Review

A Narrative Review on the Neurocognitive Profiles in Eating Disorders and Higher Weight Individuals: Insights for Targeted Interventions

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A Narrative Review on the Neurocognitive Profiles in Eating Disorders and Higher Weight Individuals: Insights for Targeted Interventions

Isabel Krug [1,*], An Binh Dang [1], Evonne Lu [2], Wenn Ooi [1], Jade Portingale [1] and Stephanie Miles [3,4,5]

[1] Melbourne School of Psychological Sciences, The University of Melbourne, Melbourne, VIC, Australia
[2] Nursing and Health Sciences, Monash Faculty of Medicine, Monash University, Melbourne, VIC, Australia
[3] Orygen, Parkville, VIC, Australia
[4] Centre for Youth Mental Health, The University of Melbourne, Melbourne, VIC, Australia
[5] Department of Psychological Sciences, Swinburne University of Technology, Hawthorn, VIC, Australia
* Correspondence: isabel.krug@unimelb.edu.au; Tel.: + 61390358551; Fax: +61393264774

Abstract: Background/Objectives: Recent research has increasingly focused on the neurocognitive characteristics associated with eating disorders (EDs), including, anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), other specified feeding or eating disorders (OSFED) and individuals of higher weight (HW). This critical narrative review summarizes recent findings on neurocognitive processes across the weight spectrum, emphasizing implications for diagnosis and treatment. **Results:** Our review highlights that individuals with AN, especially the restrictive subtype of AN, exhibit reduced cognitive flexibility and increased attention to detail. In BN and BED, cognitive profiles are also marked by cognitive rigidity as well as impaired decision-making and impulsivity, with dysregulated reward processing and attentional bias toward food-related signals and reward sensitivity. OSFED presents a diverse neurocognitive profile, necessitating more focused research to delineate its specific characteristics. Finally, neurocognitive studies in individuals with HW highlight deficits in cognitive flexibility, increased impulsivity, and altered reward processing. These cognitive impairments may contribute to the intricate interplay of factors underlying overeating behaviours and subsequent weight gain. **Conclusions:** Comparing these profiles across the weight spectrum underscores unique and shared impairments, emphasizing the complexity of neurocognitive mechanisms in relation to body mass index (BMI) and eating behaviours. Future research should prioritize longitudinal and intervention studies to elucidate causal relationships and explore cognitive training, virtual reality, and neuromodulation techniques, ultimately informing more personalized treatment strategies for EDs.

Keywords: anorexia nervosa; bulimia nervosa; binge eating disorder; otherwise specified feeding or eating disorders; higher weight; neurocognitive profile; executive functioning

1. Introduction

Neurocognitive characteristics have been increasingly recognised as significant factors in the development and maintenance of eating disorders (EDs). Deficits in executive functioning and neurocognitive symptoms such as impulsivity [1], cognitive inflexibility [2], and poor emotion regulation [3]. have been associated with several EDs. However, it remains unknow if these neurocognitive difficulties are intrinsic to EDs and part of the illness or are secondary consequences of malnutrition and changes in weight.

While EDs, including anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and other specified feeding or eating disorders (OSFED), involve distinct behavioural patterns, they all share common underlying disturbances related to eating, body image, and weight

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[4,5]. Further, when examining EDs, considering people with a higher weight (HW) can provide additional insights - not as an ED, but as a condition situated at the one end of the weight spectrum.

In this review, the term HW will refer to individuals whose body weight or body mass index (BMI) exceeds what health guidelines consider the "normal" range. Specifically, the term HW includes individuals with BMIs classified as overweight (BMI 25–29.9) or obese (BMI ≥ 30) according to the criteria set forth by the World Health Organization [6]. While HW is not classified as an ED, it is often associated with maladaptive eating behaviours that overlap with characteristics of EDs, particularly BED and BN. For instance, one previous study found that lifetime obesity was as high as 87 % in a treatment seeking BED sample [7]. HW individuals also often exhibit neurocognitive and emotional dysfunctions similar to those seen in ED populations, including impaired cognitive flexibility [8], reward processing [9], heightened impulsivity [10], and emotion processing difficulties [11].

AN and HW represent extremes of weight dysregulation, and integrating HW into a broader examination of neurocognitive functioning is both relevant and necessary for understanding the full scope of weight-related problems. While various studies assessing HW have referred to their sample as 'suffering from obesity', we use the terminology of HW throughout the review. This term is preferred in scientific and clinical contexts to reduce stigma and bias, promoting a more neutral, person-centred approach that respects body diversity and lessens the emphasis on weight as a sole health measure [12].

The current review aims to summarize the most recent research on neurocognitive processes in EDs and HW, primarily focusing on data from experimental tasks. Where experimental data are limited or unavailable, self-report data will also be considered. This review will examine neurocognitive domains in different weight categories, including underweight (AN), "normal" weight (BN, OSFED), and HW (BED, HW). By comparing neurocognitive profiles in EDs and HW, we aim to determine whether cognitive impairments are primarily associated with extreme weight (underweight in AN or overweight/HW) or are intrinsic to the disorders themselves. Clarifying this distinction is essential for improving diagnostic practices and developing treatment interventions that address both cognitive and weight-related aspects of these conditions.

2. Anorexia Nervosa

According to the DSM-5, AN is an ED characterized by a persistent restriction of energy intake, an intense fear of gaining weight or becoming fat, and a distorted body image. AN results in a significantly low body weight for the individual's age, sex, and developmental trajectory. It has two primary subtypes: the restrictive subtype (AN-R), and the binge-purging subtype (AN-BP), which is characterized by cycles of binge eating followed by purging behaviours to prevent weight gain [4,5]. AN is associated with numerous severe complications, including electrolyte imbalances, reduced bone density, cognitive impairments, and psychiatric comorbidities, notably anxiety, depression, and obsessive-compulsive traits [13,14].

Beyond the disorder's characteristic cognitive and behavioural distortions concerning food and body image, AN is also linked to neurocognitive deficits in areas such as cognitive flexibility, decision-making, and central coherence [15]. These impairments may be intrinsic to AN or could result from malnutrition and low BMI [16]. Although the extent of the role of starvation in AN is not fully understood, it is notable that some neurocognitive functions remain preserved in AN, and general intelligence typically falls within normal ranges in people diagnosed with AN [17]

2.1. Cognitive Flexibility

refers to the ability to adapt to changing task demands or environmental shifts by adjusting behaviours and/or thoughts [18] . One prominent construct within cognitive flexibility is set shifting, or the capacity to switch between different mental sets or rules. In AN, cognitive inflexibility has been proposed as a mechanism that may perpetuate symptoms and contribute to treatment resistance [2].While some studies indicate that individuals with AN exhibit poorer cognitive flexibility

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compared to healthy controls (HCs) [19–21], other research has reported no significant differences between these groups in cognitive flexibility tasks [22–24].

A systematic review by Miles et al. (2020) highlights these mixed findings, showing that adolescents with AN perform comparably to HCs on cognitive flexibility tasks, whereas adults with AN often demonstrate poorer performance on perceptual-based cognitive flexibility tasks, yet no significant group difference was supported for verbal cognitive flexibility tasks. The review further notes variability in findings for weight-restored and fully recovered AN samples, suggesting that while some aspects of cognitive flexibility may improve, certain deficits may persist even after recovery or weight restoration [2].

While research examining group differences in cognitive flexibility among individuals with AN and HCs has produced mixed results [2], other studies have investigated the relationship between cognitive flexibility and BMI in AN. A recent systematic review concluded that cognitive flexibility was not associated with BMI [17], and subsequent studies have also reported no significant link between cognitive flexibility and BMI in AN samples [25,26]. These findings suggest that impaired cognitive flexibility in AN may not be attributable to weight or starvation alone.

2.2. Central Coherence

refers to the ability to grasp complex stimuli or information, often described as "big picture" thinking, and weak central coherence is typified by an excessive focus on small details. A systematic review and meta-analysis conducted by Keegan et al. (2021) [27] concluded that participants with AN exhibited significantly poorer central coherence compared to HCs. Studies investigating the relationship between BMI and central coherence in individuals with AN have produced varied results [17]. For example, López et al. (2008) [28] found in a sample of women with AN that there was a negative association between BMI and errors on the Homograph Reading Task, a measure of central coherence, yet there were no other significant correlations between BMI and the outcomes of other central coherence tasks included in the study. Conversely, Herbrich et al. (2018) [29] observed a negative correlation between BMI and performance on the Group Embedded Figures Test in participants with AN-BP, but not in the AN-R group.

In summary, the relationship between BMI and central coherence in individuals with AN is complex and warrants further research to better understand its implications for treatment and intervention strategies.

2.3. Attentional Bias

toward and preoccupation with food stimuli is well-documented in individuals with AN [12,30]. The Stroop Task, Dot-Probe Task, in combination with eye-tracking paradigms are commonly used to assess this bias. Eye-tracking research demonstrates that individuals with AN initially display an attentional bias towards food cues which is similar to HCs, but rather than sustaining this attention, individuals with AN quickly switch to attentional avoidance [31]. Werthmann et al. (2019) [31]further found that avoidance was stronger for high-calorie food cues, especially in adults with longer AN illness durations compared to adolescents. This avoidance behaviour tends to become more entrenched as the illness progresses, potentially maintaining the fear of food or restrictive eating behaviours [32]. In terms of BMI, while some work has found no association between BMI and attentional bias[33], other research has revealed that those with a low BMI are more likely to pay attention to high-calorie foods [31].

2.4. Impulsivity

defined as a tendency to act quickly without considering potential consequences, particularly in emotionally charged situations, is frequently linked to EDs, including the AN-BP subtype [1,34]. Studies indicate that individuals with AN-BP display higher impulsivity and greater novelty-seeking behaviours than those with the AN-R subtype [34,35]. Additionally, those with AN-BP are more likely to engage in impulsive, high-risk activities, such as substance use [35] and shoplifting [36].

