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Obesity is associated with lack of inhibitory control and impaired heart rate variability reactivity and recovery in response to food stimuli

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ABSTRACT

Recent theories compare obesity with addiction in terms of lack of inhibitory control in both clinical populations. The present study hypothesized impaired inhibition in obese patients reflected both in executive functions and reduced vagal tone (indexed by a decrease in heart rate variability; HRV) in response to food stimuli. Twenty-four inpatients with obesity (19 women) and 37 controls (24 women) underwent ECG monitoring during baseline, food stimuli viewing, and a recovery phase. Tests and questionnaires assessing inhibitory control and psychopathological dispositions were also administered. As hypothesized, patients were characterized by deficits in all the tests measuring inhibitory capacities. Results also show greater HRV reduction and impaired HRV recovery in response to food stimuli in obese patients compared to controls. The drive to eat experienced by obese patients in the absence of caloric need may rely on impairments in inhibitory and vagal functioning. Results are discussed in terms of implications for therapy.

1. Introduction

The World Health Organization (WHO) defined overweight and obesity as “abnormal or excessive fat accumulation that presents a risk to health”. One of the most used measures of obesity is the body mass index (BMI). A person with a BMI of 30 or more is usually considered obese. A person with a BMI equal to or > 25 is considered overweight. Excess weight, especially obesity, is associated with severe medical conditions, seriously increased mortality, increased mood disorders, and decreased quality of life (Jia and Lubetkin, 2010). It does this through a variety of pathways, some as straight as the mechanical stress of carrying extra weight and some involving complex alterations in the hormonal system and metabolism (e.g., Mills et al., 2008). Nowadays, obesity is considered a normal response to modern society although this pathological condition has become a very serious health problem.

Although obesity is clearly related to a calorie intake exceeding calorie spending (Wood, 2006), and despite that many obese people know that they should eat less, the majority of them do not succeed in dieting (Wing and Hill, 2001; Wing and Phelan, 2005). Why are obese people so ineffective in losing weight? Why can't they resist extra-food intake?

Recently, it has been suggested that one possible contributory cause of the maintenance of unhealthy eating habits may rely on cognitive processes that make eating well difficult, such as increased food cue reactivity, weak executive functions, and attention bias (Jansen et al., 2015). Current studies on the neuropsychological profiles of patients suffering from obesity show recurring executive dysfunctions (Fagundo et al., 2012; Reinert et al., 2013). Among executive components, a deficit in inhibitory control is often reported in obese patients (Lavagnino et al., 2016). A lack of inhibitory control is revealed by impulsive behaviors such as responding before a given task is understood, answering before sufficient information is available, allowing attention to be captured by irrelevant stimuli (i.e. distractibility), or failing to correct obviously inappropriate responses (response inhibition). In obese patients, lack of inhibitory control results in automatic impulses to eat and uncontrolled eating (Hofmann et al., 2012) and overeating (Verdejo-García et al., 2008). Some authors have suggested that obesity is associated with less effective inhibitory control and proposed a causal relationship between lack of inhibition and overeating (Appelhans, 2009; Guerrieri et al., 2008; Houben and Jansen, 2014; Houben et al., 2014; Jasinska et al., 2012; Nederkoorn et al., 2006a, 2006b, 2006c; Nederkoorn et al., 2009). Also, a deficit in

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response inhibition has been linked to unsuccessful dieting in obese children (Kulendran et al., 2014; Nederkoorn et al., 2006b). Indeed, poor response inhibition has been suggested as one facet of impulsivity, although there are conflicting data as to which component of impulsivity is directly related to response inhibition (Dick et al., 2011). The specific components of impulsivity involved in overeating are still a matter of debate. In general, attentional impulsivity has been consistently associated with various measures of overeating. Less consistent but still positive associations have been found with motor impulsivity, particularly in patients with binge eating behavior. On the contrary the association between non-planning impulsivity and overeating seems weak (Meule, 2013).

All together, data on obesity and inhibitory control define a cognitive process in which low levels of inhibition lead to overeating, unhealthy behaviors, and weight gain, whereas increasing levels of inhibition may help people to lose and to maintain healthy behaviors and proper weight.

As posited by both the Polyvagal Theory (Porges, 2001, 2007) and the Neurovisceral Integration Model (Thayer and Lane, 2000), such inhibitory control is reflected in higher heart rate variability (HRV). HRV is the variability of the time periods between adjacent heart beats (Task Force, 1996) and is the result of the dynamic interplay between the fast acting parasympathetic nervous system and the relatively slower sympathetic nervous system. High tonic HRV is an index of robust parasympathetic control (i.e., via the vagus nerve) on the heart and has been associated with effective self-regulation as well as adaptive and flexible responses to meet various situational demands (e.g., Thayer et al., 2009). On the other hand, phasic HRV suppression represents the withdrawal of cardiac vagal control and the activation of defensive systems and has been conceptualized as a biomarker of stress (Park et al., 2014; Thayer et al., 1996). If phasic HRV suppression when performing a task or facing a stressor reflects the perception of the environment as unsafe and the consequent release (disinhibition) of the otherwise constantly inhibited threat response, phasic HRV enhancement reflects the exertion of successful self-regulatory effort (e.g., reappraisal) and the subsequent perception of the environment as safe (Porges, 2007; Thayer and Lane, 2009).

