

# A Pilot Study of Obesity Management: Contributions of **Cognitive-Behavioral Group Therapy to Stress, Anxiety,** and Emotional Eating

Un Estudio Piloto sobre el Manejo de la Obesidad: Contribuciones de la Terapia Cognitivo-Conductual Grupal al Estrés, la Ansiedad y la Alimentación Emocional

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Abstract. Objetive. Cognitive-Behavioral Group Therapy (CBGT) is a group approach that assesses the interconnections between thoughts, emotions, and behaviors in a group setting. This study aimed to assess the feasibility and preliminary effectiveness of a CBGT protocol focused on emotion regulation, in reducing emotional reactivity and its potential impact on components of emotion-driven eating behaviors. Method. Twenty participants underwent an 8-week intervention, with half receiving psychoeducational intervention and the other half receiving CBGT with a focus on emotional regulation. We used questionnaires to assess anxiety and eating behavior, and we measured psychophysiological changes through cortisol levels and heart rate variability. Results. After six weeks, the CBGT group had lower scores for emotional and uncontrolled eating, along with an increase in parasympathetic modulation and a decrease in cortisol levels. These results suggest that CBGT may hold potential for improving emotional regulation and reducing emotion-based eating behavior; however, further research is needed to confirm its effectiveness.

Keywords. Eating Behavior, Anxiety, Stress, Group Therapy, Obesity

Resumen. Objetivo. La Terapia Cognitivo-Conductual Grupal (TCCG) es un enfoque grupal que evalúa las interconexiones entre pensamientos, emociones y comportamientos en un entorno grupal. Este estudio tuvo como objetivo evaluar la viabilidad y la efectividad preliminar de un protocolo de TCCG enfocado en la regulación emocional, en la reducción de la reactividad emocional y su posible impacto en los componentes de los comportamientos alimentarios impulsados por las emociones. Método. Veinte participantes se sometieron a una intervención de 8 semanas, con la mitad recibiendo una intervención psicoeducativa y la otra mitad recibiendo TCCG con un enfoque en la regulación emocional. Utilizamos cuestionarios para evaluar la ansiedad y el comportamiento alimentario, y medimos cambios psicofisiológicos a través de niveles de cortisol y variabilidad de la frecuencia cardíaca. Resultados. Después de seis semanas, el grupo de TCCG presentó puntuaciones más bajas en la alimentación emocional y descontrolada, junto con un aumento en la modulación parasimpática y una disminución en los niveles de cortisol. Conclusión: Estos resultados sugieren que la TCCG puede tener potencial para mejorar la regulación emocional y reducir el comportamiento alimentario basado en emociones; sin embargo, se necesita más investigación para confirmar su efectividad.

Palabras clave. Primera Ayuda Psicológica, voluntarios, empatía, estrategias de afrontamiento.





# Introduction

The number of overweight individuals has significantly increased in the past decade, and this rise can be attributed to a combination of genetic, lifestyle, and eating-related factors (Murray et al., 2020). With respect to this, the World Health Organization (WHO, 2024) reported that in 2022, approximately 43% of the global adult population was classified as overweight, and 16% was living with obesity. Consequently, overweight and obesity are currently recognized as a risk factor for the onset of physical illness and mental disorders and a public health issue of pandemic proportions (Dassen et al., 2018; Leutner et al., 2023).

Primary clinical evidence indicates that both obesity and overweight are linked to elevated glucose levels, which exacerbate adipose tissue dysfunction. This dysfunction exacerbates insulin resistance, promotes weight gain, and results in metabolic dysfunction and inflammatory responses. These changes can be initiated in the overweight condition and become more pronounced in obesity, with obesity representing a more severe stage (Barbosa & Carvalho, 2023; Lafortuna et al., 2017). Accumulation of adipose tissue is acknowledged as a complex condition. Evidence indicates the interaction of genetic, hormonal, behavioral, and lifestyle factors, alongside an obesogenic environment and cognitive and emotional response (Bose et al., 2009; Dallman, 2010; Kollei et al., 2018; Silva, 2015).

Beyond the well-established physical health factors extensively discussed in the literature on overweight and obesity, cognitive and emotional components have emerged as critical elements in explaining obesogenic behaviors. These factors not only contribute to the etiology of increased adipose tissue accumulation but also pose significant challenges for long-term treatment, particularly in the management of glucose glycemic control, weight maintenance, and the increase in weight regain (Raman et al., 2013, 2020).

The relationship between emotional responses and eating behavior is explained by theoretical models that link cognitive and emotional vulnerabilities to human eating patterns. From this perspective, cognitive and emotional factors interact, demonstrating that individuals under stress, particularly those with emotional dysregulation, may exhibit eating behaviors influenced by emotions, disinhibition, and cognitive biases. These behaviors often serve as coping mechanisms in distressing situations (Kollei et al., 2018; Van Strien et al., 2014). The cognitive and emotional components predicted in the concept of human eating behavior, including cognitive control, disinhibition, and emotional eating, are indeed the focus of one of the most generally recognized hypotheses about the relationship between emotion and eating behavior (Arexis et al., 2023).

Emotional eating refers to the excessive consumption of food in response to negative emotions or stress, serving as a coping mechanism where highly palatable foods alleviate psychological distress (Bilici et al., 2020). In contrast, cognitive restriction involves the mental effort to limit food intake to control eating behavior (Aoun et al., 2019). Disinhibited eating is marked by overeating in response to external food cues, driven by their salience and reduced self-regulation (Esteves et al., 2012). While these eating behaviors are often considered non-psychopathological, they can become dysfunctional when associated with emotional difficulties or stress-related disorders, such as anxiety, depression, and eating disorders (Arexis et al., 2023; Waller & Osman, 1998).

Stress appears to be a critical factor as it influences emotional regulation and facilitates the occurrence of unhealthy eating behaviors, such as increased emotional eating and reward-driven eating (Verdejo-Garcia et al., 2019). In obesogenic environments, stress can often result in an increase in the consumption of hyperpalatable foods, which temporarily alleviate distress but reinforce unhealthy eating patterns and weight gain (Leigh et al., 2018; Yau & Potenza, 2013).

