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Original article

A real world study on the genetic, cognitive and psychopathological differences of obese patients clustered according to eating behaviours

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ABSTRACT

Background: Considering that specific genetic profiles, psychopathological conditions and neurobiological systems underlie human behaviours, the phenotypic differentiation of obese patients according to eating behaviours should be investigated. The aim of this study was to classify obese patients according to their eating behaviours and to compare these clusters in regard to psychopathology, personality traits, neurocognitive patterns and genetic profiles.
Methods: A total of 201 obese outpatients seeking weight reduction treatment underwent a dietetic visit, psychological and psychiatric assessment and genotyping for SCL6A2 polymorphisms. Eating behaviours were clustered through two-step cluster analysis, and these clusters were subsequently compared.
Results: Two groups emerged: cluster 1 contained patients with predominantly prandial hyperphagia, social eating, an increased frequency of the long allele of the 5-HTTLPR and low scores in all tests; and cluster 2 included patients with more emotionally related eating behaviours (emotional eating, grazing, binge eating, night eating, post-dinner eating, craving for carbohydrates), dysfunctional personality traits, neurocognitive impairment, affective disorders and increased frequencies of the short (S) allele and the S/S genotype.
Conclusions: Aside from binge eating, dysfunctional eating behaviours were useful symptoms to identify two different phenotypes of obese patients from a comprehensive set of parameters (genetic, clinical, personality and neuropsychology) in this sample. Grazing and emotional eating were the most important predictors for classifying obese patients, followed by binge eating. This clustering overcomes the idea that 'binging' is the predominant altered eating behaviour, and could help physicians other than psychiatrists to identify whether an obese patient has an eating disorder. Finally, recognising different types of obesity may not only allow a more comprehensive understanding of this illness, but also make it possible to tailor patient-specific treatment pathways.

1. Introduction

Obesity is a multi-factorial and heterogeneous illness [1] that presents a complex and bidirectional relationship with several psychiatric disorders [2,3]. Eating behaviours are important features that can help to better define obesity and its comorbidities, and can also be associated with psychological domains [4]. However, there have been only few investigations to date

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concerning the psychopathological importance of most pathological eating behaviours other than binge eating [5]. Considering that specific genetic profiles, psychopathological conditions and neurobiological systems underlie human behaviours, the identification of different phenotypes of obese patients according to eating behaviours is important.

Eating disorders (EDs) show trait-related alterations in serotonin function, which might be linked to the gene encoding the serotonin transporter (SERT) [6]. The two functional polymorphisms of the *SERT* gene, STin2 and 5-HTTLPR, have also been associated with affective disorders, suicidal behaviour [7], response to antidepressants [8], substance dependence and abuse [9]. The *SERT* gene may also be associated with the pathophysiology of "binge eating", but it is not clear how changes in 5-HT function could influence eating behaviours in obese patients [10].

On the other hand, personality and psychopathological traits seem to play an important role as risk factors in the development and maintenance of overweight and obesity [3,11–12], and recent studies have also described a pattern of impairment in the cognitive flexibility and decision-making domains [13,14] of obese patients with and without EDs.

Previous cluster-analysis studies of EDs have yielded clinical subtypes for dietary restraint and negative affect dimensions; however, to our knowledge no studies have clarified the relationship between neurobiological and behavioural variables in obese patients [15,16]. This could be useful for identifying recurrent eating patterns that could differentiate subjects and characterise different behavioural phenotypes, which have clear implications from both nosological and therapeutic/management perspectives.

Based on the above, our aim was to identify different behavioural phenotypes of obese patients by classifying obese patients according to their eating behaviours and comparing the resulting clusters for psychopathological features, personality traits, neurocognitive patterns and genetic profiles (i.e., 5-HTTLPR and STin2 serotonin polymorphism). Our hypothesis was that eating behaviours could be related to different phenotypes of obese patients and that these phenotypes have specific psychological and neurobiological associations.

2. Methods

2.1. Participants

From March 2014 to July 2016, all obese patients (n = 250; 82 males and 168 females) admitted to a department of Internal Medicine in Southern Italy for weight loss treatment were given the opportunity to participate in this cross-sectional investigation. Patients were selected according to the following eligibility criteria: body mass index (BMI) $\geq 30\,kg/m^2$, aged 18–65 years, and the capacity to answer a self-reporting questionnaire and to understand the process in which they were involved. The exclusion criteria were: aged under 18 or over 65 years, neurological or other medical conditions that might affect cognitive functioning, pharmacological treatment with the potential to induce cognitive impairment, and pregnancy or childbirth over the previous 12 months. All participants were informed of the aim of the study, the research procedures and their complete anonymity in the processing of all data. Those who accepted signed an informed consent form before any procedure took place. The Ethical Committee of the Hospital (Azienda Ospedaliera Universitaria Mater Domini) approved the protocol in September 2013. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

2.2. Measures

This study consisted of three parts: (1) a visit with a dietician, (2) psychological assessment, and (3) blood sampling.

An experienced dietician initially conducted an in-depth assessment of participants' abnormal eating behaviours (namely grazing, emotional eating, craving for carbohydrates, sweet eating, post-dinner eating, night eating, binge eating, hyperphagia and social eating) during the previous 6 months with the aid of a checklist (Supplementary Table 1). Behaviours were considered to be present when all the related items were answered "yes" and if the behaviour had caused clinically significant impairment or distress. The dietician also performed an anthropometrical evaluation (waist circumference, height and weight) with the patients wearing light indoor clothing and no shoes, after which their BMI (kg/m^2) was calculated. Body composition was estimated by bioelectrical impedance.

A trained psychiatrist subsequently administered the Structured Clinical Interview for the DSM-IV (SCID-I) [17] to make a diagnosis of psychiatric comorbidity. During the psychological assessment, patients also completed the following psychometrical batteries, the results of which were used to compare the clusters:

2.2.1. Eating psychopathology

- *Eating Disorder Inventory-2 (EDI-2)* [18,19]. The EDI-2 is a self-report questionnaire that assesses the psychopathology of EDs. Cronbach's alpha was 0.91.
- *Binge Eating Scale (BES)* [20]. This self-administered test is widely used in research to measure binge eating severity in the non-purge binge-eating population or to determine whether potential research participants meet the inclusion criteria for binge eating. Total BES scores < 17, 17–27 and > 27 respectively indicate that the risk of an individual having Binge Eating Disorder (BED) is unlikely, possible and probable. Participants who scored > 27 were considered positive to the test in this study. Cronbach's alpha was 0.89.

2.2.2. Measurement of personality traits

- *Temperament and Character Inventory-revised (TCI-R)* [21]. This 240-item questionnaire is based on Cloninger's neurobiological personality theory, which assesses personality through four temperamental and three character dimensions. Cronbach's alpha in this study was 0.646.

2.2.3. Psychopathology measures

- *Barratt Impulsiveness Scale (BIS) version 11* [22]. The BIS is a 30-item self-report questionnaire that measures impulsivity through three subscales: attentional (cognitive instability and inattention), non-planning (intolerance of cognitive complexity and lack of self-control), and motor (lack of perseverance and motor impulsiveness). The BIS also yields a total score. Cronbach's alpha was 0.858.
- *Mood Disorder Questionnaire (MDQ)* [23]. The MDQ is used to determine the lifetime presence of bipolar features and consists of three questions. The first question evaluates bipolar symptoms through 13 dichotomous ("yes"/"no") items and the last two assess family history, past diagnoses and disease severity. Participants are considered positive if they simultaneously answer "yes" to at least 7 of the first 13 items in question