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DEPARTMENT OF CLINICAL PSYCHOLOGY AND PSYCHOTHERAPY

Ph.D. THESIS SUMMARY

PSYCHOLOGICAL FACTORS IN OBESITY: ETIOPATHOGENETIC MECHANISMS

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2. All the Tables and Figures are numbered within the corresponding chapter or subchapter of the thesis.

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Keywords: obesity, obesity treatments, eating behavior, cognitive-behavioral treatment of obesity, etiopathogenetic factors.

CHAPTER I. THEORETICAL BACKGROUND

1.1. Obesity – a public health issue

Obesity is a very important public health issue that entangles major health problems and socioeconomic costs. Individuals with obesity are at risk for developing serious illnesses (National Institute for Health and Clinical Excellence, 2006), for being subject to stigma and discrimination (Carr & Friedman, 2005) and for having a reduced quality of life (Kushner & Foster, 2000). Also, obesity is clearly associated with increased mortality (Solomon & Manson, 1997) and brings important socioeconomic costs (National Institute of Diabetes and Digestive and Kidney Diseases, 2006).

1.1.1. Prevalence of obesity

The prevalence rate of obesity in adults is rapidly rising in most parts of the world. For example, in UK, 3–5% more adults are being classified as obese in national surveys repeated every 5 years. "Thus, the problems associated with weight gain are assuming ever-greater public health importance" (James, 2002).

1.1.2. Etiologies of obesity

Some genetic and environmental factors have been identified as being etiologic to obesity, but, similar to other common, complex diseases, the genetic determinants of interindividual variation in fat mass are likely to be multiple and interacting (Commuzie, 2002).Environmental factors can determine how an individual's genes are expressed. Factors pertaining to early development, ethnicity and family, diet composition and eating patterns and physical activity all contribute to obesity. Furthermore, some drugs and endocrine disorders can also cause or contribute to obesity. New research into etiologies of obesity is needed to provide new insights for understanding the disease and point to new and more effective treatments for obesity.

1.1.3. Medical consequences of obesity

Obesity is associated with increased risk for developing numerous medical problems such as type 2 diabetes, cardiovascular disease, hypertension, dyslipidemia, etc. (Pi-Sunyer, 2002). It is also associated with substantial increases in cardiovascular mortality and all-cause mortality, and, every year, obesity and its complications are responsible for 300.000 deaths (Khaodhiar & Blackburn, 2005). The reverse is also true - weight loss improves comorbidity risk factors. Research data shows that a 10% reduction in weight significantly reduces death and morbidity rates (Manson et al., 1995).

1.1.4. Obesity and quality of life

Besides the important medical consequences and risks of obesity, another important problem that afflicts obese patients' quality of life is stigma and discrimination. The evidence for stigma against obese individuals can be found in the jobs that are denied to these individuals, the disadvantages in education, the marginalization they face from health care professionals and the victimization from peers (Puhl & Brownell, 2008). All these can have a profound impact on quality of life of these individuals.

1.1.5. Socioeconomic implications and costs associated with obesity

By its implications and prevalence, obesity is a medical condition of primary interest to all international organizations involved in health. In 2006, within the WHO Ministerial Conference and the European Council the epidemic character of obesity was officially recognized, together with the remark that obesity affects the socioeconomic development of the countries that face this health problem. For example, in the U.S., it is estimated that the annual medical costs of obesity would be somewhere around 117 billion dollars, of which 61 billion

represent direct costs and 56 billion, represent indirect costs (National Institute of Diabetes and Digestive and Kidney Diseases, 2006).

1.2. Treatment of obesity

The clinical approach to the patient follows three steps used in the care of any patient with a multi-factorial, chronic disease: assessment, classification, and treatment (Calleo-Cross, Sharma & Aronne, 2005). Assessment includes determining the degree of obesity using BMI and waist circumference, and evaluating the overall health status of the patient. Information collected during the assessment is then used to classify the severity of obesity and related health problems. Conceptualizing obesity to the patient is also essential for the success of treatment - the patient has to understand the factors that contribute to the high weight and what he can change in order to reach a lower body weight.

The current treatments in the management of adult obesity are: (a) nutritional interventions, (b) behavioral therapy (c) pharmacotherapy, (d) gastric surgery (National Institutes of Health/National Heart, Lung, and Blood Institute, 1998). These treatment modalities are often combined into treatment packages.

1.2.1. Conceptualization.

Current clinical guidelines to obesity (National Institute for Health and Clinical Excellence, 2006), consider the energy imbalance as the fundamental cause of obesity, arguing that in the absence of increased food consumption and low energy expense, a person will not gain weight regardless of other genetic or physiological variables. The guide admits that this imbalance is not the ultimate cause of obesity, and that there are unknown causes of this imbalance. Currently it is considered that numerous behavioral, psychological, social and environmental factors, cause this alarming increase in obesity prevalence worldwide.

1.2.2. Assessment.

The clinical assessment of patients presenting with obesity is critical for understanding the etiology of the disorder in each, individual case and for establishing a strategy for how to respond to the disease through therapy. Recommendations regarding the assessment of the overweight or obese person refer to a multi-dimensional assessment including genetic, biological, social and psychological factors (National Institute for Health and Clinical Excellence, 2006). A particular attention is given to comorbidities and to identifying possible medical causes of obesity.

1.2.3. Treatment of obesity.

Evidence regarding the effectiveness of intervention strategies in the management of obesity shows that (National Institute for Health and Clinical Excellence, 2006):

- A hypo-caloric diet combined with exercise produces significant decreases in body weight.
- Combining a diet with exercise produces better results in weight management than diet alone.
- There is no evidence concerning the effectiveness of exercise alone (without diet).
- In specialized programs, behavior therapy combined with diet and / or exercise is effective in the short-term treatment of obesity.

1.2.3.1. Medical treatments

Pharmacotherapy. The pharmacological treatment of obesity has been associated with side effects, abuse, and relapse, leaving most practitioners reluctant to prescribe medications and most patients scared of medication to treat obesity, despite their preference for easy gained results (Aronne, 2002). Pharmacotherapy is indicated for individuals who have medical risks associated with obesity and / or have not responded to lifestyle modifications such as diet, exercise and behavioral change (Bray, 1998).

Surgery. Surgery is currently the only treatment that results in sustained (>10 years) and substantial (>15%) weight loss in obese patients (Pories & Beshay, 2002). The achieved weight loss has important positive effects on a large spectrum of medical and psychosocial consequences of obesity.

1.2.3.2. Psychosocial treatment of obesity

Behavior therapy is the treatment indicated in the list of American Psychological Associations' (APA) empirically validated treatments for obesity (David, 2006a). This approach to obesity rests on a few basic assumptions: (1) eating behaviors and exercise can influence weight; by appropriate changes in these, we can affect body weight; (2) eating and exercise are learned behaviors, thus, can be modified and new, more effective behaviors can be learned; and (3) in order to sustain long-term modification of these behaviors, changes in the environment that influences them is also necessary (Wing, 2008). Behavior therapy is probably the most disseminated treatment for obesity. Some of the key elements are self-monitoring, slowing the rate of eating, problem solving, stimulus control and cognitive restructuring. A behavioral program produces a weight loss of 5 to 10% on those who complete the program (Wing, 1998).

Nutrition. The most common diet strategies used are: balanced deficit diets, low calorie diets and very low calorie diets.

A balanced-deficit diet (BDD) reduces the daily calorie intake below the energy requirements, whilst also providing a healthy distribution of macronutrients (Fabricatore & Wadden, 2006). Patients can monitor the calorie intake, the fat intake, servings, or a combination of these (Fabricatore & Wadden, 2006).

Low-calorie diets they provide approximately 800–1200 calories / day (Bray, 1998). There are more options to low-calorie, too. These may include, low fat / high-carbohydrate, high-protein / low-carbohydrate, portion controlled, specific patterns, and restriction on one type of food or food groups, but they are not limited to this specific patterns (Bray, 1998). Low-calorie diets, combined with increased physical activity and behavior therapy provide the most successful therapy for weight loss and also weight maintenance (Fabricatore & Wadden, 2006).

Very low-calorie diets (VLCDs) are even more drastic in calorie count, providing 400 to 800 calories / day and are protein sparing modified fasts (American Dietetic Association, 2002). The VLCD is often delivered in a liquid form that is high in proteins and low in carbohydrates. These are diets promote rapid weight loss, but they should only be used in obesity and under close medical attention. They are contraindicated for patients with certain diseases as well as patients with psychological disturbances (Wadden, Itallie, & Blackburn, 1990). Patients must be informed of the risks associated with VLCDs and especially of the low chances for long-term maintenance weight achieved (Mannix et al., 2005).

Physical activity and exercise. Physical activity and exercise have a multitude of beneficial effects for the practicing person, including decreased body fat, lower blood pressure, an improved blood lipid profile, but also improved sleep, increased overall energy, increased ability to deal with stress and more (Mannix et al., 2005). A sedentary life is disadvantageous to all, but especially to obese individuals as obesity magnifies many of the medical consequences of such a lifestyle.

1.2.3.3. Treatment results

In a review of short and long-term effect on intentional weight loss on morbidity and mortality, Gregg (2008) draws the following conclusions: (1) there are strong and consistent evidence that relates intentional weight loss to a reduced incidence of diabetes. (2) intentional weight loss has also been consistently associated with improved blood pressure; (3) weight loss may reduce disability and improves mobility and functioning, especially in older individuals; (4) increasing evidence suggests that weight loss also reduces overall mortality and possibly

cardiovascular disease incidence. All these conclusions point to the fact that, although weight loss cannot always be maintained a lower weight brings about important health benefits and efforts to lose weight should not be abandoned even if a patients struggles with the maintenance of results.

1.2.3.4. Treatment in non-clinical settings

Obesity is most often addressed in non-clinical settings. These methods of treatment in non-clinical settings have been evaluated in terms of their effectiveness, with a series of studies conducted in the U.K., U.S.A. and some European countries, which have monitored their effectiveness (e.g. National Institute for Health and Clinical Excellence, 2006). The overall conclusion of this report is that weight management in non-clinical settings cannot be substantiated. The least we can say is that they do not produce negative effects. However, in light of these findings, public health campaigns should discourage overweight and obese individuals to try to loose weight in a non-clinical setting.

1.3. Problems and obstacles to treatment of obesity

The key problem in the treatment of obesity is the low maintenance of long-term results for all non-surgical treatments. On average, patients regain approximately one-third of their weight-loss obtained in treatment at 1-year follow-up and the rest over three years. Thus, behavioral effects of a non-surgical treatment are usually lost within three years (Cooper & Fairburn, 2001). Most clinicians and researchers attribute these relapses to patients' failure to adhere to the new behaviors strategies they learn in obesity treatment that promote weight loss, but, as soon as they are abandoned and the patients returns to "old habits" the weight returns as well. The most pressing matter in obesity treatment is the adaptation of current treatments of obesity in order to increase long term maintenance of results and facilitate long term weight management.

1.4. Addiction and obesity. A review of the literature.

In recent years, an interesting new hypothesis has arisen as to the question "why is obesity so difficult to treat"? Data from multiple research areas (eating disorders, obesity, animal studies, neurophysiology etc.) suggest that, in certain conditions, food can be addictive.

1.4.1. Defining addiction

The term has started to be used more and more often to refer to non-substance related disorders (Holden, 2001; Shaffer, 1999). Although there is still debate whether we should consider behaviors as addictions since a central element to the definition of addiction is "loss of control" (Potenza, 2006). If we narrow our approach to this central element, we can describe and tackle non-substance related conditions as addictions.

1.4.2. Food as a drug

One might be skeptic to the idea that food could be considered a drug, but studies from several areas of interest suggest this is a real possibility. One such study, investigating the effects of sugar on the brain function in animals, found that sugar releases opioids and dopamine, an effect similar to drugs, and, consequently, might have an addictive potential (Avena, Rada, Moise, & Hoebel, 2006). Neuroimaging studies have revealed that dopamine circuits play a role in the control of food intake (Trinko, Sears, Guarnieri, & DiLeone, 2007) as well as in drug response. Volkow and Wise (2005) argue that food and drugs have similar effects even though they may act through different mechanisms. Also palatability in itself is thought to be addictive, in the sense that it leads to an increased intake and meal size (Bobroff & Kisseleff, 1986).

1.4.3. Similarities between addiction and binge eating

Binge eating can be conceptualized as an addiction, based on the fact that there are many scientific and clinical pieces of evidence to support this point of view (Davis & Carter, 2009). Arguments supporting the addiction model of eating disorders stem primarily from observations

regarding similarities in the phenomenology of eating disorders and substance abuse, higher than expected rates of comorbidity, and clustering of these problems in families (Vandereycken, 1990).