Stress conditions may increase individuals' preference for hyperpalatable and comfort foods, as



emotional distress can trigger the consumption of such foods as a coping mechanism, activating reward pathways to alleviate emotional distress (Leigh et al., 2018; Sominsky & Spencer, 2014). Neurobiological evidence links these behaviors to alterations in hypothalamic-pituitary-adrenal (HPA) axis reactivity, potentially influencing the lateral hypothalamic area (LHA) to promote ghrelin-mediated consumption of sugar- and fat-rich foods. Additionally, the ventral tegmental area (VTA) is implicated in enhancing reward-seeking behaviors through increased dopaminergic transmission (Linders et al., 2022; Rebello & Greenway, 2016). Clinical studies further support this model, showing that cortisol secretion and sensitivity are associated with increased adipose tissue accumulation in overweight and obese patients compared to eutrophic individuals (Lengton et al., 2022; Martens et al., 2021).

The stress reaction is directly correlated with emotional expression. A set of psychophysiological and behavioral changes occurs when individuals face stressors, preparing the organism to cope with and adapt to challenging situations. However, when the stressor persists, it leads to allostatic overload, initially characterized by an imbalance in the autonomic nervous system (ANS), with increased sympathetic activity and reduced parasympathetic regulation.

This imbalance contributes to the maintenance of a state of emotional distress and predisposes individuals to emotional dysregulation and anxiety-related conditions (Bian et al., 2022; Candia-Rivera et al., 2023). ANS dysfunction, particularly in psychiatric disorders, is often observed through alterations in heart rate variability (HRV). This measure provides insights into the sympathetic and parasympathetic branches of the ANS, which are crucial for understanding stress reactivity and adaptive emotional responses in mental health conditions (Agorastos et al., 2023). In fact, clinical evidence observed through HRV parameters suggests a sympathovagal imbalance, characterized by heightened sympathetic activity and reduced parasympathetic modulation, in overweight and obese individuals compared to eutrophic weight controls (Strüven et al., 2021; Yadav et al., 2017).

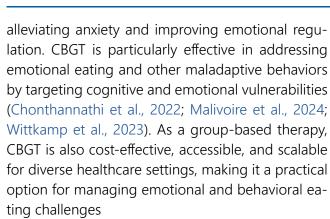
Michopoulos et al. (2015) investigated the link between emotional dysregulation and eating patterns in obese children, finding that childhood trauma contributes to emotional dysregulation, which, in turn, influences emotional eating behaviors. Neurological evidence from functional magnetic resonance imaging (fMRI) studies revealed increased limbic activity and negative emotions associated with anticipatory well-being responses to food consumption in women, a phenomenon absent in those with neutral or positive mood states (Bohon et al., 2009). These findings suggest that emotional eating activates reward pathways as a compensatory response to emotional distress (Volkow et al., 2011). Supporting this, Chua et al. (2004) found that melancholic-themed films increased negative emotions and food consumption in 42 participants. Similarly, a meta-analysis of 33 studies with 2491 participants confirmed a direct correlation between negative emotional states and increased food intake (Cardi et al., 2015).

Conventional treatments for managing overweight and obesity, such as dietary counseling and physical activity recommendations, often show limited long-term efficacy (Baker et al., 2022; Dansinger et al., 2007; Wadden et al., 2020). This limitation is largely attributed to their inability to address the emotional and behavioral factors underlying eating patterns. Emotional dysregulation and maladaptive responses to stress are key contributors to overeating and weight gain, yet they remain unaddressed in many traditional interventions.

Cognitive-Behavioral Therapy (CBT) is a promising approach for managing overweight and obesity by targeting emotional regulation and modifying eating behaviors. It promotes adherence to healthy lifestyle changes and addresses the psychological and emotional factors underlying obesogenic behaviors (Cha et al., 2020). Among CBT methods, Cognitive-Behavioral Group Therapy (CBGT) stands out as an effective, evidence-based intervention for

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This pilot study aimed to assess the feasibility and preliminary effectiveness of a CBGT protocol focused on emotion regulation, in reducing emotional reactivity and its potential impact on components of eating behavior. To achieve this, we assessed the stress response through salivary cortisol levels and sympathetic/parasympathetic balance, as indicators of emotional dysregulation. Additionally, the study explored the relationship between emotional states and emotion-driven eating behaviors, hypothesizing that improvements in emotional regulation would be associated with positive changes in eating behavior components. To evaluate the efficacy of the protocol, participants were divided into two groups: an intervention group, which received CBGT focused on emotion regulation, and a control group, which attended psychoeducational sessions addressing general dietary and health behaviors.

## Method

#### Participants

A power analysis was conducted using G\*Power (version 3.1.9) to estimate the required sample size for this pilot study, which focused on methodology and feasibility for preliminary data collection (Lancaster et al., 2004). Using an estimated power of 90%, an alpha level of 5%, and an effect size of .8, the minimum sample size required was determined to be N = 8 per group (Critical F = 5.31). Therefore, the sample size of N = 10 per group used in this study exceeds the minimum requirement, ensuring that the study hypothesis could be adequately tested while maintaining the exploratory nature of pilot studies.

Participants were recruited through social media advertisements and posters in health clinics. A total of 22 volunteers (10 females and 12 males) expressed interest and attended an initial session to receive study information and undergo screening. Inclusion criteria included being aged 18-60, having a BMI of 30 or greater, difficulty losing weight, and no history of eating disorders, depressive symptoms, or psychotropic medication use. All 22 volunteers met the criteria and were randomly assigned to either the control group (n = 10) or the CBGT group (n = 12). Both groups participated in a baseline data collection session. During the intervention, dropout criteria included starting weight loss medication, missing three consecutive sessions, or developing a physical or psychiatric condition. Two participants in the CBGT group were withdrawn for missing three consecutive sessions, leaving a final follow-up sample of 20 participants: 10 in the control group and 10 in the CBGT group (Figure 1). After completing the study, control group participants were invited to join the CBGT clinical protocol.

#### **Ethical Aspect**

This study was approved by the Ethics Committee of Universidade Potiguar (CAAE: 00336818.8.0000.5296, N° 2.955.998) and conducted in accordance with the guidelines of the National Ethics Committee (CONEP) and the principles outlined in the Declaration of Helsinki. Participants were fully informed about the study's objectives, potential benefits, and risks before their participation. All participants signed an Informed Consent Form, ensuring their understanding and voluntary agreement to take part in the research.

