

Neurocognitive impairments in eating disorders patients with and without comorbid food addiction

Received: 30 July 2025

Accepted: 1 December 2025

Published online: 08 December 2025

Cite this article as: Munguía L., Granero R., Supit K. *et al.* Neurocognitive impairments in eating disorders patients with and without comorbid food addiction. *J Eat Disord* (2025). <https://doi.org/10.1186/s40337-025-01494-6>

Lucero Munguía, Roser Granero, Kim Supit, Ignacio Lucas, Ashley N. Gearhardt, Susana Jiménez-Murcia & Fernando Fernández-Aranda

We are providing an unedited version of this manuscript to give early access to its findings. Before final publication, the manuscript will undergo further editing. Please note there may be errors present which affect the content, and all legal disclaimers apply.

If this paper is publishing under a Transparent Peer Review model then Peer Review reports will publish with the final article.

Neurocognitive impairments in Eating Disorders patients with and without comorbid Food Addiction

Lucero Munguía^{1,2,3,;} Roser Granero^{1,2,4,;} Kim Supit ^{5;} Ignacio Lucas ^{6;} Ashley N. Gearhardt^{7;} Susana Jiménez-Murcia^{1,2,3,8;} Fernando Fernández-Aranda^{1,2,3,8}

¹CIBER Fisiopatología Obesidad y Nutrición (CIBEROBn), Instituto de Salud Carlos III (ISCIII), Barcelona Spain

²Psychoneurobiology of Eating and Addictive Behaviours Group, Neurosciences Programme, Bellvitge Biomedical Research Institute (IDIBELL), Barcelona, Spain

³Clinical Psychology Service, University Hospital of Bellvitge, Barcelona, Spain

⁴Department of Psychobiology and Methodology, Autonomous University of Barcelona, Barcelona, Spain

⁵Faculty of Science, University of Amsterdam, Amsterdam, The Netherlands.

⁶Department of Basic Psychology, University of Valencia, Valencia, Spain

⁷Department of Psychology University of Michigan, Michigan, USA

⁸Department of Clinical Sciences, School of Medicine and Health Sciences, University of Barcelona, Barcelona, Spain

Correspondence should be addressed to:

*FFA ffernandez@bellvitgehospital.cat

Clinical Psychology Unit, University Hospital of Bellvitge, Hospitalet del Llobregat, Feixa Llarga s/n, 08907, Barcelona, Spain.

ABSTRACT

Background: Research into the presence of Food Addiction (FA) in Eating Disorders (EDs) has gained increasing attention due to its association with greater symptom severity and poorer treatment outcomes. While the clinical and psychopathological significance of FA in EDs is well established, its neurocognitive impact remains largely unexplored. This study had two primary aims: (1) to examine the psychopathological profile and neurocognitive performance of patients with Eating Disorders (EDs), comparing those with Food Addiction (ED FA+) to those without (ED FA–), alongside a Healthy Control (HC) group; and (2) to explore potential correlations between neurocognitive performance and clinical-psychopathological variables. **Methods:** The sample consisted of N=152 female participants that met the following conditions: *EDs FA+* (N=65), *EDs FA-* (N=24), and *HC* (N=63). All participants completed a comprehensive battery of questionnaires, including the Yale Food Addiction Scale 2.0. (YFAS-2), and others for the assessment of the eating disorder symptoms, general psychopathology, impulsivity and emotion regulation. Decision-making and inhibitory control were assessed using the Iowa Gambling Task and The Connors Continuous Performance Test II. **Results:** Neurocognitive differences were moderate, with lower IGT performance in Block 3 and greater CPT variability, especially in the ED FA+ group. Although the ED FA+ group presented a more severe clinical profile—characterized by heightened eating symptomatology and general psychopathology (ED F - vs ED FA+: EDI-2 Total score $p .001 d 0.95$; SCL-90R Global Severity Index $p .008 d 0.71$)—this was not directly associated with greater cognitive impairment. **Conclusions:** The results underscore the need to assess Food Addiction in ED patients, as its presence may exacerbate symptom severity. Additionally, the results suggest

that patients with EDs could benefit from incorporating cognitive rehabilitation into their treatment plans, regardless of FA status.

Keywords: Eating Disorders; Food Addiction; Neurocognitive profile; Inhibitory control; Decision-making.

ARTICLE IN PRESS

PLAIN ENGLISH SUMMARY:**Background:**

Food Addiction (FA) is increasingly recognized as a common issue among people with Eating Disorders (EDs). It is linked to more severe symptoms and less success in treatment. While we know FA can worsen the emotional and psychological aspects of EDs, its effect on thinking and decision-making abilities (called neurocognitive performance) has not been studied much.

What the Study Did:

This research looked at 152 women, divided into three groups: 65 with EDs and signs of Food Addiction (ED FA+); 24 with EDs but no signs of Food Addiction (ED FA-); 63 healthy individuals (HC). Participants completed several questionnaires to measure eating disorder symptoms, emotional health, impulsivity, and emotional management strategies. They also performed tasks that assess decision-making and self-control.

What the Study Found:

On the cognitive tasks, people with eating disorders—whether they had Food Addiction or not— showed some specific difficulties compared to healthy individuals. The group with Food Addiction had more severe emotional and eating-related problems, but this was not directly linked to a worse cognitive performance.

Why It Matters:

These results suggest that Food Addiction should be assessed in people with Eating Disorders, as it may worsen their symptoms. Also, since thinking and decision-making difficulties were found in both ED groups, treatments that

- 81 include cognitive rehabilitation (like exercises to improve focus, decision-
82 making, and self-control) could help improve recovery.

ARTICLE IN PRESS

BACKGROUND

Food Addiction (FA) keeps growing as an area of scientific interest. It has been described as maladaptive eating behaviours, mainly characterized by excessive consumption of ultra-processed foods, that could present phenomenological similarities with addictive disorders (1,2). Even if the construct itself has not been recognized as a clinical entity by itself, several studies continue to examine its role in the development and maintenance of other conditions, in order to increase the validity of FA (3). In addition, since FA has been found highly present in Eating Disorders (EDs), the debate whether it is a construct itself or a variant of other conditions, especially Binge Eating Disorder (BED) has been raised. Even if some symptoms seem to be overlapping above the two diagnoses (4), different underlying mechanisms have been found when comparing them, highlighting psychological, physical, and neurocognitive aspects (5–9). Moreover, FA has been found in other EDs, comorbid in behavioural addictions, and, even present in the general population (10,11).

Among EDs subtypes, those within the binge spectrum disorder (BSD), namely Bulimia Nervosa (BN) and BED are the ones with higher rates of prevalence of comorbid FA (12–16). However, it is important to mention that FA has been also reported present in restrictive EDs subtypes (particularly anorexia nervosa binge-purge subtype) (17–19). It has also been associated with psychological symptoms and distress above and beyond heightened Body mass index (BMI) (20). This reflects the importance of continuing to study this construct.

Besides the above, according to the literature, the comorbid presence of FA in EDs increases the severity of the eating related symptoms (18,21), and negatively influence the treatment outcome (22–25). Furthermore, the co-

occurrence of ED and FA is associated with a more severe clinical presentation (23,26) including higher impulsivity (27,28), higher use of maladaptive emotional regulation strategies, (29), and higher comorbidity with other psychiatric conditions, such as depressive symptoms (30,31). However, many more clinically relevant domains require additional study.

When considering other addictive processes, neurocognitive impairments have been described. Among patients with Substance Use Disorders (SUD) impulsive decision-making and lack of inhibitory control have been reported (32) both, as a possible vulnerability for the development of an addiction (33) and a factor of maintenance, by contributing to the relationship between reward processes and learning (34). When considering addictive behaviours the results are similar. Patients with Gambling Disorder (GD) exhibit inhibitory control deficits when compared with the general population (35). Additionally, impairments in decision-making and planning response have been associated with higher severity of the GD (36).

Furthermore, patients among BSD have presented impulsivity choices in neurocognitive tasks (37,38), including as well binge/purging subtype of Anorexia Nervosa (AN) as well (39). The aforementioned impairments have also been associated with a complex clinical profile in EDs, by predicting poor treatment outcome (40). Besides, when considering EDs with comorbid SUD (41) or a behavioural addiction (42), a worse clinical profile emerges.

Considering that neurocognitive impairments have been associated with both, addictive processes and EDs, it would be of interest to study if the presence of FA among EDs could imply a worse neurocognitive performance. However, there are only a few studies that have considered neurocognitive performance associated with FA, especially in patients with obesity (OBE), finding impairments in decision-making, attention (43), inhibitory control and

cognitive flexibility (44). In studies that have examined neurocognitive performance in individuals with OBE and BED no impairments associated with the co-occurrence of FA in these conditions were found (45), or the authors declared the results inconclusive (9). This highlights the need for further research in this area.

Aims in this study

Therefore, the aims of the present study were twofold: (1) to examine the psychopathological profile and neurocognitive performance of patients with Eating Disorders (EDs), comparing those with Food Addiction (ED FA+) to those without (ED FA–), alongside a Healthy Control (HC) group; and (2) to explore potential correlations between neurocognitive performance and clinical-psychopathological variables.

We hypothesize that individuals in the clinical groups will exhibit greater neurocognitive impairments compared to HC. Furthermore, given that addictive behaviors are often linked to impaired decision-making and reduced inhibitory control, we expect the ED FA+ group to show more severe clinical profile and more pronounced cognitive deficits than the ED FA– group.