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Impulsivity may contribute to cycles of disordered eating, like bingeing and purging, by making it difficult to resist urges, potentially worsening symptoms [1]. In contrast, AN-R is often characterized by more rigid and controlled behaviour, highlighting a distinct difference between the two AN subtypes [35].

2.5. Emotional Processing Difficulties

such as challenges in recognizing and being aware of different emotions, have been linked to AN [22]. Research indicates that individuals with AN often struggle with identifying and managing emotions, which may lead to maladaptive behaviours, such as restrictive eating, as a coping strategy [37,38]. *Emotion recognition* - the ability to identify and interpret others' emotional expressions - is essential for social functioning and mental health, and emerging evidence suggests that individuals with AN exhibit significant deficits in this area compared to HCs [39,40]. Several systematic reviews have emphasized impaired emotion recognition and altered responses to social-affective stimuli in individuals with EDs, including those with AN [41,42].

Research consistently indicates that individuals with AN also experience significant challenges in *emotion regulation*, manifesting as difficulties in identifying, expressing, and managing emotions - factors that contribute to the persistence of restrictive eating behaviours and heightened symptom severity [43]. Women with AN have been found to report significantly greater emotion regulation difficulties than HCs [39,40,43,44]. A central element of these emotional difficulties is alexithymia, defined by an impaired ability to recognize and describe one's emotions, which is notably prevalent in AN and intensifies emotion regulation difficulties by limiting emotional awareness [45,46]. Emotion regulation challenges, coupled with alexithymia, often lead to maladaptive coping strategies such as emotional suppression and avoidance, which further reinforce disordered eating patterns and complicate treatment [46]. Consequently, addressing emotion regulation deficits, particularly by enhancing emotional awareness and fostering adaptive coping strategies, is critical for effective AN treatment.

The relationship between emotion processing (recognition and regulation) deficits and BMI in AN is inconsistent, possibly due to studies being conducted in various phases of the illness [22]. While one study found a positive correlation between BMI and emotion processing difficulties in AN such that lower BMI was associated with fewer emotion regulation difficulties [44], others have not found a significant relationship between BMI and emotion regulation [39]. A recent 15-year longitudinal study found that as individuals with AN recovered and their BMI increased, there was no significant linear effect on their emotional processing [22]. This finding is consistent with earlier studies that showed no significant association between BMI and impaired emotional functioning, whether in the acute or recovered phases of AN [39,47,48]. From such findings, Castro et al. (2021) [22] suggested than emotional dysfunction in AN may not solely be a result of malnutrition.

Overall, the relationship between neurocognitive functioning and AN is complex and research indicates that people with AN may experience some neurocognitive deficits. While findings are varied, most studies have not found a significant association between BMI and performance on neurocognitive tasks.

3. Bulimia Nervosa

BN is an ED characterized by recurrent episodes of binge eating - during which a person consumes an unusually large amount of food within a short period (e.g. 2-hour period), accompanied by a sense of loss of control [4,5]. To prevent weight gain, individuals with BN typically engage in compensatory behaviours, such as self-induced vomiting, excessive exercise, or misuse of laxatives. Binge eating episodes and purging behaviours need to occur at least once a week for three months for a diagnosis of BN [4,5]. It has been suggested that the binging and purging behaviours reflect deficiencies in inhibitory control [49], a core executive function that overrides impulses through conscious decision-making. Exploration of neurocognition in BN has also highlighted impaired decision-making compared to those with AN [50] and HCs [51–53].

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3.1. Cognitive Flexibility

research in BN has shown mixed results. Early research suggested poor cognitive flexibility in people diagnosed with BN compared to HCs [54–57]. In contrast, another study found no significant differences in cognitive flexibility between participants with BN and HCs [58]. It also remains unknown if/how cognitive flexibility may be associated with BMI in BN. Given the limited amount of research which has investigated cognitive flexibility in BN specifically, the potential role of cognitive flexibility in treatment outcomes remains unclear.

3.2. Reward Processing

refers to the neural and psychological mechanisms that evaluate, respond to, and guide behaviour based on rewarding stimuli or outcomes [59]. It has been hypothesised that reward processing is dysregulated in BN [60]. While some studies [60,61] have reported increased hedonic processing (the brain's ability to evaluate and respond to pleasurable or rewarding experiences) and heightened sensitivity to food rewards in individuals with BN, others [62] have observed hypo-functioning of brain reward systems in BN and attributed binge eating as a means to compensate for this reward deficit.

A concept that has recently been applied to study reward processing and impulsivity in BN research is delay discounting. It refers to a phenomenon wherein the value of a reward depreciates with temporal delay in its delivery [63]. To our knowledge, only four papers to date have studied delay discounting in BN populations [64–67]. A meta-analysis of the three earliest papers showed elevated discounting of monetary rewards (i.e., greater preference for sooner and smaller amounts of money) in participants with BN compared to controls, reflecting an impulsive preference for immediate rewards over delayed gratification [68]. However, the meta-analysis comprised a small, pooled sample size of 84 BN participants, and results concerning monetary rewards may not translate to condition-specific triggers (i.e., food). A more recent study by Hagan et al. (2021) [67] extended experimental stimuli to food rewards and observed decreased delay discounting to both monetary and food rewards in women with BN compared to HCs. That is, women with BN showed a preference for larger-later over smaller-sooner amounts of food and money relative to HCs. Although this finding contradicts previous findings from Amlung et al. (2019) [68], decreased delay discounting may explain prolonged periods of food intake restriction in BN, a behaviour which necessitates a high degree of cognitive control.

3.3. Impulsivity

has been found to be a core feature of BN. Accordingly, research has consistently shown that BN individuals scored higher on measures of impulsivity compared to HCs, suggesting that heightened impulsivity may contribute to the disorder's characteristic binge-purge cycle [69]. However, recent literature has started to challenge the simplistic view of BN as a disorder of impulsivity. For instance, Neveu et al. (2018) found that women with BN used both health and taste ratings in making uncontrolled food choices, whereas HCs only used tastiness in their food choices. This finding suggests that binge-related food choices in BN are more calculated than previously thought and binge eating may not be a purely impulsive act. This theory is echoed in a systematic review by Howard et al. (2020) [1], which did not find consistent evidence supporting the simplistic view of BN as a disorder of impulsivity.

A possible explanation for the inconsistency in evidence regarding impulsivity in BN is that cognitive control impairments (i.e. impulsivity) may coexist alongside more deliberate planning and goal-oriented processes. For example, Neveu et al (2014) [64] used a computerized version of the Race Game to assess backward reasoning abilities in women with BN versus HC. The study found that planning ability based on backward reasoning was enhanced in women with BN compared to HCs when preconditioned with image cues of binge foods which were designed to induce craving [64]. Planning, such as that which was induced in this experiment, may work to reduce cravings for binge foods and extend binge refractory periods. This finding is especially relevant since some BN

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individuals deliberately plan for binge episodes in advance, and binge anticipation may decrease the motivation to attempt other coping strategies, resulting in negative reinforcement eating expectancies (i.e., the belief that eating will help mitigate distress) [70]. Thus, therapeutic strategies for BN might be more effective if they also consider the goal-oriented nature of binge eating episodes.

3.4. Emotional Processing Difficulties

have also been found in individuals with BN. Some research has shown that participants with BN self-report significantly more emotion regulation difficulties than HCs, but do not exhibit difficulties in recognising emotions [39]. A recent meta-analysis by Mourilhe et al. (2021) [71] reported a positive correlation between the amount of food ingested during a binge eating episode and depressive symptoms in BN individuals. Similarly, Davis and Smith (2023) [72] reported that positive urgency (i.e., the tendency to act impulsively in response to distress or extremely positive emotions) was associated with a greater amount of ingested food during a binge eating episode during an ad-lib meal test among women with BN. These findings may be explained by the fact that foods typically eaten during a binge eating episode are highly palatable and rewarding. Thus, the loss of control during binge episodes may be driven by the instantaneous feelings of reward related to food consumption [73].

Additionally, individuals with BN often report heightened emotional reactivity, including difficulty managing distressing emotions, which may fuel the urge to binge eat as a form of self-soothing [74]. According to Smyth et al. (2007) [75], negative mood states such as anxiety and sadness commonly precede binge eating episodes, suggesting that binge eating may serve as an emotional escape strategy. Complementing this theory, Fischer et al. (2018) [76] found that individuals with BN are more likely to binge after experiencing negative social interactions, pointing to a connection between interpersonal stress and binge eating urges. Collectively, the evidence supports the view that BN behaviours are intertwined with a maladaptive cycle of affect regulation, where binge eating serves as a temporary escape from negative emotions.

In summary, BN is characterised by dysregulated reward processing, with heightened sensitivity to food rewards, suggesting that the immediate pleasure of binge-eating plays a significant role in the loss of control. Additionally, there is some evidence to suggest that individuals with BN may struggle with emotional processing difficulties and that these difficulties drive binge-eating behaviours as coping mechanisms for stress.

4. Binge Eating Disorder

According to the DSM-5 [4,5], BED is characterised by recurrent episodes of binge eating, accompanied by a lack of control over eating during these episodes. Individuals with BED often eat more rapidly than usual, continue eating despite feeling uncomfortably full, eat large amounts without hunger, and may feel distress, guilt, or shame afterward [4,5]. Unlike BN, BED does not involve regular compensatory behaviours (e.g., purging) to counteract the binge episodes [4,5]. Recent research has shed light on the cognitive factors associated with BED in clinical populations [19] but also in non-clinical community populations [77] which may help inform our understanding of the processes involved in BED.

