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Identifying the relationship between biological, psychosocial and family markers associated with childhood obesity: Case-control "ANOBAS" study

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ABSTRACT

The recent increase in childhood obesity prevalence rates illustrates the extreme relevance of biological, psychosocial and familial factors implicated in body weight status, which at the moment remain unclear. The study aims to compare biological, psychosocial and familial markers between preadolescents with obesity and their non-overweight peers, and explore the relationship with psychiatric diagnosis on these markers. Both groups were composed of 40% of males with a mean age of 10 years, and no differences in socio-demographic variables were found between groups. No sex differences were found on bio/psycho/family markers. While 48% (n = 24) of the preadolescents with obesity presented a DSM-IV diagnosis (OGD), only 2% (n = 1) of the non-overweight peers (NG) met diagnostic criteria. Significant differences were found for all bio/psycho/family markers among obese preadolescents with the exceptions of cortisol, peptide YY and maternal state-anxiety and depression. The preadolescents with obesity without a diagnosis (OGND) presented greater levels of leptin than NG ($p = 0.01$). For psychosocial markers, statistically significant differences were found between groups in the majority of the variables ($p < 0.01$), with the exception of trait anxiety where a tendency towards significance was revealed ($p = 0.06$). For family markers, we found statistically significant differences in emotional over-involvement ($p = 0.01$), with NG mothers presenting lower scores than OGD and OGND. Include psychosocial and family factors in obesity intervention programs is necessary. Also, health professionals working with children with obesity must take care to assess the presence of a psychiatric diagnosis amongst this population.

1. Introduction

In line with global trends concerning adult obesity, childhood obesity also presents alarmingly high rates that call for more nuanced studies exploring the complex interactions between biological, psychosocial and environmental factors related to this elusive field of enquiry (Gurnani et al., 2015). Indeed, over the last three decades, numerous researchers from a wide range of disciplines have attempted to offer theoretical models to enhance our understanding of the multiple factors that influence childhood obesity (Davison and Birch, 2001; Neumark-Sztainer, 2005; McAllister et al., 2009; Tabacchi et al., 2007). Along these lines, Hofmann (2016) recently revised the concept of obesity, concluding that although it is accepted as a disease within the health care system, there is otherwise little agreement on whether it is a metabolic disease, a genetic disease, a neurochemical disease (Kopelman and Finer, 2001; Rankinen et al., 2002), a mental disorder (eating disorder or addictive disorder) (Heshka, and Allison, 2001; Devlin, 2007; Neumark-Sztainer et al., 2007; Volkow and O'Brien, 2007; Marcus and Wildes, 2009), or a combination of these concepts. Moreover, from an ecological perspective, the individual cannot be assessed without taking into consideration the relationship with their environment (Bronfenbrenner, 1979). This perspective provides a helpful starting point for linking biology, psychology, and social theories, and provides a useful grounding for a multifactorial approach.

In a pioneer publication by Bronfenbrenner (1979), the author describes a child's development as being affected by contexts that had a direct influence on the child (e.g., family, school) and others that affected the child indirectly (e.g., cultural tools/groups or social status). Bronfenbrenner and Ceci (1994) updated this model by including a biological perspective in order to account for the relationship between heritability and development, and the processes of a gene-environment interaction. Nearly a decade later, Davison and Birch (2001) adapted

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this model to explain the causes of childhood obesity and describe how children's weight status is associated with a set of environments (personal, family and community) in which that child is embedded. Harrison et al. (2011) continued to build upon this model by elaborating a *Six-Cs* model (cell, child, clan, community, country, and culture), which recognizes both environmental and hereditary influences and may be adapted to any stage of the child's development. Recently, Hemmingsson (2014) expanded this model by exploring the psychological interactions in childhood obesity that had previously been overlooked, such as the role psychological and emotional distress play in mediating the negative energy balance.

From a biological perspective, twin studies have indicated that between 30–70% of the variance in Body Mass Index (BMI) is due to genetic factors and is related to numerous physical and metabolic problems (Chesi and Grant, 2015). For example, the regulation of food consumption appears to be determined by different hormones such as adipokines, which have both metabolic and hedonic functions (Neri et al., 2015). Leptin stands out as one of the most frequently studied hormones and together with peptide YY and ghrelin, which regulate feeding behaviors and body weight homeostasis, is a key factor in maintaining the body's energy balance (Lustig, 2006). The location of leptin receptors in limbic structures suggests its direct role in the regulation of stress and emotional processes such as depression and anxiety symptoms (Roubos et al., 2012). However, their role specifically among children is unclear. One study showed that children with higher emotional problem score present higher leptin even when controlling for BMI (Kohlboeck et al., 2014). Additionally, it has been observed that obese children present higher cortisol levels (Donoho et al., 2011; Camargos et al., 2016) than their non-overweight peers, which would suggest inflammatory neuroendocrine changes from very early ages in response to psychosocial stress.