1.4.4. Similarities between addiction and obesity

Behavioral factors of addiction and obesity. A first important argument for similarities between addiction and obesity, refers to the fact that eating and drug use involve learned habits and preferences that are maintained by powerful and repetitive rewards (Volkow & Wise, 2005) and furthermore, they persist and strengthen despite the threat of catastrophic consequences (Mazza & Marano, 2009). Second, certain dysfunctional eating patterns relevant to obesity develop in a similar fashion to that of the ingestive behavior of drugs (e.g. excessive eating), or have the same effect on behavior that abstinence from drug has (e.g. restraint eating). And finally, craving of drugs and food cravings are another strong similarity. A strong sense of craving, dismal and repeated failures at giving up the habit represents one of the main features that define drug addicts (Davis & Carter, 2009). This usually leads to repeated cycles of cessation and relapse. A similar pattern can be found in obese individuals with food cravings and repeated failures to diet (Davis & Carter, 2009).

Neurophysiology of addiction and obesity. There is strong evidence that most addictive drugs affect certain common pathways in the brain and also, there seem to be shared substrates for food and drug rewards in animals (Pelchat, 2002), leading us to believe that an important mechanism of addiction is the very way our brain works, and, in some cases, many substances and behaviors that are benign for most individuals can be addictive for others. When it comes to addiction, Volkow and Wise (2005) note that it seems as if the brain responds to a drug as it would respond to food under conditions of severe deprivation. This again argues for shared pathways and mechanisms. Furthermore, food intake is regulated by multiple signals, both central and peripheral. Drug intake regulation is much simpler as it is modulated mostly by the drug's central effects. Thus, regulation of food consumption is more complex (Volkow & Wise, 2005) and we can presume, much more difficult than regulation of drug consumption. Furthermore, imaging studies showed an inverse relationship of dopamine D2 receptors availability with BMI, suggesting a potential role for dopamine signaling in the development of obesity (Trinko et al., 2007).

Genetics of addiction and obesity. Addiction and obesity can be described as multi-factorial disorders and are thought to have significant genetic components. These disorders are considered to be under polygenetic control (Volkow & Wise, 2005), even though some studies found point mutations relevant for obesity (Friedman & Leibel, 1992) and for addiction (Volkow & Li, 2004).

Environmental factors of addiction and obesity. The environment often plays an important role in shaping our behavior. Different drugs establish different levels of compulsive behavior and so do different foods. Individuals in a high fat, high-carbohydrate environment are at considerably greater risk to overeat than those in a vegetarian environment (Volkow & Wise, 2005). Stressful life events can also influence and impact the development of an addiction or obesity. Johnson, Cohen, Kasen, & Brook (2002) found that childhood stress is associated with a higher risk for weight problems during adolescence or early adulthood. The same association was found between stressful life events during childhood and a higher risk of substance abuse and addiction (Dube et al., 2003).

1.4.5. Advantages for conceptualizing obesity as an addiction

Considering the similarities between obesity and addiction, one could assume that obesity may respond to the same types of intervention that have demonstrated effectiveness in treating substance abuse (Acosta et al., 2008). More and more researchers (and clinicians as well) are

beginning to recognize the potential value of studying these two disorders together. For example, Davis and Carter (2009) argue that researchers and clinicians would benefit from considering obesity as an addiction when investigating the causes of excessive consumption, when trying to understand its phenomenology and when considering the factors that increase individual vulnerability.

Obese patients could be presented with an addiction model. Thus, patients might cope better with their problem if they understand that they may be fighting a strong neurobiological drive to overeat in an environment that exploits these urges. It may also foster a therapeutic sense of empathy and improve therapeutic outcomes as patients might have more confidence in a treatment that understands overeating in these terms and promotes learning strategies to support life-long efforts to resist overeating and prevent relapse (Davis & Carter, 2009).

Conclusions. Obesity and addiction are both disorders that involve ingestive behavior. Both affect some individuals that are exposed to certain vulnerability factors, but not all. This remains an unsolved puzzle that emphasized the fact that we do not have yet a comprehensive understanding of the etiopathogeny of obesity or addiction. Excessive food or drug intake leads to changes in behavior and brain activity that makes the behavior grow more compulsive and difficult to control. Important advantages can be drawn from studying obesity and addiction together, both for theoretical developments and clinical strategies to be applied within the field of prevention and treatment of these disorders.

1.5. The need for a change in the clinical approach to obesity

Obesity has come to be considered as an exceedingly complex group of diseases that should be characterized as a syndrome (Atkinson, 2005). Although obesity has been considered a serious public health problem for a long time now, overall we cannot say that we have an effective treatment of obesity on the medium, long and very long-term (Monteiro & Victoria, 2005). Considering the low efficiency of obesity treatment, a plausible cause is that interventions are aimed at non-essential mechanisms of obesity. We believe more research is necessary to identify relevant and powerful etiological agents for obesity.

CHAPTER II. RESEARCH AIMS AND OVERALL METHODOLOGY

This research project had two main goals: (1) investigate psychological factors that have an etiopathogenetic role in the development and treatment of obesity (chapter 3.1); (2) develop a new psychological treatment of obesity that would target etiopathogenetic factors of obesity (chapters 3.2 and 3.3).

In light of the theoretical background, we started this research project with two key questions: "***Why are long-term results of obesity treatment so poor?"*** and "***Why do individuals with obesity have so much difficulty controlling their weight and eating behavior?"*** These two questions resulted in three research directions: (1) cognitive-behavioral factors relevant to obese individuals; (2) control of eating behavior; (3) obesity as a food addiction.

For the first research direction, we focused on some general and some specific cognitive-behavioral factors which we have investigated in three studies. *Study 1* was a metaanalytic investigation into how much does eating behavior change following obesity treatment and how does this change relate to weight loss. *Study 2* focused on a specific cognitive factor– irrational food beliefs (Osberg et al., 2008) - thought to influence weight loss and weight maintenance. *Study 3* focused on self perception of obese individuals as a specific (body self esteem) and general (self esteem, unconditional self acceptance) cognitive factor.

The second research direction concerns control of eating behavior. Some researchers were suggesting eating to be an automatic behavior (Cohen & Farley, 2008). We proceeded to

test this hypothesis in an experimental investigation (*study 4*). We designed two experiments to test three specific features of automaticity, as a means to diagnose automaticity (see Moors & De Houwer, 2006): uncontrollability, unconsciousness and efficiency.

The third research direction starts from an interesting hypothesis: obesity could be the result of a food addiction. *Study 5* investigated the relationship between food addiction symptoms and BMI, while also looking at some psychological characteristics of "food addicts".

After the investigation of these three research directions and the analysis of results they provide (chapter 3.2), the next step was to design a new treatment of obesity, that would incorporate techniques and strategies aiming to modify the psychological factors we found to be relevant to obesity (chapter 3.3). This program was tested in a randomized clinical trial (*study 6*) in order to (1) investigate its efficiency, (2) investigate the mechanisms of change and (3) provide evidence for our theory of change (the etiopathogenetic character of the psychological factors). The new treatment was tested against the current standard psychosocial treatment of obesity (behavioral weight loss therapy). Finally, in chapter 4 findings were summarized and general conclusions were drawn.

CHAPTER III. ORIGINAL RESEARCH

3.1. PSYCHOLOGICAL FACTORS IN OBESITY

3.1.1. COGNITIVE BEHAVIORAL FACTORS RELEVANT TO OBESITY

Study 1. Effect of Obesity Treatments on Eating Behavior: Psychosocial Interventions versus Surgical Interventions. A Systematic Review[*]

Introduction

This review investigated the effect that obesity treatment has on eating behavior and how this relates to weight loss following treatment. How much does eating behavior change following treatment of obesity? How does this change in eating behavior relate to change in weight status? In order to achieve these objectives we searched for relevant studies in the literature – studies that investigated treatment of obesity and provided result in terms of weight loss and eating behavior change. For the final analysis 18 articles were selected and grouped into two categories: psychosocial and surgical interventions.

Method

Selection of studies. We selected studies that: (a) assessed both weight loss and eating behavior before and after treatment, (b) had as a main objective the treatment of obesity and (c) included adult participants. Potential articles were identified in PsycINFO and PubMed using keywords: "obesity", "treatment", "eating behavior" (and variations). After the initial selection steps, 43 articles were retrieved for full text examination. Articles were classified in two categories: *psychosocial interventions* versus *surgical interventions*. Eleven studies in the psychosocial interventions category and seven articles in the surgery category complied with all inclusion and exclusion criteria and were coded and analyzed for the present metaanalysis.

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[*] This study was published:
Moldovan, A.R. & David, D. (2011). Effect of Obesity Treatments on Eating Behavior: Psychosocial Interventions versus Surgical Interventions. A Systematic Review. *Eating Behaviors*, *12* (3), 161-167.
The authors have contributed to the manuscript as follows:

- Moldovan, A.R. : data collection, data analyses and writing the manuscript
- David, D.: study design.

Derivation of effect sizes. Effect sizes were calculated according to published procedures (Hunter & Schmidt, 1990). Effect sizes (Glass's *d*) for two outcomes were computed: weight measured by BMI and eating behavior (EB) for each category of the selected articles – psychosocial and surgical treatments. Mean differences between pretreatment and post treatment values of targeted outcomes were calculated for each study and then divided by the standard deviation of the pretreatment values. For psychosocial interventions we also had follow-up data so mean differences between pretreatment and follow-up were computed. To estimate the overall effect of interventions, the 95% confidence interval (CI) for the difference between pretreatment and post treatment (or follow-up) values was calculated and then compared to zero. If the 95% CI included zero, there would be no significant effect of the intervention. We also conducted fail-safe analysis for all outcomes, in each study category, to check for the reliability of results.

Results

Psychosocial interventions. Post-treatment analysis.

A total of 39 effect sizes were derived from the 11 studies (1284 subjects) in the psychosocial interventions category for the EB outcome. A large and significant mean effect size of $d = .73$ (CI = .66, .90) was found for EB.

For weight outcome (BMI), 17 effect sizes were derived from the 11 studies (1284 subjects). The mean effect size was significant, but small $d = .32$ (CI = .28, .36). Psychosocial interventions for obesity yield modest weight losses, of about 10 % of the initial body weight. However, we have to mention that this effect, although small statistically, clinically and normatively is an important one, as studies indicate that a 10 % loss in weight is associated with important gains in health, as long as this outcome can be maintained (National Institute for Health and Clinical Excellence, 2006).

We also conducted a fail safe analysis: for EB outcome, the fail-safe number is 254.7 and for BMI, the fail-safe number is 37.2.

Psychosocial interventions. Follow up Analysis.

We computed 29 effect sizes for EB and 9 effect sizes for BMI at follow-up. Psychosocial interventions have a significant, medium mean effect size on EB($d = .47$, CI = (.45, .49)) and a small effect size on BMI ($d = .37$, CI = .18, .56)). *Fail-safe analysis* for follow-up show that 107 studies on EB showing no results, would be necessary to invalidate our results, while 24 studies on BMI, showing no effect of psychosocial interventions, would dismiss our results.

Surgical interventions.

For the surgical interventions studies a total of 18 effect sizes (853 subjects) were derived from the 7 studies for EB outcome. The overall effect size for EB following surgical intervention is 1.84 (CI = 1.26, 2.42), very large and significant. For BMI outcome 7 effect sizes (853 subjects) were derived to calculate the mean effect size. The overall mean effect size for BMI was 1.40 (CI = 1.25, 1.65) indicating a large and significant effect size. The *fail-safe analysis* yielded 154.8 for EB outcome, so 155 studies necessary to invalidate our results. Finally, for BMI outcome 109 studies showing no results would change our results. Table 4 summarizes the significant results of this metaanalysis.

Table 4
Summary of results

	Lifestyle Interventions		Surgical Interventions
	Post-treatment	Follow up	
Eating behavior	.73 (CI = .66, .90)	.47 CI = (.45, .49)	1.84 (CI = 1.26, 2.42)

Weight	.32 (CI = .28, .36)	.37 (CI = .18, .56)	1.40 (CI = 1.25, 1.65)

Discussion

Our results show that EB changes significantly after treatment of obesity. The change is greater for surgical treatment than for psychosocial ones. The change in eating EB seems to correspond to a change in BMI if we look at surgical treatments, but, if we look at psychosocial treatments, there seems to be a lower correspondence between the two outcomes. The relationship between EB and BMI should be clarified in future studies. If EB is an etiopathogenetic factor for weight (and obesity), then interventions should seek to obtain more significant changes in EB and, thus, lead to a more significant weight loss (even if not at the same fast pace as surgery does). If EB is not an etiopathogenetic factor of obesity, but a symptom, alongside a high BMI, then efforts to obtain better and longer lasting results for obesity should look elsewhere.