4.1. Cognitive Flexibility

has been investigated in a small number of studies and found to be consistently impaired in individuals with BED compared to HCs [19,78–82]. Studies have shown that individuals with BED struggle to adapt their behaviour and cognitive strategies when faced with changing contingencies or rules [19,82]. Moreover, compared to patients with AN, the cognitive profile of BED has been shown to be characterised by poorer cognitive flexibility [19]. Cognitive rigidity in BED may contribute to the persistence of maladaptive eating patterns by hindering their ability to engage in adaptive problem solving [54], such as modifying established habits and routines surrounding food and eating.

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Specific set-shifting deficits in BED may be linked to the escape from awareness theory [83], which proposes that binge eating results from an individual's effort to draw attention away from emotional distress by narrowing their focus on the immediate environment (food) rather than aversive self-perceptions [84]. Consequently, individuals with BED find it challenging to shift their focus away from food-related stimuli, leading to increased preoccupation with food, heightened cravings, and ultimately, binge eating episodes.

4.2. Impulsivity

particularly impaired response inhibition, has been consistently and robustly observed in individuals with BED (for reviews see [85–87]). The Go/No-Go Task and the Stop Signal Reaction Time Task are commonly used to assess response inhibition. Studies have found that individuals with BED exhibit increased commission errors on the Go/No-Go task and longer stop signal reaction times on the Stop Signal Reaction Time Task [85–87]. These errors and poor reaction times are indicative of impulsivity and poor response inhibition. An impaired ability to inhibit pre-potent responses may contribute to the difficulty in resisting the urge to binge eat, particularly in the presence of tempting or palatable stimuli. This automatic tendency to consume foods despite the individual's awareness of the potential adverse outcomes, may result in the initiation or continuation of binge eating episodes [85–87].

4.3. Emotional Processing Difficulties

have been implicated in the onset and maintenance of BED[88], with greater emotion related difficulties observed in BED patients compared to HCs (for a review see [3]). Researchers have theorised that negative emotions, particularly the inability to effectively process emotions, result in heightened emotional distress, triggering episodes of binge eating as a means of escape or self-soothing [83,88].

In summary, deficits in cognitive flexibility, response inhibition and emotional processing difficulties have been consistently observed in people diagnosed with BED. These cognitive impairments may contribute to the persistence of maladaptive eating patterns and the difficulty in regulating eating behaviour by hindering the ability to modify established habits, redirect attention away from food-related cues, and resist the urge to binge eat.

5. Otherwise Specified Feeding or Eating Disorders

OSFED encompass a range of disordered eating behaviours that do not fully meet the criteria for AN, BN or BED. This category is important as it captures the experiences of many individuals who struggle with significant eating and body image disturbances but do not fit neatly into established diagnostic categories. Among the subtypes of OSFED, five prominent forms include: atypical anorexia nervosa (AAN), purging disorder (PD), night eating syndrome (NES) and BN and BED of low frequency and/or duration (sub-BN and sub-BED) [4,5].

Most research on OSFED has assessed the disorder as a whole OSFED group, treating it as a single entity despite its heterogeneous nature. This approach overlooks the possibility that distinct neurocognitive profiles may exist within OSFED's specific subtypes. Because studies on neurocognitive experimental tasks specific to the OSFED subtypes are limited, we also include neuroimaging findings in the subsequent section, which offer valuable insights into the neural underpinnings of these cognitive processes [89,90]. Neuroimaging studies allow us to examine the activation patterns in brain regions related to reward processing, cognitive control, and emotional regulation, providing a more comprehensive understanding of the neurocognitive characteristics of OSFED [91].

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5.1. Overall OSFED

The investigation of neurocognitive factors in OSFED remains limited, as evidenced by a review focusing on socio-cognitive factors and EDs in young people which found that only four out of the included 38 studies were on OSFED [92]. Two studies highlighted that individuals with OSFED exhibited hyperactivation in reward, attention, and visual processing regions in response to high-calorie food cues, while showing hypo-activation in cognitive control areas, indicating difficulties in regulating responses to food-related stimuli [93,94]. However it needs to be acknowledged that while the study by Bartholfy et al. (2019) [94] assessed a binge purging presentation sample, the study by Wang et al. (2016) [93] comprised a restrictive OSFED sample. Given the heterogeneous OSFED presentations in these two studies and the small number of participants, drawing clear conclusions on neurocognition in OSFED is challenging.

A further finding of the Prince et al. (2022) [92] review was that OSFED participants demonstrated increased neural responses in the prefrontal circuitry and cerebellum when exposed to food images, correlating with heightened obsessive-compulsive symptoms. Finally, Bodell et al, (2018) [95] also included in the Prince et al. (2022) [92] review found that alterations in reward-related neural circuitry were concurrently and prospectively associated with binge eating in a community-based sample of adolescent girls presenting with OSFED. Taken together, these findings suggest that individuals with OSFED may experience unique cognitive and emotional responses to food cues, which could impact treatment approaches and necessitate further research to develop targeted interventions.

Finally, a recent study by Wang et al. (2021) [96], not included in the Prince et al. (2022) [92] review, assessed female adolescents in a residential ED programme who had been diagnosed with AN, BN, or OSFED. Findings showed that, in comparison to a historical sample of adolescent HCs, adolescents with EDs had significantly greater levels of cognitive rigidity and attention to detail, with both findings presenting with large effect sizes. Even after adjusting for age, length of illness, and BMI, there was still a substantial correlation between these neurocognitive deficits and the severity of the EDs (assessed through a clinical ED screening tool). However, in this study the OSFED sample was very small and not separated from the other EDs. Given these limitations, the findings of Wang et al. (2021) [96] should be interpreted with caution.

5.1.1. AAN

is characterized by the same behavioural patterns and cognitive features as AN, but individuals diagnosed with this ED maintain a weight within or above the normal range [4,5]. Often, people with AAN are initially at a higher weight when they develop their ED. Although they may experience a dramatic weight loss, they do not drop to a BMI below 18 and are not classified as underweight [89,90].

People with AAN often experience severe cognitive distortions related to body image, including a pervasive fear of gaining weight and a preoccupation with food and dieting [97]. It is likely that cognitive rigidity and central coherence deficiencies are prevalent in this group, mirroring those observed in individuals with threshold AN. However, research that has assessed neurocognition in AAN and compared ANN to AN and/or other EDs or other comparison groups is currently lacking.

One study [98] found that in a sample of female adolescents with AAN and HCs, there was evidence for increased functional connectivity within the somatosensory network in response to food images in the AAN group. In addition, there was evidence for decreased functional connectivity in the ANN group in the brain networks linked with salience, attention, and inhibitory control, and in the brain areas involved in food cue processing and appetite regulation [98]. The findings suggest that high caloric food is associated with increased somatosensory processing in AAN, yet low caloric food is given greater salience and is considered more engaging [98]. This connectivity pattern may play a key role in the unique challenges AAN individuals face regarding food reward processing [98]. Conversely, another study by the same author found that AAN in newly diagnosed adolescents was not associated with structural changes in the brain [99]. That is, adolescents with AAN and HCs did not significantly differ in brain grey matter volume [99]. This finding contrasts with similar work

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in adult and adolescent AN (for a review see [100]), which often shows alterations in brain volume and structure, possibly due to factors like malnutrition and prolonged illness. Future research is required to disentangle these findings to clarify whether AAN may or may not involve the similar or distinct neurobiological impacts on brain structure as seen in AN.

5.2.2. PD

refers to individuals who engage in purging behaviours - such as self-induced vomiting or excessive exercise - without the binge-eating episodes characteristic of BN [4,5]. Negative urgency - the propensity to act impulsively when experiencing negative emotions - along with other dimensions of impulsivity, has been investigated in individuals with PD. A study by Davis et al. (2020) [101] found that individuals with PD exhibited significantly higher levels of negative urgency compared to HC, although these levels were lower than those observed in individuals with BN. However, no significant differences were detected between the PD and HC groups or between the PD and BN groups on additional impulsivity traits, including lack of premeditation, lack of perseverance, and sensation seeking [101]. Consistent with these findings, a more recent study examining a larger sample of individuals with PD reported no significant differences in sensation seeking compared to controls. Furthermore, individuals with PD were characterized by lower reward dependence and novelty seeking but exhibited higher persistence scores compared to those with BN and BED [102]. It is possible that people with PD have a tendency toward impulsive actions under emotional distress which may reinforce the use of purging behaviours as a maladaptive coping strategy complicating symptom management and intervention approaches for PD [101]. However, the currently limited and contradictory findings in the literature need to be first clarified before such conclusions can be made.

5.2.3. NES

another subtype of OSFED, involves recurrent episodes of significant food intake after the evening meal or during nighttime awakenings, often causing distress [4,5]. Though neurocognitive research is sparse, individuals with NES may have altered reward sensitivity and emotional regulation difficulties, potentially exacerbating night eating and leading to weight gain [103,104]. Like the other OSFEDs, NES also appears to be influenced by emotional fluctuations, with some individuals using night eating as a temporary coping mechanism for stress, which disrupts circadian rhythms and further heightens distress [105,106]. It is also possible that impulsivity may increase susceptibility to night binge eating episodes by weakening self-control though this theory requires further investigation.

5.2.4. Sub-BN/BED

Sub-BN involves recurrent binge eating episodes followed by compensatory behaviours occurring less than once a week or for less than three months, while sub-BED features binge eating episodes with distress which occurring less frequently than required for a full BED diagnosis [4,5]. In a study by Darcy et al. (2012) [107], neurocognitive assessments in adolescents with BN, sub-BN, and HCs revealed no significant group differences in set-shifting abilities, with small effect sizes. Neurocognitive factors in sub-BED have also been assessed [108,109]. For instance, Manasse et al. (2015) [108] found that individuals with sub-BED exhibited similar deficits in inhibitory control as those with full-threshold BED.

Overall, these observations suggest that neurocognitive factors may play a role in the development and maintenance of binge eating and purging behaviours along a continuum, rather than being specific to the clinical BN/BED diagnosis. These cognitive impairments may contribute to the perpetuation of maladaptive eating patterns and the potential escalation of binge eating/purging behaviours over time.