The hyperactivation of the hypothalamic-pituitary-adrenal (HPA) axis during stressful periods (e.g., being a victim of bullying) leads to exposure to greater levels of cortisol, an increase in appetite and a higher percentage of abdominal fat (Donoho et al., 2011). Previous research has found that among preadolescents with obesity, there is a direct, significant relationship between alterations in the HPA axis and anxiety and depression symptoms (Pervanidou et al., 2013). Furthermore, research suggests that people often change their eating behaviors when they experience stress, or are under persistent external interpersonal, financial or other strains. In fact, roughly 40% or more increase, and 40% or less decrease, caloric intake when under stress (Torres and Nowson, 2007).

From a psychological perspective, emotional eating can be considered a maladaptive coping strategy which serves to help the child suppress negative emotions, but which leads to chronic stress, appetite up-regulation, low-grade inflammation and reduced basal metabolism (Hemmingsson, 2014). Psychological distress in children with obesity is well documented, with greater body weight associated with more negative physical self-perception, lower self-esteem, anxiety, depression, poor social skills and weight related teasing (Van Geel et al., 2014; Rankin et al., 2016).

Childhood obesity may also be influenced by familial factors. Not only does the family play an important role in the child´s first stages of socialization, it is also an environment where the interaction of genetic and environmental factors can be found. The family therefore, may be responsible not only for the origin of many of the genetically transmitted characteristics of childhood obesity, but also aspects related to the child's psychosocial structure (Cervera et al., 2003). Numerous studies have argued that parental psychopathology (mainly anxiety and depression) coexist or even precede the pathology of their children with obesity (Vila et al., 2004; Rankin et al., 2016) and possibly contribute to the development and/or maintenance of the child's obesogenic environment. Furthermore, parental socioeconomic disadvantages may facilitate a conflictive family environment that can generate an ample range of personal frustrations, unmet emotional needs, hopelessness, depression, anxiety and general insecurity, that, in turn, may impact on the child's well-being (Hemmingsson, 2014).

To the best of our knowledge, no previous study has explored the relationship between biological, psychosocial and familial markers and childhood obesity, nor has the influence of the child´s psychiatric diagnosis on these markers been assessed. With this in mind, the underlying objective of the current study was to identify the biological, psychosocial and family markers which were associated with childhood obesity. For this, three specific aims were identified for this study: (1) To compare biological, psychosocial and familial markers in a sample of preadolescents with obesity (OG) with their non-overweight peers (NG), (2) Evaluate the frequency and type of psychiatric diagnoses amongst these children using a semi-structured diagnostic interview and to assess differences between the two groups of children, (3) To explore the influence of a psychiatric diagnosis on the bio/psycho/familial markers. The hypotheses are the following: a) The OG children will present greater impairment in the biomarkers than the NG children, b) The OG children will present greater rates of psychiatric diagnoses than the NG children, c) The presence of a psychiatric disorder in the OG children will be associated with greater impairment levels in biological, psychosocial and familial variables when compared to the OG children without a psychiatric disorder and the NG children.

2. Method

2.1. Participants and recruitment procedure

The ANOBAS study (PSI2011-23127) is a cross-sectional case-control study designed to assess early risk factors for childhood obesity, such as biological, environmental, familial and individual factors, in a representative sample of preadolescents aged 8 to 12 years from the region of Madrid, Spain. Details of the recruitment and sample selection procedure have been published elsewhere (Blanco et al., 2017). The total sample consisted of 100 preadolescents. Fifty of these children had a BMI > 97th percentile (obesity group, OG) according to the age- and sex-specific cut-off points proposed by Cole et al. (2000), were recruited from the "Daroca" Primary Health Center and none of the obese patients was seeking for treatment at that time.

The non-overweight group (NG) ($n = 50$) was made up of families recruited from thirteen primary schools (both public and state-subsidized) in Madrid through direct response flyers. Of the 160 families expressing an interest in participating, 50 families were matched by age, sex and socioeconomic status (SES) according to Hollingshead's index (Hollingshead, 1975) with children from the OG (1:1). For the NG, children who were currently overweight or underweight (above the 85th percentile or below the 15th percentile) were excluded. The sample size was calculated with the Power Calculator software (http://calculators.stat.ucla.edu /powercalc).