Study 2. Irrational food beliefs and eating behavior in obese individuals

Introduction

This study aims to explore the potential utility of irrational food beliefs for weight management, their relationship with dysfunctional eating behavior and their diagnostic utility in identifying obese individuals at a higher risk for poor weight management.

Method

Participants. Participants were 381 individuals (68 % women, 32 % men) from the general population of Cluj-Napoca, Romania, aged 18 to 66 (mean age 36). They were classified based on their BMI into for categories: underweight (53), normal weight (206), overweight (62) and obese (59).

Measures. Participants were asked to provide weight and height (to compute BMI) and complete the following measures: Three Factors Eating Questionnaire Revised 21 (Cappeleri et al, 2009); Irrational Food Beliefs Scale (Osberg et al., 2008); General Attitudes and Beliefs Scale – short version (Lindner, Kirkby, Wertheim, & Birch, 2007).

Results

There is a significant, although small correlation ($r(379)=.226$, $p=.000$) between BMI and Irrational Food Beliefs (IFB), and a nonsignificant correlation between BMI and Rational Food Beliefs (RFB). BMI and general irrational beliefs did not correlate significantly ($r(379) = .010$, $p = .854$ for Irrationality subscale, and $r(379) = .030$, $p = .562$ for Rationality subscale). IFB and general irrational beliefs correlate significantly, but the correlation is small ($r(379) = .283$, $p<.000$) showing that the two concepts are linked, but they do not measure the same concept.

Our next objective was to see if IFB would distinguish the obese individuals from the other weight categories. The one-way ANOVA, $F(3,377) = 8.452$, $p = .000$, demonstrated that IFB differentiate between the four groups (underweight, normal weight, overweight and obese). Post hoc analyses, using Scheffe post hoc criterion for significance, indicated that the average score was significantly higher in the obese group as compared to the normal weight group (MD[†] = 9.28, SE = 2.56, $p=.000$, $d = .63$) and to the underweight group (MD = 10.89, SE = 2.56, $p=.000$, $d = .72$).

[†] MD = Mean Difference between groups.

Then we wanted to see if IFBS discriminates obese individuals from normal weight individuals and overweight ones. Analysis was performed for the discriminative ability of the IFB subscale between obese and normal weight individuals. Data revealed that IFBS discriminates obese individuals from normal weight ones, but only with a low accuracy (AUC=.68).

Our next objective was to see how irrational food beliefs relate to dysfunctional eating behavior. IFB correlate with emotional eating ($r(379)=.474$, $p=.000$) and uncontrolled eating ($r(379)=.568$, $p = .000$), but not with cognitive restraint ($r(379)= -.098$, $p = .224$). Mediation analysis revealed that relationship between irrational food beliefs and BMI is mediated completely by emotional eating.

Discussion

Results indicate that IFB are a significant factor for obesity. Although the correlation and strength of association have small values, we have to consider that the criterion we are talking about is one influenced by many factors (BMI is largely genetically determined, and its heritability can be as high as 80% in Western populations and it is also influenced by gender (Schousboe et al., 2003), social economic status (Dykes et al., 2003) and other factors). For a specific psychological variable, to be able to predict BMI, in a general population (not a treatment seeking one), is not something to easily disregard. IFB also seem to be a predictor for emotional eating, and its influence on BMI seems to be of a greater magnitude (explaining about 15 % of the variation in BMI).

IFB are also linked to general irrational beliefs, supporting the authors' claims that the construct was inspired by Ellis's concept of irrationality. However, general irrational beliefs have no predictive value for BMI. Obese individuals are not more irrational in general, but they are more irrational when it comes to food. This is what interventions need to tackle. Concerning the possible diagnostic use of the Irrational Food Beliefs Scale, data currently indicates low accuracy of the scale, but it could be improved by a further examination of the scale and refinement of items that could bring better sensitivity and especially a higher specificity.

Our results show that IFB are relevant and could be useful to the study and clinical practice of obesity. Further research has to establish better their role in weight maintenance and also their relationship with other factors relevant to obesity. To conclude, cognitive-behavioral factors offer potential benefits for the assessment and therapy of obese individuals, and further studies should investigate if changing such factors in the treatment of obesity could improve long-term results for these patients.

Study 3. Self-esteem, body self-esteem and unconditional self-acceptance. Relations with body mass and depression.

Introduction

Our primary objective for this study was to investigate which measure of self-perception (self-esteem, unconditional self-acceptance and body self-esteem) is best predicted by high body weight. Do obese individuals have just lower body self-esteem or can we find problems with general self-esteem and self-acceptance as BMI increases? As secondary objectives, we were interested to see any mediating relationships between the variables that will prove to be significantly predicted by body mass. Another objective was to see if any of these indicators mediates the relationship between high BMI and depression.

Method

Participants. A total of 350 individuals, aged 18 to 65 (mean age 28) took part in this study. 73 % were women, and 27 % men. 220 were normal weight (BMI below 25) and 131 were overweight and obese (BMI>25).

Measures. Participants were asked to provide weight and height (to compute BMI) and complete the following measures: Body self-esteem scale (Franzoi & Shields, 1984); Unconditional Self-acceptance Questionnaire (Chamberlain & Haaga, 2007); Rosenberg Self-esteem Scale (Rosenberg, 2007); Beck Depression Inventory II.

Results

Results show a negative, small but significant correlation between BMI and unconditional self-acceptance ($r(348)=-.187$, $p=.000$), a nonsignificant correlation between self-esteem and BMI ($r(348)=-.024$, $p=.660$), and a negative, large and significant correlation between body self-esteem and BMI ($r(348)=-.586$, $p=.000$). It seems that the higher the BMI, the lower the body self-esteem, and to some extent, the lower unconditional self-acceptance is.

Our secondary objective was to see any mediating relationships between the variables that prove to be in a significant relationship with body mass, on the one hand and depression on the other hand. The only significant result of the mediation analyses showed a complete and significant mediation effect (Sobel test = 2.96, $p = .000$) of body self-esteem as a mediator between BMI and depression.

Discussion

Results indicate that a high BMI is associated with low body self-esteem and low levels of unconditional self-acceptance, in a sample of general population. We did not find a significant relationship between BMI and self-esteem. Our results show body self-esteem and unconditional self-acceptance to be relevant to body weight. Studies investigating the efficiency of obesity treatment should try to establish if these might be mechanisms of change. Future studies should also investigate the potential causal role of body self-esteem and unconditional self-acceptance on the development or maintenance of obesity.

3.1.2. EATING AS AN AUTOMATIC BEHAVIOR

Study 4. Features of automaticy in eating behavior

Introduction

Our main objective for this study is to investigate eating behavior in an experimental design and see if it meets the characteristics of automaticity. We decided to test three features of automaticity described by Moors & De Houwer (2006):

(1) *Uncontrollability*. There are more situations where we can consider a behavior to be uncontrollable in terms of goal: (a) the goal is absent, and the effect may be absent; (b) the goal is present, but the effect is absent; (c) the effect is present, but the goal is not the cause of the effect. We expect the two latter situations can be found in relation to eating behavior.

(2) Unconsciousness. Consciousness or unconsciousness may apply to: (a) the stimulus input that evokes the response; (b) the output of a process; (c) the process itself; (d) the consequences of a process. It is important to make explicit what one considers to be unconscious when one uses the term (Moors & De Houwer, 2006). We believe that eating is an unconscious process in terms of stimulus that evokes the response (people generally do not realize that certain environmental cues influences them and will justify their behavior in terms of conscious goals and intentions) and also the process itself (once started, eating continues efficiently without conscious control).

(3) (Non)efficiency. A behavior is efficient when it consumes little or no processing resources (Moors & De Houwer, 2006). An automatic behavior will be efficient when allowed to continue naturally and nonefficient when other processes are involved (inhibition) – i.e. effort is required to refrain from eating when food is present (Cohen & Farley, 2008).

[‡]Experiment 1: features of uncontrollability and unconsciousness in eating behavior.

The main objective of the first experiment was to test the characteristic of uncontrollability and unconsciousness. If eating behavior is to fulfill the characteristic of uncontrollability in terms of goal independence then we have to find a set of conditions that is sufficient for the occurrence of the process (eating) and that the goal of engaging in it is absent from the set, or, if the goal is present it is not necessary.

1. Consumption of food will be prompted by the behavior of an accomplice.
2. Consumption of food will be predicted by individuals' self-control (the lower the self-control the higher the probability of consumption).
3. Consumption of food will be predicted by individuals' impulsivity (the higher the self-control the higher the probability of consumption).
4. Consumption of food will be predicted by individuals' eating behavior (the more dysfunctional their eating behavior, the higher the probability of consumption).
5. Consumption of food will be predicted by individuals' food addiction symptoms (the more symptoms they have, the higher the probability of consumption).

This is the set of conditions that we expect will be sufficient to provoke the process of eating, and we expect that subjects' intention to eat or the instruction they will receive (to eat or not) will have no impact on their eating behavior.

If eating behavior is unconscious in terms of stimulus input that evokes the response then:

1. Individuals will justify their behavior in terms of goal.
2. Individuals will offer alternative justifications to explain the lack of congruence between their goal and their behavior.
3. Individuals will be unaware of stimuli / factors that make them eat.

Apart from these main objectives, we wanted to see if obese individuals would behave different from normal weight individuals. Would they consume more often than normal weight individuals would?

Method

Participants were 100 first year psychology students (aged 18 to 37, mean age 21) from the Babeş-Bolyai University, Romania. BMI varied between 16.6 and 40.2 with a mean BMI of 22.8. Only 18 individuals could be classified as obese. Most were female (81), with only a small sample of male participants (19).

Experimenters. The experimental procedure was carried out by MA level students. For each participant there was a team of four experimenters: one as the experimenter who gave instructions, an accomplice who acted as a partner for the participant, and two more accomplices, who were observers and acted as another team that was participating in the study.

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The authors have contributed to the manuscript as follows:

- Moldovan, A.R. : study design, data collection, data analyses and writing the manuscript
- David, D.: study design.

Procedure and design. Participants were asked to take part in a study that aims to identify cognitive and collaborative strategies used to solve a cognitive task by two persons who have never met before. The subject was instructed that he was randomly assigned to a partner and they would have 30 minutes to complete the task (solving a puzzle). The subject was then taken into the study room and introduced to his partner (accomplice). There was food on the table where the task had to be solved. There were two conditions: (1) with interdiction - participant is invited to eat, but only after finishing the task, as eating might interfere with task resolution; (2) without interdiction – participant is invited to eat if he/she wishes to. As participants were left alone, the accomplice and the subject started solving the puzzle. Four minutes into the task the "accomplice" starts to eat. The accomplice and observers secretly recorded the participants' behavior. The *independent variables* were (1) the interdiction to eat (received and not); (2) participants' self-reported goal (to eat or not to eat). The *dependent variable* was *consumption* - if subjects ate or not (dichotomous variable).

Measures. After the completion of the task, participants were informed about the true objectives of the experiment and asked to complete the following measures: Three Factors Eating Questionnaire Revised 21 (Cappeleri et al., 2009); Self-control Scale (Grasmick, Tittle, Bursik & Arneklev, 1993); The Barratt Impulsiveness Scale, Version 11 (Patton, Stanford, & Barratt, 1995); Yale Food Addiction Scale (Gearhardt, Corbin, & Brownell, 2009). Also participants were asked to provide height and weight (to compute BMI) and complete a questionnaire assessing phenomenological aspects of the experiment. The questionnaire had several questions assessing: participants' goal, participants' reasons to eat or not; if they knew or suspected the real goal of the study prior to being informed; if they took the interdiction seriously, or if they disregarded it.

Results

Our first objective was to investigate the feature of goal independence as a mark of uncontrollability. We expected that consumption of food cannot be predicted by the subjects' intention to eat or to refrain themselves (their self-reported goal). Thus we computed Pearson chi-square test (χ^2) and Phi (Φ) to test for the association between subjects' self-reported goal (to eat or not to eat) and their behavior (they consumed or not). Results show no significant relationship between the two nominal variables ($\chi^2(3)= 1.941$, $p = .523$; $\Phi = -.153$, $p = .164$). Then we looked at the instruction subjects were given. Results showed, again, no association between the given instruction and eating behavior ($\chi^2(3) = .653$, $p = .419$; $\Phi = .083$, $p = .419$).