Despite the emergence of research on neurocognitive factors associated with specific OSFED subtypes, the literature remains limited compared to that of AN, BN, and BED. The heterogeneity of

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OSFED presents challenges in establishing consistent neurocognitive profiles. Factors such as varying symptomatology, comorbid conditions, and developmental influences further complicate the analysis of cognitive impairments across the different OSFED subtypes [89]. Moreover, the lack of standardised diagnostic assessment tools specifically designed for OSFED [90] restricts our understanding of cognitive functioning in this population. Future studies should prioritize examining cognitive and emotional processes across various OSFED subtypes to inform tailored interventions, ultimately improving treatment outcomes for this underrepresented population.

6. Higher Weight

Neurocognitive profiles of HW individuals highlight deficiencies in executive function, reward processing, reward sensitivity, alongside marked changes in neuroplasticity and the cognitive function in brain regions modulating reward, learning and decision-making[10,110]. A HW and neurocognitive alterations (such as impulsivity and cognitive rigidity) have been found to exhibit a bi-directional relationship; neurocognitive patterns are suggested to influence susceptibility to overconsumption, and reciprocally, weight status and diet-elicited HW promotes changes in the prefrontal cortex structure and functionality, worsening executive functions and episodic memory [111–113].

6.1. Cognitive Flexibility

has also been assessed in those with HW and a meta-analysis of 25 studies found that cognitive flexibility was significantly poorer in HW participants compared to HCs [114]. Supporting this meta-analysis, a recent study also found significantly poorer flexibility in patients with HW compared to HCs [8]. Given this consistent finding Testa et al. (2021) [8] suggested that cognitive rigidity may contribute to the persistence of unhealthy eating habits and difficulties in behaviour change. In a cognitive control task, HW participants were found to perform significantly poorer than HCs for food stimuli but not neutral stimuli (flowers) [115]. This result could indicate an impairment in cognitive flexibility, with greater cognitive resource demands in response to food stimulus[115].

6.2. Impulsivity

has been suggested to be elevated in those at a HW. A meta-analysis by Yang et al. (2018) [114] revealed that HW participants performed worse than HCs on inhibition tasks and decision-making tasks. Another systematic review by Lavagnino et al. (2016) [86] concluded that HW participants exhibit decreased inhibitory response performance in tasks when compared to HCs. Contrarily, Testa et al. (2021) [8] found greater impulsivity in participants with both HW and Type 2 Diabetes compared to HCs, but no significant difference in impulsivity in those with HW compared to HCs. In a more recent study by Reyes et al. (2024) [10], BMI was correlated with task performance such that higher BMI was associated with lower inhibitory control. The literature suggests that there may be a relationship between the inclination to immediate reward despite potential future loss and a deficit in the ability to maintain self-control and regulate responses to reward and punishment [8,116].

6.3. Reward Processing

Appraisal of reward and subsequent capacity to inhibit gratification are believed to guide individual eating behaviours [117]. People at a HW appear to show marked changes in reward sensitivity to food cues [118]. In a study comparing HW women to HCs, HW participants were shown to be more likely to choose immediate reward and gratification and forgo future gains, i.e., participants with a HW displayed lower delay discounting compared to HCs[9]. This low delay discounting is proposed to reflect eating habits of HW individuals, whereby individuals may preferentially elect for immediate reward over a potential future greater benefit of improved health [9].

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6.4. Emotional Processing Difficulties

have been implicated in the development of maladaptive eating behaviours contributing to HW, with a common trend of low levels of recognition, self-reporting, and regulation of emotions [11,119]. In a systematic review and meta-analysis, Fernades et al. (2018) [11] found that compared to HCs, participants at a HW had a greater difficulty in identifying emotions, higher alexithymia, lower emotional awareness, and greater difficulties using emotion regulation strategies. However, there was no research to support a hypothesis that people at a HW exhibit and impaired ability to recognise the emotions of others or express their emotions [11]. Although further research is required, it appears that emotion processing in HW is nuanced and that HW is not characterised by a general emotion processing deficit [11].

To summarise, neurocognitive research reveals that HW is linked to deficiencies in cognitive flexibility, impulsivity, and reward processing. There is also some evidence of specific emotion processing difficulties. These neurocognitive factors may contribute to the complex interplay of processes driving overeating and weight gain in people at a HW.

7. Clinical Implications

7.1. Classification

Neurocognitive knowledge can significantly enhance the classification of EDs by identifying cognitive profiles that differentiate these conditions beyond the DSM-5 [4,5] or ICD-11 [120] diagnostic criteria. The variations in cognitive flexibility, central coherence, attentional biases, emotional processing, and reward processes/impulsivity outlined above can aid in refining diagnostic boundaries, offering a more granular classification system that accounts for cognitive profiles alongside behavioural symptoms (e.g. binge eating behaviour, restriction) [121]. Importantly, some of these neurocognitive factors, such as cognitive flexibility, appear to be transdiagnostic, meaning they may be present across multiple ED subtypes. By incorporating these cognitive traits into future classification models, we can enhance the identification of distinct subtypes within and across different EDs. This approach would shift towards a dimensional framework that emphasizes underlying neurocognitive features, rather than relying solely on traditional diagnostic categories [8,68]. This model does not imply collapsing all ED diagnoses into a single group, but rather focusing on the neurocognitive profiles that might cut across different ED subtypes, helping to tailor treatment based on individual cognitive characteristics.

7.2. Treatment

An important question in understanding neurocognitive factors in EDs is whether potential deficits in cognitive functioning represent stable traits or whether they are state factors associated with the illness. Are these deficits likely to remit on their own with recovery, or do they require targeted intervention [122] ? Addressing this issue is crucial for developing effective treatment strategies. While some cognitive impairments may improve as individuals recover from the disorder, others may persist and need to be specifically targeted in treatment to optimize outcomes. Understanding the nature of these deficits - whether trait-like or state-dependent - can help clinicians decide whether to focus on cognitive remediation as part of the therapeutic process or whether to simply monitor cognitive functioning throughout recovery [122]. Presently, it remains unclear if neurocognitive impairments in EDs are traits or state factors and further research is needed to understand this issue.

Understanding the neurocognitive profiles in individuals with AN, BN, BED, OSFED, and HW is crucial for developing effective treatment interventions. In a very recent Delphi consensus paper [123], improving cognitive flexibility and impulse control were identified by researchers, clinicians, careers, and those with lived experience as key targets for ED treatments. In AN, potential deficits in cognitive flexibility, attentional biases, and emotional processing difficulties reveal specific targets for therapy. Cognitive Remediation Therapy (CRT, e.g., [91]) has been developed to help patients

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improve cognitive flexibility and central coherence, in addition to preparing patients to participate in other therapies [124]. However, a recent meta-analysis of randomized controlled trials on CRT for AN showed no significant improvement in central coherence over control treatments at the end of treatment, but this finding was based on only three studies [125]. Cognitive Remediation and Emotion Skills Training (CREST) builds on CRT by integrating cognitive training with emotion skills training. This combination has shown promising results in AN patients, with significant improvements in social anhedonia, emotional labelling, and patients' confidence in their capacity for change ([126]. Finally, it is also worth noting that there are aspects of traditional cognitive behaviour therapy (CBT) which encourage adaptive and flexible thinking [127]. Additional research is therefore required to further improve CRT/CREST and develop additional treatments which can target poor cognitive flexibility across the different ED subtypes.

To address attentional biases in AN, exposure-based therapies can gradually desensitize ED individuals to anxiety-provoking food cues [30]. For instance, through repeated exposure to food-related scenarios, patients can learn to tolerate these cues without resorting to avoidance or restrictive behaviours. Furthermore, emotional processing challenges can be tackled using emotion regulation training, such as dialectical behaviour therapy (DBT) [128], which teaches ED patients to identify and manage their emotions more effectively. Skills like mindfulness [129] and distress tolerance training [130] can also enhance emotional resilience in ED individuals, potentially lowering the risk of relapse.

In BN and BED, cognitive rigidity and attentional biases may manifest in food preoccupations and impulsive behaviours. Recently the Transdiagnostic Cognitive Remediation Therapy (TCRT), a new adaptation of CRT for EDs has been developed, which addresses common cognitive difficulties across ED diagnoses (i.e., cognitive flexibility, central coherence, and impulsivity). A recent qualitative study of thirteen patients with restrictive or binge/purge EDs and concurrent cognitive difficulties examined the impact of TCRT through semi-structured interviews [131]. Eleven participants viewed the treatment favourably, highlighting its relevance and the insights it provided into their thinking styles. Seven participants noted TCRT as a foundation for initiating changes and adopting new strategies. Engagement appeared to be enhanced by experiencing challenges directly related to their cognitive difficulties.

Other treatment modalities to consider for BN and BED, similar to AN, include attentional bias modification/ training techniques, that may redirect attention away from food stimuli, helping to decrease cravings and impulsivity associated with binge episodes [30]. Exposure-response prevention techniques, adapted from anxiety treatments, can help BN/BED patients build tolerance to high-craving environments [132]. Finaly, adapted DPT interventions, such as Dialectical Behaviour Therapy for Binge Eating Disorder (DBT-BED) [133] aimed to reduce binge eating by improving adaptive emotion-regulation skills has been found to also help BED patients to regulate their emotion regulation difficulties.

For OSFED, the diverse presentations necessitate flexible and integrative interventions, as research on its classification, subtypes, and effective treatments is still in its infancy. This diagnostic category includes various symptom patterns that do not fully align with the other EDs, requiring approaches that address both the cognitive and emotional challenges specific to each presentation in addition to ED symptoms [89,90]. Combining elements from CRT/CREST, broader emotion regulation training, and attentional bias modification may effectively target these unique needs. As our understanding of OSFED develops, refining such targeted interventions will be essential for enhancing treatment outcomes and providing tailored support.