METHODS

Participants

The total sample consisted of N=152 female participants, divided into three groups. The clinical sample included N=89 individuals diagnosed with EDs (M_{age}=31.75 years), further categorized based on the presence (N=65 ED FA+) or absence (N=24 ED FA–) of FA. The remaining group consisted of healthy controls (HC) (N=63 HC / M_{age}=33.59 years). Among the clinical groups, most of Eating Disorder subtypes were present: 40 participants were

diagnosed with Anorexia Nervosa (AN), and 49 with binge-spectrum disorders (BSD, 12 with BN and 37 with BED). General inclusion criteria (for both, clinical and control group) required participants to be female and over 18 years old, and to provide signed informed consent. For the clinical groups, the specific inclusion criteria was to be diagnosed with an ED by a specialized psychologist using a face-to-face, semi-structured clinical interview based on DSM-5 criteria, whereas exclusion criteria included the presence of an organic mental disorder, intellectual disability, neurodegenerative disease (e.g., Parkinson's disease), or an active psychotic disorder. For the HC group, the same exclusion criteria applied, with the additional requirement of having current or past symptoms related to Eating Disorders, assessed through self-response questionnaires (see the measures section).

Measures

Psychological assessment

The Yale Food Addiction Scale 2.0 (YFAS2.0) (46) validated in the Spanish population (47), is a self-report questionnaire for measuring FA, based on DSM-5 Criteria. It consist of 35-items answered on a 8-point Likert scale and assesses 11 symptoms with which it establishes severity cut-offs: mild (2-3 symptoms), moderate (4-5 symptoms), and severe (6-11 symptoms). Besides, the scale produce two measurements: (a) a continuous symptom count score that reflects the number of fulfilled diagnostic criteria and (b) a food addiction threshold based on the number of symptoms (at least 2) and self-reported clinically significant impairment or distress. This final measurement allows the binary classification of food addiction (present versus absent). The internal consistency of the total scale for our sample was 0.98 (coefficient alpha).

The Eating Disorder Inventory-2 (EDI-2) (48), is a 91-item self-report questionnaire that assesses ED-related symptoms, across eleven subscales: Drive for thinness, body dissatisfaction, bulimia, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, maturity fears, asceticism, impulse regulation, and social insecurity. Items are rated on a 6-point Likert scale. The internal consistency values (Cronbach's alpha) varied between .70 and .90 for the EDI-2 subscales, with a value of .97 for the total score.

The Symptom Checklist-Revised (SCL-90-R) (49), validated in the Spanish population (50), is a self-report questionnaire used to assess psychological and psychopathological symptoms in nine dimensions: somatization, obsession-compulsion, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism. It also includes a global severity index (GSI), a positive symptom distress index (PST), and a positive symptom total (PSDI). The internal consistency values (Cronbach's alpha) ranged from .83 to .95, with a value of .98 for the global index.

The Impulsive behaviour scale (UPPS-P) (51), validated in the Spanish population (52), is a 59 item self-report questionnaire developed to assess impulsive behaviour through five subscales: negative urgency, positive urgency, lack of premeditation, lack of perseverance and sensation seeking. The internal consistency values (Cronbach's alpha) ranged from .82 to .91, with a value of .92 for the total score.

The Difficulties in Emotion Regulation Scale (DERS), (53), validated in the Spanish population (54), is a 36-item self-report questionnaire that assesses difficulties in emotion regulation across six subscales: strategies,

non-acceptance, awareness, impulse, goals and clarity. Items are rated on a 5-point Likert scale. The internal consistency values (Cronbach's alpha) ranged from .88 to .93, with a value of .97 for the total score.

Neurocognitive measures

The Iowa Gambling Task (IGT) (55) is a computerised task to evaluate decision-making. It involves 100 turns distributed across four decks of cards (A, B, C and D). Each time the participant selects a deck, a specified amount of play money is awarded, the interspersed rewards among these decks are probabilistic punishments (monetary losses with different amounts). Participants are instructed that the final aim of the task is to win as much money as possible and to avoid losing as much money as possible. Decks A and B are not advantageous as the final loss is higher than the final gain; however, decks C and D are advantageous since the punishments are smaller. The IGT is divided into five blocks. The first blocks are supposed to assess the decision-making process under ambiguity conditions while the last blocks (after the 40th selection) are used to assess the decision-making capabilities under risk conditions due to the fact that the rules have been figured out at this point (56). Several indices could be used to analyse performance in the IGT. The total score for each block of 20 cards is usually reported, being the first block being thought as measures of decision making under ambiguity. The IGT total score is the difference among the total number of cards selected from Decks A and B (disadvantageous ones) and those selected from Decks C and D (advantageous ones), higher scores indicate better performance on the task. Finally the IGT learning and risk scores could be obtained. The IGT learning score is the difference among the net score in the last two blocks (rules figured out) and the net score in the first two (decisions made under

ambiguity), while the IGT risk score is the score of the last two blocks added together (a measure of decision making under risk or certainty) (57).

The Connors Continuous Performance Test II (CPT) (58) is a digital tool designed to evaluate selective attention and inhibitory control. During the test, participants are instructed to press the space bar whenever a target letter appears on the screen (GO), except when the non-target letter "X" is displayed (NO-GO). To assess inhibitory control, the non-target stimulus (NO-GO) is presented 10% of the time, requiring participants to refrain from responding. Performance of the task includes the hit rate (responding to target stimuli) the number of omissions (missed targets), and the number of commissions (responses to non-target stimuli); measures of reaction time and reaction time variability could also be obtained. A high number of commissions is associated with poor inhibitory control.

Procedure

Participants from the clinical groups voluntarily sought treatment at the Eating Disorders Unit, of the Clinical Psychology Department of the University Hospital of Bellvitge in Barcelona. They accepted to be part of the study after researchers of the unit presented to them the aims of the research and the specifications of their participation. The HC was recruited from the same catchment area, by display information about the study and received the specifications of their participation by the involved researchers; their participation was voluntary, and they did not receive any type of compensation. Clinical participants were diagnosed by a specialized psychologist, using a face-to-face semi-structured clinical interview based on DSM-5 criteria (59). Sociodemographic information (e.g., age, sex, marital status, educational level, etc.) was self-reported during the initial assessment.

All participants completed the psychometric questionnaires in a one-hour session, followed by a second visit for the neurocognitive assessment. For the clinical groups, all the assessments were conducted prior to the start of treatment.

Ethics

The study was approved by the Clinical Research Ethics Committee, of Bellvitge University Hospital (Ref. PR146714; PR205/17; PR319/20). According to the Declaration of Helsinki, signed informed consent to participate in the study was obtained from both, control and clinical groups, before completing the assessment.

Data analysis

Stata18 for Windows was used for the data analysis (60). The comparison between the groups for the sociodemographic variables was done through chi-square tests (χ^2) for categorical measures and analysis of variance (ANOVA) for quantitative measures. The comparison for the clinical variables between the groups was done with analysis of covariance (ANCOVA), adjusted by the participants' age, education level and ED subtype. The estimation of the effect size of the differences between the groups was calculated with the standardized Cohen's-d coefficient for the pairwise comparisons estimated in the ANOVA / ANCOVA tests (mild-moderate effect size was considered for $|d| > 0.50$ and large-high effect size for $|d| > 0.80$), and with the standardized Cramer's-V coefficient for the chi-square tests (mild-moderate effect size was considered for $C-V > 0.20$ and large-high effect size for $C-V > 0.60$). Given the small sample size of one of the study groups (ED FA-, $n = 24$) and the resulting limited statistical power to detect potential associations among

variables, we adopted a broader criterion for interpreting the results. Specifically, associations were considered “relevant” not only when statistical significance was achieved, but also when effect sizes reached at least the moderate range, as these may indicate meaningful patterns despite reduced power. Additionally, Finner’s correction was employed to control the increase in Type-I error due to the use of multiple significance statistical tests.

The path analysis was conducted using Structural Equation Modelling (SEM) with an exploratory approach. The model was specified based on prior theoretical frameworks and empirical evidence, ensuring that the relationships included were both conceptually meaningful and clinically relevant. This exploratory SEM allowed for the examination of potential direct and indirect associations among variables while maintaining interpretability within the context of eating disorder research. All paths were defined a priori based on established hypotheses, supporting the validity and clinical applicability of the findings, while also allowing the model to reveal novel patterns and relationships that could inform future research. With the aim to obtain the most parsimonious model, only significant parameters were retained and interpreted in the final model. Additionally, due to the large amount of information in the SEM and the relatively low sample size, a latent variable was defined for the main cognitive measures analysed in the study. The maximum likelihood estimation was employed, free-estimated parameters were allowed, and the goodness-of-fit was evaluated with standard criteria (61): root mean square error of approximation $RMSEA < 0.08$, Bentler’s Comparative Fit Index $CFI > 0.90$, Tucker-Lewis Index $TLI > 0.90$, and standardized root mean square residual $SRMR < 0.10$. The global predictive capacity was assessed with the coefficient of determination (CD).

RESULTS

Description of the sample

The description for the sociodemographic variables, the onset and duration of the ED, and the YFAS-2 screening group are displayed in Table 1. This table also contains a statistical comparison between the HC versus the ED subsamples, and the AN versus the BSD subtypes.

--- Insert Table 1 ---

Compared to HC, ED patients reported lower education levels and lower social position indexes. Compared to BSD, AN patients showed a higher prevalence of being single, higher education levels, lower social position index, lower mean chronological age, and lower prevalence of patients within the positive YFAS-2 screening group. Therefore, the statistical analysis was adjusted by ED subtype and age.

Comparison of the sociodemographic profiles

The comparison of the sociodemographic profile between the three groups is displayed in Table 2. Compared to the clinical groups, the HC participants reported higher education levels, the higher likelihood of being employed, and the higher social position index. ED FA+ group was characterized by including the highest proportion of married patients and the highest proportion of individuals with lower education levels. Therefore, the statistical analysis was adjusted by educational levels. Compared to the other two groups, ED FA- included patients with the youngest chronological mean age.

--- Insert Table 2 ---

Comparison of the clinical profiles

Tables 3 and 4 contains the comparison between the groups for the clinical and the neurocognitive measures registered in the study, through ANCOVA adjusted by the participants' sex, education level and ED-subtype. Overall, the worst clinical profile was associated with ED FA+. These patients registered the highest ED symptom levels (in all the EDI-2 scales), the worst psychological state (the highest mean scores in the SCL-90R), the highest impulsivity, and most difficulties in the emotion regulation. In terms of BMI, the HC and ED FA- groups did not differ significantly, whereas participants with ED FA+ exhibited a higher mean BMI relative to the other two groups.

