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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**A R T I C L E  I N F O**

Article history:
Received 21 August 2017
Received in revised form 30 October 2017
Accepted 21 November 2017
Available online xxx

Keywords:
Obesity
Eating behaviour
Cluster
Genetics
Cognitive functioning
Affective disorder

**A B S T R A C T**

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.

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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
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 identifi-
cation of different phenotypes of obese patients according to
eating behaviours is important.

Eating disorders (EDs) show trait-related alterations in seroto-
nin function, which might be linked to the gene encoding the
serotonin transporter (SERT) [6]. The two functional polymor-
phisms of the SERT gene, SThn2 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 pathophysiolo-
ogy 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
neurocognitive 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; how-
ever, 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 SThn2 serotonin polymorphism). Our hypothesis was that
eating behaviours could be related to different phenotypes of
obese patients and that these phenotypes have specific psycho-
logical 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) ≥ 30 kg/m2, 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/m2) was calculated. Body composition was estimated
by bioelectrical impedance.

A trained psychiatrist subsequently administered the Struct-
ured 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 psychomet-
rical 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 poten-
tial research participants meet the inclusion criteria for binge
eating. Total BES scores < 17, 17–27 and > 27 respectively indi-
cate 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 neurological
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 symp-
toms 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
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