Then we looked at associations between consumption and the psychological variables we measured to check our hypotheses that these would better predict consumption. We computed logistic regression to correlate the nominal variable (consumption – yes or no) with the numerical variables (dysfunctional eating behaviors, self-control, impulsivity, symptoms of food addiction). There was only one variable that predicted consumption – eating behavior, specifically cognitive restraint (B=-.109, $p = .042$). Cognitive restraint predicted almost equally both consumption (accuracy 62.2 %) and non-consumption (61.9%). Thus, individuals with high scores on cognitive restraint were more likely to consume food than individuals with low scores were. This is consistent with our hypothesis of automaticity.

We then examined individuals' phenomenological experience, to test the characteristic of unconsciousness of the automatic behavior. We asked participants to justify their behavior in relation to their goal. Participants were asked to declare their goal, and then their goal was confronted with their behavior and asked to justify their behavior. A questionnaire provided a few standardized answers and the possibility to freely express other reasons. Results showed that participants justified their behavior in terms of goal, and when their goal and their behavior did not coincide, they found justifications. Our model shows that eating behavior is not predicted by

the goal individuals declare, but by cognitive restraint. All participants were under the impression they were trying to achieve their goal and had good justifications for when they did not.

Results so far indicate that eating behavior fulfills two characteristics of automaticity: uncontrollability (i.e. goal independence) and unconsciousness.

Our next objective was to look at eating behavior, comparing obese individuals and normal weight individuals. First, we computed Pearson chi-square test and Phi to test for the association between BMI classification (obese and normal weight) and consumption (yes or no). Results were significant ($\chi^2(3) = 15.976$, $p = .00$; $\Phi = .412$, $p = .00$) showing an effect in the favor of the obese group, whose members consumed significantly less frequently than those in the normal weight group. However, results concerning this objective are severely limited by the small sample size in our study.

Discussion

Results support our hypothesis, that eating behavior is an automatic behavior – uncontrollable (in terms of goal independence) and unconscious (in terms of the stimulus that evokes the response). Results supported the hypothesis that referred to eating behavior. Individuals' success in controlling their behavior does not depend on their goal or the instruction received. Those who tend to control their food intake on a regular basis (cognitively restrained), are more likely to fail to do so when faced with a situation similar to the one described here. Results can be explained by the fact that controlling an automatic behavior is resource consuming and success to do so is limited in time.

An interesting find was that individuals with obesity did not consume food. We expected obese participants to consume more often either because they found food more palatable or because they need to control their diet on a regular basis, and in such a context they would fail to do so. One explanation could be that obese individuals refrained themselves from eating as a function of social stigma on obesity. In addition, obese individuals might be "better trained" at refraining themselves from food (especially in some high-risk situations) than normal weight individuals. They might just be somewhat better at the task of controlling eating for a short period of time.

Results also showed that participants justified their behavior in terms of goal, and when their goal and their behavior did not coincide, they found justifications. It is interesting that individuals whose goal was not to eat and ended up eating justified their behavior in terms of hunger, craving and difficulty to resist, while none of the participants who ate, but did not have a goal to refrain from food justified their behavior in these terms. This shows, in our opinion, that individuals do not have insight into their behavior and, when confronted with having to explain it, they quickly improvise an explanation to excuse their behavior, as the behavior is automatic and individuals are unconscious to the stimulus input that evokes the response and the process itself.

Experiment 2: Evidence of nonefficiency of eating behavior under conditions of voluntary control.

The objective of experiment 2 was to test the characteristic of (non) efficiency. For eating behavior to be an automatic behavior that does not require effort to unravel, but does require effort to refrain from it, the following hypothesis should be met:

1. Participants will have a better performance (more correctly identified differences and fewer errors) in the no-interdiction condition than in the interdiction condition.
2. Participants will spend more time trying to solve the task in the no-interdiction condition than in the interdiction condition (the effort of refraining will make them stop sooner).

Besides this objective, we were interested in the differences between the two groups: normal weight individuals and obese individuals. Would their performance be similar or different in the two conditions?

Method

Participants were 40 first year psychology students from the Babes Bolyai University, Romania and 40 candidates for participation in a randomized clinical trial of obesity treatment. The students were all normal weight, their mean age was 21 (ranging from 19 to 27). Most participants were female (30) with only a small sample of male participants (10). The patients were all obese, with a mean age of 33 (ranging from 18 to 54), 27 were women and the rest men.

Experimenters. The experimental procedure was carried out by MA level students. They explained the task to participants and were present in the room while participants resolved the task, ensuring the experimental manipulation.

Procedure and design. Participants were informed that investigators want to compare cognitive strategies used in a task, under some particular conditions (related to eating behavior). Participants had to find differences between two very similar images (same degree of difficulty) and they were placed in two conditions: (1) with food present and allowed to eat while performing the task (no-interdiction) and (2) with food present but not allowed to eat (interdiction). Each participant went through each condition on two consecutive days. The order of the two conditions was randomized. The *independent variable* was the manipulation of the interdiction to eat: (1) interdiction condition – food was present, but there was an interdiction to consume the food prior to finishing the task; (2) no-interdiction condition – food was present and participants could eat at any time, if they wished to. The *dependent variables* (measures of interference of the effort to control eating behavior) were:

(1) Differences between images (correct differences). The more effort needed to control eating behavior, the fewer the resources available for the task, the fewer differences between the two images will be identified.
(2) Errors (number of incorrect differences). The more resources needed to control the automatic response, the more errors there will be in trying to solve the task.
(3) Duration spent on the task – participants were able to stop working on the task anytime, when they thought they had completed the task (they were not informed of the number of differences between the two images). The more effort needed to complete the task and control eating, the more difficult and unpleasant the task will be, so the sooner they will escape from it.

Measures. We asked participants for self-reported height and weight (to compute BMI). In addition, we had a questionnaire to insure the effect of the manipulation (participants where manipulation was unsuccessful were excluded from the analysis).

Results

Our first objective was to see if eating behavior would fulfill the (non)efficiency feature of automaticity. First, we compared the whole sample to see if there were differences between the two conditions. Table 2 summarizes results.

Table 2
Comparison of performance in the two conditions, total sample (N=77).

	Condition	M (SD)	*F*	Sig.	Cohen's *d*
Differences between images	No interdiction	10.20 (2.79)	15.217	.000	.37
	Interdiction	9.31 (1.91)			
Errors	No interdiction	.38 (.77)	3.852	.054	.23
	Interdiction	.22 (.55)			

Time spent on task	No interdiction	11.58 (6.01)	18.266	.000	.33
	Interdiction	9.75 (4.77)			

Results are significant between the two conditions for the whole sample, showing a small to medium effect size of the interdiction on participant's performance. When individuals try to control their eating behavior, they find fewer differences, make more errors and spend less time in the task as compared to the situation where they can eat.

Next we checked if the performance is different between obese and normal weight individuals. We checked for the effect of weight status for the three dependent variables. Results show normal weight individuals have a better performance when it comes to differences between images that they identify (($F(1, 75) = 7.375$, $p = .008$) and they spend more time on the task ($F(1, 75) = 18.735$, $p = .001$) then obese individuals do. When it comes to errors committed during the task, the differences were not significant ($F(1, 75) = 3.895$, $p = .053$).

Finally, we looked at the performance considering the effect of the interaction between the condition and weight. Results were significant for differences between images ($F(1, 75) = 6.250$, $p = .015$) and time spent on the task ($F(1,75) = 12.369$, $p = .001$), but not for errors ($F(1,75) = .778$, $p = .381$). What these results show us is that individuals behave differently in the two conditions, and this behavior is moderated by weight.

We were interested to see how individuals behave in each condition considering their weight status. So, we computed paired samples t test to see exactly how each group (normal weight versus obese) behaves in each condition (interdiction and no interdiction). Results are displayed below in Table 3 and Table 4.

Table 3

Comparison of performance in the two conditions, normal weight group (n=39).

	Condition	*M (SD)*	*t*	*Sig.*	*Cohen's d (if t sig.)*
Differences between images	No interdiction	11.07 (2.82)	4.251	.000	.58
	Interdiction	9.69 (1.76)			
Errors	No interdiction	.52 (.92)	1.954	.058	-
	Interdiction	.31 (.66)			
Time spent on task	No interdiction	14.38 (5.78)	5.884	.000	.57
	Interdiction	11.25 (5.08)			

Note: M (SD) = Mean and Standard Deviation; t=student t test; Sig. = significance level; Cohen's d = size effect.

Table 4

Comparison of performance in the two conditions, obese group (n=38).

	Condition	*M (SD)*	*t*	*Sig.*	*Cohen's d*
Differences between images	No interdiction	9.18 (2.41)	1.222	.270	-
	Interdiction	8.87 (2.02)			
Errors	No interdiction	.16 (.37)	1.000	.327	-
	Interdiction	.08 (.27)			
Time spent on task	No interdiction	8.28 (4.44)	.564	.577	-
	Interdiction	7.98 (3.73)			

Note: M (SD) = Mean and Standard Deviation; t=student t test; Sig. = significance level; Cohen's d = size effect.

Analyzing the two tables, we can see that the difference in the two conditions is accounted for by normal weight individuals, and we cannot see a significant difference for the obese group. The effect size of the manipulation for differences identified and time spent in task actually

increases if we look only at the normal weight sample. If we look at the obese group, we can see that their performance is quite similar in both conditions.

Results so far indicate that obese individuals spend significantly less time trying to solve a task when food is present and their performance is significantly lower as compared to that of normal weight individuals in similar conditions.

Discussion

Results support our main objective and hypotheses, that eating seems to be an automatic behavior and that trying to control it is resource consuming. Effort to regulate eating affects performance in a cognitive task and makes individuals quit sooner at trying to solve it. An impairment effect on cognitive processes can be observed in individuals that are confronted with food, but kept from eating, for a short period of time (individuals spent a minimum of 5 minutes and a maximum of 24 minutes on the task).

The interdiction to eat during the resolution of a cognitive task has a medium to large effect on normal weight individuals' performance. The same interdiction seems to have no effect on obese individuals, but their performance is equally bad in both conditions, and closer to that of the normal weight individuals' in the interdiction condition. Obese individuals have a much worse performance in both conditions when compared to normal weight individuals, so it appears that the presence of food triggers their need for controlling the behavior, irrespective of an exterior interdiction.

General discussions and conclusions to experimental evidence for eating as an automatic behavior

Results of the two experiments support our general objective and hypotheses. Eating has features of automatic behavior, thus can be diagnosed as being automatic. Our results bear important implications for the research and clinical practice in the field of eating behaviors. Changing the way we think about eating behavior (as automatic, not voluntary) could have an impact on multiple levels of the management of obesity. First, at a more general, public health level where food policies have concentrated on giving people information that would make them follow a healthy diet. Simply knowing what you should eat does not ensure a healthy diet or a healthy weight. Secondly, at a more individual level, treatment of obesity should also change. Eating behavior can be controlled, but, by means of voluntary effort, it is resource consuming and can only be maintained for short periods of time. Obese individuals manage to lose weight, but fail to maintain it. The most plausible factor that explains this vicious circle is the poor control that individuals have over their *automatic* eating behavior. For control of eating behavior to be efficient, new strategies that minimize effort and make control possible for longer periods of time is needed, especially for long-term management of obesity.

3.1.3. OBESITY AS A RESULT OF A FOOD ADDICTION

Study 5. Food addiction symptoms and body weight

Introduction

This study aims to investigate the concept of food addiction on a sample from a general population and see if there is an association between BMI and food addiction indicators. Such an association was found in an imaging study that has demonstrated that dopamine D2 receptor availability is inversely correlated with BMI (Wang et al., 2001). Thus, our main objective was

to explore an association between BMI and specific food addiction symptoms. We were also interested to see if a clinical diagnosis of food addiction would be more frequent among obese individuals then among normal weight ones. As a secondary objective, we wanted to compare individuals that could be considered as food addicted to those that are not, in terms of certain psychological variables mentioned as relevant in the literature (impulsivity, emotional regulation, self control, dysfunctional eating behavior).

Method

Participants were153 individuals from a general population (98 normal weight, 54 obese). Their age ranged between 18 and 55 (mean age 31). There were more women than men (62 % versus 38%).

Measures. Participants were asked to provide height and weight (for BMI) and to complete the following measures: Yale Food Addiction Scale (Gearhardt et al., 2009); The Barratt Impulsiveness Scale, Version 11 (Patton et al., 1995); Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004); Self-control Scale (Grasmick et al., 1993); Three Factors Eating Questionnaire Revised 21 (Cappeleri et al, 2009).

Results

Results show a small, although significant correlation between the two variables (r (150) = .175, p = .032). It seems that the number of food addiction symptoms increases as BMI increases. However, a clinical diagnosis of food addiction is not more frequesnt among obese individuals ($\chi^2(3) = 2.763$, p =.199) and Phi (Φ =.134, p =.118).