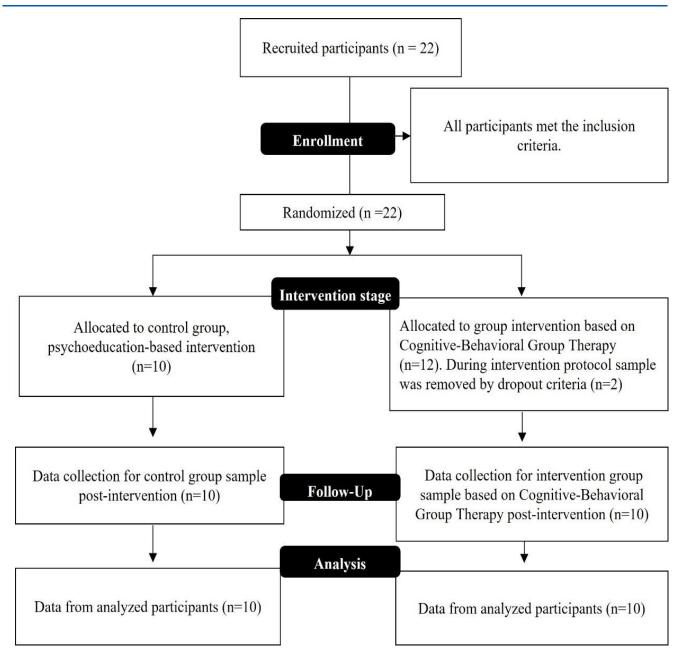
## **Clinical Screening**

#### **Clinical Interview**

To determine eligibility based on inclusion criteria, all volunteers participated in a session conduc-



Figure 1. Flowchart illustrating the recruitment, randomization, and follow-up of participants in the intervention and control group



*Note.* Flowchart illustrating the recruitment, randomization, intervention, and follow-up process for participants. A total of 22 participants were recruited, all of whom met the inclusion criteria. Participants were randomly assigned to the control group (n = 10) or the intervention group (n = 12). During the intervention stage, 2 participants from the intervention group were removed based on dropout criteria, resulting in a final analyzed sample of 20 participants.



ted by the research team and a clinical psychologist from the University Psychology Service-School. During this session, a structured questionnaire was initially administered, collecting sociodemographic data and general information on physical health, lifestyle, eating habits, sleep habits and medical history, with a particular focus on the clinical history of obesity. Screening for the presence of previous eating disorders, depressive disorders, and other psychiatric conditions was performed using the Structured Clinical Interview for DSM-5 (SCID-5-CV, Osório et al., 2019). Additionally, the Beck Depression Inventory (BDI-II) was applied to precisely assess depressive symptomatology.

#### Anthropometric Measurements

During the interview, anthropometric parameters such as weight and height were measured to calculate the Body Mass Index (BMI). The BMI was calculated as BMI =  $W/H^2$ , where W represents the participant's current weight in kilograms and  $H^2$  represents the participant's height in meters squared. Weight was measured with an electronic scale and height with a traditional stadiometer. We used the eligibility criterion for BMI > 30.0kg/m<sup>2</sup> (obesity), following the classification of the World Health Organization (Oliveira et al., 2012).

#### Instruments

The Three Factor Eating Questionnaire (TFEQ-R21)

The TFEQ-R21 is a globally recognized and revised instrument that is designed to evaluate components of human eating behavior using the three-factor model: cognitive restraint (CR), emotional eating (EE), and uncontrolled eating (UE). The scale consists of 21 items on a Likert scale ranging from 1 to 4, with scores calculated on a scale ranging from 0 to 100 points and raw scores converted to percentages. The revised questionnaire was proposed by Tholin et al. (2005), and the Brazilian translation and psychometric validation were conducted by De Me-

deiros et al. (2017). The Brazilian version replicated the hypothetical model with good fit indices ( $\chi^2$ /df = 2.24, CFI = .97, TLI = .96, and RMSEA = .05), as observed in confirmatory factor analyses. Additionally, reliability was assessed using Cronbach's alpha, demonstrating adequate internal consistency for the three domains (UE:  $\alpha$  = .83, EE:  $\alpha$  = .92, and CR:  $\alpha$  = .83). This measure was designed to assess the participants' eating behaviors.

#### Beck Depression Inventory (BDI-II)

A self-report instrument consisting of 21 questions designed to assess depression symptoms in adults (Beck et al., 1996). For the Brazilian population version, the Cronbach's alpha coefficient for this instrument in the community sample was .93, indicating a high level of internal consistency (Gomes-Oliveira et al., 2012). It is suggested for people between the ages of 17 and 80. Because the absence of depressive symptoms was one of the inclusion criteria, this measure was used for screening and eligibility purposes in the study, with a cutoff score of >13 points indicating a clinical sample, according to the instrument's technical manual.

#### The State-Trait Anxiety Inventory (STAI)

The STAI is a self-report instrument commonly used to measure trait (STAI-T) and state anxiety (STAI-S). It is frequently applied in clinical research and settings to assess and diagnose anxiety-related disorders (Spielberger, 2010). The two scales each have 20 Likert scale items. The goal of this measure was to assess the occurrence of anxious symptoms in the current state as well as an anxiety-prone functional profile. The instrument asks the participant to describe how they feel "at this moment" to measure the emotional state factor, whereas the trait factor considers the participant's response to how they "usually feel". The Brazilian version used in this study was based on the adaptation by Fioravanti et al. (2006), which demonstrated good internal consistency of the items analyzed using Cronbach's Alpha, with a score of .89 for state anxiety (STAI-S), re-



flecting current worry states, and .88 for trait anxiety (STAI-T), associated with a predisposition to anxious thoughts and a stable mood state. Evidence for trait anxiety was further tested by Andrade et al. (2001), who observed that the STAI-T items consistently infer a pattern of anxiety maintained by neuroticism and a predisposition to mood-worrying dimension.

#### A daily record of food intake

The study implemented a self-monitoring food diary for the CBGT group, a key strategy in weight loss cognitive-behavioral therapy, to help participants track cognitive patterns and emotions influencing eating behavior. A customized diary was developed for recording meals, behaviors, emotions, thoughts, motivations, binge episodes, and session goals. It was incorporated as a qualitative self-monitoring tool, adapted from standard CBT guidelines (Gormally et al., 1982; Lindgreen et al., 2018; Schumacher et al., 2021; Wilson & Vitousek, 1999).

#### Heart rate variability measures

Following the standardization by Esco and Flatt (2014), heart rate variability (HRV) was measured using an ECG finger monitor (ithlete Ltd, Southampton, UK) for 15 minutes under resting conditions to assess autonomic nervous system (ANS) activity through R-R interval analysis, allowing inferences about the balance between the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). Sympathetic modulation was evaluated using SDNN (standard deviation of normal RR intervals), which reflects overall heart rate variability with sympathetic predominance, and LF (low frequency, .04 - .15 Hz), which primarily reflects sympathetic activity with minimal vagal influence. Parasympathetic modulation was assessed using RMSSD (root-meansquare of successive differences), reflecting shortterm PNS activity; PNN50 (percentage of adjacent R-R intervals differing by more than 50 ms), which indicates parasympathetic predominance; and HF (high frequency, .15 - .4 Hz), a marker of vagal tone that reflects parasympathetic activity (Table 1). Measurements were conducted in a controlled, quiet environment during the pre-intervention and follow-up sessions, prior to administering other instruments. HRV parameters were selected based on existing literature (Georgieva-Tsaneva, 2019; Hartmann et al., 2019; Shaffer & Ginsberg, 2017).