--- Insert Table 3 ---

Significant group differences were found only in Block 3, but not in total net, learning, or risk scores. On the CPT, differences were modest but moderate effects were observed in reaction time variability, especially in the ED FA+ group.

--- Insert Table 4 ---

--- Insert Figure 1 ---

Path analysis

Figure 2 shows the path diagram with the standardized coefficients obtained in the SEM and Table 5 the complete results. Adequate goodness-of-fit was achieved: $\chi^2 = 24.07$ ($p=.239$); RMSEA=0.048; CFI=0.973; TLI=0.952; SRMR=0.057. The global predictive capacity was around 49% ($CD=.486$).

--- Insert Table 5 ---

--- Insert Figure 2 ---

The latent variable defined by neurocognitive variables (labelled in the path diagram as “cognition”) achieved positive significant contributions for IGT total and the CPT perseverations, and negative significant contributions for the CPT omissions. Therefore, the higher the scores in this latent variable, the better the neurocognitive performance.

Higher impairment in the cognitive domain impacted on the higher impulsivity and emotion dysregulation levels. A profile characterized by higher impulsivity and higher emotion regulation difficulties also contributed to increasing the FA severity. Regarding the ED severity, higher levels in this variable were directly associated to higher FA level and higher impairments in emotion regulation. FA was also a mediational link into the relationships between impulsivity and emotion regulation with ED severity.

DISCUSSION

The present study aimed to analyse the clinical and neurocognitive profiles in patients with EDs with and without comorbid Food Addiction (FA), and to explore potential correlations between neurocognitive performance and other clinical variables. The results partially supported our hypotheses. As expected, patients in the ED FA+ group exhibited a more severe clinical profile; however, they did not show greater neurocognitive impairment on the cognitive tasks compared to those without FA.

A complex clinical ED profile, when FA is present, has already been described (23,26), specially associated with impulsivity and emotional regulation. Among impulsivity traits, consistent with our results, negative urgency has been raised as an important differential trait between ED FA+ and ED FA- (28), highlighting that the FA presence is directly associated with emotional and affective impairments, which can lead us to consider FA symptoms as a

way to cope with the emotions (62). These findings align with previous research, as the ED FA+ group showed higher levels of general psychological distress and greater difficulties in emotion regulation compared to the ED FA– group.

Regarding neurocognitive performance, our results revealed specific rather than generalized differences between groups. Both ED groups showed lower IGT scores in Block 3 (see figure 1), suggesting decision-making difficulties during specific phases of the task, while overall task indices were comparable. On the CPT, moderate effects were observed mainly for reaction time variability, particularly in the ED FA+ group, indicating subtle attentional fluctuations (see table 4). These findings are consistent with previous research showing that cognitive alterations in ED with comorbid FA often appear in specific domains rather than across the board. According to the existing literature (45) only two FA diagnosis criteria were related to emotional control, flexibility and planning (Eating for longer durations and higher amounts than intended and continuing to eat despite knowing the negative consequences). Therefore, cognitive differences were modest and should be interpreted with caution (9,63), since present results do not substantially support the hypothesis of a worst cognitive profile related to FA.

Considering the associations between variables, the path analysis suggests that neurocognitive factors may play an indirect role in ED severity via impulsivity and emotion regulation, warranting further research before drawing firm clinical implications. The link between executive functions and emotions has been widely studied, for example, impairments in flexibility can be associated with lack of cognitive reappraisal as a way to cope with negative emotions (64), implying even depressive symptoms (65) and a risk factor for ED development (66,67). On the other hand, lack of inhibitory

control has been found in patients with ED predicting worse treatment outcome (68). Furthermore, the strong link reported between FA and negative urgency (28) may lead to consider that the cognitive impairments might emerge for the FA+ group under the context of high negative affect (but not in less affectively changed states). The aforementioned helps to explain the contribution of FA to the ED severity, by having a mediational role, between FA, higher impulsivity and emotion dysregulation. A complex relation that seems to be related to cognitive impairments.

Therefore, even the present study constitutes a first approach to the associations of cognitive impairments and FA, it follow the line to other researchers point of view (3). To continue exploring neural processes observed in BSD and other addictive processes such as FA may be helpful to use or develop other treatment approaches (rather than the usual CBT approach) to target maintenance and severity associated factors to the ED.

Cognitive rehabilitation could be potentially beneficial for patients with ED. Cognitive Remediation Therapy (CRT), through Computer training programs, has been used to induce modification of the attentional bias toward food in ED, as well as helping to increase inhibitory control (69). As an alternative, integrating executive function (EF) training with cognitive-behavioral therapy (CBT) has shown improvements in individuals with BED (70). When considering ED and comorbid FA, special attention should be placed on enhancing the development of emotional regulation skills. Dialectical behavior therapy (DBT) has shown positive results in the treatment of ED, especially in reducing binge episodes by targeting the food intake to manage negative emotions (71,72). There is also growing interest in the variant of radically open DBT, which addresses disorders characterized by non-externalization of emotions (73) and aims to increase receptivity and

flexibility of the emotions. Also, the use of mindfulness as a complementary training in ED treatments is shown to improve results in treating ED symptomatology, potentially through enhanced emotional regulation skills (74,75).

Limitations and future studies

The present study has several strengths, since, to our very own knowledge, it is one of the few studies that considers neurocognitive performance associated with FA and EDs. However, some aspects should be taken into account. The sample was exclusively constituted by women, even though this is justified by a higher prevalence of FA among women, the study of the relationship of the variables may change when considering men, especially regarding emotion regulation and FA. The study was also performed in a very specific geographical area, limiting the generalisation of the results. Furthermore, other cognitive processes will be of interest to be explored in patients with EDs and comorbid FA. Also, considering the results of the study, we can suggest that investigating cognitive performance in the presence of negative affect may be an important area for future studies. Finally, the sample size can also be considered a limitation affecting the ability to identify true relationships among the study variables. One group included only $n = 24$ patients (ED FA+); therefore, the relevance of associations was assessed based on both statistical significance and effect size estimates. On the other hand, the size of the clinical sample ($n = 89$) could also be considered relatively small for conducting a path analysis. In this regard, it should be noted that smaller samples may reduce the stability and generalizability of the estimated parameters, as well as the statistical power to detect indirect or complex relationships among variables. However, the sample size should

not be viewed as a barrier to performing SEM, because there is no clear consensus regarding the minimum sample size required to conduct these analyses. Recommendations in the literature vary widely, depending on factors such as model complexity, the number of estimated parameters, the strength of the relationships among variables, and the distributional properties of the data. While some authors suggest simple rules of thumb (e.g., a minimum of 5-10 participants per estimated parameter or an overall sample of at least 200 cases), others argue that these guidelines are overly simplistic and that the adequacy of sample size should be evaluated based on model-specific characteristics and estimation methods. Therefore, determining the appropriate sample size for SEM remains an ongoing methodological debate, and researchers are encouraged to consider both empirical and theoretical criteria when assessing the adequacy of their sample. In this study, the path analysis was conducted for exploratory purposes. In such cases, path analysis can be performed with relatively small sample sizes, as the primary goal is to identify potential relationships and generate hypotheses rather than to test a fully specified theoretical model. Since the focus is on exploring patterns and possible pathways, smaller samples can still yield meaningful insights; however, the results should be interpreted cautiously and regarded as preliminary. Another limitation concerns the ecological validity of the neurocognitive tasks employed. The study used general neurocognitive tasks (IGT and CPT), which may not capture disorder-specific impairments. Incorporating food- or body-related stimuli in future studies could enhance ecological validity and sensitivity to clinical differences.

CONCLUSIONS

The findings of this study indicate that neurocognitive profiles did not differ significantly between ED patients with and without comorbid Food Addiction (FA). However, some results are worthy of being mentioned. First of all, the relationship between Food Addiction (FA) and cognitive impairment may be mediated by underlying factors such as impulsivity and emotional dysregulation. In addition, those with FA exhibited notably more severe eating disorder symptoms and general psychopathology. These results highlight the clinical value of early FA assessment in individuals with EDs, as its presence may signal a more complex and severe presentation. Furthermore, since both ED FA+ and ED FA– groups demonstrated neurocognitive impairments compared to healthy controls, incorporating cognitive rehabilitation into treatment plans may be beneficial for all ED patients, regardless of FA status.

LIST OF ABBREVIATIONS

AN	Anorexia Nervosa
ANCOVA	Analysis of covariance
ANOVA	Analysis of variance
BED	Binge Eating Disorder
BMI	Body mass index
BN	Bulimia Nervosa
BSD	Binge spectrum disorder
CBT	Cognitive-behavioral therapy
CPT	Connors Continuous Performance Test II

518	CRT	Cognitive Remediation Therapy
519	DBT	Dialectical behavior therapy
520	DERS	Difficulties in Emotion Regulation Scale
521	DSM-5	Diagnostic and Statistical Manual of Mental Disorders,
522	Fifth Edition	
523	EDs	Eating disorders
524	ED FA+	Eating disorders and Food addiction present
525	ED FA-	Eating disorders and Food addiction absent
526	EF	Executive function
527	EDI-2	Eating Disorder Inventory-2
528	FA	Food addiction
529	GD	Gambling Disorder
530	GSI	Global severity index
531	HC	Healthy Control
532	IGT	Iowa Gambling Task
533	OBE	Obesity
534	PSDI	Positive symptom total
535	PST	Positive symptom distress index
536	SCL-90-R	Symptom Checklist-Revised

537	SD	Standard deviation
538	SEM	Structural Equations Model
539	SUD	Substance Use Disorders
540	UPPS-P	Impulsive behaviour scale
541	YFAS-2	Yale Food Addiction Scale 2.0.

