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

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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 071)*—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

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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.

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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.

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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

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

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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 validly 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-

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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 off 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 emerge.

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

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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

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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).

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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.

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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,

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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

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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.

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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

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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).

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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

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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 an 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: χ^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 ---

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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

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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

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

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

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

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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 take 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

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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

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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

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CRT	Cognitive Remediation Therapy
DBT	Dialectical behavior therapy
DERS	Difficulties in Emotion Regulation Scale
DSM-5	Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
EDs	Eating disorders
ED FA+	Eating disorders and Food addiction present
ED FA-	Eating disorders and Food addiction absent
EF	Executive function
EDI-2	Eating Disorder Inventory-2
FA	Food addiction
GD	Gambling Disorder
GSI	Global severity index
HC	Healthy Control
IGT	Iowa Gambling Task
OBE	Obesity
PSDI	Positive symptom total
PST	Positive symptom distress index
SCL-90-R	Symptom Checklist-Revised

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SD Standard deviation

SEM Structural Equations Model

SUD Substance Use Disorders

UPPS-P Impulsive behaviour scale

YFAS-2 Yale Food Addiction Scale 2.0.

DECLARATIONS

Ethics

The present study was approved by the Clinical Research Ethics Committee of the 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. Participants did not receive any form of compensation for their participation in this study.

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Data availability statement

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

Conflicts of interest/Competing interests

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

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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.

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938 ***Table 1*** Descriptive for the sample

		HC *(n = 63)*		ED *(n = 89)*			AN *(n = 40)*		BSD *(n = 49)*		
		n	*%*	*n*	*%*	*p*	*n*	*%*	*n*	*%*	*p*
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%	
		Mean	*SD*	*Mean*	*SD*	*p*	*Mean*	*SD*	*Mean*	*SD*	*p*
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		---	---	---	---	
		n	*%*	*n*	*%*	*p*	*n*	*%*	*n*	*%*	*p*
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).

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

		HC (*n = 63*)		ED FA− (*n = 24*)		ED FA+ (*n = 65*)		HC *vs* ED FA−		HC *vs* ED FA+		ED FA− *vs* ED FA+	
		n	*%*	*n*	*%*	*n*	*%*	*p*	*C-V*	*p*	*C-V*	*p*	*C-V*
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 %						
	Divorced	5	7.9%	0	0.0%	3	4.6%						
Education	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		13	20.6 %	10	41.7 %	19	29.2 %	**.047 ***	**.213 †**	.262	.099	.267	.118
	Unemployed												
	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 %						
		Mean	*SD*	*Mean*	*SD*	*Mean*	*SD*	*p*	*/d/*	*p*	*/d/*	*p*	*/d/*
Age (years-old)		33.59	10.89	26.38	9.04	33.74	11.50	**.007 ***	**0.72 †**	.938	0.01	**.005 ***	**0.71 †**
Onset of ED (years-old)		---	---	17.63	6.697	20.78	10.02	---	---	---	---	.157	0.37
Duration of ED (years-old)		---	---	7.067	8.374	12.5	8.94	---	---	---	---	**.011 ***	**0.63 †**

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

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

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

	HC *(n = 63)*		ED FA− *(n = 24)*		ED FA+ *(n = 65)*		HC *vs* ED FA−		HC *vs* ED FA+		ED FA− *vs* ED FA+	
	Mean	*SD*	*Mean*	*SD*	*Mean*	*SD*	*p*	*/d/*	*p*	*/d/*	*p*	*/d/*
BMI (kg/m²)	21.81	2.21	23.20	11.27	33.29	13.46	.844	0.17	**.001***	**1.19†**	**.001***	**0.81†**
EDI-2 Drive for thinness	2.19	2.90	10.29	6.17	13.46	4.99	**.001***	**1.68†**	**.001***	**2.76†**	**.014***	**0.57†**
EDI-2 Body dissatisfaction	4.00	5.29	12.75	7.30	18.71	6.40	**.001***	**1.37†**	**.001***	**2.50†**	**.001***	**0.87†**
EDI-2 Interoceptive awarenes	1.60	2.26	9.58	6.49	13.18	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	13.15	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	23.90	16.68	75.92	37.71	109.98	34.28	**.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	27.90	18.08	56.63	19.44	69.26	11.39	**.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	21.43	4.40	22.21	5.70	24.15	5.55	.818	0.15	**.012***	**0.54†**	.286	0.35
UPPS-P Lack of perseverance	19.56	4.97	22.75	5.25	23.86	5.63	**.046***	**0.62†**	**.001***	**0.81†**	.681	0.20