#### Salivary cortisol concentrations

Salivary cortisol concentrations were measu-

Time Domain	Parameters	Units	ANS analysis	
SDNN	Standard Deviation of N-N intervals	ms	SNS	
rMSSD	Root mean square of successive R-R interval	ms	PNS	
PNN50	Percentage of successive RR intervals that differ by more than 50 ms	%	SNS/PNS†	
Frequency Dor	nain			
HF	Power of high-frequency range .15 Hz a .4 Hz	%	PNS	
LF	Power of low-frequency range .04 HZ a .15 HZ	%	SNS	

#### Table 1. Heart rate variability assessment parameters for time and frequency domains

*Note.* This table presents heart rate variability (HRV) parameters analyzed in the time and frequency domains. SDNN reflects overall variability (SNS), rMSSD and HF indicate parasympathetic activity (PNS), and PNN50 represents a balance between SNS and PNS. LF reflects sympathetic activity (SNS), corresponding to the low-frequency range (.04 - .15 Hz), while HF corresponds to the high-frequency range (.15 - .4 Hz) and parasympathetic activity (PNS).



red before the intervention and 6 weeks later (follow-up). Participants were instructed to collect saliva immediately upon awakening in the morning, no later than 8:00 a.m., to assess baseline cortisol concentrations upon awakening. Participants were also advised not to drink, eat, or brush their teeth for at least 60 minutes prior to collection. Saliva samples were collected in Salivette® tubes (Sarstedt, Germany) and stored at -80°C until analysis. Before analysis, samples were centrifuged at 10,000g for 20 minutes, and cortisol concentrations were determined using a commercia-Ily available kit (DSL-10-671000 ACTIVE® cortisol enzyme immunoassay - EIA). This study opted to assess baseline morning cortisol as a practical and reliable measure of stress response. Interventions affecting stress or basal HPA axis functioning are often associated with flatter cortisol slopes, which can be evaluated using waking morning salivary cortisol levels (Adam & Kumari, 2009). The 6-week gap for salivary cortisol measurement was implemented to minimize potential acute influences of the intervention on this parameter.

#### Procedures

After screening and obtaining participants' consent, a second data collection session was scheduled. Participants received the salivary cortisol collection kit along with general instructions for proper sample collection and information regarding the scheduled data collection session. On the designated day, the salivary cortisol samples were checked, and individual data collection sessions were conducted according to a predefined schedule. The data collection process began with the assessment of heart rate variability, followed by the application of the anxiety scale and the eating behavior questionnaire. Participants were then randomly divided into two groups: a control group (CG), which focused on psychoeducation, and a Cognitive-Behavioral Group Therapy (CBGT) group focused on emotional regulation. Participants were instructed

to attend weekly sessions but were not informed about their group allocation.

The CG received an eight-session intervention with a script of activities centered on psychoeducation in thematic areas linked to an emotional reaction, emotional eating, and healthy eating behaviors. Following that, six weeks after the previous meeting, follow-up data were collected.

In the CBGT group, the intervention performed was cognitive-behavioral psychotherapy focused on emotional regulation and elements of cognitive restructuring, with eight weekly meetings, lasting two hours each, and pre-established interventions. The intervention was structured into sessions organized around thematic axes, focusing on psychoeducation, cognitive restructuring, skills training, and strategies for recognizing, monitoring, and managing situations related to emotions and eating behaviors. The sessions included skills training for emotional regulation, behavioral techniques such as graduated exposure and behavioral activation, as well as relapse prevention strategies (Table 2). Techniques and activities were adapted from different protocols focused on emotional regulation and stress management to meet the specific needs of the participants (Castelnuovo et al., 2017; Guerrini Usubini et al., 2022; Neufeld et al., 2021; Saranapala et al., 2022; Torres et al., 2020).

The study opted to collect data on psychophysiological measures and self-reported measures of anxiety and eating behavior 6 weeks after the intervention. This decision was made to observe longterm effects while avoiding the immediate impact of the therapeutic intervention on these measures and minimizing potential bias associated with post-intervention effects (Clarke et al., 2018).

#### **Statistical Analysis**

The Kolmogorov-Smirnov test was used to determine the data's normality. Data from the Three Factor Eating Questionnaire - R21 (TFEQ-R21) assessments, the State-Trait Anxiety Inventory (STAI), heart rate variability, and circulating cortisol con-



Session	Theme	Techniques
01	<ul> <li>Group presentation, objectives, and the therapeutic contract</li> <li>Introduction to Cognitive-Behavioral Group Therapy</li> <li>Presentation of the daily food intake record</li> </ul>	<ul> <li>Psychoeducation</li> <li>Socratic questioning</li> <li>Daily record of food intake</li> </ul>
02	- Cognitive-Behavioral Model of Eating Behaviors - Stress and obesity	- Psychoeducation - Cognitive restructuring - Guided discovery
03	<ul> <li>Distinction between losing weight and maintaining weight</li> <li>Difficulties losing weight</li> <li>Automatic negative thoughts</li> <li>Emotion regulation and stress coping</li> </ul>	<ul> <li>Psychoeducation</li> <li>Cognitive restructuring</li> <li>ABC functional analysis</li> <li>Reframe negative thoughts</li> <li>Guided discovery</li> </ul>
04	- Core and Intermediate Beliefs	<ul> <li>Psychoeducation</li> <li>Cognitive restructuring</li> <li>ABC functional analysis</li> <li>Reframe negative thoughts</li> <li>Guided discovery</li> </ul>
05	- Cognitive Behavioral Approach to Emotional Eating	<ul> <li>Psychoeducation</li> <li>ABC functional analysis</li> <li>Daily record of food intake</li> <li>Guided discovery</li> </ul>
06	- Recognizing and Managing Emotions - Emotional regulation and emotional eating	<ul> <li>Relaxation and stress reduction techniques</li> <li>Fact-checking</li> <li>Successive approximation</li> <li>Mindfulness eating</li> <li>The downward arrow</li> </ul>
07	<ul> <li>The role of healthy habits (physical activity, coping with stress, and nutritional education)</li> <li>Relapse prevention strategies</li> </ul>	<ul> <li>Psychoeducation</li> <li>Coping cards</li> <li>Fact-checking</li> <li>Mindfulness eating</li> <li>Fact-checking</li> </ul>
08	- Ending stage of the group and feedback	- Feedback

#### Table 2. Description of the intervention performed in the experimental group (CBGT)

*Note.* The intervention protocol was conducted over 8 consecutive weeks, with each session structured around specific themes and corresponding cognitive-behavioral techniques.



