beck Depression Inventory (Beck A.T, 1972)), BMI > 27, current cigarette smoking,

history of substance abuse and current use of a central nervous system active medication, any history of neurological, psychiatric or eating disorders, being currently on a weight-reducing diet, and food restrictions due to allergies, intolerance or vegetarianism. The study was approved by the Montreal Neurological Institute Research Ethics Board. Each subject signed an approved informed consent form and received compensation for participation.

2.2. Experimental Design

Participants underwent fMRI on two occasions: once during the final exam period, and once during a non-exam period of the academic year. For the academic stress session, participants were required to have a minimum of three written final exams, two of which had to be scheduled within 5 days following the scanning day. The order of the scanning sessions was counterbalanced across participants and all of the scans started in the afternoon between 2:00 p.m. and 5:00 p.m. to account for diurnal changes in hormone levels and appetite. Scanning sessions were scheduled one month apart at the same time of day for each participant. Female participants were scanned during their luteal phase. On the scan days, participants were asked to refrain from exercise and caffeine, and were instructed not to eat anything for four hours prior to the experimental session. At arrival in the lab, they were given the same standard lunch (turkey and cheese sandwich, yogurt and fruit, 535 kcal). Participants were instructed to finish their meal, but three participants did not completely consume it in one session. Participants underwent fMRI two hours after eating. Blood samples were obtained immediately prior to and immediately following the fMRI scan to assess plasma levels of ghrelin (Fig. 1).

2.3. Psychological measures

Participants filled out the Three Factor Eating Questionnaire (TFEQ), a measure of eating styles (Stunkard & Messick, 1985), as well as the BIS/BAS Scale (Carver & White, 1994b). The BIS scores reported here are the mean of the two sessions. Scores showed good test-retest reliability across sessions ($r = 0.85$).

The self-report Perceived Stress Scale (PSS) was administered at the start of each session (Cohen, Kamarck, & Mermelstein, 1983). It is a measure of an individual's perception of chronic stress during the last month. In addition, acute stress and hunger levels were assessed using Visual Analog Scales (VAS) throughout the scanning session. On the VAS, we asked the participants "On a scale from 0 to 10 how hungry/stressed do you feel now?"

2.4. Hormone measurements

Saliva samples were collected to measure diurnal cortisol levels for 4 days per session (exam and non-exam). For each session, subjects were instructed to collect their own saliva samples using salivettes[®] (Sarstedt Inc., Rommelsdorf, Germany) on two weekdays and one weekend day in the week preceding the scan, as well on the day of the scan (Fig. 1). Sampling was conducted five times on each day: upon awakening, 30 min after awakening, at lunch, at dinner, and at bedtime (Supplemental Table 4). They were asked to refrigerate the samples immediately after collection, in containers provided. Levels of salivary cortisol were measured using a time-resolved fluorescence immunoassay (Kirschbaum & Hellhammer, 1994) and log-transformed. We measured cortisol awakening response (CAR) which is characterized by a sharp increase within the first 30 min after awakening. CAR provides a marker of current allostatic stress load (J. C. Pruessner et al., 1997). It can be calculated using the area under the curve (AUC) method that yields measures with respect to ground (AUC_g) and baseline (AUC_i) values (Jens C

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