542 **DECLARATIONS**

543 ***Ethics***

544 The present study was approved by the Clinical Research Ethics Committee
 545 of the Bellvitge University Hospital (Ref. PR146714; PR205/17; PR319/20).
 546 According to the Declaration of Helsinki, signed informed consent to
 547 participate in the study was obtained from both, control and clinical groups,
 548 before completing the assessment. Participants did not receive any form of
 549 compensation for their participation in this study.

550 ***Data availability statement***

551 Study data will not be made available in order to protect the confidentiality
 552 of the participants of the study.

553 ***Conflicts of interest/Competing interests***

554 FFA and SJM received consultancy honoraria from Novo Nordisk. The rest of
 555 the authors declare no conflict of interest. The funders had no role in the
 556 design of the study; in the collection, analyses, or interpretation of data; in
 557 the writing of the manuscript; or in the decision to publish the results.

558 ***Fundings and Acknowledgements***

This study was supported by the European Union through the: eprObes project (GA 101080219), BIOREXIA project (EPPermed2024-618), connect care project (PCI2025-163265), and cofounded by FEDER funds/European Regional Development Fund (ERDF), a way to build Europe. CIBEROBn is an initiative of ISCIII. We thank CERCA Programme/Generalitat de Catalunya for guarantee institutional support. Additional funded by Ministerio de Ciencia e Innovación (PDI2021-124887OB-I00) and Instituto de Salud Carlos III (ISCIII) (Exp: FIS22053 - Ref: DTS22/00072 and Exp: FIS23069 - Ref: FORT23/00032_2). FFA is supported by the Catalan Institution for Research and Advanced Studies (ICREA-Academia, 2024- Programme). RG is supported by the Catalan Institution for Research and Advanced Studies (ICREA-Academia, 2021- Programme).

Authors' contributions

Conceptualization: LM, FF-A; Methodology: RG; Formal analysis: RG; Investigation: LM, IL, KS; Resources: FF-A, SJM; Data curation: RG; Writing - original draft preparation: LM; Writing - review and editing: LM, IL, ANG, SJ-M, FF-A; Supervision: SJM, FF-A; Funding acquisition: FF-A, SJM. All authors have read and agreed to the published version of the manuscript.

Authors' information

LM lmunguia@idibell.cat <https://orcid.org/0000-0002-9751-810X>

RG roser.granero@uab.cat <https://orcid.org/0000-0001-6308-3198>

IL ignacio.lucas@uv.es <https://orcid.org/0000-0001-9426-5082>

584 KS kimsupit@gmail.com <https://orcid.org/0009-0000->
585 7812-3148

586 ANG agearhar@umich.edu <https://orcid.org/0000-0003->
587 3843-5731

588 SJM sjimenez@bellvitgehospital.cat <https://orcid.org/0000-0002->
589 3596-8033

590 FFA ffernandez@bellvitgehospital.cat <https://orcid.org/0000-0002->
591 2968-989

592 **References**

- 593 1. Imperatori C, Fabbriatore M, Vumbaca V, Innamorati M, Contardi A,
594 Farina B. Food Addiction: definition, measurement and prevalence in
595 healthy subjects and in patients with eating disorders. Riv Psichiatr
596 [Internet]. 2016 Mar 1 [cited 2025 Jul 17];51(2):60–5. Available from:
597 <https://pubmed.ncbi.nlm.nih.gov/27183510/>
- 598 2. Meule A, Gearhardt AN. Food addiction in the light of DSM-5. Nutrients
599 [Internet]. 2014 Sep 16 [cited 2025 Jul 17];6(9):3653–71. Available
600 from: <https://pubmed.ncbi.nlm.nih.gov/25230209/>
- 601 3. Treasure J, Leslie M, Chami R, Fernández-Aranda F. Are trans
602 diagnostic models of eating disorders fit for purpose? A consideration
603 of the evidence for food addiction. Eur Eat Disord Rev [Internet]. 2018
604 Mar 1 [cited 2025 Oct 24];26(2):83–91. Available from:
605 <https://pubmed.ncbi.nlm.nih.gov/29341400/>
- 606 4. Ratković D, Knežević V, Dickov A, Fedrigolli E, Čomić M. Comparison of
607 binge-eating disorder and food addiction. J Int Med Res [Internet].
608 2023 Apr 1 [cited 2025 Oct 27];51(4). Available from:
609 <https://pubmed.ncbi.nlm.nih.gov/37115520/>
- 610 5. Carter JC, Van Wijk M, Rowsell M. Symptoms of ‘food addiction’ in
611 binge eating disorder using the Yale Food Addiction Scale version 2.0.
612 Appetite. 2019 Feb 1;133:362–9.
- 613 6. Carbone EA, Aloï M, Rania M, de Filippis R, Quirino D, Fiorentino TV, et
614 al. The relationship of food addiction with binge eating disorder and
615 obesity: A network analysis study. Appetite. 2023 Nov 1;190.

7. Aloï M, Liuzza MT, Rania M, Carbone EA, De Filippis R, Gearhardt AN, et al. Using latent class analysis to identify different clinical profiles according to food addiction symptoms in obesity with and without binge eating disorder. *J Behav Addict* [Internet]. 2024 Mar 26 [cited 2025 Oct 27];13(1):262–75. Available from: <https://pubmed.ncbi.nlm.nih.gov/38276994/>
8. Linardon J, Messer M. Assessment of food addiction using the Yale Food Addiction Scale 2.0 in individuals with binge-eating disorder symptomatology: Factor structure, psychometric properties, and clinical significance. *Psychiatry Res*. 2019 Sep 1;279:216–21.
9. Iceta S, Rodrigue C, Legendre M, Daoust J, Flaudias V, Michaud A, et al. Cognitive function in binge eating disorder and food addiction: A systematic review and three-level meta-analysis. *Prog Neuropsychopharmacol Biol Psychiatry* [Internet]. 2021 Dec 20 [cited 2025 Jul 24];111. Available from: <https://pubmed.ncbi.nlm.nih.gov/34256024/>
10. Etxandi M, Baenas I, Munguía L, Mestre-Bach G, Granero R, Gómez-Peña M, et al. Clinical Features of Gambling Disorder Patients with and Without Food Addiction: Gender-Related Considerations. *J Gambl Stud* [Internet]. 2022 Sep 1 [cited 2025 Oct 27];38(3):843–62. Available from: <https://pubmed.ncbi.nlm.nih.gov/34585341/>
11. Munguía L, Jiménez-Murcia S, Valenciano-Mendoza E, Granero R, Gaspar-Pérez A, Guzmán-Saldaña RME, et al. Risk patterns in food addiction: a Mexican population approach. *Eating and Weight Disorders* [Internet]. 2022 Apr 1 [cited 2025 Jul 17];27(3):1077–87. Available from: <https://pubmed.ncbi.nlm.nih.gov/34143405/>
12. Jobin A, Gingras F, Beaupré J, Legendre M, Bégin C. Clinical Relevance of Food Addiction in Higher Weight Patients across the Binge Eating Spectrum. *Behavioral Sciences* [Internet]. 2024 Aug 1 [cited 2025 Jul 17];14(8). Available from: <https://pubmed.ncbi.nlm.nih.gov/39199041/>
13. Aloï M, Liuzza MT, Rania M, Carbone EA, De Filippis R, Gearhardt AN, et al. Using latent class analysis to identify different clinical profiles according to food addiction symptoms in obesity with and without binge eating disorder. *J Behav Addict* [Internet]. 2024 Mar 26 [cited 2025 Jul 17];13(1):262–75. Available from: <https://pubmed.ncbi.nlm.nih.gov/38276994/>
14. Gearhardt AN, White MA, Masheb RM, Morgan PT, Crosby RD, Grilo CM. An examination of the food addiction construct in obese patients with binge eating disorder. *International Journal of Eating Disorders* [Internet]. 2012 Jul [cited 2025 Jul 17];45(5):657–63. Available from: <https://pubmed.ncbi.nlm.nih.gov/22684991/>

- 657 15. Meule A, Von Rezori V, Blechert J. Food addiction and bulimia nervosa.
 658 European Eating Disorders Review [Internet]. 2014 [cited 2025 Jul
 659 17];22(5):331–7. Available from:
 660 <https://pubmed.ncbi.nlm.nih.gov/24995543/>
- 661 16. de Vries SK, Meule A. Food Addiction and Bulimia Nervosa: New Data
 662 Based on the Yale Food Addiction Scale 2.0. European Eating Disorders
 663 Review [Internet]. 2016 Nov 1 [cited 2025 Jul 17];24(6):518–22.
 664 Available from: <https://pubmed.ncbi.nlm.nih.gov/27578243/>
- 665 17. Tran H, Poinso P, Guillaume S, Delaunay D, Bernetiere M, Bégin C, et
 666 al. FOOD ADDICTION AS A PROXY FOR ANOREXIA NERVOSA SEVERITY:
 667 NEW DATA BASED ON THE YALE FOOD ADDICTION SCALE 2.0.
 668 Psychiatry Res [Internet]. 2020 Nov 1 [cited 2025 Jul 17];293.
 669 Available from: <https://pubmed.ncbi.nlm.nih.gov/33007684/>
- 670 18. Granero R, Hilker I, Agüera Z, Jiménez-Murcia S, Sauchelli S, Islam MA,
 671 et al. Food addiction in a Spanish sample of eating disorders: DSM-5
 672 diagnostic subtype differentiation and validation data. European
 673 Eating Disorders Review [Internet]. 2014 Nov 1 [cited 2025 Jul
 674 17];22(6):389–96. Available from:
 675 <https://pubmed.ncbi.nlm.nih.gov/25139680/>
- 676 19. Sanchez I, Lucas I, Munguía L, Camacho-Barcia L, Giménez M,
 677 Sánchez-González J, et al. Food addiction in anorexia nervosa:
 678 Implications for the understanding of crossover diagnosis. European
 679 Eating Disorders Review [Internet]. 2022 May 1 [cited 2025 Jul
 680 17];30(3):278–88. Available from:
 681 <https://pubmed.ncbi.nlm.nih.gov/35306714/>
- 682 20. Horsager C, Færk E, Bruun JM, Lauritsen MB, Østergaard SD. Food
 683 Addiction Is Strongly Associated With Psychopathology and Reduced
 684 Psychological Well-Being Among Adults Irrespective of BMI. Eur Eat
 685 Disord Rev [Internet]. 2025 Jul 14 [cited 2025 Jul 17]; Available from:
 686 <https://pubmed.ncbi.nlm.nih.gov/40658368/>
- 687 21. Gearhardt AN, Boswell RG, White MA. The association of “food
 688 addiction” with disordered eating and body mass index. Eat Behav
 689 [Internet]. 2014 [cited 2025 Jul 17];15(3):427–33. Available from:
 690 <https://pubmed.ncbi.nlm.nih.gov/25064294/>
- 691 22. Hilker I, Sánchez I, Steward T, Jiménez-Murcia S, Granero R, Gearhardt
 692 AN, et al. Food Addiction in Bulimia Nervosa: Clinical Correlates and
 693 Association with Response to a Brief Psychoeducational Intervention.
 694 European Eating Disorders Review [Internet]. 2016 Nov 1 [cited 2025
 695 Jul 17];24(6):482–8. Available from:
 696 <https://pubmed.ncbi.nlm.nih.gov/27593963/>
- 697 23. Munguía L, Gaspar-Pérez A, Jiménez-Murcia S, Granero R, Sánchez I,
 698 Vintró-Alcaraz C, et al. Food Addiction in Eating Disorders: A Cluster