Finally, results show food addicts are more impulsive ($t(151) = 3.000$, p = .000, d = .52), less self-controlled ($t(151) = -2.104$, p = .034, d = -.35), have more difficulties in emotional regulation ($t(151) = 2.969$, p = .004, d = .55) and also higher levels of uncontrolled eating ($t(151) = 4.050$, p = .000, d = .66) when compared to non-food addicts.

Discussion

Our results show an association between BMI and food addiction symptoms. Although it is a small correlation, we believe it is not a negligible one, as BMI is a multi-factorial indicator. An association between a variable that is also affected by genetic and biological factors and a psychological construct is an important one, even though the effect size might be small. For the time being, we can influence our behavior to a greater extent (through psychotherapy) than we can influence our genes (through genetic alterations) and sometimes the physiological processes of our body (through pharmacotherapy). The finding that obese individuals are not clinically addict in a significantly higher proportion than normal weight individuals is somewhat surprising. We expected, based on our literature review and studies from other areas (imaging, neurophysiology) to find more food addicts in the obese sample. We consider this result might be due to the fact that our sample was not a treatment seeking sample.

Our results have shown that food addicted individuals, irrespective of their body weight, have higher levels of impulsivity, emotional regulation difficulties and uncontrolled eating and lower levels of self-control. These factors should be taken into account when working with individuals that are obese, as changes in these variables could lead to better results in the status of their “food addiction” problem.

The question of weather food can or cannot lead to an addiction has important implications, especially for clinical practice. Serious research is needed on this topic to ensure that food addiction will be addressed through evidence based treatments, and not just popular programs with little or no effectiveness, as the concept of food addiction is appealing to the public and already intensely marketed.

3.2. DESIGNING AN ETIOPATHOGENETIC PSYCHOLOGICAL TREATMENT FOR OBESITY

3.2.1. Clinical Implications of Results

Results of the metaanalysis (study 1) point to a superior efficiency of surgical interventions for the treatment of obesity and dysfunctional eating patterns over the psychosocial interventions. However, Bond et al. (2009), studying people who have lost a significant (and comparable) amount of weight through surgical and non-surgical methods, found that weight-loss maintenance comparable with that after bariatric surgery can be accomplished through non-surgical methods with more intensive behavioral efforts. The problem is that a very small sample of individuals actually achieve a weight loss through behavioral efforts comparable to that obtained through surgical methods, and it is this maintenance of results that is the current "Achilles' heel" for obesity treatment, especially psychosocial interventions. This is a challenge for all psychosocial interventions: improve treatment attendance, rate of success after treatment and especially long-term maintenance of results. These are all goals we hope to achieve in our new, enhanced treatment of obesity.

Results of study 2 indicated an association of irrational food beliefs (IFB) to BMI. We considered this association an important one taking into account that BMI is largely determined genetically, with a heritability as high as 80% in Western populations and it is also influenced by gender, social economic status as well as other factors (Keksitalo et al., 2008). IFB were also found to be a predictor for emotional eating, thus targeting IFB, together with trying to reduce emotional eating, might yield better results in the treatment of obesity.

In study 3, results indicated that a high BMI is associated with low body self-esteem and low unconditional self-acceptance. Also, we found that the association between BMI and depression was mediated by body self-esteem. Approaching unconditional self-acceptance in the treatment of obesity might lead to better results for the treatment of obesity as suggested by other authors as well (Wilson, 1996) so these results offered us some other factors to consider in the design of the enhanced treatment: emotional distress (depression in particular), body self-esteem and unconditional self-acceptance.

What these three studies had told us so far is that cognitive-behavioral factors offer promising benefits for the psychological treatment of obesity and they should be considered in the treatment for obesity in the attempt to improve long-term results for these patients.

Study 4 was comprised of two experiments designed to test for features of automaticity (uncontrollability, efficiency and unconsciousness). Results showed that eating has all these features and it could be diagnosed as automatic. Individuals' success in controlling their behavior does not depend on their goal or on a received instruction not to eat. Those who were cognitively restraint were more likely to fail at trying to refrain from eating. Trying to control eating is expensive in terms of cognitive resources. Obese individuals manage to lose weight, but fail to maintain it. The most plausible factor that explains this vicious circle is this effort and expenditure needed to control the *automatic* eating behavior that cannot be sustained for long periods of time. For control of eating behavior to be efficient, new strategies that minimize effort and make control possible for longer periods of time is needed, especially for long-term management of obesity. Our results raise the need for new therapeutic approaches to automatic behaviors.

Study 5, food addiction symptoms and body weight, showed that, the higher the body mass, the more symptoms of food addiction we can find. Obese food addicts might have different outcomes in the treatment of obesity, as they may have different needs and other associated psychological problems that should be dealt with. We also found an association

between food addiction symptoms and emotional regulation difficulties, impulsivity, and uncontrolled eating. These factors should be taken into account when working with individuals that are obese, as changes in these variables could lead to better results in the status of their "food addiction" problem.

To summarize all these results, we found a series of psychological factors relevant to individuals with obesity. Analyzing all these factors, we decided to focus on three variables as outcomes of our obesity treatment: weight, food addiction and depression. The enhanced treatment we design will have to produce relevant changes for these outcomes.

Then, considering the associations we found between outcome variables and the other factors we have investigated, we considered the following clusters of variables as potential mechanisms of change. We found significant relationships between weight and eating behavior, irrational food beliefs and unconditional self-acceptance, so these variables might be mechanisms for change related to weight. Next, we found associations between food addiction symptoms and emotional regulation difficulties, impulsivity, self-control and uncontrolled eating, so we tested their role as mechanisms of change for food addiction. Finally, we found depression to be associated with body self-esteem so we will also test this as a mechanism of change for depression.

3.2.2. Designing a new enhanced cognitive-behavioral treatment of obesity

3.2.2.1. Behavioral weight loss treatment.

Behavioral weight loss (BWL) treatment is regarded as a necessary component to any obesity treatment program (Wilson & Brownell, 2002). Three features are characteristic to BWL (Wadden & Foster, 2000): (a) goal-orientation – clear goals are specified in terms that can easily be measured; (b) process-orientation – the focus is on learning a set of skills necessary to manage weight; (c) advocating small rather than large changes (based on the principle of shaping). Alongside these basic characteristics, behavioral treatment has evolved the last decades into a package that includes several components such as self-monitoring, slowing down the rate of eating, physical activity, nutrition education, stimulus control, problem solving, relapse prevention (Wadden & Butryn, 2003). The typical duration of BWL is 16 to 26 weeks (Wing, 1998). Sessions of 60 to 90 minutes provided in group format by psychologists, dieticians or other health professionals is also typical (Wadden & Butryn, 2003). The group format seems to enhance the efficiency of the treatment (Renjilian et al., 2001) and is also more cost-effective (Wadden et al, 2005). A program respecting all these steps and principles of behavioral weight loss therapy constitutes the reference treatment in our randomized clinical trial.

3.2.2.2. Developing an etiopathogenetic weight loss program

Innovative components to a new, enhanced cognitive-behavioral treatment of obesity.

a) Rational emotive behavior therapy

Participants will learn to identify faulty thinking that undermines their long-term goals of losing weight and maintaining weight. Specific irrational beliefs, pertaining to food and eating, alongside other more general beliefs (relevant to emotional and behavioral consequences) will be targeted in the program.

b) Implementation intention and rational anticipation techniques

Implementation intention refers to planning in advance in order to increase chances of success. It refers to establishing, in detail, the specific behaviors that will ensure the achievement of a goal, as well as other details related to the context in which the goal has to be achieved. Implementation intention will be used by participants in our program in order to better control their eating behavior (e.g. avoid overeating, avoid eating unhealthy food, deal with tempting situations) and to increase physical activity.

Rational anticipation techniques (David, 2006a) try to anticipate the influence that faulty information has on our behavior. Once acknowledged, these influences can be blocked. Specific beliefs about food and eating can impact individual's eating behavior. By becoming aware of these beliefs and their impact on their behavior, patients can prepare behavioral responses that will "override" these beliefs for the specific moments when they cannot be aware of these influences.

c) Elements from addiction therapy

The enhanced treatment will aim to increase the value of nonfood reinforcers (e.g. activities that do not involve food). In addition, the enhanced treatment will specifically approach the notion of "food addiction" and include this component in the conceptualization of weight control. Each participant will identify his or her "addictive food" ("drug's of choice"), if such foods exist. The therapist and participant will work together to find specific strategies to avoid them. Also, participants will be matched into pairs and encouraged to be each other's sponsor / motivator, as a useful strategy for tempting moments and times of relapse.

d) *Strategies aimed to enhance maintenance of results*.

The enhanced treatment will encourage the group members to keep in touch, and an Internet based discussion group will be created toward the end of treatment. The therapists working with the group will also be part of this discussion group in the beginning (then gradually retreat) and will provide helpful tips and information to participants in order to help maintain results and promote further change. Studies have suggested that groups of obese individuals can be taught how to run their own peer support groups and such groups can help to maintain the lost weight and prevent regain (Perri, 2002).

More details of the enhanced treatment program are presented in the next section.

3.3. A RANDOMIZED CLINICAL TRIAL OF AN ENHANCED COGNITIVE-BEHAVIORAL TREATMENT FOR OBESITY.

Introduction

The main problem with obesity treatment is poor long-term results. This is precisely what we aimed in the design of the new, enhanced treatment we test in this study: improving results and especially long-term results for weight loss by acting on certain psychological factors which we hypothesize to be etiopathogenetic mechanisms.

General objectives

Our main objectives for this randomized controlled trial were:

I. To test the effectiveness of a new enhanced treatment of obesity against a standard behavioral treatment for obesity..
II. To explore the mechanisms of change responsible for our results..

For the first objective we have conducted an outcome study. Our main outcome was weight (expressed as BMI). We also had two secondary outcomes: food addiction symptoms and depression. The treatment we have designed aims to modify these outcomes, and, through the specific strategies we have included in the treatment (see chapter 3.2.) we expect that these outcomes will be significantly better in the enhanced treatment condition as opposed to the standard treatment.

Our hypotheses related to the outcomes were:

1. Participants in the enhanced group will have a lower BMI as compared to participants in the standard group, at the end of treatment and follow-up.
2. Participants in the enhanced group will have fewer food addiction symptoms as compared to participants in the standard group, at the end of treatment and follow- up.

3. Participants in the enhanced group will have fewer depression symptoms as compared to participants in the standard group, at the end of treatment and follow-up.

For the second objective we have conducted a theory of change study. Identifying factors that lead to or facilitate change is extremely important for the development of etiopathogenetic treatments. We expect changes in our main outcome (BMI) to be related to changes in eating behavior, irrational food beliefs and unconditional self-acceptance. Previous studies we have conducted (see chapter 3.1.) showed significant relationships between BMI and these variables. Thus, our hypotheses, based on these previous results, for weight outcome are:

1. For participants in the enhanced treatment group, the significant changes in BMI, as compared to the standard treatment group, will be related to significant changes in eating behavior.
2. For participants in the enhanced treatment group, the significant changes in BMI, as compared to the standard treatment group, will be related to significant changes in irrational food beliefs.
3. For participants in the enhanced treatment group, the significant changes in BMI as compared to the standard treatment group will be related to significant changes in unconditional self-acceptance.

For food addiction symptoms, we found associations with uncontrolled eating, emotional regulation difficulties, self-control and impulsivity, irrespective of body weight. Thus, we expect for changes in food addiction symptoms to be related to these variables. Our hypotheses are:

1. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in uncontrolled eating.
2. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in emotional regulation difficulties.
3. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in self-control.
4. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in impulsivity.

Our study on body self-esteem, unconditional self-acceptance and self-esteem in obese individuals showed that only body self-esteem is a strong predictor for depression in obese individuals. However, according to REBT (see David, 2006a), depression is the consequence of certain irrational beliefs, so we also measured general irrational beliefs and considered them as a potential mediating variable. Thus, our hypotheses for the mechanisms of change related to depression were:

1. For participants in the enhanced treatment group, the significant changes in depression, as compared to the standard treatment group, will be related to significant changes in body self-esteem.
2. For participants in the enhanced treatment group, significant changes in depression, as compared to the standard treatment group, will be related to significant changes in general irrational beliefs.