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UPPS-P Sensation seeking	24.87	5.04	24.83	7.07	25.06	8.14	1.000	0.01	.988	0.03	.990	0.03
UPPS-P Positive urgency	21.29	7.11	26.08	9.63	28.72	8.48	.052	**0.57 †**	**.001 ***	**0.95 †**	.400	0.29
UPPS-P Negative urgency	23.76	6.01	30.88	5.50	34.28	6.16	**.001 ***	**1.24 †**	**.001 ***	**1.73 †**	.063	**0.58 †**
UPPS-P Total score	113.32	20.38	126.75	21.05	136.08	22.62	**.036 ***	**0.65 †**	**.001 ***	**1.06 †**	.195	0.43
DERS Non acceptance	10.51	4.69	18.33	6.07	22.15	5.57	**.001 ***	**1.44 †**	**.001 ***	**2.26 †**	**.012 ***	**0.66 †**
DERS Goal directed behaviours	11.60	3.35	16.63	4.00	18.00	4.26	**.001 ***	**1.36 †**	**.001 ***	**1.67 †**	.332	0.33
DERS Diff. impulse control	8.90	2.49	15.17	6.05	17.51	5.61	**.001 ***	**1.35 †**	**.001 ***	**1.98 †**	.113	0.40
DERS Lack of awareness	12.94	3.78	16.75	3.63	19.58	4.83	**.001 ***	**1.03 †**	**.001 ***	**1.53 †**	**.022 ***	**0.66 †**
DERS Limited access emotions	13.52	4.75	25.04	6.99	27.32	6.38	**.001 ***	**1.93 †**	**.001 ***	**2.45 †**	.269	0.34
DERS Lack of emotional clarity	8.63	3.16	15.00	5.32	16.29	3.67	**.001 ***	**1.46 †**	**.001 ***	**2.23 †**	.362	0.28
DERS Total score	66.11	14.62	107.63	23.68	120.71	21.30	**.001 ***	**2.11 †**	**.001 ***	**2.99 †**	**.019 ***	**0.58 †**

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

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

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

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

	HC (*n = 63*)		ED FA− (*n = 24*)		ED FA+ (*n = 65*)		HC *vs* ED FA−		HC *vs* ED FA+		ED FA− *vs* ED FA+	
	Mean	*SD*	*Mean*	*SD*	*Mean*	*SD*	*p*	*/d/*	*p*	*/d/*	*p*	*/d/*
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	21.61	-3.43	15.96	0.05	19.10	.236	0.43	.479	0.23	.484	0.20
IGT Learning	6.79	15.25	4.24	12.85	4.52	11.65	.587	0.18	.618	0.17	.934	0.02
IGT Risk	4.41	15.32	1.29	11.86	1.31	12.18	.510	0.23	.501	0.22	.994	0.00
CTP Omissions-Tgeneral	60.87	41.35	56.57	31.19	46.85	8.48	.682	0.12	.171	0.47	.197	0.43
CTP Commissions-Tgeneral	53.70	12.53	57.77	8.74	56.00	9.18	.273	0.38	.524	0.21	.502	0.20
CTP HitRT-Tgeneral	50.07	10.43	50.29	9.84	48.08	10.22	.946	0.02	.534	0.19	.347	0.22
CTP HitSE-Tgeneral	52.11	11.91	48.22	12.34	50.17	11.09	.311	0.32	.602	0.17	.477	0.17
CTP VarSE-Tgeneral	50.39	10.08	48.45	8.24	51.60	9.52	.552	0.21	.703	0.12	.178	0.35
CTP DPrime-Tgeneral	53.56	10.58	55.72	5.80	54.82	6.73	.465	0.25	.661	0.14	.670	0.14
CTP Beta-Tgeneral	47.89	6.58	47.60	3.57	48.46	8.39	.906	0.06	.815	0.07	.629	0.13
CTP Perseverations-Tgeneral	56.95	15.84	53.61	9.59	58.62	25.48	.631	0.26	.806	0.08	.315	0.26
CTP HitRTBlock-Tgeneral	47.88	11.70	48.92	5.59	50.86	7.04	.745	0.11	.339	0.31	.398	0.31
CTP HitSEBlock-Tgeneral	49.90	12.07	55.20	7.77	57.56	8.72	.157	**0.52 †**	**.036 ***	**0.73 †**	.378	0.28
CTP HitRTIsi-Tgeneral	53.32	12.21	47.75	8.22	48.70	9.43	.131	**0.53 †**	.197	0.42	.716	0.11
CTP HitSEIsi-Tgeneral	51.57	11.46	48.83	7.86	50.64	11.38	.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.

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

		B	SE	z-stat	p	95% CI B	
Structural							
DERS total	Cognition	-0.2917	0.1451	-2.01	.044	-0.6478	-0.0644
UPPS-P total	Cognition	-0.3145	0.1484	-2.12	.034	-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	-0.1164	0.0588	-1.98	.048	-0.5076	0.2748
	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

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

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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: χ^2 = 24.07 (p=.239); RMSEA=0.048; CFI=0.973; TLI=0.952; SRMR=0.057