Finally, in HW populations, impulsivity, reward processing difficulties, and emotional processing difficulties may perpetuate cycles of overeating and negatively impact body image. Mindfulness-based exercises can enhance focus and control over food-related impulses [134], while strategies from acceptance and commitment therapy and DBT [135] can help individuals reframe their relationship with food. Acceptance and commitment therapy encourages acceptance of cravings without acting on them, fostering healthier responses to food cues and reducing binge eating behaviour [136]. Together, these interventions can foster a more adaptive relationship with food,

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reduce the impact of impulsivity, reward processing difficulties, and emotional biases, and enhance overall well-being.

7.2.1. Virtual Reality

Virtual reality (VR) might be specifically useful in targeting neurocognitive deficits in EDs across the weight spectrum [137]. For attentional biases, VR can offer controlled, immersive environments where exposure-based therapies are more precisely tailored and engaging [138]. For instance, VR exposure sessions can simulate real-life food-related scenarios, allowing patients to practice exposure techniques in a safe, yet realistic setting. By engaging with these VR-based scenarios, patients could incrementally increase their tolerance for anxiety-provoking cues without using avoidance or restrictive behaviours and coping mechanisms, leading to sustained desensitization [137].

In addressing emotional processing challenges, VR can complement emotion regulation training [139] and enhance interventions like DBT [140]. By replicating challenging social or emotional contexts, VR scenarios could help individuals practice distress tolerance and mindfulness in simulated situations which they might find stressful in real life. The immersive nature of VR allows for an experiential form of learning, where patients can safely explore emotional responses, develop resilience, and refine coping skills [139] [140]. This integration could lower the risk of relapse by fostering emotional resilience and enhancing self-regulation skills that are essential for recovery. In addition, given the widespread adoption of VR technologies, these skills could be practised by patients in their home outside of medical appointments with clinicians.

7.2.2. Brain Stimulation

Different brain stimulation techniques can also offer a promising approach to improving cognitive deficiencies in individuals with EDs and those with HW by directly targeting the neural circuits associated with cognitive flexibility, emotional regulation, and/or impulse control. Techniques such as transcranial magnetic stimulation [141] and transcranial direct current stimulation [142,143] can modulate neural activity in areas of the brain implicated in these cognitive domains, potentially enhancing the effectiveness of traditional therapies.

Cognitive flexibility has largely been associated with functioning in the prefrontal cortex [144], the anterior cingulate cortex [145] and the orbitofrontal cortex [146]. Stimulating these areas may increase activity in these brain regions, neural pathways, and neural plasticity, potentially improving cognitive flexibility and reducing rigid, rule-bound thinking. Similarly, individuals with BN and BED, who may exhibit heightened impulsivity and attentional biases toward food cues, could benefit from brain stimulation targeting the dorsolateral prefrontal cortex to improve inhibitory control and reduce impulsive behaviours [147,148].

In individuals with HW, brain stimulation may address impairments in reward processing and executive function, which are often associated with challenges in regulating food intake. Stimulating or inhibiting activity in the prefrontal cortex could enhance impulse control and support better decision-making in food-related contexts, potentially leading to healthier eating patterns [149,150].

7. Limitations

While our narrative review encompasses a broad range of studies examining neurocognitive and related factors in EDs and HW individuals, it may not be comprehensive. We recognize the necessity for additional structured systematic reviews and/or meta-analyses to further explore this topic, as these approaches would provide a more rigorous synthesis of the literature, including standardized inclusion criteria and quantitative pooling of results. The decision to conduct a narrative review, rather than a systematic review, was based on the exploratory nature of this topic and the need to provide a broad overview of diverse studies across multiple domains of neurocognitive processes. A narrative review allows for a more flexible and comprehensive discussion of varied study designs, methodologies, and findings, which may be less feasible in the structured format of a systematic review.

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Another limitation of this review is the exclusion of studies on avoidant/restrictive food intake disorder (ARFID) and other eating disorders in childhood [4,5]. This decision is in line with previous reviews [89] given that these disorders typically onset in early childhood and are clinically distinct in presentation to AN, BN, BED and OSFED [4,5]. Future research should address these gaps to ensure a more comprehensive understanding of neurocognitive factors across the full spectrum of EDs, including early-onset disorders, which may present unique developmental and cognitive challenges.

There are also various limitations within the reviewed studies that need to be considered. First, the diversity of cognitive assessment tools used across studies makes direct comparisons difficult and limits the ability to draw consistent conclusions. Studies employed different tasks to assess cognitive domains such as flexibility, attentional bias, and emotion regulation, creating challenges in identifying shared or distinct impairments specific to each disorder. Further, OSFED is underrepresented in the literature, and this lack of data makes it difficult to determine whether its neurocognitive profile aligns with other EDs or HW individuals.

Second, the variability in ED subtypes and illness stages among participants, which affects neurocognitive findings across disorders complicates the comparison of studies. In AN, differences have been found between AN-R and AN-BP in neurocognitive profiles [29], yet most studies have lumped the two groups into one overall AN group due to small sample sizes. The transdiagnostic nature of ED symptoms further blurs the lines between disorders, as features like impulsivity and reward sensitivity, commonly observed in AN-BP, BN and BED, may represent transdiagnostic vulnerabilities rather than disorder-specific traits [8,68]. Additionally, there is likely substantial heterogeneity in cognitive deficits across EDs and within cognitive domains - some deficits may be more prominent in one ED subtype than another, reflecting variability in the nature and intensity of these impairments.

Third, it is important to acknowledge the influence of weight and nutritional status on neurocognitive function, and highlight that BMI alone is insufficient to capture the full scope of malnutrition and starvation and the impact that they might have on cognition [16]. For instance, cognitive rigidity and set shifting difficulties have been found in AN, yet BMI does not account for this complexity in understanding the neurocognitive profile of AN (for a review see [17].

Fourth, the reliance on cross-sectional studies restricts understanding of how neurocognitive deficits develop and progress across different stages of ED, its recovery, and HW. The limited amount of longitudinal research available on EDs and cognition restricts insights into whether neurocognitive impairments endure over time or improve with treatment and recovery. For example, longitudinal tracking of cognitive flexibility in AN could help clarify whether such deficits improve with weight restoration and recovery or if they persist as chronic features of the disorder. Similarly, observing attentional biases in BN and BED across treatment phases could reveal whether these biases diminish as part of recovery or if they remain stable, potentially contributing to relapse risk.

Finally, a critical aspect to consider in the context of the existing research is the trait vs. state nature of neurocognitive deficits. Without longitudinal data, it remains uncertain whether observed impairments represent stable traits or if they are transitory, linked to specific phases of the ED.

8. Future Research Directions

Future research should focus on refining our understanding of neurocognitive profiles in AN, BN, BED, and OSFED through detailed longitudinal studies. These studies should track cognitive changes over time, capturing the dynamic nature of these disorders and their recovery phases. This approach can clarify whether observed neurocognitive deficits are temporary and related to current illness stage/severity or if they are stable traits that persist beyond weight restoration. This distinction is crucial, as trait-like deficits may necessitate targeted interventions even post-recovery, while state-dependent deficits may be more responsive to traditional treatment approaches and may not require ongoing treatment [122].

Investigating physiological markers, such as brain structure/function and nutritional biomarkers, is crucial for understanding the interplay between weight, nutritional status, and neurocognitive function. Neuroimaging studies, like functional magnetic resonance imaging, can

elucidate changes in brain activity related to cognitive flexibility, reward processing, and other executive functions in individuals with EDs and HW. Such findings could pave the way for targeted interventions that focus on ameliorating any cognitive deficits through cognitive training or exposure therapies. Additionally, examining nutritional biomarkers, such as omega-3 fatty acids [151] or vitamin D levels [152], may help determine their role in cognitive performance among individuals with EDs and HW. Understanding these relationships could lead to integrative treatment approaches that combine nutritional support with cognitive interventions.

Lastly, the inclusion of the different OSFED presentations in future research is essential for comprehensively assessing the full range of cognitive impairments and their connection to emotional and behavioural patterns in individuals with disordered eating. By expanding the research focus to encompass OSFED, we can better understand how people with these illnesses navigate their cognitive challenges and thus can develop targeted interventions that address their unique needs [89,90].

9. Conclusion

The current narrative review on the neurocognitive profiles associated with AN, BN, BED, OSFED, and HW reveals significant insights into the cognitive and emotional challenges across the weight spectrum. Understanding that cognitive rigidity and attentional biases are prevalent in AN highlights the necessity for tailored interventions that specifically address these cognitive patterns. In BN and BED, the interplay between cognitive rigidity, emotional processing difficulties, heightened reward sensitivity and impulsivity may exacerbate binge-eating/purging behaviours, underscoring the need for therapeutic strategies that consider the complex relationship between reward/emotions and eating. Additionally, individuals with HW experience neurocognitive challenges that impair decision-making and executive function. These insights across EDs and HW underscore the critical need for individualized, transdiagnostic approaches in treatment. Future research should prioritize longitudinal studies to explore the evolution of these neurocognitive profiles over time and include investigations into OSFED subtypes, ensuring the development of effective, tailored interventions that improve recovery outcomes across diverse ED and HW populations.

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References

1. Howard, M.; Gregertsen, E.C.; Hindocha, C.; Serpell, L. Impulsivity and compulsivity in anorexia and bulimia nervosa: A systematic review. *Psychiatry Res* **2020**, *293*, 113354, doi:10.1016/j.psychres.2020.113354.
2. Miles, S.; Gnatt, I.; Phillipou, A.; Nedeljkovic, M. Cognitive flexibility in acute anorexia nervosa and after recovery: A systematic review. *Clin Psychol Rev* **2020**, *81*, 101905, doi:10.1016/j.cpr.2020.101905.
3. Kittel, R.; Brauhardt, A.; Hilbert, A. Cognitive and emotional functioning in binge-eating disorder: A systematic review. *Int J Eat Disord* **2015**, *48*, 535-554, doi:10.1002/eat.22419.
4. Association, A.P. *Diagnostic and Statistical Manual of Mental Disorders.* APA: Wahington, DC, 2013.
5. Association., A.P. *Diagnostic and Statistical Manual of Mental Disorders*. APA: Washington, DC, 2022; https://doi.org/10.1176/appi.books.9780890425787.
6. Lim, Y.; Boster, J. Obesity and Comorbid Conditions. In *StatPearls*, Treasure Island (FL), 2024.