Clinical interviews (K-SADS-PL) were carried out by several trained psychologists (coauthor: M.B. and colleagues: M.P., S.S., T. L., T.M.) and the battery of questionnaires was completed by parents and children at Primary Health Center. A small number of the clinical interviews were carried out at the child's respective school. All children were supervised by the study coordinator (A.R.S.). Psychosocial reports and blood results were personally delivered to the parents of the children and to their pediatricians. Children presenting with a psychiatric diagnosis were referred to mental health services.

Nurses carried out anthropometric measurements of the preadolescents, with weight collected using a Seca digital (Type 799 and 769) weighing scale (kg). Nurses also recorded the child´s Tanner stage (Marshall and Tanner, 1969, 1970). Following clinical interviews, clinicians provided the families with an appointment for a blood draw, as well as additional information regarding the procedure. Blood draws took place at Niño Jesus Children´s Hospital (supervised by M.G.) and the samples were analyzed at Institute of Frio, Institute of Food Science, Technology and Nutrition (ICTAN) (supervised by A.M.).

The study received ethical approval by the Niño Jesus Children´s Hospital (Nº Ref. 0009/10, Central Committee of Research), Primary Care Commission (Ref. 11/12), and the corresponding University Committee (UAM) (CEI 27–673). Participation was voluntary and informed assent and consent was obtained by each participating family.

2.2. Measures

2.2.1. Evaluation of biological markers

Fasting venous blood samples were collected between 8–9 AM from patients and controls in EDTA-K3E Vacutainer (BD Biosciences) tubes. Plasma was obtained by centrifugation during 15 min. at 1300 *g* and 4 °C. Aliquots were frozen at -80 °C until analysis. Plasma cortisol was measured by a commercial immunoassay. Plasma levels of leptin and PYY were measured by multiplex immunobead assay and xMAP technology (Human Metabolic Hormone panel and Human Adipokine panel, respectively, from Millipore Corp.; and Magpix analytical test instrument, Luminex Corp., Austin, TX). The soluble leptin receptor was measured by ELISA (Abnova, Walnut, CA, USA).

Concentrations were determined by interpolation in a standard curve plotting fluorescence intensity versus standard concentrations. Two quality controls (high and low) were run within each assay. For PYY, undetectable values were found for a significant number of samples, which were not taken into account in the statistical analysis. The minimum detectable concentration for each analyze is as follows: leptin, 27 pg/mL; PYY, 28 pg/mL; leptin receptor, 0.04 ng/mL. All analyses were performed in different aliquots to avoid freeze-thawing repetitions.

2.2.2. Evaluation of psychosocial markers

The Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version (K-SADS-PL) (Kaufman et al., 1997; De la Peña et al., 2002) is a semi-structured diagnostic interview designed to assess current and lifetime psychopathology in preadolescents. Scores include 1 (absence of diagnosis), 2 (probably diagnosis) and 3 (definitive clinical diagnosis). For the current study, only definitive clinical diagnoses were used and the rest were coded as non-diagnoses.

The State-Trait Anxiety Inventory for Children (STAIC) (Spielberger et al., 1973) is composed of two separate anxiety scales, made up of 20 items each, with four alternatives (0 to 3) The state-anxiety (SA) scale measures transient levels of anxiety, whereas the trait-anxiety (TA) scale measures dispositional, or more stable, levels of anxiety. Higher scores show higher levels of anxiety (SA or/and TA). Cronbach´s alpha was 0.89 for state-anxiety and 0.85 for trait-anxiety in the Spanish version of the scale (Seisdedos, 1990).

The Child Depression Inventory (CDI) (Kovacs, 1992) consists of 27 items with three response options (0 to 2) and aims to measure the cognitive, affective and behavioral signs of depression. The total score of the scale ranges from 0 to 54. The internal reliability of the Spanish version was 0.69 (Davanzo et al., 2004).

The Perception of Teasing Scale (POTS-S) (Thompson et al., 1995) assesses the frequency of experiencing teasing among children and young adults. While the original scale consists of 11 items, the Spanish version includes 9 of these items and the same two subcales (weight related teasing (WRT) and competency teasing (CT), and has demonstrated good psychometric properties. Participants respond to each item on a Likert scale ranging from 1 to 5. For the Spanish version, the WRT scale (4 items) has a Cronbach's alpha of 0.86 and the CT scale (5 items) has an alpha of 0.76 (López-Guimerá et al., 2012).

2.2.3. Evaluation of familial markers

The State Trait Anxiety Inventory (STAI) (Seisdedos, 1990). This inventory is comprised of 40 items equally distributed in two subscales: State-Anxiety (SA) and Trait-Anxiety (TA), and scored on a four-point Likert scale (from 1 to 4). Higher scores indicate higher levels of anxiety. The Spanish validation reported a Cronbach's alpha of 0.90 for TA and 0.94 for SA (Guillén-Riquelme and Buela-Casal, 2011).