- 699 Analysis Approach and Treatment Outcome. *Nutrients* [Internet]. 2022
 700 Mar 1 [cited 2025 Jul 17];14(5). Available from:
 701 <https://pubmed.ncbi.nlm.nih.gov/35268059/>
- 702 24. Romero X, Agüera Z, Granero R, Sánchez I, Riesco N, Jiménez-Murcia
 703 S, et al. Is food addiction a predictor of treatment outcome among
 704 patients with eating disorder? *European Eating Disorders Review*
 705 [Internet]. 2019 Nov 1 [cited 2025 Jul 17];27(6):700–11. Available
 706 from: <https://pubmed.ncbi.nlm.nih.gov/31637816/>
- 707 25. Wiedemann AA, Ivezaj V, Gueorguieva R, Potenza MN, Grilo CM.
 708 Examining self-weighting behaviors and associated features and
 709 treatment outcomes in patients with binge-eating disorder and obesity
 710 with and without food addiction. *Nutrients* [Internet]. 2021 Jan 1 [cited
 711 2025 Jul 17];13(1):1–11. Available from:
 712 <https://pubmed.ncbi.nlm.nih.gov/33374870/>
- 713 26. Jiménez-Murcia S, Agüera Z, Paslakis G, Munguia L, Granero R,
 714 Sánchez-González J, et al. Food addiction in eating disorders and
 715 obesity: Analysis of clusters and implications for treatment. *Nutrients*
 716 [Internet]. 2019 Nov 1 [cited 2025 Jul 17];11(11). Available from:
 717 <https://pubmed.ncbi.nlm.nih.gov/31684127/>
- 718 27. Murphy CM, Stojek MK, MacKillop J. Interrelationships among impulsive
 719 personality traits, food addiction, and Body Mass Index. *Appetite*
 720 [Internet]. 2014 Feb 1 [cited 2025 Jul 17];73:45–50. Available from:
 721 <https://pubmed.ncbi.nlm.nih.gov/24511618/>
- 722 28. Wolz I, Granero R, Fernández-Aranda F. A comprehensive model of
 723 food addiction in patients with binge-eating symptomatology: The
 724 essential role of negative urgency. *Compr Psychiatry* [Internet]. 2017
 725 Apr 1 [cited 2025 Jul 17];74:118–24. Available from:
 726 <https://pubmed.ncbi.nlm.nih.gov/28160693/>
- 727 29. Li J, Wang L, Xia F, Pan L, Liu JF. The impact of emotional dysregulation
 728 on food addiction: Examining the intervention role of gender. *Acta*
 729 *Psychol (Amst)* [Internet]. 2025 Apr 1 [cited 2025 Jul 17];254. Available
 730 from: <https://pubmed.ncbi.nlm.nih.gov/39923548/>
- 731 30. Burrows T, Kay-Lambkin F, Pursey K, Skinner J, Dayas C. Food
 732 addiction and associations with mental health symptoms: a systematic
 733 review with meta-analysis. *Journal of Human Nutrition and Dietetics*
 734 [Internet]. 2018 Aug 1 [cited 2025 Jul 17];31(4):544–72. Available
 735 from: <https://pubmed.ncbi.nlm.nih.gov/29368800/>
- 736 31. Zielińska M, Łuszczki E, Bartosiewicz A, Oleksy Ł, Stolarczyk A, Dereń
 737 K. Relationship Between BMI, Self-Rated Depression, and Food
 738 Addiction—A Cross-Sectional Study of Adults in Postpandemic Poland.
 739 *Depress Anxiety* [Internet]. 2024 [cited 2025 Jul 17];2024(1). Available
 740 from: <https://pubmed.ncbi.nlm.nih.gov/40226725/>

- 741 32. Cavicchioli M, Movalli M, Bruni A, Terragni R, Bellintani S, Ricchiuti A,
742 et al. The Complexity of Impulsivity Dimensions among Abstinent
743 Individuals with Substance Use Disorders. *J Psychoactive Drugs*
744 [Internet]. 2023 [cited 2025 Jul 17];55(4):471–82. Available from:
745 <https://pubmed.ncbi.nlm.nih.gov/35998223/>
- 746 33. Verdejo-Garcia A, Albein-Urios N. Special issue on vulnerabilities to
747 substance abuse impulsivity traits and neurocognitive mechanisms
748 conferring vulnerability to substance use disorders.
749 *Neuropharmacology* [Internet]. 2021 Feb 1 [cited 2025 Jul 17];183.
750 Available from: <https://pubmed.ncbi.nlm.nih.gov/33189766/>
- 751 34. Poulton A, Hester R. Transition to substance use disorders: Impulsivity
752 for reward and learning from reward. *Soc Cogn Affect Neurosci*
753 [Internet]. 2020 Oct 1 [cited 2025 Jul 17];15(10):1182–91. Available
754 from: <https://pubmed.ncbi.nlm.nih.gov/31848627/>
- 755 35. Mestre-Bach G, Steward T, Granero R, Fernández-Aranda F, Mena-
756 Moreno T, Vintró-Alcaraz C, et al. Dimensions of Impulsivity in
757 Gambling Disorder. *Sci Rep* [Internet]. 2020 Dec 1 [cited 2025 Jul
758 17];10(1). Available from: <https://pubmed.ncbi.nlm.nih.gov/31941903/>
- 759 36. Mestre-Bach G, Steward T, Balodis IM, DeVito EE, Yip SW, George TP,
760 et al. Discrete Roles for Impulsivity and Compulsivity in Gambling
761 Disorder. *Front Psychiatry* [Internet]. 2021 Dec 7 [cited 2025 Jul
762 17];12. Available from: <https://pubmed.ncbi.nlm.nih.gov/34950074/>
- 763 37. Lozano-Madrid M, Granero R, Lucas I, Sánchez I, Sánchez-González J,
764 Gómez-Peña M, et al. Impulsivity and compulsivity in gambling
765 disorder and bulimic spectrum eating disorders: Analysis of
766 neuropsychological profiles and sex differences. *European Psychiatry*
767 [Internet]. 2023 [cited 2025 Jul 17];66(1). Available from:
768 <https://pubmed.ncbi.nlm.nih.gov/37855168/>
- 769 38. Perpiñá C, Segura M, Sánchez-Reales S. Cognitive flexibility and
770 decision-making in eating disorders and obesity. *Eating and Weight*
771 *Disorders* [Internet]. 2017 Sep 1 [cited 2025 Jul 17];22(3):435–44.
772 Available from: <https://pubmed.ncbi.nlm.nih.gov/27744608/>
- 773 39. Steward T, Mestre-Bach G, Vintró-Alcaraz C, Agüera Z, Jiménez-Murcia
774 S, Granero R, et al. Delay Discounting of Reward and Impulsivity in
775 Eating Disorders: From Anorexia Nervosa to Binge Eating Disorder.
776 *European Eating Disorders Review* [Internet]. 2017 Nov 1 [cited 2025
777 Jul 17];25(6):601–6. Available from:
778 <https://pubmed.ncbi.nlm.nih.gov/29057603/>
- 779 40. Testa G, Granero R, Misiolek A, Vintró-Alcaraz C, Mallorqui-Bagué N,
780 Lozano-Madrid M, et al. Impact of Impulsivity and Therapy Response in
781 Eating Disorders from a Neurophysiological, Personality and Cognitive
782 Perspective. *Nutrients* [Internet]. 2022 Dec 1 [cited 2025 Jul