Results

naire)

centrations were then examined using repeated measurements. To support the assumption of equal variance of differences between conditions, ANO-VA and Mauchly's sphericity test were performed. Partial eta squared (np2) was used to calculate the size of the effect. The reference values for effect size in this study were classed as small (.20), moderate (.50), and large (.80; Cohen, 1988).

Using scores from the TFEQ-R21 instrument, multiple linear regression models were used to examine the post-intervention predictive effect of salivary cortisol, sympathetic modulation (SDNN and HF frequency parameters), and parasympathetic modulation (rMSSD parameters, PNN50, and LF frequency) on emotion-based eating behavior domains. With a p-value of .05, all differences were judged statistically significant. Data are expressed as Mean ± Standard Deviation (*SD*) or Standard Error of the Mean (SEM). Statistica 7.0 for Windows was used to execute statistical procedures.

**TFEQ-R21 (Three Factor Eating Question-**

Statistical analysis of the emotional eating (EE)

domain indicated a group effect [F(1,18) = 7.285, p= .01,  $\eta_p^2$  = .28], a session effect [*F*(1,14) = 31.277, p = .001,  $\eta_p^2 = .69$ ], and a group × session interaction effect [ $F(1,14) = 6.39, p = .02, \eta_p^2 = .44$ ]. A post-hoc Tukey correction applied to the group × session interaction effect revealed that the control and CBGT groups did not differ statistically at baseline. However, post-hoc analysis revealed that the CBGT group showed a significant reduction in EE scores during the follow-up compared to their baseline scores. Additionally, when comparing EE scores between the CBGT and control groups at the follow-up phase, the CBGT group demonstrated lower scores, indicating that participants who underwent the emotional regulation intervention reported improvements in emotion-based eating behavior (see Table 3).

The statistical analysis for the domain of uncontrolled eating (UE) found no group effect [*F*(1,18) = 0.54, *p* = .40], but did find a session effect [*F*(1,18) = 20.52, *p* = .002,  $\eta_p^2$  = .52] and a group × session interaction effect [*F*(1,18) = 13.50, *p* = .001;  $\eta_p^2$  = .42]. The post-hoc Tukey test for group × session interaction demonstrated that the CBGT group significantly reduced self-reported UE scores when comparing fo-

							0 Weeks)
Instruments		Baseline ( $t = 0$ )		Follow-up ( $t = 6$ weeks)			
		CG	CBGT	р	CG	CBGT	р
TFEQ-R21	EE	20.2 ± 2.1	20.5 ± 2.5	.901	18.4 ± 2,6	14.7 ± 1.8*	.005
	UE	23 ± 2.1	26.4 ± 4.9	.458	20 ± 2.4	16.2 ± 2.2*	.001
	CR	14.1 ± 2.6	12.8 ± 2.4	.677	17.8 ± 1.1*	18.5 ± 1.6*	.001
STAI	STAI-S	52.1 ± 3.7	45.2 ± 5.16	.102	44.4 ± 5.7	38.8 ± 4.6	.437
	STAI-T	51.9 ± 3.5	53.2 ± 4.1	.912	50.8 ± 6.2	50.2 ± 3.6	.967

**Table 3.** The anxiety and emotional eating behavior dimensions at baseline (t = 0) and 6 weeks post-intervention (t = 6 weeks)

*Note.* Repeated measures ANOVA was used, followed by Tukey's correction. Statistical significance was set at \*p < .005 for comparisons within the CBGT group and between the CBGT and control groups (CG) in EE scores 6 weeks post-intervention. For UE scores, a significant level of \*p = .001 indicated statistical differences when comparing the CBGT group at follow-up versus baseline. Additionally, differences in UE scores were observed between the CBGT and CG groups at follow-up. For CR scores, a significant level of \*p = .001 indicated statistical differences when comparing both groups at follow-up versus baseline. The p values in the table represent the significant levels for interaction effects in EE and UE, as well as session effects in CR. The data is presented as mean  $\pm$  *SD*.

llow-up to baseline. Additionally, significant differences were observed when comparing the CBGT group to the CG at the follow-up phase, indicating that the reduction in UE scores was specifically related to the group that underwent the intervention focused on emotional regulation (see Table 3).

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The repeated measures ANOVA did not demonstrate a group effect [F(1,18) = 0.94, p = .34] or a group × session interaction effect [F(1,18) = 1.56, p = .22] in the analysis of the last domain, cognitive restraint (CR), but a session effect was detected [F(1,18) = 38.12, p = .001,  $\eta_p^2 = .67$ ]. As a result, a change in this factor is proposed over the sessions for both groups (see Table 3).

#### State-Trait Anxiety Inventory (STAI)

The STAI instrument was used in this study to evaluate the effect of intervention on anxiety. Repeated measures ANOVA revealed a significant effect of sessions on STAI-S [F(1,18) = 12.55, p = .001,  $\eta_p^2 = .41$ ], but no significant effects were observed for groups [F(1,18) = 3.00, p = .10] or the group × session interaction [F(1,18) = 0.65, p = .82]. These results indicate that all participants, regardless of group, experienced a reduction in state anxiety following the intervention. For STAI-T, no significant effects were found for groups [F(1,18) = 0.08, p = .77], sessions [F(1,18) = 2.98, p = .10], or the group × session interaction [F(1,18) = 0.84, p = .36, see Table 3].

#### Heart Rate variability

Heart rate variability measures were employed in the study as an indicator of sympathetic/parasympathetic modulation in emotional reaction. For the time domain, considering the SDNN parameter, the statistical analysis did not show any effect of the group [F(1,14) = 0.08, p = .77], sessions [F(1,14)= 0.19, p = .66], or groups × sessions interaction [F(1,14) = 2.25, p = .15].

The statistical analysis of the rMSSD parameter revealed no influence of group [*F*(1,14) = 1.33, *p* = .26], but it was able to notice an effect of sessions [*F*(1,14) = 5.11; *p* = .04,  $\eta_p^2$  = .26] and of groups

× sessions interaction [F(1,14) = 11.55, p = .004;  $\eta_p^2 = .45$ ]. The Tukey post-hoc correction test revealed that the CBGT group showed a statistically significant increase in the rMSSD parameter at follow-up compared to baseline. Additionally, a higher rMSSD was observed in the CBGT group compared to the CG at follow-up, suggesting that this measure only varied for the CBGT group (see Table 4).