Beck Depression Inventory (BDI-II) (Beck et al., 1996). This self-report scale contains 21 items measuring somatic and cognitive-affective symptoms. For each item the respondent selects the response on a four-point Likert scale (from 0 to 3) that best describes how he/she felt during the past 14 days. Higher scores indicate higher levels of depression. The BDI-II has demonstrated high internal consistency in the Spanish validation ($\alpha = 0.87$) (Sanz et al., 2003).

Family Questionnaire (FQ) (Wiedemann et al., 2002). This questionnaire is composed of 20 items that evaluate family members' levels of Expressed Emotion (EE). Responses to items are given on a four-point Likert scale (from 1 to 4). The FQ is made up of two subscales: Critical Comments (CC) and Emotional Over-involvement (EOI). Higher scores indicate high EE. Acceptable reliability coefficients have been reported for the Spanish version ranging from 0.72 to 0.83 for each subscale (Sepulveda et al., 2014).

2.3. Statistical analysis

Data were analyzed using the statistical software package SPSS 21.0 for Windows. Data are presented with the median (ME) and interquartile range (IR), and percentages were used to describe the categorical variables. BMI standard deviation scores (BMI z-scores) were computed by comparing the subjects' body mass index with the ideal BMI of the general population of the same sex and age (Sobradillo et al., 2004). As each NG subject was matched to a specific OG case and the data had a non-parametric distribution, the comparison between two groups were analyzed using the Wilcoxon signed rank test. A series of one-way multiple analyses of covariance (ANCOVAs) were carried out to assess the effect of diagnoses group (OGD: Obesity group with diagnoses; OGND: Obesity group without a diagnosis; NG: Non-overweight group without a diagnosis) using the logarithmic values on the psychosocial (STAIC, CDI, POTS), familial markers (STAI, BDI, FQ) and biomarkers (cortisol, leptine, R-leptine, and PYY), to normalize the distributions, sex, age, BMI z-score, Tanner stage and SES were controlled for. Post-hoc Bonferroni comparisons were used. Only one case of NG presented a definitive psychiatric disorder (Generalized Anxiety Disorder) in the moment of evaluation and this case was not excluded from the initial analysis, but was excluded for the ANCOVA analyses (NG, n = 49).

3. Results

3.1. Anthropometric and sociodemographic characteristics of the sample

The anthropometric parameters characteristics are summarized in Table 1a and Table 1b. The OG had a significantly higher BMI z-scores and waist circumference (WC) compared with NG children. The OG children were 40% males with a median age of 10 years ($IR = 2$). No significant differences between groups were found in Tanner stage ($p > 0.05$). No sex differences for OG and NG were found on bio/psycho/familial markers ($p > 0.05$). We found significant differences ($p < 0.05$) between the mothers' BMI, as the OG mothers presented a median BMI of 24.95 ($IR = 5.90$) and the NG mothers presented a median BMI of 24.10 ($IR = 5.40$). However, no significant differences were identified between groups in terms of children´s sex and age, or parent´s socioeconomic status (SES; Table 1a). No anthropometric differences were found between OGD and OGND (Table 1b).

4. Differences for biomarkers

Significant differences were found between the two groups on leptin and leptin receptor levels ($p < 0.001$). The OG preadolescents had higher leptin levels and lower leptin receptor levels than NG preadolescents ($p < 0.001$). No differences were found between cortisol and PYY in obese and non-overweight preadolescents ($p > 0.05$; Table 2).

Table 1a
Anthropometric and Bio/Psychological/Familial markers of obese and non-overweigh group.

	NG (N = 50) Over all	OG (N = 50) Over all	t/χ^2
Age [Me (IR)]	10 (3)	10 (2)	1.18 n.s.
Male [N (%)]	20 (40)	20 (40)	0.01 n.s.
BMI z-score [Me (IR)]	0.21 (1.07)	2.85 (1.61)	**13.94*****
WC [Me (IR)]	60.75 (9)	85 (14.05)	**13.72*****
Tanner Stage [N (%)]			0.16 n.s.
II	30 (60)	28 (56)	
III	20 (40)	22 (44)	
Mother´s Age [Me (IR)]	43 (6)	42 (8)	−1.40 n.s.
Mother´s BMI [Me (IR)]	24.10 (5.40)	24.95 (5.90)	**2.41***
Socioeconomic status (SES) [N (%)]			1.07 n.s.
I ($_{lowest}$)	2 (4)	2 (4)	
II	6 (12)	6 (12)	
III	25 (50)	28 (56)	
IV	16 (32)	12 (24)	
V ($_{highest}$)	1 (2)	2 (4)	

Note. *$p < .05$, **$p < 0.01$, *** $p < 0.001$; n.s.= non-significant.
OG, Obesity group; NG, Non-overweight group; IR, Interquartil Range; BMI, Body Mass Index; BMI z-scores indicate BMI accounting for differences among growing preadolescents based on their age and sex Sobradillo et al. (2004); WC, Waist circumference.