- 783 17];14(23). Available from:
784 <https://pubmed.ncbi.nlm.nih.gov/36501041/>
- 785 41. Lozano-Madrid M, Bryan DC, Granero R, Sánchez I, Riesco N, Mallorquí-
786 Bagué N, et al. Impulsivity, emotional dysregulation and executive
787 function deficits could be associated with alcohol and drug abuse in
788 eating disorders. *J Clin Med* [Internet]. 2020 Jun 1 [cited 2025 Jul
789 17];9(6):1–13. Available from:
790 <https://pubmed.ncbi.nlm.nih.gov/32575816/>
- 791 42. Munguía L, Lucas I, Jiménez-Murcia S, Mora-Maltas B, Granero R,
792 Miranda-Olivos R, et al. Executive functions in binge spectrum eating
793 disorders with comorbid compulsive buying. *European Eating*
794 *Disorders Review* [Internet]. 2021 Nov 1 [cited 2025 Jul 24];29(6):854–
795 67. Available from: <https://pubmed.ncbi.nlm.nih.gov/34322952/>
- 796 43. Steward T, Mestre-Bach G, Vintró-Alcaraz C, Lozano-Madrid M, Agüera
797 Z, Fernández-Formoso JA, et al. Food addiction and impaired executive
798 functions in women with obesity. *European Eating Disorders Review*
799 [Internet]. 2018 Nov 1 [cited 2025 Jul 17];26(6):574–84. Available
800 from: <https://pubmed.ncbi.nlm.nih.gov/30159982/>
- 801 44. Rodrigue C, Ouellette AS, Lemieux S, Tchernof A, Biertho L, Bégin C.
802 Executive functioning and psychological symptoms in food addiction: a
803 study among individuals with severe obesity. *Eating and Weight*
804 *Disorders* [Internet]. 2018 Aug 1 [cited 2025 Jul 17];23(4):469–78.
805 Available from: <https://pubmed.ncbi.nlm.nih.gov/29947017/>
- 806 45. Blume M, Schmidt R, Hilbert A. Executive functioning in obesity, food
807 addiction, and binge-eating disorder. *Nutrients* [Internet]. 2019 Jan 1
808 [cited 2025 Jul 17];11(1). Available from:
809 <https://pubmed.ncbi.nlm.nih.gov/30597858/>
- 810 46. Gearhardt A, Corbin W, Brownell K. Development of the Yale Food
811 Addiction Scale Version 2.0. *Psychology of Addictive Behaviors*.
812 2016;30:113–21.
- 813 47. Granero R, Jiménez-Murcia S, Gerhardt AN, Agüera Z, Aymamí N,
814 Gómez-Peña M, et al. Validation of the Spanish version of the Yale
815 Food Addiction Scale 2.0 (YFAS 2.0) and clinical correlates in a sample
816 of eating disorder, gambling disorder, and healthy control participants.
817 *Front Psychiatry* [Internet]. 2018 [cited 2020 Feb 20];9:321. Available
818 from: <http://www.ncbi.nlm.nih.gov/pubmed/29887808>
- 819 48. Garner DM. *Inventario de Trastornos de la Conducta Alimentaria (EDI-*
820 *2)—Manual*. Madrid, Spain: TEA; 1998.
- 821 49. Derogatis LR. *SCL-90-R: Symptom Checklist-90-R. Administration,*
822 *Scoring and Procedures Manual—II for the Revised Version*. Towson,
823 MD, USA; Clinical Psychometric Research; 1994.

- 824 50. Derogatis LR. SCL-90-R. Cuestionario de 90 Síntomas-Manual. TEA,
825 editor. Madrid, Spain; 2002.
- 826 51. Whiteside SP, Lynam DR. The five factor model and impulsivity: Using
827 a structural model of personality to understand impulsivity. *Pers*
828 *Individ Dif*. 2001 Mar 1;30(4):669-89.
- 829 52. Verdejo-García A, Lozano Ó, Moya M, Alcázar MÁ, Pérez-García M.
830 Psychometric properties of a spanish version of the UPPS-P impulsive
831 behavior scale: Reliability, validity and association with trait and
832 cognitive impulsivity. *J Pers Assess* [Internet]. 2010;92(1):70-7.
833 Available from: <https://pubmed.ncbi.nlm.nih.gov/20013458/>
- 834 53. Gratz KL, Roemer L. Multidimensional assessment of emotion
835 regulation and dysregulation: Development, factor structure, and
836 initial validation of the difficulties in emotion regulation scale. Vol. 26,
837 *Journal of Psychopathology and Behavioral Assessment*. 2004. p. 41-
838 54.
- 839 54. Wolz I, Agüera Z, Granero R, Jiménez-Murcia S, Gratz KL, Menchón JM,
840 et al. Emotion regulation in disordered eating: Psychometric properties
841 of the Difficulties in Emotion Regulation Scale among Spanish adults
842 and its interrelations with personality and clinical severity. *Front*
843 *Psychol* [Internet]. 2015 [cited 2019 Sep 27];6:907. Available from:
844 <http://www.ncbi.nlm.nih.gov/pubmed/26175710>
- 845 55. Bechara A, Damasio AR, Damasio H, Anderson SW. Insensitivity to
846 future consequences following damage to human prefrontal cortex.
847 *Cognition* [Internet]. 1994 [cited 2021 Feb 9];50(1-3):7-15. Available
848 from: <https://pubmed.ncbi.nlm.nih.gov/8039375/>
- 849 56. Brand M, Labudda K, Markowitsch HJ. Neuropsychological correlates of
850 decision-making in ambiguous and risky situations. *Neural Networks*
851 [Internet]. 2006 Oct [cited 2021 Apr 9];19(8):1266-76. Available from:
852 <https://pubmed.ncbi.nlm.nih.gov/16942857/>
- 853 57. Giannunzio V, Degortes D, Tenconi E, Collantoni E, Solmi M,
854 Santonastaso P, et al. Decision-making impairment in anorexia
855 nervosa: New insights into the role of age and decision-making style.
856 *European Eating Disorders Review* [Internet]. 2018 Jul 1 [cited 2021
857 Apr 9];26(4):302-14. Available from:
858 <https://pubmed.ncbi.nlm.nih.gov/29665149/>
- 859 58. Conners CK& MS (Eds.). *Conners' Continuous Performance Test II:*
860 *Computer Program for Windows Technical Guide and Software Manual*.
861 Mutli-Health Systems.; 2000.
- 862 59. American Psychiatric Association. *DIAGNOSTIC AND STATISTICAL*
863 *MANUAL OF DSM-5™*. 5th Editio. 2013.

- 864 60. StataCorp. Stata Statistical Software: Release 18. College Station,
865 Texas: Stata Press Publication (StataCorp LLC).; 2023.
- 866 61. Barrett P. Structural equation modelling: Adjudging model fit. *Pers*
867 *Individ Dif.* 2007;42(5):815–24.
- 868 62. Carlson L, Steward T, Agüera Z, Mestre-Bach G, Magaña P, Granero R,
869 et al. Associations of food addiction and nonsuicidal self-injury among
870 women with an eating disorder: A common strategy for regulating
871 emotions? *European Eating Disorders Review* [Internet]. 2018 Nov 1
872 [cited 2025 Jul 24];26(6):629–37. Available from:
873 <https://pubmed.ncbi.nlm.nih.gov/30318670/>
- 874 63. Villa FM, Crippa A, Rosi E, Nobile M, Brambilla P, Delvecchio G. ADHD
875 and eating disorders in childhood and adolescence: An updated
876 minireview. *J Affect Disord* [Internet]. 2023 Jan 15 [cited 2025 Jul
877 24];321:265–71. Available from:
878 <https://pubmed.ncbi.nlm.nih.gov/36356347/>
- 879 64. Fombouchet Y, Lannegrand L, Lucenet J. Relationships between
880 emotion regulation strategies and executive functions in adolescence:
881 Exploring the effects of discrete emotions and age. *J Adolesc*
882 [Internet]. 2024 Aug 1 [cited 2025 Jul 24];96(6):1239–48. Available
883 from: <https://pubmed.ncbi.nlm.nih.gov/38693714/>
- 884 65. Zhu X, Wang Y, Gao S, Liu X, Zhao F, Zhao X. Associations between
885 executive functions and negative emotions: A moderated mediation
886 analysis of emotion regulation and gender. *J Affect Disord* [Internet].
887 2025 Oct 1 [cited 2025 Jul 24];386. Available from:
888 <https://pubmed.ncbi.nlm.nih.gov/40441638/>
- 889 66. Pappaianni E, Barona M, Doucet GE, Clark C, Frangou S, Micali N.
890 Neurocognitive Endophenotypes for Eating Disorders: A Preliminary
891 High-Risk Family Study. *Brain Sci* [Internet]. 2023 Jan 1 [cited 2025 Jul
892 24];13(1). Available from: <https://pubmed.ncbi.nlm.nih.gov/36672080/>
- 893 67. Pappaianni E, Borsarini B, Berchio C, Aicoboaie S, Konstantopoulou S
894 V., Van de Van de ville D, et al. Neurobiology and Cognition in Girls at
895 High-Risk of Eating Disorders: Exploring Imaging-Derived Trait
896 Markers. *European Eating Disorders Review* [Internet]. 2025 [cited
897 2025 Jul 24]; Available from:
898 <https://pubmed.ncbi.nlm.nih.gov/40275482/>
- 899 68. Mallorquí-Bagué N, Lozano-Madrid M, Granero R, Mestre-Bach G,
900 Vintró-Alcaraz C, Sánchez I, et al. Cognitive and clinical gender-related
901 differences among binge-spectrum eating disorders: Analysis of
902 therapy response predictors. *European Eating Disorders Review*
903 [Internet]. 2023 May 1 [cited 2025 Jul 24];31(3):377–89. Available
904 from: <https://pubmed.ncbi.nlm.nih.gov/36482806/>