Alterations in autonomic nervous system function have been previously described in eating disorders (Faris et al., 2006). Indeed, individuals with eating disorders are prone to cardiovascular complications (Casiero and Frishman, 2006), and epidemiological studies have shown that obesity is an independent risk factor for cardiovascular morbidity and mortality (Lavie et al., 2009).

When resting HRV has been considered, results seem contradictory as higher HRV has been found in bulimia nervosa and anorexia compared to healthy controls (Peschel et al., 2016; Petretta et al., 1997). Compensating behaviors such as binging have been proposed as a possible explanation for such counterintuitive results. As expected, children and adults with obesity are characterized by reduced resting HRV, which can be increased by a successful weight reduction program (Mazurak et al., 2016; Poirier et al., 2003).

In the pathology of eating disorders, however, responses to stress seem to play a more important role compared to resting state autonomic function (e.g., Hilbert et al., 2011). For example, individuals who engage in binge eating appraise challenging situations as more stressful and show greater difficulties managing the emotional consequences of stress, in comparison to subjects without an eating disorder (Gluck, 2006). Previous studies on HRV responses to stress in individuals with eating disorder found inconsistent results. Some studies found HRV reduction during mental stressors such as the Stroop task, (e.g., Friederich et al., 2006), whereas others found no changes in HRV in response to this task in non-binge obese patients (e.g., Messerli-Bürgy et al., 2010). Most studies found delayed physiological recovery from such stressors in eating disorders (e.g., Vögele et al., 2009).

In our opinion, such inconsistencies may be possibly due to the fact that previous studies did not use idiosyncratically relevant stress stimuli. Accordingly, Cozzolino et al. (2014) found impaired cardiac autonomic regulation in response to a mixed meal in obese children and adolescents. Whereas for most people food represents a motivational stimulus, in obese patients it may become an important source of stress leading to reduced HRV and lack of inhibition (e.g., Thayer and Lane, 2000). To our knowledge, this is the first study to combine the use of vagally mediated HRV in response to food stimuli with neuropsychological and psychopathological indices of inhibitory capacities in obese patients and controls. We hypothesized that the drive to eat experienced by obese patients in the absence of true caloric need would be associated with impairments in inhibitory functions and vagal responses to food stimuli indexed by HRV. We administered behavioral and psychopathological scales, two ecologic tests assessing the inhibitory components of executive functions, and a food stimuli-viewing paradigm with simultaneous HRV assessment. First, we hypothesized to find differences between patients with obesity and controls in inhibitory functions. We expected such differences to be mirrored by crucial dissimilarities in self-rating scales assessing impulsivity and eating behaviors. Second, we hypothesized that obese patients would show stronger HRV reduction and impaired HRV recovery in response to food stimuli compared to controls.

2. Method

2.1. Participants

In order to determine the sample size, we first reviewed previous research on the same topic, finding an average $n = 32$ for studies using physiological assessment. For the analyses regarding neuropsychological and psychopathological indices of inhibitory capacities, the G*Power software (Faul et al., 2007) has been used to calculate the optimal sample size linked to a statistical power = 0.80. In this case, given a Cohen's $d = 1$, the power analysis for one-way ANOVA revealed a total sample size of 56 (α err prob = 0.02; Critical $F = 3.37$).

Twenty-five subjects admitted in a clinic specialized for treatments of eating disorder were enrolled in the study. After exclusion of one patient due to poor signal quality causing artifacts on the ECG, the final sample consisted of 24 patients (19 females and 5 males). The diagnosis of obesity was done by a psychiatrist expert in eating disorders, according to the International Classification of Diseases (ICD)-10: E66.0. Seventeen subjects had a familial pathological condition linked to weight (e.g. diabetes, overweight, obesity). Three patients regularly took medications for symptoms of anxiety and depression (alprazolam and quetiapine).

Forty-nine healthy control subjects were recruited from the general population by word of mouth and the use of flyers distributed in the university area (e.g., bookshops, cafeterias, and public library). After exclusion of 12 subjects (8 women) due to bad quality of the data (ECG electrodes fell off during the session leading to poor quality ECG traces or participants did not complete the questionnaires), the control group was composed of 37 participants (24 females and 13 males).

Exclusionary criteria, assessed during a pre-screening semi-structured interview, were: diagnosis of heart disease, substance abuse/dependence and pregnancy or childbirth within the last 12 months. All participants were Caucasian.

2.2. Procedure

The protocol was approved by the local Ethics Committee and all participants gave written informed consent. Subjects were asked to refrain from: a) drinking alcohol, tea, or coffee, b) eating, and c) strenuous exercise 2-h preceding the scheduled appointment. First, participants were evaluated for possible inclusion in the study by means of clinical assessment and the administration of a semi-structured interview, aimed at assessing weight history and eating behaviors. Then, participants were instrumented for cardiovascular monitoring