Table 1b
Anthropometric and Bio/Psychological/Familial markers of OGD and OGND group.

	OGND (N = 25) Over all	OGD (N = 24) Over all	t/χ^2
Age [Me (IR)]	9 (2)	11 (1)	**2.73*****
Male [N (%)]	10 (40)	10 (40)	0.01 n.s.
BMI z-score [Me (IR)]	3.22(1.50)	2.90 (1.26)	0.63 n.s.
WC [Me (IR)]	85 (12.75)	85.50 (13.90)	0.93 n.s.
Mother´s Age [Me (IR)]	39 (10)	43 (8)	1.62 n.s.
Mother´s BMI [Me (IR)]	25.71 (5.90)	25.50 (6.20)	0.41 n.s.
Socioeconomic status (SES)[N (%)]			8.01 n.s.
I ($_{lowest}$)	2 (8)	0 (0)	
II	1 (4)	5 (20)	
III	14 (56)	14 (56)	
IV	8 (32)	4 (16)	
V ($_{highest}$)	0(0)	2 (8)	

Note. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$; n.s.= non-significant.
OGD, Obesity group with a psychiatric diagnosis; OGND, Obesity group without a psychiatric diagnosis; IR, Interquartil Range; BMI z-scores indicate Body Mass Index.

4.1. Differences for psychosocial markers

K-SADS-PL diagnoses at the evaluation are shown in Table 2. While 48% (n = 24) of the OG preadolescents presented a DSM-IV-TR diagnosis, only 2% (n = 1) of the NG preadolescents met diagnostic criteria. A total of 26% (n = 13) of the children in the OG presented a diagnosis of an anxiety disorder, followed by 10% (n = 5) with a mood disorder, 8% (n = 4) with attention deficit or disruptive behavior disorders and 4% (n = 2) with an eating disorder (see Table 2). Significant differences were found between the two groups on psychosocial markers. The OG preadolescents reported greater state and trait anxiety, higher levels of depression and greater frequency of both types of teasing ($p < 0.05$).

4.2. Differences for familial markers

When familial markers were analyzed, we found significant differences between the two groups. OG mothers reported significantly greater trait-anxiety (z = 2.64; $p = 0.01$), and a tendency towards significant differences in levels of state-anxiety (z = 1.87; $p = .06$) and depression (z = 1. 73; $p = 0.08$). We also found significant differences between both groups in expressed emotion ($p < 0.001$). In terms of critical comments, the OG presented higher scores ($Me = 21$; $IR = 9$) than the NG ($Me = 17$; $SD = 6.25$) and regarding emotional over-involvement, the OG again presented higher scores ($Me = 25$; $IR = 9$) than the NG ($M3 = 20$ $SD = 8$) (Table 2).

4.3. Differences for the Bio/Psycho/Familial markers according to the clinical diagnosis

In order to determine whether the presence of a psychiatric diagnosis was related to bio/psycho/familial markers, ANCOVA analyses were carried out, controlling for sex, age, BMI z-score, Tanner stage and SES. The independent variables were the following groups: OG children with a psychiatric diagnoses (OGD), OG children without a psychiatric diagnosis (OGND) and NG children. In regards to biomarkers, univariate tests confirmed that there were statistically significant differences between the OGND and NG on Leptin parameters ($p = 0.01$). For psychosocial markers, statistically significant differences were found between groups in nearly all variables ($p < 0.01$), with the exception of trait-anxiety where only a tendency towards significance ($p = 0.06$) was found. For familial markers, we found statistically significant differences in emotional over-involvement ($p = 0.01$), with NG mothers presenting lower scores ($Me = 3.02$; $IR = 0.36$) than OGD ($Me = 3.33$; $IR = 0.37$) and OGND ($Me = 3.17$; $SD = 0.30$). Results from the post hoc analyses, are summarized in Table 3.