69. Eichen DM, Matheson BE, Appleton-Knapp SL, Boutelle KN. Neurocognitive Treatments for Eating Disorders and Obesity. *Curr Psychiatry Rep* [Internet]. 2017 Sep 1 [cited 2025 Jul 24];19(9). Available from: <https://pubmed.ncbi.nlm.nih.gov/28744627/>
70. Eichen DM, Strong DR, Twamley EW, Boutelle KN. Adding executive function training to cognitive behavioral therapy for binge eating disorder: A pilot randomized controlled trial. *Eat Behav* [Internet]. 2023 Dec 1 [cited 2025 Jul 24];51. Available from: <https://pubmed.ncbi.nlm.nih.gov/37660487/>
71. Klein AS, Skinner JB, Hawley KM. Adapted group-based dialectical behaviour therapy for binge eating in a practicing clinic: Clinical outcomes and attrition. *European Eating Disorders Review* [Internet]. 2012 May [cited 2025 Jul 24];20(3). Available from: <https://pubmed.ncbi.nlm.nih.gov/22367862/>
72. Safer DL, Telch CF, Agras WS. Dialectical behavior therapy for bulimia nervosa. *American Journal of Psychiatry* [Internet]. 2001 [cited 2025 Jul 24];158(4):632–4. Available from: <https://pubmed.ncbi.nlm.nih.gov/11282700/>
73. Hempel R, Vanderbleek E, Lynch TR. Radically open DBT: Targeting emotional loneliness in Anorexia Nervosa. *Eat Disord* [Internet]. 2018 Jan 2 [cited 2025 Jul 24];26(1):92–104. Available from: <https://pubmed.ncbi.nlm.nih.gov/29384459/>
74. Butryn ML, Juarascio A, Shaw J, Kerrigan SG, Clark V, O’Planick A, et al. Mindfulness and its relationship with eating disorders symptomatology in women receiving residential treatment. *Eat Behav* [Internet]. 2013 Jan [cited 2025 Jul 24];14(1):13–6. Available from: <https://pubmed.ncbi.nlm.nih.gov/23265395/>
75. Sala M, Vanzhula I, Roos CR, Levinson CA. Mindfulness and Eating Disorders: A Network Analysis. *Behav Ther* [Internet]. 2022 Mar 1 [cited 2025 Jul 24];53(2):224–39. Available from: <https://pubmed.ncbi.nlm.nih.gov/35227400/>

938 **Table 1** Descriptive for the sample

		HC (<i>n</i> = 63)		ED (<i>n</i> = 89)			AN (<i>n</i> = 40)		BSD (<i>n</i> = 49)		
		<i>n</i>	%	<i>n</i>	%	<i>p</i>	<i>n</i>	%	<i>n</i>	%	<i>p</i>
Marital	Single	47	74.6%	66	74.2%	.384	38	95.0%	28	57.1%	<.001*
	Married	11	17.5%	20	22.5%		2	5.0%	18	36.7%	
	Divorced	5	7.9%	3	3.4%		0	0.0%	3	6.1%	
Education	Primary	10	15.9%	42	47.2%	<.001*	10	25.0%	32	65.3%	<.001*
	Secondary	18	28.6%	30	33.7%		21	52.5%	9	18.4%	
	University	35	55.6%	17	19.1%		9	22.5%	8	16.3%	
Employed											
	Unemployed	13	20.6%	29	32.6%	.105	16	40.0%	13	26.5%	.177
	Employed or student	50	79.4%	60	67.4%		24	60.0%	36	73.5%	
Social index	High	6	9.5%	1	1.1%	.005*	0	0.0%	1	2.0%	.025*
	Mean-high	17	27.0%	12	13.5%		5	12.5%	7	14.3%	
	Mean	6	9.5%	13	14.6%		1	2.5%	12	24.5%	
	Mean-low	15	23.8%	16	18.0%		7	17.5%	9	18.4%	
	Low	19	30.2%	47	52.8%		27	67.5%	20	40.8%	
		<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>p</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>p</i>
Age (years-old)		33.59	10.89	31.75	11.33	.319	23.23	6.41	38.71	9.58	<.001*
Onset of ED (years-old)		---	---	19.93	9.32		---	---	---	---	
Duration of ED (years-old)		---	---	11.03	9.07		---	---	---	---	
		<i>n</i>	%	<i>n</i>	%	<i>p</i>	<i>n</i>	%	<i>n</i>	%	<i>p</i>
YFAS-2	Positive	0	0%	65	73.0%	<.001*	24	60.0%	41	83.7%	.012*
	Negative	63	100%	24	27.0%		16	40.0%	8	16.3%	

939 *Note.* HC: healthy controls. ED: eating disorder. AN: anorexia. BSD: Binge spectrum disorder.
 940 SD: standard deviation.

941 *Bold: significant comparison (.05 level).

Table 2 Comparison between the groups for the sociodemographic profiles, onset and duration of ED

		HC (<i>n</i> = 63)		ED FA ⁻ (<i>n</i> = 24)		ED FA ⁺ (<i>n</i> = 65)		HC <i>vs</i> ED FA ⁻		HC <i>vs</i> ED FA ⁺		ED FA ⁻ <i>vs</i> ED FA ⁺	
		<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>p</i>	<i>C-V</i>	<i>p</i>	<i>C-V</i>	<i>p</i>	<i>C-V</i>
Marital	Single	47	74.6	22	91.7	44	67.7	.173	.201	.323	.133	.068	.246
			%		%		%		†				†
	Married	11	17.5	2	8.3%	18	27.7						
Education	Divorced	5	7.9%	0	0.0%	3	4.6%						
	Primary	10	15.9	9	37.5	33	50.8	.001	.393	.001	.408	.136	.212
			%		%		%	*	†	*	†		†
	Secondary	18	28.6	12	50.0	18	27.7						
			%		%		%						
	University	35	55.6	3	12.5	14	21.5						
Employment			%		%		%						
	Unemployed	13	20.6	10	41.7	19	29.2	.047	.213	.262	.099	.267	.118
			%		%		%	*	†				
	Employed	50	79.4	14	58.3	46	70.8						
			%		%		%						
	Social index												
High		6	9.5%	0	0.0%	1	1.5%	.036	.343	.040	.280	.785	.139
			%		%		%	*	†	*	†		
	Mean-high	17	27.0	2	8.3%	10	15.4						
			%		%		%						
	Mean	6	9.5%	3	12.5	10	15.4						
			%		%		%						
	Mean-low	15	23.8	4	16.7	12	18.5						
			%		%		%						
	Low	19	30.2	15	62.5	32	49.2						
			%		%		%						
		<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>p</i>	<i> d </i>	<i>p</i>	<i> d </i>	<i>p</i>	<i> d </i>
Age (years-old)		33.5	10.8	26.3	9.04	33.7	11.5	.007	0.72			.005	0.71
		9	9	8		4	0	*	†	.938	0.01	*	†
Onset of ED (years-old)		---	---	17.6	6.69	20.7	10.0	---	---	---	---	.157	0.37
				3	7	8	2						
Duration of ED (years-old)		---	---	7.06	8.37	12.5	8.94	---	---	---	---	.011	0.63
				7	4							*	†

Note. HC: healthy controls. ED: eating disorder. FA: food addiction. SD: standard deviation.

*Bold: significant comparison (.05 level). †Bold: effect size within mild-moderate to large-high range.

947 **Table 3** Comparison between the groups for clinical profiles

	HC (<i>n</i> = 63)		ED FA $\square$ (<i>n</i> = 24)		ED FA+ (<i>n</i> = 65)		HC <i>vs</i> ED FA $\square$		HC <i>vs</i> ED FA+		ED FA $\square$ <i>vs</i> ED FA+	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>p</i>	<i> d </i>	<i>p</i>	<i> d </i>	<i>p</i>	<i> d </i>
BMI (kg/m ²)	21.8 1	2.21	23.2 0	11.2 7	33.2 9	13.4 6	.844	0.17	.001 *	1.19 †	.001 *	0.81 †
EDI-2 Drive for thinness	2.19	2.90	10.2 9	6.17	13.4 6	4.99	.001 *	1.68 †	.001 *	2.76 †	.014 *	0.57 †
EDI-2 Body dissatisfaction	4.00	5.29	12.7 5	7.30	18.7 1	6.40	.001 *	1.37 †	.001 *	2.50 †	.001 *	0.87 †
EDI-2 Interceptive awareness	1.60	2.26	9.58	6.49	13.1 8	6.03	.001 *	1.64 †	.001 *	2.54 †	.011 *	0.57 †
EDI-2 Bulimia	0.44	1.09	4.04	4.95	8.09	5.19	.001 *	1.00 †	.001 *	2.04 †	.001 *	0.80 †
EDI-2 Interpersonal distrust	1.86	2.78	5.13	4.13	6.52	5.08	.005 *	0.93 †	.001 *	1.14 †	.366	0.30
EDI-2 Ineffectiveness	1.17	2.00	8.92	6.50	13.1 5	6.12	.001 *	1.61 †	.001 *	2.63 †	.002 *	0.67 †
EDI-2 Maturity fears	4.25	3.61	5.58	3.11	8.42	5.73	.483	0.40	.001 *	0.87 †	.038 *	0.61 †
EDI-2 Perfectionism	3.62	3.26	4.50	3.13	6.32	4.13	.603	0.28	.001 *	0.73 †	.115	0.50 †
EDI-2 Impulse regulation	0.75	1.61	3.96	5.35	5.95	5.12	.006 *	0.81 †	.001 *	1.37 †	.129	0.38
EDI-2 Ascetic	1.78	1.45	5.00	3.38	7.69	4.05	.001 *	1.24 †	.001 *	1.94 †	.002 *	0.72 †
EDI-2 Social insecurity	2.19	3.32	6.17	4.93	8.52	4.65	.001 *	0.95 †	.001 *	1.57 †	.067	0.51 †
EDI-2 Total score	23.9 0	16.6 8	75.9 2	37.7 1	109. 98	34.2 8	.001 *	1.78 †	.001 *	3.19 †	.001 *	0.95 †
SCL-90R Somatization	0.55	0.50	1.78	0.76	2.04	0.84	.001 *	1.89 †	.001 *	2.15 †	.296	0.33
SCL-90R Obsessive	0.70	0.52	1.60	0.79	2.08	0.69	.001 *	1.34 †	.001 *	2.26 †	.008 *	0.65 †
SCL-90R Sensitivity	0.53	0.50	1.65	0.93	2.23	0.80	.001 *	1.50 †	.001 *	2.56 †	.004 *	0.67 †
SCL-90R Depressive	0.62	0.56	2.04	0.91	2.62	0.67	.001 *	1.88 †	.001 *	3.26 †	.002 *	0.73 †
SCL-90R Anxiety	0.47	0.43	1.42	0.68	1.89	0.80	.001 *	1.66 †	.001 *	2.20 †	.013 *	0.62 †
SCL-90R Hostility	0.33	0.44	1.16	0.98	1.34	0.93	.001 *	1.09 †	.001 *	1.40 †	.597	0.20
SCL-90R Phobic anxiety	0.13	0.28	0.74	0.66	1.11	0.85	.001 *	1.22 †	.001 *	1.55 †	.063	0.48
SCL-90R Paranoia	0.45	0.58	1.10	0.75	1.59	0.78	.001 *	0.96 †	.001 *	1.66 †	.014 *	0.64 †
SCL-90R Psychotic	0.25	0.36	1.15	0.58	1.51	0.75	.001 *	1.87 †	.001 *	2.15 †	.040 *	0.54 †
SCL-90R GSI	0.48	0.39	1.51	0.66	1.95	0.60	.001 *	1.90 †	.001 *	2.91 †	.003 *	0.70 †
SCL-90R PST	27.9 0	18.0 8	56.6 3	19.4 4	69.2 6	11.3 9	.001 *	1.53 †	.001 *	2.74 †	.005 *	0.79 †
SCL-90R PSDI	1.47	0.39	2.19	0.44	2.53	0.51	.001 *	1.73 †	.001 *	2.34 †	.008 *	0.71 †
UPPS-P Lack of premeditation	21.4 3	4.40	22.2 1	5.70	24.1 5	5.55	.818	0.15	.012 *	0.54 †	.286	0.35
UPPS-P Lack of perseverance	19.5 6	4.97	22.7 5	5.25	23.8 6	5.63	.046 *	0.62 †	.001 *	0.81 †	.681	0.20