7. Villarejo, C.; Fernandez-Aranda, F.; Jimenez-Murcia, S.; Penas-Lledo, E.; Granero, R.; Penelo, E.; Tinahones, F.J.; Sancho, C.; Vilarrasa, N.; Montserrat-Gil de Bernabe, M., et al. Lifetime obesity in patients with eating disorders: increasing prevalence, clinical and personality correlates. *Eur Eat Disord Rev* **2012**, *20*, 250-254, doi:10.1002/erv.2166.
8. Testa, G.; Mora-Maltas, B.; Camacho-Barcia, L.; Granero, R.; Lucas, I.; Aguera, Z.; Jimenez-Murcia, S.; Banos, R.; Bertaina-Anglade, V.; Botella, C., et al. Transdiagnostic Perspective of Impulsivity and Compulsivity in Obesity: From Cognitive Profile to Self-Reported Dimensions in Clinical Samples with and without Diabetes. *Nutrients* **2021**, *13*, doi:10.3390/nu13124426.
9. Miranda-Olivos, R.; Steward, T.; Martinez-Zalacain, I.; Mestre-Bach, G.; Juaneda-Segui, A.; Jimenez-Murcia, S.; Fernandez-Formoso, J.A.; Vilarrasa, N.; Veciana de Las Heras, M.; Custal, N., et al. The neural correlates of delay discounting in obesity and binge eating disorder. *J Behav Addict* **2021**, *10*, 498-507, doi:10.1556/2006.2021.00023.
10. Reyes, S.; Peirano, P.; Gahagan, S.; Blanco, E.; Algarin, C. Neurocognitive factors predicting BMI changes from adolescence to young adulthood. *Obesity (Silver Spring)* **2024**, *32*, 768-777, doi:10.1002/oby.23978.
11. Fernandes, J.; Ferreira-Santos, F.; Miller, K.; Torres, S. Emotional processing in obesity: a systematic review and exploratory meta-analysis. *Obes Rev* **2018**, *19*, 111-120, doi:10.1111/obr.12607.
12. Ralph, A.F.; Brennan, L.; Byrne, S.; Caldwell, B.; Farmer, J.; Hart, L.M.; Heruc, G.A.; Maguire, S.; Piya, M.K.; Quin, J., et al. Management of eating disorders for people with higher weight: clinical practice guideline. *J Eat Disord* **2022**, *10*, 121, doi:10.1186/s40337-022-00622-w.
13. Keski-Rahkonen, A.; Mustelin, L. Epidemiology of eating disorders in Europe: prevalence, incidence, comorbidity, course, consequences, and risk factors. *Curr Opin Psychiatry* **2016**, *29*, 340-345, doi:10.1097/YCO.0000000000000278.
14. Baenas, I.; Etxandi, M.; Fernandez-Aranda, F. Medical complications in anorexia and bulimia nervosa. *Med Clin (Barc)* **2024**, *162*, 67-72, doi:10.1016/j.medcli.2023.07.028.
15. Stedal, K.; Broomfield, C.; Hay, P.; Touyz, S.; Scherer, R. Neuropsychological functioning in adult anorexia nervosa: A meta-analysis. *Neurosci Biobehav Rev* **2021**, *130*, 214-226, doi:10.1016/j.neubiorev.2021.08.021.
16. Phillipou, A.; Rossell, S.L.; Castle, D.J. Anorexia nervosa or starvation? *Eur J Neurosci* **2018**, *48*, 3317-3318, doi:10.1111/ejn.14158.
17. Fuglset, T.S. Is set-shifting and central coherence in anorexia nervosa influenced by body mass index, anxiety or depression? A systematic review. *BMC Psychiatry* **2021**, *21*, 137, doi:10.1186/s12888-021-03120-6.
18. Deak, G.O. The development of cognitive flexibility and language abilities. *Adv Child Dev Behav* **2003**, *31*, 271-327, doi:10.1016/s0065-2407(03)31007-9.
19. Aloi, M.; Rania, M.; Caroleo, M.; Bruni, A.; Palmieri, A.; Cauteruccio, M.A.; De Fazio, P.; Segura-Garcia, C. Decision making, central coherence and set-shifting: a comparison between Binge Eating Disorder, Anorexia Nervosa and Healthy Controls. *BMC Psychiatry* **2015**, *15*, 6, doi:10.1186/s12888-015-0395-z.
20. Nakazato, M.; Hashimoto, K.; Schmidt, U.; Tchanturia, K.; Campbell, I.C.; Collier, D.A.; Iyo, M.; Treasure, J. Serum glutamine, set-shifting ability and anorexia nervosa. *Ann Gen Psychiatry* **2010**, *9*, 29, doi:10.1186/1744-859X-9-29.
21. Buzzichelli, S.; Marzola, E.; Amianto, F.; Fassino, S.; Abbate-Daga, G. Perfectionism and cognitive rigidity in anorexia nervosa: Is there an association? *Eur Eat Disord Rev* **2018**, *26*, 360-366, doi:10.1002/erv.2591.
22. Castro, T.F.; Miller, K.; Araujo, M.X.; Brandao, I.; Torres, S. Emotional processing in recovered anorexia nervosa patients: A 15 year longitudinal study. *Eur Eat Disord Rev* **2021**, *29*, 955-968, doi:10.1002/erv.2858.

23. Giel, K.E.; Wittorf, A.; Wolkenstein, L.; Klingberg, S.; Drimmer, E.; Schonenberg, M.; Rapp, A.M.; Fallgatter, A.J.; Hautzinger, M.; Zipfel, S. Is impaired set-shifting a feature of "pure" anorexia nervosa? Investigating the role of depression in set-shifting ability in anorexia nervosa and unipolar depression. *Psychiatry Res* **2012**, *200*, 538-543, doi:10.1016/j.psychres.2012.06.004.
24. Konstantakopoulos, G.; Tchanturia, K.; Surguladze, S.A.; David, A.S. Insight in eating disorders: clinical and cognitive correlates. *Psychol Med* **2011**, *41*, 1951-1961, doi:10.1017/S0033291710002539.
25. Dougherty, E.N.; Bottera, A.R.; Forester, G.; Schaefer, L.M.; Forbes, E.E.; Wildes, J.E. Prospective associations between cognitive flexibility and eating disorder symptoms in anorexia nervosa and bulimia nervosa. *Psychiatry Res* **2024**, *332*, 115717, doi:10.1016/j.psychres.2024.115717.
26. Hemmingsen, S.D.; Daugaard, N.; Sjogren, M.; Lichtenstein, M.B.; Gudex, C.; Piil, F.; Stoving, R.K. Cognitive Flexibility in Hospitalized Patients with Severe or Extreme Anorexia Nervosa: A Case-Control Study. *J Pers Med* **2023**, *13*, doi:10.3390/jpm13061000.
27. Keegan, E.; Tchanturia, K.; Wade, T.D. Central coherence and set-shifting between nonunderweight eating disorders and anorexia nervosa: A systematic review and meta-analysis. *Int J Eat Disord* **2021**, *54*, 229-243, doi:10.1002/eat.23430.
28. Lopez, C.; Tchanturia, K.; Stahl, D.; Booth, R.; Holliday, J.; Treasure, J. An examination of the concept of central coherence in women with anorexia nervosa. *Int J Eat Disord* **2008**, *41*, 143-152, doi:10.1002/eat.20478.
29. Herbrich, L.; Kappel, V.; van Noort, B.M.; Winter, S. Differences in set-shifting and central coherence across anorexia nervosa subtypes in children and adolescents. *Eur Eat Disord Rev* **2018**, *26*, 499-507, doi:10.1002/erv.2605.
30. Stott, N.; Fox, J.R.E.; Williams, M.O. Attentional bias in eating disorders: A meta-review. *Int J Eat Disord* **2021**, *54*, 1377-1399, doi:10.1002/eat.23560.
31. Werthmann, J.; Simic, M.; Konstantellou, A.; Mansfield, P.; Mercado, D.; van Ens, W.; Schmidt, U. Same, same but different: Attention bias for food cues in adults and adolescents with anorexia nervosa. *Int J Eat Disord* **2019**, *52*, 681-690, doi:10.1002/eat.23064.
32. Garcia-Burgos, D.; Wilhelm, P.; Vogele, C.; Munsch, S. Food Restriction in Anorexia Nervosa in the Light of Modern Learning Theory: A Narrative Review. *Behav Sci (Basel)* **2023**, *13*, doi:10.3390/bs13020096.
33. Gilon Mann, T.; Hamdan, S.; Bar-Haim, Y.; Lazarov, A.; Enoch-Levy, A.; Dubnov-Raz, G.; Treasure, J.; Stein, D. Different attention bias patterns in anorexia nervosa restricting and binge/purge types. *Eur Eat Disord Rev* **2018**, *26*, 293-301, doi:10.1002/erv.2593.
34. Claes, L.; Nederkoorn, C.; Vandereycken, W.; Guerrieri, R.; Vertommen, H. Impulsiveness and lack of inhibitory control in eating disorders. *Eat Behav* **2006**, *7*, 196-203, doi:10.1016/j.eatbeh.2006.05.001.
35. Steward, T.; Mestre-Bach, G.; Vintro-Alcaraz, C.; Aguera, Z.; Jimenez-Murcia, S.; Granero, R.; Fernandez-Aranda, F. Delay Discounting of Reward and Impulsivity in Eating Disorders: From Anorexia Nervosa to Binge Eating Disorder. *Eur Eat Disord Rev* **2017**, *25*, 601-606, doi:10.1002/erv.2543.
36. Miyamoto, E.; Okumura, Y.; Maruo, K.; Kitani, S. Shoplifting Behavior Among Patients With an Eating Disorder at a Medical Correctional Center in Japan: A Cross-Sectional Study. *Front Psychiatry* **2022**, *13*, 767170, doi:10.3389/fpsyt.2022.767170.
37. Hatch, A.; Madden, S.; Kohn, M.R.; Clarke, S.; Touyz, S.; Gordon, E.; Williams, L.M. In first presentation adolescent anorexia nervosa, do cognitive markers of underweight status change with weight gain following a refeeding intervention? *Int J Eat Disord* **2010**, *43*, 295-306, doi:10.1002/eat.20695.