5. Discussion

In order to strengthen both intervention and prevention efforts aimed at children with, or at risk of developing, obesity, it is necessary to increase our understanding of the risk factors associated with it (Oude-Luttikhuis et al., 2009). Along these lines, and to the best of our knowledge, this is the first study that has simultaneously investigated the biological, psychosocial and familial markers associated with childhood obesity (97th percentile), from an ecological framework. Furthermore, this study allows us to explore the role of psychiatric comorbidity on the variables that were assessed.

Our first hypothesis was partially confirmed. We found significant differences between the groups, with the obesity group presenting greater impairment on the majority of the biological, psychosocial and familial markers. In terms of *biological* markers, higher leptin and leptin receptor levels were found among children with obesity. This finding mirrors previous studies and is thought to be associated with the relationship between leptin and adipose tissue (Wang et al., 2013; Azab et al., 2014; Olza et al., 2014; Pires et al., 2014). In contrast, levels of cortisol and peptide YY were similar between two groups. We expected higher levels of cortisol in obese children because it is associated with activation of the HPA axis and exposure to stressful situations (Dockray et al., 2009; Donoho et al., 2011). Wilson and Sato (2014) pointed out that the relationship between stress and obesity in childhood is poorly understood, but suggested that in where family conflicts may lead to stress, which the children may manage by engaging in emotional eating. However, this relationship could not be proved with the present study. One possible explanation for the current findings could be based on the fact that the assessment was done early in the morning, as other studies have indicated that serum cortisol levels taken early in the morning may not reflect the production of cortisol during the entire 24 h, where many stressful situations could occur (Guzzetti et al., 2014). We also expected lower PYY levels due to its relationship with satiety and the fact that previous studies found that compared to normal-weight peers, children with obesity demonstrate a blunted postprandial response of PYY (Roth et al., 2005). However, varying results may be found regarding basal PYY values in obese children (Wojcicki, 2012). Our results may be better understood when taken into

Table 2
Bio/Psychosocial/Familial markers in NG and OG.

	NG (N = 50)		OG (N = 50)	Z/χ^2	OR	Confidence Interval (95%CI)
	Over all		Over all			
Biomarkers [Me (IR)]						
Cortisol(μg/dL)	8.15 (4.2)		8.10 (4.5)	0.93 n.s.	1.05	0.94-1.17
Leptine (ng/mL)	2.76 (3.8)		14.2(12.5)	7.49***	1.58***	**1.32-1.91**
R_Leptine (ng/mL)	21.30 (8.7)		12.8 (5.5)	−8.70***	0.75***	**0.67-0.83**
PYY (ng/mL)	9 (73.1)		9 (85.5)	1.41 n.s.	1.01	0.99-1.01
Psychosocial markers						
Clinical Diagnosis (K-SADS-PL) [N (%)]	1 (2%)		24 (48%)	31.57***	10.19***	**3.92-26.51**
Anxiety Disorder		1 (2%)	13 (26%)	14.94***		
Mood Disorder		0 (0%)	5 (10%)	5.26*		
Eating Disorder		0 (0%)	2 (4%)	2.04 n.s.		
Attention Deficit or Disruptive Behavior Disorder		0 (0%)	4 (8%)	4.16*		
State-Anxiety (STAIC-S)	24 (5)		25 (10)	2.39**	1.12**	**1.02-1.22**
Trait-Anxiety (STAIC-T)	30 (8.5)		32 (9)	2.49**	1.08**	**1.01-1.15**
Depression (CDI)	7 (4)		8 (10)	2.18*	1.10**	**1.01-1.19**
Weight Teasing (POTS-WRT)	4 (0)		6 (6)	5.66***	3.98***	**1.77-8.95**
Competency Teasing (POTS-CT)	5 (2)		7 (6)	4.05***	1.30***	**1.10-1.54**
Familial Markers [Me (IR)]						
State-Anxiety (SA)	16.5(13.5)		20 (17)	1.87	1.03	0.99-1.07
Trait-Anxiety (TA)	15 (13.25)		21 (13)	2.64**	1.05*	**1.01-1.10**
Depression (BDI-II)	6 (8.25)		8.50(12.3)	1.73	1.05	0.99-1.11
Family- Critical comments (FQ-CC)	17 (6.25)		21 (9)	2.96**	1.11**	**1.02-1.20**
Family- Over-involvement (FQ-EOI)	20 (8)		25 (9)	5.21***	1.23***	**1.11-1.36**

Note. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$. IR, Interquartile Range; OG, Obesity group; NG, Non-overweight group; OR, Odd Ratios; peptide YY. Statistically significant confidence intervals are highlighted in bold font. BMI z-scores indicate BMI accounting for differences among growing preadolescents based on their age and sex Sobradillo et al. (2004); WC, Waist circumference.

consideration with the suggestion that PYY levels are not related to satiety in children with obesity and therefore any observed differences between obese and normal-weight peers may not actually be contributing to the development of obesity (Lomenick et al., 2008). Future studies are needed to determine if it is possible that during preadolescence, biological markers are not as decisive, but become more evident as the child progresses through puberty.