Method

Participants. A total of 51 patients participated in this randomized clinical trial (25 in the enhanced group, 26 in the control group). Patients were from the Cluj and Mureş districts, Romania. Their age ranged between 18 and 61, with a mean age of 34. The majority of

participants were female (42), with only a small sample of male participants (9). An initial sample of 76 participants was assessed for inclusion and exclusion criteria. Twelve patients did not meet these criteria and were referred to community care. The remaining 64 patients were assessed at baseline, randomized to either standard (*N*=31) or enhanced treatment (*N*=33) and had to wait for inclusion in treatment group. At the start of treatment we had 25 patients in the enhanced treatment group and 26 in the control one. We had 4 dropout patients in the enhanced group and 8 dropouts in the standard treatment group. Drop out patients were defined as having participated in at least one session and less than seven weeks (middle of treatment).

Therapists and evaluators. Nine therapists with a formal training in CBT provided treatment in both conditions. Therapists were randomly assigned to provide either behavioral treatment or the enhanced treatment and were only trained in one treatment protocol. Manuals describing detailed guidelines for the two treatment conditions were available for each therapist. In addition to the therapists, assessments were carried out by four psychologists, who were independent from the study and blinded to treatment assignment.

Procedure. Participants were recruited via Internet announcements and referred by their G.P. to take part in a randomized clinical trial for the treatment of obesity. Inclusion criteria referred to: (a) adult participant; (b) BMI $\geq$ 30. Exclusion criteria referred to: (a) serious complications from obesity; (b) severe psychological disorders. Participants who met these criteria were administered a full set of questionnaires and were randomized to treatment.

Treatment conditions. Participants were included in a 16 weeks intervention program. They were randomly assigned to either a control, standard behavioral treatment or to a new, enhanced cognitive behavioral treatment.

Treatment duration and format. Both treatments were delivered in a both individual and group format. The first three sessions were individual, followed by seven group sessions, then another two individual sessions and three more group sessions. Each session lasted 90 minutes.

Treatment components. Both treatments included:

(a) A dieting component. Participants in both groups were provided with materials on healthy food and assisted to make up a personal, healthy diet.

(b) A physical activity component. This component was discussed from the beginning of the treatment program, but participants were encouraged to start exercising when they felt ready.

(c) A counseling component. Individuals were counseled about their lifestyle. This is the component where the two treatments were most different.

Standard behavioral weight loss treatment. The protocol included: (a) self-monitoring of daily food intake and physical activity; (b) nutrition education; (c) stimulus control techniques to help patients manage cues associated with inappropriate eating; (d) slowing the rate of eating; (e) physical activity education; (f) relapse prevention; (g) cognitive restructuring.

Table 1

Standard behavioral weight loss treatment structure

Week	Session Theme	Format
1	Initial assessment	Individual
2	Conceptualization of weight control and eating behavior.	Individual
3	Establishing goals and the problem list.	Individual
4	Getting ready to change. Enhancing motivation for change, acknowledging the advantages and disadvantages of change.	Group
5	Nutritional education. General principles of healthy eating (I).	Group
6	Nutritional education. General principles of healthy eating (II).	Group
7	Changing the behavior and environment I	Group
8	Changing the behavior and environment II	Group
9	Changing the behavior and environment III	Group

10	Working harder to lose weight (physical activity and "correct" dieting).	Group
11	Intermediary assessment (How is the program working?)	Group
12	Emotional problems and their impact on eating. Finding better solution to emotional situations (distraction, involvement in more adaptive behaviors, identifying and correcting dysfunctional thoughts).	Individual
13	Body image concerns and their impact on weight control - identifying and correcting dysfunctional thoughts.	Individual
14	Dealing with difficult moments, temptations and exceptions.	Group
15	Long-term maintenance. Long-term goals.	Group
16	Final assessment and booster session.	Individual

Enhanced cognitive behavioral weight loss treatment. This program included all the components of the standard treatment described above. There were major differences for the cognitive restructuring component. The cognitive component was a rational emotive behavior therapy based on Ellis's principles (Ellis, 1962 as cited by David, 2006a). Thus, participants' general irrational beliefs, their specific irrational food beliefs and other irrational beliefs relevant to weight loss and maintenance (irrational beliefs about physical activity, body concerns etc.) were identified and disputed according to REBT techniques and principles. In addition to these differences in cognitive restructuring, the program also incorporated a few more innovations:

- Conceptualizing eating as an automatic behavior which can be controlled better through certain techniques than through "will power"; restructuring the myth of "will power" as the determinant of success in weight loss.
- Rational anticipation (David, 2006a) and implementation intention (Gollwitzer & Brandstatter, 1997) as techniques to better control eating as an automatic behavior.
- Introducing the notion of "food addiction in the conceptualization of weight control; identifying "addictive food" and coming up with specific strategies to avoid them.
- Assigning participants to a "sponsor" similar to addiction programs. This strategy also aims to be a mechanism to ensure maintenance of results.

Table 2

Enhanced Treatment Structure

Week	Session Theme	Format
1	Initial assessment	Individual
2	Conceptualization of weight control and eating behavior. Fundamentals of rational emotive behavioral therapy. Eating as an automatic behavior.	Individual
3	Establishing goals and the problem list. Food addiction.	Individual
4	Getting ready to change. Identifying motivations to change the lifestyle. The problems of the motivation to lose weight in order to "look better" – the problem of body self-esteem and advantages of unconditional self-acceptance of body shape and weight.	Group
5	Nutritional education. General principles of healthy eating (I).	Group
6	Nutritional education. General principles of healthy eating (II).	Group
7	Changing the behavior and environment I	Group
8	Changing the behavior and environment II. Eating as an automatic behavior - Rational anticipation techniques and implementation intention.	Group
9	Changing the way we think about food and ourselves. Irrational beliefs and how they affect our emotions and behavior.	Group
10	Working harder to lose weight (physical activity and dieting). Restructuring irrational beliefs and self-defeating thoughts related to physical activity and movement.	Group
11	Intermediary assessment (how is the program working?)	Group
12	Emotional problems and their impact on eating. Identifying and disputing relevant irrational beliefs.	Individual
13	Body image concerns and their impact on weight control. Identifying and disputing relevant irrational beliefs.	Individual

14	Dealing with difficult moments, temptations and exceptions. Rational anticipation techniques and implementation intention.	Group
15	Long-term maintenance, long-term goals. Exercising your rational beliefs. Keeping in touch with your group and your therapist.	Group
16	Final assessment and booster session.	Individual

Measures: (a) Body weight (on a standardized digital scale) and height in order to compute BMI; (b) Yale Food Addiction Scale (Gearhardt et al., 2009); (c) Beck Depression Inventory II; (d) Three Factors Eating Questionnaire Revised 21 (Cappeleri et al, 2009); (e) Irrational Food Beliefs Scale (Osberg et al., 2008); (f) General Attitudes and Beliefs Scale – short version (Lindner et al., 2007); (g) Unconditional Self-acceptance Questionnaire (Chamberlain & Haaga, 2007); (h) Body self-esteem scale (Franzoi & Shields, 1984); (i) Self-control Scale (Grasmick et al., 1993); (j) The Barratt Impulsiveness Scale, Version 11 (Patton et al., 1995); (k) Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004). Participants completed these measures at four points in time: (a) baseline; (b) middle of treatment; (c) end of treatment; (d) follow-up.

Outcome study

Our main outcome was weight (measured as BMI). We also had two secondary outcomes: food addiction symptoms and depression. Our hypotheses related were:

1. Participants in the enhanced group will have a lower BMI, as compared to participants in the standard group, at the end of treatment and follow-up.
2. Participants in the enhanced group will have fewer food addiction symptoms, as compared to participants in the standard group, at the end of treatment and follow- up.
3. Participants in the enhanced group will have fewer depression symptoms, as compared to participants in the standard group, at the end of treatment and follow- up.

Besides these hypotheses related to outcomes, we also expect that the new treatment will result in lower dropout rates. We designed the new treatment to help prevent high dropout rates in the first sessions, so we wanted to check if our strategies were successful.

Results

All analyses were conducted using the intent-to-treat principle. All randomized patients were included in the analyses, regardless of their subsequent withdrawal from treatment. The last available score, on each measure, was used as the score at the end of treatment for dropouts. Follow-up analyses were conducted for patients who completed treatment (defined as receiving at least 12 of the 16 sessions of treatment). We compared the two treatments at baseline, middle of treatment and end of treatment, for all outcomes.

Baseline. Pretreatment group differences were assessed and we found no significant pretreatment differences among conditions for BMI ($t(49) = -.951$, $p = .346$), food addiction ($t(49) = -.514$, $p = .610$) or depression ($t(49) = -1.232$, $p = .224$).

Middle treatment (outcomes at 8 weeks). Data analysis at 8 weeks showed no significant difference for BMI ($t(49) = -1.352$, $p = .183$) or food addiction symptoms ($t(49) = -1.079$, $p = .286$). There is a significant difference though, for depression ($t(49) = -2.242$, $p = .030$) indicating a large effect size ($d = -.64$) for the enhanced treatment by 8 weeks already. Results show the enhanced treatment has started to impact significantly on depression already by the middle of treatment.

Post treatment (outcome at 16 weeks). At the end of treatment we find, again, no significant difference between the two groups for BMI ($t(49) = -1.250$, $p = .217$) or food addiction symptoms ($t(49) = -.411$, $p = .683$). The only significant difference is for depression ($t(49) = -2.729$, $p = .009$, $d = -.76$). For food addiction we also checked for significant

differences in dichotomous score (food addicts versus non-food addicts). The chi-square test showed no significant differences between the two treatments ($\chi^2(3)= .233$, $p = .629$; $\Phi^{\S} = -.068$, $p = .629$).

These results show that, at the end of the 16 weeks of treatment, the enhanced treatment is superior to the standard one only in reducing depressive symptoms in obese participants. We also wanted to see how the outcomes change in each group, so we performed repeated measures ANOVAs for differences at the three points in time (baseline to middle to end of treatment) for each group.

For the enhanced treatment group we found significant differences between pre, middle and post-treatment for BMI ($F(2,22) = 51.677$, $p = .000$), food addiction symptoms ($F(2,22) = 14.767$, $p = .000$) and depression ($F(2,22) = 23.172$, $p = .000$).

For the standard treatment group, we also found significant differences between pre, middle and post-treatment for all outcomes: BMI ($F(2,23) = 23.222$, $p = .000$), food addiction symptoms ($F(2,23) = 10.007$, $p = .000$) and depression ($F(2,23) = 7.435$, $p = .005$).

We computed paired samples t test, with a Bonferroni correction, to see at what point in time we can see results. Results are presented for each treatment group.

Weight outcome. For the enhanced group, there were significant differences from baseline to middle of treatment ($t(24) = 7.577$, $p = .000$) with a small effect size (d=.29). Differences were also significant from middle of treatment to end of treatment ($t(24) = 3.624$, $p = .001$, $d = .08$) but the effect size is negligible. Overall, the effect size from baseline to end of treatment for the enhanced group is a small to medium one as indicated by Cohen's $d = .37$ ($t(24) = 7.593$, $p = .000$). For the standard group there were significant differences from baseline to middle of treatment ($t(25) = 4.563$, $p = .000$, d=.18), but only a very small effect size. Differences were also significant from middle of treatment to end of treatment ($t(25) = 4.367$, $p = .000$, $d = .09$) but the effect size is negligible. Overall, the effect size from baseline to end of treatment for the standard treatment group is a small one - $d = .27$ ($t(25) = 5.012$, $p = .000$).

Food addiction outcome. For the enhanced treatment group, we have significant differences from baseline to middle of treatment ($t(24) = 3.858$, $p = .001$, $d = .57$) but no significant difference from middle to end of treatment ($t(24) = 1.432$, $p = .166$). Overall, the effect size from baseline to post treatment is a large one - $d = .80$ ($t(24) = 5.192$, $p = .000$). For the standard treatment group we have significant differences from baseline to middle of treatment ($t(25) = 2.697$, $p = .013$, $d = .55$) and also middle to end of treatment ($t(25) = 3.361$, $p = .003$, d=.37). Overall, the effect size from baseline to post treatment is a very large one - $d = .86$ ($t(25) = 3.699$, $p = .001$).

Depression outcome. For the standard group, we can find significant differences from baseline to middle treatment ($t(24) = 4.625$, $p = .000$, d=.53), and also significant differences from middle treatment to post treatment ($t(24) = 4.456$, $p = .000$, $d = .41$). The overall effect of the enhanced treatment on depression is a large one - $d = .90$ ($t(24) = 5.214$, $p = .000$). For the standard group, we can find significant differences from baseline to middle treatment ($t(25) = 2.727$, $p = .012$, d=.39), but no significant difference from middle treatment to post treatment ($t(25) = 1.871$, $p = .074$). The overall effect of the standard treatment on depression is a medium one - $d = .66$ ($t(25) = 2.942$, $p = .007$).