In the evaluation of the PNN50 parameter, the repeated measures ANOVA did not show a group effect [*F*(1,14) = 0.06, *p* = .79], but a session effect was observed [*F*(1,14) = 21.77, *p* = .001,  $\eta_p^2$  = .57], as well as a groups × sessions interaction effect [*F*(1,14) = 10.95, *p* = .003,  $\eta_p^2$  = .37]. For the group × session interaction effect, the Tukey post-hoc correction test revealed that the CBGT group was the only group to show a significant increase in this parameter at follow-up compared to baseline. Additionally, higher PNN50 scores were observed in the CBGT group compared to the CG at follow-up (see Table 4).

The parameters LF% and HF% were employed in the study to evaluate frequency domains. For LF%, a measure of the sympathetic modulation, the repeated measures ANOVA revealed only a group effect  $[F(1,14) = 10.04, p = .005, \eta_p^2 = .35]$ , with no statistical differences for the session effect [F(1,14) = 0.06, p = .96], as well as the interaction between groups and sessions [F(1,14) = 0.19, p = .66]. The data indicate that the CBGT group had a higher frequency of sympathetic modulation than expected by the LF% parameter in general, although there was no decline during the sessions (see Table 4).

Regarding the parameter of HF%, an indicator of parasympathetic modulation, statistical analysis revealed a group effect [*F*(1,14) = 46.09, *p* = .001,  $\eta_p^2$  = .71], a session effect [*F*(1,14) = 13.52, *p* = .001,  $\eta_p^2$  = .42], and a group × session interaction [*F*(1,14) = 13.94; *p* = .001,  $\eta_p^2$  = .42]. The Tukey correction test observed a general increase in HF% for the CBGT group. Furthermore, the test for the group × session interaction effect revealed that the CBGT group showed a significant increase in HF% when comparing follow-up to baseline. Additionally, higher HF%

**Table 4.** The parameters of heart rate variability at baseline (t = 0) and 6 weeks after the intervention ended (t = 6 weeks)

Parameters			Baseline ( $t = 0$ )			Follow-up (t= 6 weeks)		
			CG	CBGT	р	CG	CBGT	р
TIME	SNS	SDNN	37.5 ± 6.4	36.2±11.1	.310	34.3 ± 5.5	34.5 ± 4.3	.630
DOMAIN	modulation							
	PNS	rMSSD	32.8 ± 3.8	29.2 ± 5.6	.803	30.5 ± 2.4	37.5 ± 5.4*	.004
	modulation	PNN50%	24.3 ± 2.9	17.3 ± 11.3	.227	26.8 ± 2.6	32.0 ± 8.9*	.003
FREQUENCY DOMAIN	SNS modulation	LF%	53.1 ± 5.8	69.3 ± 11.8*	.062	55.3 ± 7.5	66.4 ± 22.1*	.005
	PNS modulation	HF%	32.2 ± 3.3	38.3 ± 8.7	.132	31.6 ± 3.8	55.6 ± 11.7*	.001

*Note.* Repeated measures ANOVA followed by Tukey's correction was used. For SDNN, no significant differences were observed. For rMSSD, statistical significance was found (\*p < .004) when comparing the CBGT group at follow-up versus baseline and CBGT versus CG at follow-up. For PNN50%, significance was observed (\*p < .001) when comparing the CBGT group at follow-up versus baseline and CBGT versus CG at follow-up. For LF%, a group effect was observed, with generally higher LF% in the CBGT group (\*p < .005). For HF%, significant differences were observed (\*p < .001) when comparing the CBGT group at follow-up versus baseline and CBGT versus CG at follow-up. For LF%, a group effect was observed, with generally higher LF% in the CBGT group (\*p < .005). For HF%, significant differences were observed (\*p < .001) when comparing the CBGT group at follow-up versus baseline and CBGT versus CG at follow-up. The p values in the table represent the level of significance for interaction effect (rMSSD, PNN50%, and HF%) and group effect for LF%. All data are presented as mean ± *SD*.

was observed in the CBGT group compared to CG at follow-up (see Table 4). These findings suggest that the increase in this parasympathetic modulation parameter occurred exclusively in the CBGT group.

#### Salivary Cortisol Levels

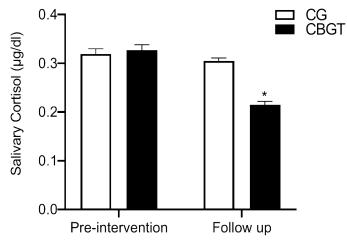
The study used repeated measures ANOVA to evaluate the effect of treatment interventions on waking basal cortisol expression. The analysis revealed a significant group effect [F(1,14) = 6.75, p = .01,  $\eta_p^2 = .24$ ], session effect [F(1,14) = 57.52; p = .001;  $\eta_p^2 = .76$ ], and group × session interaction [F(1,14) = 35.56; p = .001;  $\eta_p^2 = .66$ ]. Tukey's post-hoc correction indicated that the CBGT group showed a statistically significant reduction in cortisol levels at follow-up compared to baseline, as well as when compared to the CG at follow-up. This suggests that only the CBGT group experienced a reduction in waking basal cortisol expression during the waking phase (see Figure 2).

# The predictive effect of emotional response on eating behavior

The study's goal was to predict the efficacy of therapy models on emotional response and its impact on emotional-based eating behavior domains (disinhibition and emotional eating). The predictive influence of basal cortisol and sympathetic and parasympathetic modulation was investigated using multiple linear regression models.

Multiple-linear regression analysis for the control group revealed no predictive model of salivary cortisol on the domains of uncontrolled and emotional eating [F(1,8) = 0.043, p = .84,  $R^2 = .05$ ], nor a predictive effect of sympathetic modulation [F(2,7)= 3.40, p = .09,  $R^2 = .49$ ] or parasympathetic modulation [F(3,6) = 2.85, p = .12,  $R^2 = .58$ ]. As a result, the multiple-linear regression models used in this study could not predict the occurrence of uncontrolled and emotional eating behaviors. However, a tendency toward sympathetic modulation was





**Figure 2.** DWaking salivary cortisol concentrations at baseline (t = 0) and 6 weeks after the intervention ended (t = 6 weeks).