Regarding *psychosocial* markers, the results of the semi-structured diagnostic interview supported the hypothesis that there would be a higher rate of psychiatric disorders diagnosed among the preadolescents with obesity as compared to the non-overweight group (48% vs. 2%). The most frequent diagnoses for this group were anxiety disorders, which mirrors the results of Vila et al. (2004), who using the K-SADS, found that among 155 children with obesity, 58% presented at least one DSM-IV diagnosis, with

Table 3
One-way ANCOVAs of the relationship between diagnoses K-SADS in obese group and non-overweight group and Biological, Psychosocial and Familial markers.

	Obesity Group		Non-overweight group				
	OGD (N = 24) Me (IR)	OGND (N = 25) Me (IR)	NG (N = 49) Me (IR)	Overall *p*-value	OGND vs OGD *p*	OGND vs NG *p*	OGD VS. NG *p*
Biomarkers							
Cortisol	2.11 (0.55)	2.07 (0.53)	2.10 (0.52)	0.32	0.90	0.52	1.00
Leptine	9.53 (0.82)	9.56 (0.87)	7.92 (1.28)	**0.01**	0.48	**0.01**	0.25
R_Leptine	2.44 (0.55)	2.59 (0.37)	3.05 (0.40)	0.16	1.00	0.21	0.26
PYY	2.19 (1.97)	2.25 (2.66)	2.19(2.21)	0.67	1.00	1.00	1.00
Psychosocial markers							
State- Anxiety (STAIC-S)	3.40 (0.40)	3.22 (0.28)	3.17 (0.20)	**0.01**	**0.02**	0.44	**0.01**
Trait- Anxiety (STAIC-T)	3.43 (0.32)	3.46 (0.33)	3.40 (0.30)	0.06	1.00	0.08	0.09
Depression (CDI)	2.39 (0.84)	1.94 (0.92)	1.94 (0.69)	**0.01**	0.21	0.07	**0.01**
Weight Teasing (POTS-WRT)	1.79 (0.80)	1.79 (1.10)	1.39 (0.01)	**0.01**	1.00	**0.01**	**0.01**
Competency Teasing (POTS-CT)	1.94 (0.96)	1.94 (0.69)	1.61 (0.34)	**0.01**	0.43	**0.04**	**0.01**
Familial markers							
State-Anxiety (SA)	3.09 (0.78)	2.89 (0.85)	2.83 (0.92)	0.08	0.57	0.54	0.07
Trait-Anxiety (TA)	3.13 (0.52)	3.04 (0.55)	2.77 (0.84)	0.09	0.31	1.00	0.14
Depression *(BDI-II)*	2.30 (1.26)	2.30 (1.45)	2.01 (1.32)	0.79	1.00	1.00	1.00
Family- Criticism (FQ-CC)	3.13 (0.50)	2.99 (0.34)	2.89 (0.35)	0.11	0.65	0.63	0.11
Family- Overinvolvement (FQ-EOI)	3.33 (0.37)	3.17 (0.30)	3.02 (0.36)	0.01	0.91	**0.03**	0.01

The median and interquartile range was presented as logarithm. Logarithm transformations were performed to be used in ANCOVA analyses. OGD, Obesity group with a psychiatric diagnosis; OGND, Obesity group without a psychiatric diagnosis; NG, Non-overweight group with non diagnoses.
[p] < 0.05 values or values with a tendency towards significance ($p =$ 0.05-0.10) for differences between the groups after adjusting for age, sex, BMI z-score, Tanner stage and socioeconomic status are highlighted in bold font.

anxiety disorders also being the most prevalent diagnoses. In terms of the preadolescents' depression and anxiety, our study found differences between the two groups, with the OG presenting both higher levels of self-reported anxiety and depression than the NG. These results were also confirmed by findings from the diagnostic interview. Prior studies have also revealed that anxiety and mood disorders are associated with obesity in childhood and adolescence, and have suggested that an increase in BMI may be associated with psychosocial distress, thereby increasing the risk for obesity and the maintenance of an obese state into adulthood (Vila et al., 2004; Pulgarón, 2013; Van Geel et al., 2014). The study also evaluated the presence of teasing with the OG presenting more frequency of WRT and CT than the NG group. In a review by Rankin et al. (2016), the authors reported that psychosocial problems such as stigma, teasing, and bullying in obese children and adolescents were associated with emotional and physical health consequences when compared with their non-overweight group.