38. Oldershaw, A.; Lavender, T.; Sallis, H.; Stahl, D.; Schmidt, U. Emotion generation and regulation in anorexia nervosa: a systematic review and meta-analysis of self-report data. *Clin Psychol Rev* **2015**, *39*, 83-95, doi:10.1016/j.cpr.2015.04.005.
39. Harrison, A.; Sullivan, S.; Tchanturia, K.; Treasure, J. Emotional functioning in eating disorders: attentional bias, emotion recognition and emotion regulation. *Psychol Med* **2010**, *40*, 1887-1897, doi:10.1017/S0033291710000036.
40. Saure, E.; Raevuori, A.; Laasonen, M.; Lepisto-Paisley, T. Emotion recognition, alexithymia, empathy, and emotion regulation in women with anorexia nervosa. *Eat Weight Disord* **2022**, *27*, 3587-3597, doi:10.1007/s40519-022-01496-2.
41. Caglar-Nazali, H.P.; Corfield, F.; Cardi, V.; Ambwani, S.; Leppanen, J.; Olabintan, O.; Deriziotis, S.; Hadjimichalis, A.; Scognamiglio, P.; Eshkevari, E., et al. A systematic review and meta-analysis of 'Systems for Social Processes' in eating disorders. *Neurosci Biobehav Rev* **2014**, *42*, 55-92, doi:10.1016/j.neubiorev.2013.12.002.
42. Oldershaw, A.; Hambrook, D.; Stahl, D.; Tchanturia, K.; Treasure, J.; Schmidt, U. The socio-emotional processing stream in Anorexia Nervosa. *Neurosci Biobehav Rev* **2011**, *35*, 970-988, doi:10.1016/j.neubiorev.2010.11.001.
43. Racine, S.E.; Wildes, J.E. Dynamic longitudinal relations between emotion regulation difficulties and anorexia nervosa symptoms over the year following intensive treatment. *J Consult Clin Psychol* **2015**, *83*, 785-795, doi:10.1037/ccp0000011.
44. Brockmeyer, T.; Febry, H.; Leiteritz-Rausch, A.; Wunsch-Leiteritz, W.; Leiteritz, A.; Friederich, H.C. Cognitive flexibility, central coherence, and quality of life in anorexia nervosa. *J Eat Disord* **2022**, *10*, 22, doi:10.1186/s40337-022-00547-4.
45. Nowakowski, M.E.; McFarlane, T.; Cassin, S. Alexithymia and eating disorders: a critical review of the literature. *J Eat Disord* **2013**, *1*, 21, doi:10.1186/2050-2974-1-21.
46. Westwood, H.; Kerr-Gaffney, J.; Stahl, D.; Tchanturia, K. Alexithymia in eating disorders: Systematic review and meta-analyses of studies using the Toronto Alexithymia Scale. *J Psychosom Res* **2017**, *99*, 66-81, doi:10.1016/j.jpsychores.2017.06.007.
47. Haynos, A.F.; Utzinger, L.M.; Lavender, J.M.; Crosby, R.D.; Cao, L.; Peterson, C.B.; Crow, S.J.; Wonderlich, S.A.; Engel, S.G.; Mitchell, J.E., et al. Subtypes of Adaptive and Maladaptive Perfectionism in Anorexia Nervosa: Associations with Eating Disorder and Affective Symptoms. *J Psychopathol Behav Assess* **2018**, *40*, 691-700, doi:10.1007/s10862-018-9672-8.
48. Oldershaw, A.; DeJong, H.; Hambrook, D.; Broadbent, H.; Tchanturia, K.; Treasure, J.; Schmidt, U. Emotional processing following recovery from anorexia nervosa. *Eur Eat Disord Rev* **2012**, *20*, 502-509, doi:10.1002/erv.2153.
49. Skunde, M.; Walther, S.; Simon, J.J.; Wu, M.; Bendszus, M.; Herzog, W.; Friederich, H.C. Neural signature of behavioural inhibition in women with bulimia nervosa. *J Psychiatry Neurosci* **2016**, *41*, E69-78, doi:10.1503/jpn.150335.
50. Liao, P.C.; Uher, R.; Lawrence, N.; Treasure, J.; Schmidt, U.; Campbell, I.C.; Collier, D.A.; Tchanturia, K. An examination of decision making in bulimia nervosa. *J Clin Exp Neuropsychol* **2009**, *31*, 455-461, doi:10.1080/13803390802251378.
51. Boeka, A.G.; Lokken, K.L. The Iowa gambling task as a measure of decision making in women with bulimia nervosa. *J Int Neuropsychol Soc* **2006**, *12*, 741-745, doi:10.1017/S1355617706060887.

52. Brand, M.; Franke-Sievert, C.; Jacoby, G.E.; Markowitsch, H.J.; Tuschen-Caffier, B. Neuropsychological correlates of decision making in patients with bulimia nervosa. *Neuropsychology* **2007**, *21*, 742-750, doi:10.1037/0894-4105.21.6.742.
53. Matsumoto, J.; Hirano, Y.; Numata, N.; Matzuzawa, D.; Murano, S.; Yokote, K.; Iyo, M.; Shimizu, E.; Nakazato, M. Comparison in decision-making between bulimia nervosa, anorexia nervosa, and healthy women: influence of mood status and pathological eating concerns. *J Eat Disord* **2015**, *3*, 14, doi:10.1186/s40337-015-0050-6.
54. Roberts, M.E.; Tchanturia, K.; Stahl, D.; Southgate, L.; Treasure, J. A systematic review and meta-analysis of set-shifting ability in eating disorders. *Psychol Med* **2007**, *37*, 1075-1084, doi:10.1017/S0033291707009877.
55. Tchanturia, K.; Anderluh, M.B.; Morris, R.G.; Rabe-Hesketh, S.; Collier, D.A.; Sanchez, P.; Treasure, J.L. Cognitive flexibility in anorexia nervosa and bulimia nervosa. *J Int Neuropsychol Soc* **2004**, *10*, 513-520, doi:10.1017/S1355617704104086.
56. Tchanturia, K.; Davies, H.; Roberts, M.; Harrison, A.; Nakazato, M.; Schmidt, U.; Treasure, J.; Morris, R. Poor cognitive flexibility in eating disorders: examining the evidence using the Wisconsin Card Sorting Task. *PLoS One* **2012**, *7*, e28331, doi:10.1371/journal.pone.0028331.
57. Vall, E.; Wade, T.D. Trail making task performance in inpatients with anorexia nervosa and bulimia nervosa. *Eur Eat Disord Rev* **2015**, *23*, 304-311, doi:10.1002/erv.2364.
58. Galimberti, E.; Martoni, R.M.; Cavallini, M.C.; Erzegovesi, S.; Bellodi, L. Motor inhibition and cognitive flexibility in eating disorder subtypes. *Prog Neuropsychopharmacol Biol Psychiatry* **2012**, *36*, 307-312, doi:10.1016/j.pnpbp.2011.10.017.
59. Nusslock, R.; Alloy, L.B. Reward processing and mood-related symptoms: An RDoC and translational neuroscience perspective. *J Affect Disord* **2017**, *216*, 3-16, doi:10.1016/j.jad.2017.02.001.
60. Simon, J.J.; Skunde, M.; Walther, S.; Bendszus, M.; Herzog, W.; Friederich, H.C. Neural signature of food reward processing in bulimic-type eating disorders. *Soc Cogn Affect Neurosci* **2016**, *11*, 1393-1401, doi:10.1093/scan/nsw049.
61. Radeloff, D.; Willmann, K.; Otto, L.; Lindner, M.; Putnam, K.; Leeuwen, S.V.; Kaye, W.H.; Poustka, F.; Wagner, A. High-fat taste challenge reveals altered striatal response in women recovered from bulimia nervosa: A pilot study. *World J Biol Psychiatry* **2014**, *15*, 307-316, doi:10.3109/15622975.2012.671958.
62. Bohon, C.; Stice, E. Negative affect and neural response to palatable food intake in bulimia nervosa. *Appetite* **2012**, *58*, 964-970, doi:10.1016/j.appet.2012.02.051.
63. Matta, A.D., Gonçalves, F. L., & Bizarro, L. . Delay discounting: Concepts and measures. *Psychology & Neuroscience* **2012**, *5*, 135-146.
64. Neveu, R.; Neveu, D.; Barsumian, F.; Fouragnan, E.; Carrier, E.; Lai, M.; Sultan, J.; Nicolas, A.; Coricelli, G. Improved planning abilities in binge eating. *PLoS One* **2014**, *9*, e105657, doi:10.1371/journal.pone.0105657.
65. Kekic, M.; Bartholdy, S.; Cheng, J.; McClelland, J.; Boysen, E.; Musiat, P.; O'Daly, O.G.; Campbell, I.C.; Schmidt, U. Increased temporal discounting in bulimia nervosa. *Int J Eat Disord* **2016**, *49*, 1077-1081, doi:10.1002/eat.22571.
66. Bartholdy, S.; Rennalls, S.; Danby, H.; Jacques, C.; Campbell, I.C.; Schmidt, U.; O'Daly, O.G. Temporal Discounting and the Tendency to Delay Gratification across the Eating Disorder Spectrum. *Eur Eat Disord Rev* **2017**, *25*, 344-350, doi:10.1002/erv.2513.
67. Hagan, K.E.; Jarmolowicz, D.P.; Forbush, K.T. Reconsidering delay discounting in bulimia nervosa. *Eat Behav* **2021**, *41*, 101506, doi:10.1016/j.eatbeh.2021.101506.