As a more secondary objective of our study, we were interested to check the association of dropout rates between the two treatments. We did have 4 dropouts in the enhanced group and 8 in the standard group, but when we performed the chi square test for the association between

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treatment condition (enhanced versus standard) and dropout condition (completer versus dropout), results were not significant ($\chi^2(3)= .778$, $p = .523$; $\Phi = .124$ $p = .378$).

Follow-up analysis. Our second set of hypotheses was that we would find significant differences between the two groups at follow-up. We conducted a 6 months follow-up. Table 4 displays these results.

Table 4

Comparisons of the enhanced (n=18) and standard treatment (n=18) groups at 6 months follow-up.

	Treatment group	M (SD)	*t*	*p*	*d*
BMI	Enhanced	30.49 (5.38)	-2.048	.048	-0.68
	Standard	34.16 (5.35)			
Food addiction symptoms	Enhanced	1.94 (.93)	-.368	.715	-
	Standard	2.05 (.87)			
Depression	Enhanced	4.33 (5.24)	-2.536	.016	-0.38
	Standard	8.88 (5.52)			

Results show significant differences in BMI and depressive symptoms. Thus, at 6 months follow up, participants in the enhanced treatment group have a significantly lower BMI compared to that of participants in the standard treatment. The effect size is a medium one. Also, depression continues to be significantly lower at follow-up for participants in the enhanced treatment group. The effect size is again a small one.

We also looked at the evolution of outcomes for each group. For the standard treatment group, there is a significant evolution of BMI from end of treatment to follow up indicating that the participants from the standard treatment group are beginning to regain weight (BMI $t(17) =-3.485$, $p=.003$). And also, participants in this group are also becoming more depressed from end of treatment to follow up ($t(17) = -3.173$, $p= .006$). There were no significant differences from end of treatment to follow-up for food addiction symptoms ($t(17) = -.809$, $p = .430$). For the enhanced group, there is no significant difference from end of treatment to follow-up for BMI ($t(17) = 1.788$, $p = .092$) suggesting that these participants are roughly maintaining their post treatment weight. There were significant differences for depression ($t(17) = 2.360$, $p =.031$) as participants in the enhanced group seem to be less depressed at follow up as compared to post treatment. And there is no significant difference in terms of food addiction symptoms ($t(17) = -.516$, $p = .619$).

Looking at these results together, it seems that the between group differences at follow up for weight outcome is accounted not by a continued weight loss by the enhanced treatment group, but by a weight regain in the standard treatment group and a maintenance of results in the enhanced group.

Discussion

As a conclusion so far, we can say that some superior effect of the enhanced program over the standard one can be observed at the end of treatment for the secondary outcomes; first of all, for depression that improves considerably. For addiction we cannot see superiority of the enhanced treatment in better effects, but in effects obtained sooner, and this can be important in clinical practice (for both efficacy and cost-effectiveness of treatment). For the main outcome, weight, results at 6 months follow up show that participants in the enhanced treatment maintain their weight, but these results need to be confirmed at longer follow-ups.

Theory of change study

Our hypotheses related to BMI were:

1. For participants in the enhanced treatment group, the significant changes in BMI, as compared to the standard treatment group, will be related to significant changes in eating behavior.
2. For participants in the enhanced treatment group, the significant changes in BMI, as compared to the standard treatment group, will be related to significant changes in irrational food beliefs.
3. For participants in the enhanced treatment group, the significant changes in BMI, as compared to the standard treatment group, will be related to significant changes in unconditional self-acceptance.

Our hypotheses related to food addiction were:

1. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in uncontrolled eating.
2. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in emotional regulation difficulties.
3. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in self-control.
4. For participants in the enhanced treatment group, the significant changes in food addiction symptoms, as compared to the standard treatment group, will be related to significant changes in impulsivity.

Finally, for depression, our hypotheses were:

1. For participants in the enhanced treatment group, the significant changes in depression, as compared to the standard treatment group, will be related to significant changes in body self-esteem.
2. For participants in the enhanced treatment group, significant changes in depression, as compared to the standard treatment group, will be related to significant changes in general irrational beliefs.

Results

Weight Outcome

Step 1: The Efficacy Test. Results in the outcome study showed no differences between groups for weight, but they showed significant differences between pretreatment and post treatment, for both treatments. Considering both treatments were active conditions, we proceed to the next steps in analyzing potential mediators for change.

Step 2. Intervention Specificity. Here we assess the relationship between treatment and change in the candidate mediators. We compared pretreatment and post treatment scores on these measures on the total sample, for each treatment condition, using paired sample *t* tests. As it can be seen from table 6, all results are significant, for both treatment conditions, with size effects ranging from medium to large and even very large.

Table 6

Paired samples t tests and effect sizes for pretreatment - post treatment values on hypothesized variables, for each treatment condition.

	Treatment	*t*	*Sig.*	Cohen's *d*
UE	Enhanced	4.885	.000	1.00
	Standard	3.929	.001	.90
EE	Enhanced	7.213	.000	1.13
	Standard	3.654	.001	.72
CR	Enhanced	-2.870	.008	-.62
	Standard	-5.974	.000	-1.38
IFB	Enhanced	7.809	.000	1.31
	Standard	3.329	.003	.89
USA	Enhanced	-9.381	.000	-1.56
	Standard	-3.391	.002	-.73

Note: UE = uncontrolled eating; EE= emotional eating; CR = cognitive restraint; IFB = irrational food beliefs; USA = unconditional self-acceptance

Then we checked for between group differences on post treatment scores on each variable, with pretreatment score on BMI serving as covariate. Results show significant group differences for: emotional eating ($F(1,49) = 5.507$, $p = .023$), irrational food beliefs ($F(1,49) = 10.002$, $p = .003$) and unconditional self-acceptance ($F(1,49) = 7.109$, $p = .011$). For uncontrolled eating ($F(1,49) = .243$, $p = .624$) and cognitive restraint ($F(1,49) = 1.623$, $p = .209$) there were no significant between group differences. What these results show, so far, is that the enhanced treatment led to a more significant change in emotional eating, irrational food beliefs and unconditional self-acceptance, as compared to the standard treatment.

Step 3. *Psychopathology Test.* Residual change scores from pretreatment to post treatment for BMI were correlated with those for each hypothesized mediator. Results show the changes in BMI for both treatment groups are related to changes in uncontrolled eating, emotional eating, irrational food beliefs and unconditional self-acceptance. The only difference is for changes in cognitive restraint, which relates to changes in BMI only for the standard group.

Table 7

Correlations between change score (pre to post) between weight outcome and hypothesized variables, for each treatment group.

		BMI	
		Enhanced (*N*=25)	Standard (*N*=26)
UE	*r*	.446**	.565**
	Sig.	.025	.003
EE	*r*	.417**	.497**
	Sig.	.038	.010
CR	*r*	.038	-.792**
	Sig.	.858	.000
IFB	*r*	.561**	.435**
	Sig.	.004	.030
USA	*r*	-.478**	-.429**
	Sig.	.018	.033

Note: UE = uncontrolled eating; EE= emotional eating; CR = cognitive restraint; IFB = irrational food beliefs; USA = unconditional self-acceptance.

Step 4: Mediation Test. We were not able to run a full mediation analysis as we had no significant differences between the treatment conditions at the end of treatment for weight outcome. We found significant differences between treatments for three of the five hypothesized mediators: emotional eating, irrational food beliefs and unconditional self-acceptance, but as the outcome study has revealed, there are no differences between groups for BMI. Therefore,

following Jacobson et al. (1996) and Szentagotai, David, Lupu and Cosman (2008), we formulated conclusions regarding the mechanisms of change on the basis of steps one to three (see the discussion section).

Food Addiction Outcome

Step 1. The Efficacy Test. The outcome study has revealed no significant differences between groups for food addiction symptoms. However, there were significant differences between pretreatment and post treatment. Considering our treatments were both active treatments we proceed to the next steps in analyzing potential mediators for change.

Step 2. Intervention test. This step tests weather treatment affects the mechanism of action supposed to produce intervention effects. We examined the degree to which each treatment resulted in decreased uncontrolled eating, difficulties in emotional regulation and impulsivity, on one hand and increased self-control, on the other hand. We compared pretreatment and posttreatment scores for measures on the total sample, for each treatment condition. All results are significant, for both treatment conditions, except for the score on emotional regulation difficulties which does not change significantly in the standard group treatment (table 9).

Table 9

Paired samples t tests and effect sizes for pretreatment- post treatment values on hypothesized variables, for each treatment condition.

	Treatment group	*T*	*Sig.*	Cohen's *d*
UE	Enhanced	4.885	.000	1.00
	Standard	3.929	.001	.90
DERS	Enhanced	3.636	.001	.18
	Standard	1.584	.126	-
CTRL	Enhanced	-2.941	.007	-.35
	Standard	-2.981	.006	-.73
BIS	Enhanced	3.112	.005	.30
	Standard	2.519	.019	.53

Note: UE = uncontrolled eating; DERS = emotional regulation; CTRL = self-control; BIS = impulsivity.

Then we checked for between group differences on post treatment scores on each hypothesized mediator, with pretreatment score on food addiction symptoms serving as covariate. Results were not significant for all variables, so it seems the two treatments share similar mechanisms when it comes to food addiction outcome.

Step 3. Psychopathology test. We correlated change scores for food addiction symptoms to that of the hypothesized mediating variables. Results show the changes in food addiction symptoms, for the enhanced treatment group, are related only to changes in emotional regulation difficulties. For the standard treatment group, changes in food addiction symptoms are related to changes in emotional regulation strategies and also self-control (table 10).

Table 10

Correlations between change score (pre to post) between weight outcome and hypothesized variables, for each treatment group.

		YFAS	
		Enhanced (*N*=25)	Standard (*N*=26)
UE	*r*	.064	.369
	Sig.	.762	.063
DERS	*r*	.637**	.485**
	Sig.	.001	.014
CTRL	*r*	.014	-.553**
	Sig.	.947	.003

BIS	*r*	-.059	.385
	Sig.	.782	.058

Note: YFAS = food addiction; UE = uncontrolled eating; DERS = emotional regulation; CTRL = self-control; BIS = impulsivity.

Step 4. Mediation Analysis. As with the previous analysis for weight outcome, we had no significant differences in food addiction symptoms and the hypothesized mediators between treatment conditions at posttreatment, so we were not able to run a full mediation analysis.

Depression Outcome

Step 1: The Efficacy Test. As we have seen in the outcome study there were significant within group and between groups differences for depression, so we can proceed to the next steps.

Step 2. Intervention test. Here we check if treatment affects the mechanism of action supposed to produce intervention effects. We examined the degree to which each condition resulted in decreased irrational beliefs and increased body self-esteem. We compared pretreatment and post treatment scores on these measures, for each treatment condition, using paired sample *t* tests. All results are significant for both treatment conditions, except for body self-esteem in the standard treatment group (table 12). Effect sizes range from medium to large.

Table 12

Paired samples t test and effect size for pretreatment - post treatment values on hypothesized mediating variables, for each treatment condition.

	Treatment group	*t*	p	Cohen's *d*
BSE	Enhanced	-4.771	.000	.69
	Standard	-1.959	.062	-
GABS	Enhanced	7.108	.000	1.13
	Standard	2.182	.039	.52

Note: BSE = body self-esteem ; GABS = general irrational beliefs.

Afterwards, we checked for between group differences on post treatment scores on body self-esteem and general irrational beliefs, with pretreatment score on depression serving as covariate. Results show significant group differences for irrational beliefs ($F(1,49) = 17.256$, $p = .000$) and no significant between group differences for body self-esteem ($F(1,49) = .458$, $p = .502$).

Step 3: Psychopathology Test. We tried to establish a temporal relationship between changes in particular mechanisms and outcomes The residual change score from pretreatment to post treatment for depression was correlated with the change score on body self-esteem and general irrational beliefs (table 13).

Table 13

Correlations between change score between depression outcome and hypothesized variables, for each treatment group.

		BDI	
		Enhanced (*N*=25)	Standard (*N*=26)
BSE	*r*	.050	-.803**
	Sig.	.814	.000
GABS	*r*	.562**	.453**
	Sig.	.003	.026

Note: BSE = body self-esteem; GABS = general irrational beliefs.

Step 4. Mediation Analysis.

Results so far showed significant differences between the two treatments, and also significant differences between the two treatments in irrational beliefs. For this relationship we

ran a full mediation analysis. Results show that, when controlling for irrational beliefs, the regression coefficient between treatment and depression (B = -.363, p =.009) is reduced (B=-.285), but it is still significant (p=.023). A Sobel Test indicated that the mediation effect was not significant (.19, p = .841).