Regarding *familial* markers, several studies have shown a direct relationship between parental socioeconomic status and child overweight/obesity (Shrewsbury and Wardle, 2008; Semmler et al., 2009). In our study these relationships were not analyzed as these variables had already been controlled for in the study design. However, previous research has also found maternal psychopathology to be a predictor of child obesity in their offspring (Benton et al., 2015). Consistent with these studies, we found that a noticeable percentage of the OG mothers were suffering from anxiety (state and trait) and also reported higher overall EE, and greater levels of EOI and CC than the NG mothers. Although this is the first study to explore the role of parental EE in this population, findings from previous studies suggest that these parents may respond to their children´s eating and weight with emotional over-involvement or criticism. For example, previous studies have reported that the parents of obese children tend to present either a lack of concern or excess control over their children's feeding behavior (Moens et al., 2007; Berge, 2009) and frequent critical comments regarding their child's weight and physical competence, all of which was associated with reports of low self-esteem and distress by their children (Berge, 2009).

Attending to the high rates of psychiatric disorders in this sample of children with obesity, the third objective of this study was to explore the influence of a psychiatric diagnosis on the *bio/psycho/familial* markers that were assessed. We hypothesized that the presence of a psychiatric disorder would be related to worse scores on the psychosocial, family and biomarkers, with preadolescents with obesity and a psychiatric diagnosis (OGD) presenting greater impairment than the other two groups. This hypothesis was partially supported. For *biological* markers, only leptin values presented differences between the groups. Children with obesity without a psychiatric disorder reported greater levels of leptin than their non-overweight peers. However, no differences were found between the leptin levels of children with obesity and a psychiatric disorder and children with obesity without a psychiatric disorder. In terms of *psychosocial* markers, differences were found between the three groups on state-anxiety, depression and teasing. The OGD group presented worse scores than the NG on all variables that were assessed, with the exception of trait-anxiety. However, the only difference observed between the OGD and OGND group was that children with obesity with a psychiatric diagnosis presented greater levels of state anxiety. Thus, it appears that the presence of a psychiatric disorder could be related to notable psychological distress, as well as teasing, in preadolescents with obesity. Finally, in regard to *familial* markers, only emotional over-involvement (FQ-EOI) showed significant differences between groups. The OGD had higher scores on FQ-EOI than the two other groups, which may be related to the fact that emotional over-involvement is thought to be a common reaction by a family member when their relative is ill (Koutra et al., 2014).

6. Limitations

The present study is not without its limitations. For example, the cortisol concentrations were measured in one moment in time and may not have detected the variations that could result from exposure to stressful situations. Furthermore, the family variables were assessed with self-report questionnaires and are therefore subject to the shortcoming inherent in this type of assessment, such as socially desirable responding.

Future studies are needed to continue to explore the variety of variables associated with the obesogenic environment. Finally, longitudinal studies would allow us to elucidate possible causal relationships related to the children´s psychological health and familial factors.

7. Conclusion

Given that these results are intended to add support to the ecological model of Harrison et al. (2011) and Hemmingsson (2014), it would be important to develop preventive programs for childhood obesity and long-term interventions that take into account the complex nature of the problem. These findings point to the importance of taking into consideration psychosocial and family factors in the development of childhood obesity intervention programs. Such programs could teach parents skills to improve communication and manage stress, and provide children with coping skills for dealing with teasing, which may improve their anxiety and depression symptoms. Furthermore, closely working with their pediatrician to assess for psychiatric diagnoses at early stages of obesity may also help to improve the course and outcome of the child's health. Finally, although these interventions may target certain aspects of this problem on an individual or familial level, larger scale prevention is needed in order to implement sustained solutions that aim to target the obesogenic environment as a whole (Fuemmeler et al., 2016).

One particular strength of the present study is that information on children's psychological distress was collected both via self-report questionnaires, filled out by the children themselves, and a semi-structured clinician administered interview with questions directed at the child and confirmed with the parent. In a review of prior studies assessing psychological correlates of childhood obesity, Puder and Munsch (2010) expressed the need for future studies to include both child and parent reports of children´s mental health, given the fact that prior studies have often included only one parent reports, which can affect the detection of more emotional symptoms. We also believe that a mention must be made regarding the sample recruitment. This endeavor was particularly difficult, as the study design required matching by sex, age and SES a sample of non-clinical patients with obesity (1:1) with a group of non-overweight preadolescents. Finally, a longitudinal design could provide a global perspective of maintaining obesity factors in each stage of child's development.

Declaration of Competing Interest

We wish to confirm that there are no known conflicts of interest associated with this publication.

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