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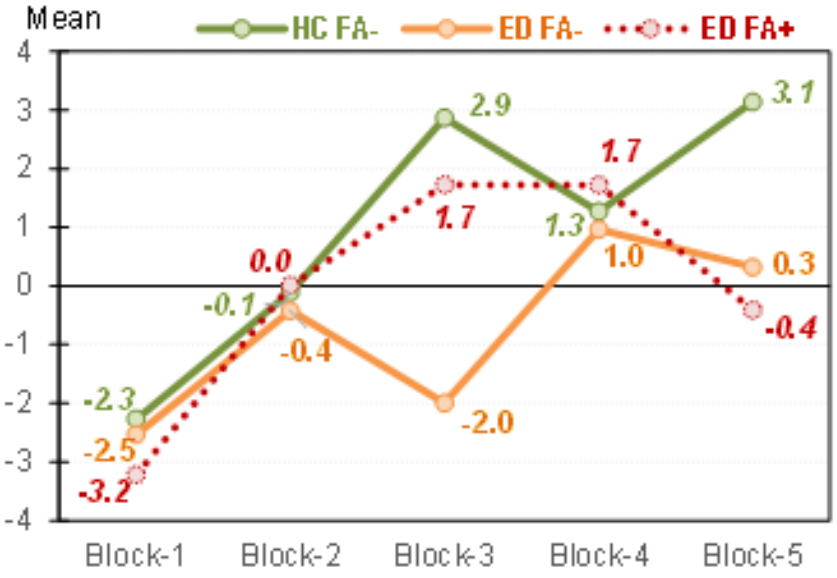
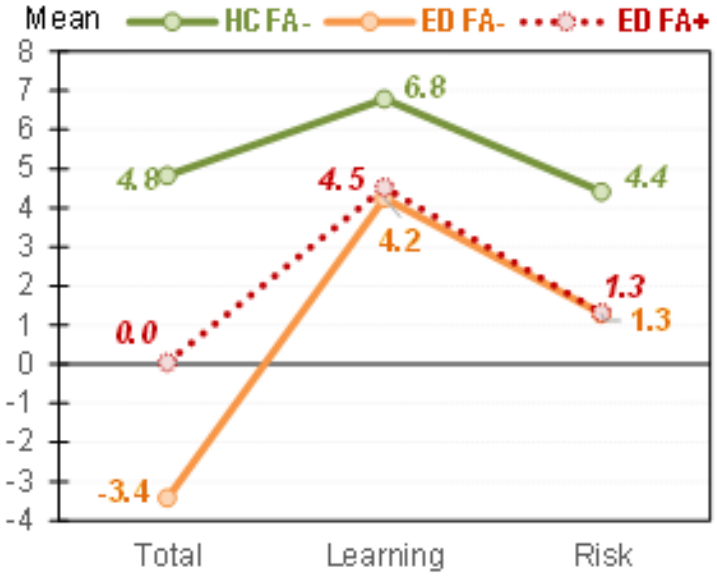
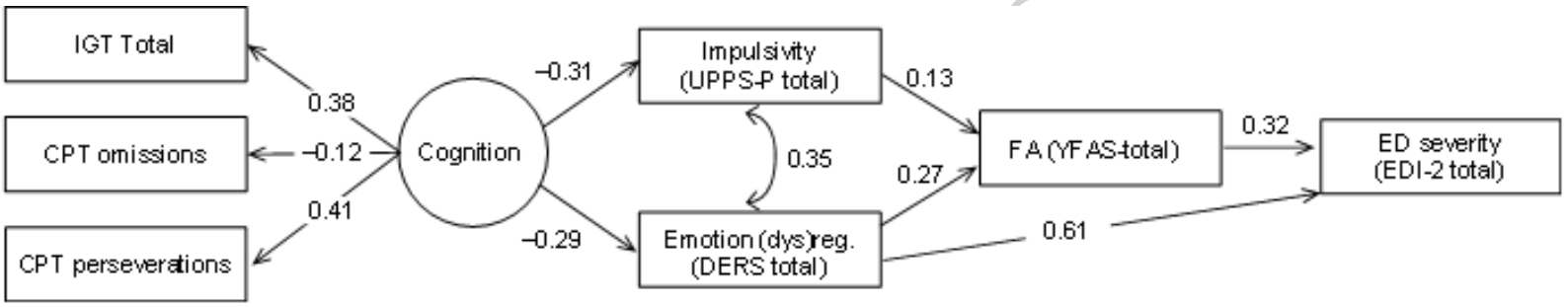

Mean
HC FA-
ED FA-
ED FA+
4
3
2
1
0
-1
-2
-3
-4
-2.3
-2.5
-3.2
-0.1
0.0
2.9
-0.4
1.7
-2.0
1.3
1.7
1.0
3.1
0.3
-0.4
Block-1
Block-2
Block-3
Block-4
Block-5


Mean
HC FA-
ED FA-
ED FA+
8
7
6
5
4
3
2
1
0
-1
-2
-3
-4
4.8
6.8
4.4
0.0
4.5
4.2
1.3
1.3
-3.4
Total
Learning
Risk


IGT Total
CPT omissions
CPT perseverations
0.38
−0.12
0.41
Cognition
−0.31
−0.29
Impulsivity
(UPPS-P total)
Emotion (dys)reg.
(DERS total)
0.35
0.13
0.27
FA (YFAS-total)
0.32
ED severity
(EDI-2 total)
0.61