UPPS-P Sensation seeking	24.8 7	5.04	24.8 3	7.07	25.0 6	8.14	1.00 0	0.01	.988	0.03	.990	0.03
UPPS-P Positive urgency	21.2 9	7.11	26.0 8	9.63	28.7 2	8.48	.052	0.57 †	.001 *	0.95 †	.400	0.29
UPPS-P Negative urgency	23.7 6	6.01	30.8 8	5.50	34.2 8	6.16	.001 *	1.24 †	.001 *	1.73 †	.063	0.58 †
UPPS-P Total score	113. 32	20.3 8	126. 75	21.0 5	136. 08	22.6 2	.036 *	0.65 †	.001 *	1.06 †	.195	0.43
DERS Non acceptance	10.5 1	4.69	18.3 3	6.07	22.1 5	5.57	.001 *	1.44 †	.001 *	2.26 †	.012 *	0.66 †
DERS Goal directed behaviours	11.6 0	3.35	16.6 3	4.00	18.0 0	4.26	.001 *	1.36 †	.001 *	1.67 †	.332	0.33
DERS Diff. impulse control	8.90	2.49	15.1 7	6.05	17.5 1	5.61	.001 *	1.35 †	.001 *	1.98 †	.113	0.40
DERS Lack of awareness	12.9 4	3.78	16.7 5	3.63	19.5 8	4.83	.001 *	1.03 †	.001 *	1.53 †	.022 *	0.66 †
DERS Limited access emotions	13.5 2	4.75	25.0 4	6.99	27.3 2	6.38	.001 *	1.93 †	.001 *	2.45 †	.269	0.34
DERS Lack of emotional clarity	8.63	3.16	15.0 0	5.32	16.2 9	3.67	.001 *	1.46 †	.001 *	2.23 †	.362	0.28
DERS Total score	66.1 1	14.6 2	107. 63	23.6 8	120. 71	21.3 0	.001 *	2.11 †	.001 *	2.99 †	.019 *	0.58 †

948 *Note.* HC: healthy controls. ED: eating disorder. FA: food addiction. SD: standard deviation.
 949 BMI: body mass index.

950 *Bold: significant comparison (.05 level). †Bold: effect size within mild-moderate to large-high
 951 range.

952 Results for the EDI-2, SCL-90R, UPPS-P and DERS obtained with ANCOVA adjusted by the
 953 participants' sex, education levels and ED subtype.

954 **Table 4** Comparison between the groups for neurocognitive
 955 measures

	HC (<i>n</i> = 63)		ED FA ⁻ (<i>n</i> = 24)		ED FA ⁺ (<i>n</i> = 65)		HC <i>vs</i> ED FA ⁻		HC <i>vs</i> ED FA ⁺		ED FA ⁻ <i>vs</i> ED FA ⁺	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>p</i>	<i> d </i>	<i>p</i>	<i> d </i>	<i>p</i>	<i> d </i>
IGT Block-1	-2.27	4.94	-2.53	3.55	-3.22	3.90	.862	0.06	.521	0.21	.529	0.18
IGT Block-2	-0.11	4.57	-0.42	2.26	0.01	4.01	.826	0.09	.929	0.03	.668	0.13
IGT Block-3	2.87	7.41	-2.00	4.67	1.73	6.17	.036 *	0.79 †	.611	0.17	.024 *	0.68 †
IGT Block-4	1.27	7.82	0.97	5.24	1.72	7.94	.909	0.05	.861	0.06	.691	0.11
IGT Block-5	3.14	9.66	0.32	7.39	-0.41	6.49	.311	0.33	.190	0.43	.713	0.11
IGT Total	4.83	21.6 1	-3.43	15.9 6	0.05	19.1 0	.236	0.43	.479	0.23	.484	0.20
IGT Learning	6.79	15.2 5	4.24	12.8 5	4.52	11.6 5	.587	0.18	.618	0.17	.934	0.02
IGT Risk	4.41	15.3 2	1.29	11.8 6	1.31	12.1 8	.510	0.23	.501	0.22	.994	0.00
CTP Omissions-Tgeneral	60.8 7	41.3 5	56.5 7	31.1 9	46.8 5	8.48	.682	0.12	.171	0.47	.197	0.43
CTP Commissions-Tgeneral	53.7 0	12.5 3	57.7 7	8.74	56.0 0	9.18	.273	0.38	.524	0.21	.502	0.20
CTP HitRT-Tgeneral	50.0 7	10.4 3	50.2 9	9.84	48.0 8	10.2 2	.946	0.02	.534	0.19	.347	0.22
CTP HitSE-Tgeneral	52.1 1	11.9 1	48.2 2	12.3 4	50.1 7	11.0 9	.311	0.32	.602	0.17	.477	0.17
CTP VarSE-Tgeneral	50.3 9	10.0 8	48.4 5	8.24	51.6 0	9.52	.552	0.21	.703	0.12	.178	0.35
CTP DPrime-Tgeneral	53.5 6	10.5 8	55.7 2	5.80	54.8 2	6.73	.465	0.25	.661	0.14	.670	0.14
CTP Beta-Tgeneral	47.8 9	6.58	47.6 0	3.57	48.4 6	8.39	.906	0.06	.815	0.07	.629	0.13
CTP Perseverations-Tgeneral	56.9 5	15.8 4	53.6 1	9.59	58.6 2	25.4 8	.631	0.26	.806	0.08	.315	0.26
CTP HitRTBlock-Tgeneral	47.8 8	11.7 0	48.9 2	5.59	50.8 6	7.04	.745	0.11	.339	0.31	.398	0.31
CTP HitSEBlock-Tgeneral	49.9 0	12.0 7	55.2 0	7.77	57.5 6	8.72	.157	0.52 †	.036 *	0.73 †	.378	0.28
CTP HitRTIsi-Tgeneral	53.3 2	12.2 1	47.7 5	8.22	48.7 0	9.43	.131	0.53 †	.197	0.42	.716	0.11
CTP HitSEIsi-Tgeneral	51.5 7	11.4 6	48.8 3	7.86	50.6 4	11.3 8	.475	0.28	.801	0.08	.510	0.18

956 *Note.* HC: healthy controls. ED: eating disorder. FA: food addiction. SD: standard deviation.

957 *Bold: significant comparison (.05 level). †Bold: effect size within mild-moderate to large-high
 958 range.

959 Results obtained with ANCOVA adjusted by the participants' sex, education levels and ED
 960 subtype.

Table 5 Complete results obtained in the SEM (standardized coefficients)

		B	SE	z- stat	p	95% CI B	
Structural							
DERS total	Cognition	-	0.1451	-	.044	-	-
		0.2917		2.01		0.6478	0.0644
UPPS-P total	Cognition	-	0.1484	-	.034	-	-
		0.3145		2.12		0.7489	0.1198
FA (YFAS-total)	DERS total	0.2749	0.0849	3.24	.001	0.1088	0.4411
	UPPS-P total	0.1333	0.0670	1.99	.047	0.0358	0.3023
EDI-2 total	DERS total	0.6102	0.0616	9.91	<.001	0.4895	0.7310
	FA (YFAS-total)	0.3190	0.0706	4.52	<.001	0.1808	0.4572
Measurement	IGT total	0.3811	0.1886	2.02	.043	0.0113	0.7508
	CPT omissions	-	0.0588	-	.048	-	0.2748
		0.1164		1.98		0.5076	
	CPT perseverations	0.4132	0.1996	2.07	.038	0.0223	0.8041
Covariances	DERS total ; UPPS-P total	0.3523	0.1119	3.15	.002	0.1334	0.5713

Note. B: standardized coefficient. SE: standard error. 95%CI: 95% confidence interval. Sample size: N = 89 ED patients.

Figure 1 Learning curve in the IGT task

Note. HC: healthy controls. ED: eating disorder. FA: food addiction.

Figure 2 Path diagram: standardized coefficients obtained in the SEM within the clinical subsample.

Note. Only significant coefficients retained in the final model. Results adjusted by age and education level. N = 89 ED patients.

Fit statistics: $\chi^2 = 24.07$ ($p=.239$); RMSEA=0.048; CFI=0.973; TLI=0.952; SRMR=0.057

ARTICLE IN PRESS