*Note.* Salivary cortisol concentrations for the control group (CG) and experimental group (CBGT) at baseline and 6 weeks post-intervention. \*p < .05 indicates a significant reduction in cortisol levels for the CBGT group at follow-up compared to baseline and versus the CG at follow-up. Only the CBGT group showed a reduction in waking basal cortisol expression. All data expressed as Mean  $\pm$  SEM.

Models		Emotional eating			
Model I (psychoeducation)	β	F	р	$R^2$	
Salivary Cortisol	.251	0.43	.84	.05	
SNS modulation (SDNN+LF)	.257	0.40	.09	.49	
PNS modulation (sMSSD + PNN50% + HF)	.457	0.85	.12	.58	
Model II (Psychotherapy focused on emotion regulation)	β	F	р	$R^2$	
Salivary Cortisol	23	0.431	.71	.19	
SNS modulation (SDNN+LF)	56	2.03	.20	.36	
PNS modulation (sMSSD + PNN50% + HF)	71	5.15	.04*	.72	

Table 5.	The predictive effect	of emotional response	e on the emotional eating domain

*Note.* Multiple-linear regression model. Statistical significance of \*p < .05 for the predictive effect of parasympathetic modulation on emotional eating behavior.





observed, though it did not reach statistical significance (see Table 5). This trend warrants further investigation with a larger sample size to confirm its potential significance.

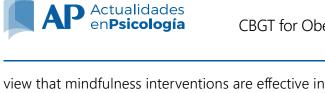
For the CBGT group, considering cortisol as a predictor, multiple-linear regression showed no association between salivary cortisol response and any of the domains of uncontrolled eating and emotional eating  $[F(1,8) = 0.469, p = .71, R^2 = .19]$ . In addition, for sympathetic modulation, the analysis did not observe statistical differences for predictive effect on the eating behavior domains analyzed by the instrument  $[F(2,7) = 2.03, p = .20, R^2 = .36]$ , but it was possible to observe a predictive effect of parasympathetic modulation on the emotional-based eating behavior domains  $[F(3,6) = 5.15, p = .04, R^2$ = .72], suggesting that parasympathetic activation may predict the occurrence of this behavior for the experimental therapeutic group. According to the model, an increase in parasympathetic activation can predict a decrease in emotional eating and uncontrolled eating occurrences (Table 5).

## Discussion

The main findings of this study for participants in the Experimental Therapeutic Group (CBGT) focused on emotional regulation, 6 weeks after the intervention, were: (1) a reduction in emotional eating and uncontrolled eating domain scores; (2) a reduction in state-anxiety domain scores, indicating an improvement in emotional response; and (3) the findings highlight improvements in HRV parameters (rMSSD, PNN50%, and HF%) and a reduction in basal cortisol levels upon awakening 6 weeks post-intervention in the group undergoing the intervention focused on emotional regulation. Additionally, multiple linear regression analysis suggested that increases in these parameters could predict a reduction in emotion-based eating behaviors.

In our study, we observed that the intervention based on a protocol focused on emotional regulation was effective in reducing eating behaviors associated with emotional expression. Some studies in the literature have looked into the relationship between emotional state and behavioral changes, particularly compensatory behaviors for emotional discomfort and stress. Casagrande et al (2020), for example, discovered a positive correlation between an increase in alexithymia symptoms and difficulties in emotional regulation, as well as both positively correlating with body mass index in a clinical sample of individuals diagnosed with alexithymia, a psychiatric condition marked by difficulties in perceiving emotional states, feelings, and bodily sensations. Another study, which looked at the relationship between emotional dysregulation and impulsive eating patterns in a group of bariatric surgery patients, discovered binge eating and overeating during times when the participants reported difficulty with emotional regulation (Benzerouk et al., 2020).

Along with other studies with similar sample characteristics (Debeuf et al., 2020; Torres et al., 2020), our participants had high pre-intervention scores in emotional eating and food disinhibition domains, indicating the occurrence of these eating patterns in this sample of obese individuals. After the intervention, individuals in the psychotherapy group focused on emotional regulation showed a reduction in these areas, suggesting that this intervention model may represent a promising approach to addressing emotional eating behavior. However, it is important to note that the observed effects could also be influenced by other factors. For example, the regulation of eating patterns may be indirectly affected by CBT through improvements in self-esteem and body dissatisfaction, which have a direct impact on weight control (Neufeld et al., 2012). Additionally, the direct effects of applied techniques on components such as self-regulation, motivation, and self-efficacy may also suggest other variables that could influence the observed outcomes (Intarakamhang & Intarakamhang, 2015). Other intervention strategies, such as those focusing on mindfulness for emotional regulation, also support these findings, as O'Reilly et al. (2014) discovered in a systematic re-



reducing obesity-related eating behaviors such as binge eating and emotional eating. In this analysis, the efficacy of these therapies was 87%, and around 63% of the analyzed studies used emotional regulation measures to minimize emotional eating.

Glisenti and Strodl (2012) compared CBT and Dialectical Behavior Therapy (DBT) for the treatment of emotional eating in a group of obese people. Both therapies received 22 sessions throughout the research. The CBT intervention included features such as recognizing beliefs and behaviors related to weight reduction issues, setting therapy goals to address these ideas and behaviors, and providing psychoeducation on healthy eating and increased physical activity. DBT therapies, on the other hand, included emotion regulation, stress, and anxiety management, and skills training to help recognize and control unpleasant emotions. After 8 weeks of follow-up, the DBT group lost 10.1% and 7.6% of their initial body weight, respectively, whereas the CBT group lost 0.7% and 0.6% of their initial body weight. Furthermore, the DBT group demonstrated a reduction in emotional response indicators, including stress reaction, anxiety, and emotional eating.

Similarly to the previously stated study, we were unable to identify an effect of the psychoeducation intervention model on lowering emotion-based eating behavior (see Table 3), despite an increase in cognitive restraint domain scores in both groups. Because the cognitive restraint domain evaluates an individual's effort and use of cognitive resources to regulate food intake and maintain body weight (Bond et al., 2001), this evidence could be explained as a possible influence of psychoeducation on this component of eating behavior. Identical findings have been reported in other investigations. Cash and Hrabosky (2003), for example, investigated the efficacy of psychoeducation in an intervention model aimed at changing body image, which resulted in increased self-esteem, healthy eating attitudes, and decreased anxiety. Another finding demonstrated the effect of psychoeducation on

the eating behavior of anorexic and bulimic patients (Tatham et al., 2016).