Considering these results, we thought there might be other mediators that might explain the significant reduction in depressive symptoms in the enhanced group (see also Weersing and Weisz, 2002). The most likely candidate, would be unconditional self-acceptance. So, we decided to do a supplementary analysis on the potential mediating role of unconditional self-acceptance for depression. Results show significant between group differences for both depression (t(49) =-2.729, p = .009) and unconditional self-acceptance (t(49) = 2.774, p =.008) even when controlling for the pretreatment score on depression (F *(1, 49)* = 6.526), p = .014). Also, there is a strong correlation between changes in depression and change in unconditional self-acceptance (r(49) = -.627, p = .000). Considering these results which correspond to steps 1 to 3 described above, we were able to run a full mediation analysis. Results show that, when controlling for unconditional self-acceptance, the relationship between depression and treatment was no longer significant (r(49)=-.174, p = .194). The mediation is complete and it has a significant effect (Sobel test statistic = 2.12, p = .034).

Discussion

Mechanisms of change for weight

For weight we can say that, in general, we have failed to identify specific mechanisms of change for the enhanced treatment. We did not find any differences between the two groups at BMI post treatment. However, we did find significant differences between groups for emotional eating, irrational food beliefs and unconditional self-acceptance. Participants in the enhanced group have reduced their emotional eating and their irrational food beliefs significantly more than participants in the standard treatment group. In the same manner, they have increased their unconditional self-acceptance. However, these superior changes do not find themselves in a lower body weight at the end of the program. These potential mediators remain promising candidates for results at follow-up and might be related with weight maintenance. These long-term results at follow-up will clarify if these variables are mediators for long-term weight maintenance or simply variables that wax and wane with variations in BMI, but are not causal to weight loss or weight regain.

Mechanisms of change for food addiction

Results show that changes in uncontrolled eating and impulsivity are not related to changes in food addiction. Changes in self-control seem to be related to changes in food addiction only for the standard group. Emotional regulation difficulties relate to changes in food addiction for both groups. However, due to the lack of between group differences in terms of food addiction symptoms and hypothesized mediators, we cannot say for sure these variables are mechanisms of change. Further studies are needed to explore the relationship between emotional regulation difficulties and food addiction.

Mechanisms of change for depression

We hypothesized body self-esteem would be a significant predictor of change in light of our previous findings. Changes in body self-esteem did correlate with changes in depression, but only for the standard treatment group, where reduction in depressive symptoms was significantly lower to that of the enhanced group. So, it seems body self-esteem might be a mechanism of change for the standard treatment, but not for the enhanced treatment. Indeed, one part of the enhanced treatment was changing the motivation to lose weight in order to look good, with the motivation of losing weight for health reasons and unconditionally accepting oneself, irrespective of body weight and shape.

So, it seems that the superior results of the enhanced treatment in reducing depression might be due to the other mediator we had hypothesized – irrational beliefs. Our data showed a mediation effect, but it was not significant, so it seemed results might be due to other psychological factors. Based on the REBT theory, we checked the mediating role of unconditional self-acceptance. Results show this mediates completely and significantly the relationship between treatment condition and depression. However, due to the fact that this was not hypothesized a priori, further confirmation of results, in other studies, is needed.

General discussions and conclusions of the randomized clinical trial

Our clinical study showed that the enhanced treatment we have designed is not superior to the standard treatment in the outcome for weight at the end of treatment. Follow-up results indicate that it might be superior in the results obtained at 6 months after treatment. Further follow-up assessments, at longer intervals will establish more clearly this potential effect. The mechanisms of change analysis indicated that the hypothesized mechanisms changed significantly in both groups, following treatment. And, although participants in the enhanced group reduced their emotional eating and their irrational food beliefs significantly more than participants in the standard treatment group and increased their unconditional self-acceptance, these superior changes do not find themselves in a lower body weight at the end of the program. It might be that some mechanisms are common for both treatments in what weight loss is concerned and the superior results of the enhanced treatment might be in terms of weight maintenance. Long term follow up results will confirm if this is so.

In what food addiction is concerned, our treatment has also failed to produce superior results in the reduction of these symptoms at the end of treatment. This is somewhat surprising as the concept of food addiction was specifically approached and discussed in the enhanced treatment unlike the standard treatment. However, the theory of change analysis indicated as a potential common mechanism emotional regulation difficulties, and self control as a specific mechanism for the standard treatment. In light of our finding that eating could be considered an automatic behavior and efforts to control it voluntary are doomed to fail on the long-term, we expect that this change in self-control, that is related to the change in addiction symptoms at the end of treatment, to be a potential risk factor for relapse. Thus, long term follow-up results might shed light on this hypothesis as well.

Depression was clearly reduced significantly more in the enhanced treatment. We had hypothesized that such a change would be due to the elements of REBT that would produce more significant changes in the way individuals interpret activating events. Our results showed that changes in irrational beliefs do not account for the between groups differences in depression, as this change is significant in both enhanced and standard treatment. Other studies comparing cognitive therapy (CT) to REBT for depression (Szentagotai et al. 2008) also found that both CT and REBT produce significant changes in irrational food beliefs, although only REBT hypothesizes irrational beliefs as a specific mechanism of change. Supplementary analysis showed that the difference between the two treatments in reducing depression can be accounted for by unconditional self-acceptance. Unconditional self acceptance was also related to change in BMI, so it might be a common mechanism for both depression and weight, in what weight maintenance is concerned. Long-term follow-up studies will confirm or disconfirm this hypothesis.

Overall, we can say that the enhanced treatment has not proven superior results at the end of treatment in what body mass and food addiction is concerned. But there seem to be some significant differences in psychological factors we have hypothesized as mediators of change that might affect these outcomes at follow-up. This remains a hypothesis to be tested. The enhanced

treatment has proven superior results in what depression is concerned, and unconditional self-acceptance seems to be a mechanism of change specific to the enhanced treatment. Further analysis and future studies will investigate if this reduction in depression might be a protective factor against relapse that might help maintain a healthy weight for a longer period of time.

There is also another way to look at our results. More researchers are questioning the focus of health professionals on weight as an outcome for obesity treatment. For example, Foster and Kendall (1994) argue that treatment success should be measured by a wider range of indicators (e.g. medical, psychological and behavioral) not just by weight loss. Similarly, Tomiyama and Mann (2008) propose that the research community shifts the focus of obesity research toward direct measures of health, such as blood pressure or insulin resistance, and away from weight, which is an imperfect indicator of current or future health problems. Similarly, other researchers have proposed considering new criteria for assessing success after obesity treatment. Eating behavior was proposed to be such a criteria (Perri, 1998) – if obesity treatments yield changes in eating patterns of obese individuals than it should be considered a successful treatment. If we take this perspective on our results and consider the variables we have hypothesized as mediators to be outcomes, we can reach some different conclusions. From this perspective, both treatments bring about modest weight losses, but important psychological health benefits in terms of: reduced levels of food addiction symptoms, depression, dysfunctional eating behavior, irrational food beliefs, impulsivity and increased self-control and body self-esteem.

From another perspective, our results have shown that, the enhanced treatment produces most changes in outcomes by the middle of treatment (especially for depression, but also food addiction). This means that the treatment duration could be shortened and we would obtain results similar to those obtained through standard treatment. Thus, the enhanced treatment could be more cost-effective. This is an important aspect for health care, as medical services are costly, and a treatment that offers similar results with fewer costs, is clearly superior.

To conclude, we can say that our results so far have failed to prove the etiopathogenetic character of the enhanced treatment for weight loss. Results have however proven that the enhanced treatment leads to a better quality of life. Long-term follow-up results will confirm if these better results in quality of life lead to a better weight maintenance or even promote further weight loss. Should this be the case, we will be able to consider the enhanced treatment an etiopathogenetic treatment for weight maintenance. Otherwise, the enhanced treatment is just a superior symptomatic treatment. This is also important, but not enough, from our point of view. Also, the enhanced treatment could be designed to be more cost-effective than the standard treatment, an important advantage not to be reckoned with. Future research in all areas related to obesity needs to focus on finding etiopathogenetic treatments, with increased effectiveness and cost-effectiveness.

CHAPTER IV. GENERAL CONCLUSIONS AND DISCUSSION

This research project had two major goals: (1) to identify psychological factors with an etiopathogenetic role in obesity and (2) to develop an etiopathogenetic treatment for obesity. The first goal describes the theoretical developments of this research project and the second goal refers to the practical implications of the research. We present both of these, concisely, in what follows.

Theoretical developments. The most noticeable contribution this research project has brought, in our view, is the evidence that eating behavior is an automatic one, difficult to control for everyone (not just obese individuals). In obese individuals, the failure to control eating

behavior is obvious, as the other factors that lead to obesity (e.g. genetic, dietary patterns etc.) act and force them to attempt to control this behavior. Lean people are not much better at controlling this behavior, it is just they do not have to do this on a daily basis.

Another important finding is related to the association between BMI and food addiction symptoms. The concept of food addiction needs much more research for its value and clinical utility to be established, but this association is an important and encouraging one for the field, especially considering our results were obtained on a non-treatment seeking sample.

Last, but not least, we have highlighted important relationships between BMI and certain cognitive-behavioral factors (irrational food beliefs, dysfunctional eating behaviors, body self esteem, unconditional self acceptance). While it is not clear yet if these factors are etiopathogenetic or not it is clear they are predictors of high BMI and research has to focus on these factors and clarify weather they are relevant to the etiopathogenesis of obesity or mere symptoms.

Practical implications. This thesis also resulted in important practical contributions to the field. In our studies we found a series of psychological factors relevant to individuals with obesity. Analyzing all these factors, we developed a new, enhanced treatment of obesity. The enhanced treatment we have designed based on all these results stems from the behavioral treatment of obesity, but addresses its main limitations. This program introduces elements of rational emotive behavior therapy to restructure irrational food beliefs (that lead to dysfunctional eating behavior) and general irrational beliefs (that lead to dysfunctional emotions and other relevant dysfunctional behaviors, i.e. lack of physical activity). Also, the enhanced treatment conceptualizes eating as an automatic behavior and introduces implementation intention and rational anticipation techniques as means of improving control over automatic behavior. In addition, elements from addiction therapy have been incorporated in the treatment not only to ensure good results as far as food addiction symptoms are concerned, but also to ensure long lasting results. Other techniques and facilities of the program (e.g. participants' discussion group) also aim to ensure long lasting results.

This new treatment aims, first of all, for better long term maintenance of results. Follow up results are encouraging, as data at 6 months showed that participants in the enhanced group maintain their weight, while participants in the standard group start to regain weight, but they need to be confirmed at longer follow ups. In addition to this main goal of long term maintenance of results, the new enhanced treatment proved to be superior to the standard treatment in results concerning quality of life (e.g. lower depression rates, lower dysfunctional eating behavior etc.). And also, patients who underwent the new treatment obtained results sooner in treatment than patients in the standard treatment, suggesting that a shorter version of the treatment could be designed that could ensure the same results, but that would be more cost-effective.

Limitation and future directions. Important questions remain. First, the etiological role of irrational food beliefs, emotional eating and unconditional self-acceptance for weight must be confirmed at long-term follow-up. Also, it remains to be seen if the significant changes we have obtained for the enhanced treatment group (in terms of depression) will contribute to long-term quality of life (in the absence of further weight loss) or if, apart from quality of life, it may have an impact on weight maintenance. Then we have to see if the differences in hypothesized mediators of change that we have obtained between the two treatments (irrational eating, emotional eating, cognitive restraint, self-control, unconditional self-acceptance, general irrational beliefs) prove to be mechanisms of change for a better quality of life, and if they impact on weight maintenance or not.

Future research should investigate the role of cognitive restraint and self-control in the management of weight. It is quite clear that low levels of both are detrimental to weight management. High levels of cognitive restraint have been shown to be predictors of eating disorders and poor control of eating behavior. Also, research into automatic behaviors and their control is welcome. We need more efficient techniques to improve long-term control of automatic behaviors. More results are needed to clarify the features and clinical utility of food addiction. Based on such results, obesity treatment should be enriched with useful techniques from addiction therapy or new techniques. New developments into automatic behaviors and their long-term control will probably impact on addiction and control of addictive behaviors, too.

Obesity remains a field of extreme importance for public health. We have tried to answer some questions with relevance for a psychological, etiopathogenetic approach to obesity. We believe we have answered some important questions, while also raising new ones. Research in all areas related to obesity needs to focus on finding more effective, etiopathogenetic treatments. This research project has brought significant findings related to eating behavior and has suggested some psychological factors that are potential etiological mechanisms for obesity. Further research is needed to confirm this role and the validity of the etiopathogenetic treatment that rests upon them.

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