In this study, it was determined whether the group intervention model focusing on emotional regulation would have direct effects on emotional response components and, as a result, on features of emotional eating. Anxiety markers, autonomic nervous system modulation, and basal cortisol awakening response were used to accomplish this. The study used an instrument capable of analyzing anxiety characteristics as a trait phenotype inherent in the subject's temperament and an anxious state, a more instantaneous measure of emotional response, to analyze participants' anxiety profiles (Bieling et al., 1998).

In terms of trait anxiety, individuals had a consistent anxiety profile before the intervention that did not differ regardless of the therapeutic paradigm used (see Table 2). This points to the occurrence of an anxious endophenotype, which is unlikely to be altered by the recommended treatment interventions. According to some studies, trait anxiety is a personality dimension based on neuroticism, with a strong tendency to experience emotional patterns associated with psychological discomfort caused by attentional biases to negative stimuli, distress, worries, and anxiety in various aversive events (Debeuf et al., 2020; Mielimąka et al., 2017; Morsy, 1983; San-Antolín et al., 2020). Evidence suggests that brain correlates for trait anxiety exist, which can be a risk factor for mental diseases (Bishop & Forster, 2013; Liu et al., 2021; Mitchell & Kumari, 2016; Saviola et al., 2020). In addition, numerous research has found a link between state anxiety and emotional eating in a sample of obese people (Ostrovsky et al., 2013; Schneider et al., 2010).

Our findings observed a reduction in state anxiety during the follow-up period across both groups, indicating that both intervention models effectively reduced scores in this domain. This result aligns with findings from Moltrecht et al. (2021), whose meta-analysis of 21 studies on emotional regulation interventions demonstrated efficacy in managing



psychiatric disorders in young populations. Similarly, psychoeducation models have shown benefits in controlling anxiety symptoms (Dolan et al., 2021; Chillemi et al., 2020; Norr et al., 2017). A possible explanation lies in psychoeducation's ability to transfer knowledge about emotional and behavioral processes, integrating emotional awareness, motivation, and behavioral strategies to manage stress. This empowers individuals to regulate their emotional responses and adopt functional coping behaviors. These findings highlight the potential of combining emotional regulation interventions and psychoeducational approaches in weight management programs to address the emotional drivers of eating behaviors. However, larger and more controlled studies are needed to clarify the specific effects of each approach.

The study assessed emotional state components using heart rate variability (HRV) to examine sympathetic/parasympathetic balance and basal salivary cortisol upon awakening. These psychophysiological indicators reflect emotional response changes mediated by the Sympathetic-Adreno-Medullary (SAM) and Hypothalamic-Pituitary-Adrenal (HPA) axes, key systems in stress and emotional regulation. While sympathetic modulation remained unchanged in both groups, participants in the emotional regulation intervention exhibited increased parasympathetic modulation.

Preliminary findings suggest that the emotional regulation strategies, such as cognitive restructuring and relaxation techniques, may have influenced HPA axis excitability and reactivity. The intervention could have balanced HPA axis activity by enhancing parasympathetic activation and reducing stress-induced sympathetic dominance. Lower basal cortisol levels in the CBGT group may reflect improved stress coping or emotional regulation, though these assumptions remain speculative due to the study's methodological limitations, which preclude definitive conclusions. Supporting evidence comes from a meta-analysis by Mikkelsen et al. (2021), which reported favorable effects of CBT on cortisol expression, while mindfulness-based interventions have shown inconsistent results (O'Leary et al., 2016). Our study adds to the growing evidence linking emotional regulation interventions to physiological and behavioral improvements, such as increased parasympathetic modulation, reduced cortisol levels, and decreases in emotional eating. However, these findings should be interpreted cautiously, as stress effects on emotion and behavior are influenced by multiple factors, Including stress intensity, individual variability, social conditions, and genetic or neural vulnerabilities (Flores-Kanter et al., 2021). Further research with more robust methodologies is needed to confirm these results and assess their longterm impact.

A linear regression model was used to test the hypothesis of the predictive effect of emotional response on emotional eating behavior in both groups. HRV variables (sympathetic and parasympathetic modulation) and salivary cortisol were used as predictor variables on aspects of emotional eating. We found no predictive influence of any of the emotional response variables on emotional eating domains in the model I. In model II, however, a substantial negative linear connection was identified for the parasympathetic modulation predictor variable, indicating that an increase in parasympathetic excitation can predict a reduction in emotional eating for the intervention group focused on emotional regulation. Although regression models showed predictive effects, these may be due to individual heterogeneity in responses or the short follow-up time, emphasizing the need for longitudinal approaches to properly capture these dynamics. Replicating this intervention in populations with clinically relevant stress conditions or across age and gender groups could reveal emotional and behavioral changes predicted by the diathesis-stress model, which emphasizes individual vulnerabilities and environmental stressors.

Certain limitations to this study should be considered. Variables such as neuroendocrine loop characteristics for women during their menstrual



cycle, resilience traits, socioeconomic indicators, and educational level were not controlled for in the study and could have directly influenced the examined parameters. Furthermore, because this was a community-based sample, the participants might represent a motivated and self-selected group seeking therapy. Although the overall results were promising in certain explored aspects, various potential moderating factors of treatment response, such as a possible placebo effect due to participants' belief in being part of a therapeutic group focused on emotional regulation, could not be ruled out. Additionally, the anthropometric variables adopted, such as BMI, may not accurately reflect adipose tissue accumulation or metabolic alterations related to obesity. More precise measures, such as body composition analysis, should be considered in future research to enhance the validity of these findings. Similarly, more accurate assessments of cortisol response, such as the Cortisol Awakening Response (CAR), could provide deeper insights into stress reactivity and its relationship with emotion-based eating patterns. The small sample size also limits the generalizability of the findings, emphasizing the need for future studies with larger samples, including clinical populations with eating disorders, to better evaluate the effects and applicability of the intervention. Finally, other variables were not controlled during the follow-up period, which may have influenced the observed outcomes.

In conclusion, the 8-week group-based intervention focused on emotional regulation demonstrated preliminary efficacy in reducing aspects of emotional reactivity with a potential impact on components of emotion-driven eating behaviors. The effects of the CBGT intervention were more pronounced compared to the psychoeducation group, suggesting its potential as a promising approach. These findings highlight the feasibility and safety of implementing group-based therapeutic interventions for obese individuals while addressing key psychophysiological and behavioral variables such as emotional eating. However, given the methodological limitations, including the small sample size and lack of clinical populations with eating disorders, these results should be interpreted cautiously. Further studies with larger samples and controlled designs are necessary to validate the intervention's long-term effectiveness and explore its broader applicability in managing dysfunctional eating behaviors.

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