68. Amlung, M.; Marsden, E.; Holshausen, K.; Morris, V.; Patel, H.; Vedelago, L.; Naish, K.R.; Reed, D.D.; McCabe, R.E. Delay Discounting as a Transdiagnostic Process in Psychiatric Disorders: A Meta-analysis. *JAMA Psychiatry* **2019**, *76*, 1176-1186, doi:10.1001/jamapsychiatry.2019.2102.
69. Waxman, S.E. A systematic review of impulsivity in eating disorders. *Eur Eat Disord Rev* **2009**, *17*, 408-425, doi:10.1002/erv.952.
70. Manasse, S.M.; Murray, H.B.; Parker, M.; Forman, E.M.; Juarascio, A.S. A characterization of binge planning behavior in individuals with binge-spectrum eating disorders. *Eat Weight Disord* **2020**, *25*, 1099-1103, doi:10.1007/s40519-019-00697-6.
71. Mourilhe, C.; Moraes, C.E.; Veiga, G.D.; F, Q.d.L.; Pompeu, A.; Nazar, B.P.; Coutinho, E.S.F.; Hay, P.; Appolinario, J.C. An evaluation of binge eating characteristics in individuals with eating disorders: A systematic review and meta-analysis. *Appetite* **2021**, *162*, 105176, doi:10.1016/j.appet.2021.105176.
72. Davis, H.A.; Smith, G.T. Examining the role of urgency in predicting binge size in bulimia nervosa. *Front Psychol* **2023**, *14*, 1166119, doi:10.3389/fpsyg.2023.1166119.
73. Donnelly, B.; Touyz, S.; Hay, P.; Burton, A.; Russell, J.; Caterson, I. Neuroimaging in bulimia nervosa and binge eating disorder: a systematic review. *J Eat Disord* **2018**, *6*, 3, doi:10.1186/s40337-018-0187-1.
74. MacDonald, D.E., Solomon-Krakus, S., Jewett, R., Liebman, R. E., & Trottier, K. . *Emotion regulation in bulimia nervosa and purging disorder. In Eating Disorders* Cham: Springer International Publishing.: 2023.
75. Smyth, J.M.; Wonderlich, S.A.; Heron, K.E.; Sliwinski, M.J.; Crosby, R.D.; Mitchell, J.E.; Engel, S.G. Daily and momentary mood and stress are associated with binge eating and vomiting in bulimia nervosa patients in the natural environment. *J Consult Clin Psychol* **2007**, *75*, 629-638, doi:10.1037/0022-006X.75.4.629.
76. Fischer, S.; Wonderlich, J.; Breithaupt, L.; Byrne, C.; Engel, S. Negative urgency and expectancies increase vulnerability to binge eating in bulimia nervosa. *Eat Disord* **2018**, *26*, 39-51, doi:10.1080/10640266.2018.1418253.
77. Carr, M.M.; Wiedemann, A.A.; Macdonald-Gagnon, G.; Potenza, M.N. Impulsivity and compulsivity in binge eating disorder: A systematic review of behavioral studies. *Prog Neuropsychopharmacol Biol Psychiatry* **2021**, *110*, 110318, doi:10.1016/j.pnpbp.2021.110318.
78. Banca, P.; Harrison, N.A.; Voon, V. Compulsivity Across the Pathological Misuse of Drug and Non-Drug Rewards. *Front Behav Neurosci* **2016**, *10*, 154, doi:10.3389/fnbeh.2016.00154.
79. Dingemans, A.E.; Vanhaelen, C.B.; Aardoom, J.J.; van Furth, E.F. The influence of depressive symptoms on executive functioning in binge eating disorder: A comparison of patients and non-obese healthy controls. *Psychiatry Res* **2019**, *274*, 138-145, doi:10.1016/j.psychres.2019.02.033.
80. Kittel, R.; Schmidt, R.; Hilbert, A. Executive functions in adolescents with binge-eating disorder and obesity. *Int J Eat Disord* **2017**, *50*, 933-941, doi:10.1002/eat.22714.
81. Kollei, I.; Rustemeier, M.; Schroeder, S.; Jongen, S.; Herpertz, S.; Loeber, S. Cognitive control functions in individuals with obesity with and without binge-eating disorder. *Int J Eat Disord* **2018**, *51*, 233-240, doi:10.1002/eat.22824.
82. Mobbs, O.; Iglesias, K.; Golay, A.; Van der Linden, M. Cognitive deficits in obese persons with and without binge eating disorder. Investigation using a mental flexibility task. *Appetite* **2011**, *57*, 263-271, doi:10.1016/j.appet.2011.04.023.
83. Heatherton, T.F.; Baumeister, R.F. Binge eating as escape from self-awareness. *Psychol Bull* **1991**, *110*, 86-108, doi:10.1037/0033-2909.110.1.86.

84. Dingemans, A.E.; Visser, H.; Paul, L.; van Furth, E.F. Set-shifting abilities, mood and loss of control over eating in binge eating disorder: An experimental study. *Psychiatry Res* **2015**, *230*, 242-248, doi:10.1016/j.psychres.2015.09.001.
85. Giel, K.E.; Teufel, M.; Junne, F.; Zipfel, S.; Schag, K. Food-Related Impulsivity in Obesity and Binge Eating Disorder-A Systematic Update of the Evidence. *Nutrients* **2017**, *9*, doi:10.3390/nu9111170.
86. Lavagnino, L.; Arnone, D.; Cao, B.; Soares, J.C.; Selvaraj, S. Inhibitory control in obesity and binge eating disorder: A systematic review and meta-analysis of neurocognitive and neuroimaging studies. *Neurosci Biobehav Rev* **2016**, *68*, 714-726, doi:10.1016/j.neubiorev.2016.06.041.
87. Schag, K.; Schonleber, J.; Teufel, M.; Zipfel, S.; Giel, K.E. Food-related impulsivity in obesity and binge eating disorder--a systematic review. *Obes Rev* **2013**, *14*, 477-495, doi:10.1111/obr.12017.
88. Gianini, L.M.; White, M.A.; Masheb, R.M. Eating pathology, emotion regulation, and emotional overeating in obese adults with Binge Eating Disorder. *Eat Behav* **2013**, *14*, 309-313, doi:10.1016/j.eatbeh.2013.05.008.
89. Dang, T.B.; Hughes, E.K.; Dang, A.B.; Lai, H.Y.; Lee, J.; Liu, S.; Portingale, J.; Fuller-Tyszkiewicz, M.; Krug, I. Taking a Deeper Dive Into OSFED Subtypes: A Meta-Analysis and Systematic Review. *Int J Eat Disord* **2024**, *57*, 2006-2040, doi:10.1002/eat.24280.
90. Krug, I.; Dang, A.B.; Hughes, E.K. There is nothing as inconsistent as the OSFED diagnostic criteria. *Trends Mol Med* **2024**, *30*, 403-415, doi:10.1016/j.molmed.2024.01.006.
91. Tchanturia, K.; Lloyd, S.; Lang, K. Cognitive remediation therapy for anorexia nervosa: current evidence and future research directions. *Int J Eat Disord* **2013**, *46*, 492-495, doi:10.1002/eat.22106.
92. Prince, T.; McLoughlin, L.; Lagopoulos, J.; Elwyn, R.; Hermens, D.F. The neural correlates of socio-cognitive factors and eating disorders in young people: A systematic review. *J Psychiatr Res* **2022**, *156*, 647-659, doi:10.1016/j.jpsychires.2022.10.058.
93. Wang, Y.; Dong, D.; Todd, J.; Du, J.; Yang, Z.; Lu, H.; Chen, H. Neural correlates of restrained eaters' high susceptibility to food cues: An fMRI study. *Neurosci Lett* **2016**, *631*, 56-62, doi:10.1016/j.neulet.2016.08.017.
94. Bartholdy, S.; O'Daly, O.G.; Campbell, I.C.; Banaschewski, T.; Barker, G.; Bokde, A.L.W.; Bromberg, U.; Buchel, C.; Quinlan, E.B.; Desrivieres, S., et al. Neural Correlates of Failed Inhibitory Control as an Early Marker of Disordered Eating in Adolescents. *Biol Psychiatry* **2019**, *85*, 956-965, doi:10.1016/j.biopsych.2019.01.027.
95. Bodell, L.P.; Wildes, J.E.; Goldschmidt, A.B.; Lepage, R.; Keenan, K.E.; Guyer, A.E.; Hipwell, A.E.; Stepp, S.D.; Forbes, E.E. Associations Between Neural Reward Processing and Binge Eating Among Adolescent Girls. *J Adolesc Health* **2018**, *62*, 107-113, doi:10.1016/j.jadohealth.2017.08.006.
96. Wang, S.B.; Gray, E.K.; Coniglio, K.A.; Murray, H.B.; Stone, M.; Becker, K.R.; Thomas, J.J.; Eddy, K.T. Cognitive rigidity and heightened attention to detail occur transdiagnostically in adolescents with eating disorders. *Eat Disord* **2021**, *29*, 408-420, doi:10.1080/10640266.2019.1656470.
97. Johnson-Munguia, S.; Negi, S.; Chen, Y.; Thomeczek, M.L.; Forbush, K.T. Eating disorder psychopathology, psychiatric impairment, and symptom frequency of atypical anorexia nervosa versus anorexia nervosa: A systematic review and meta-analysis. *Int J Eat Disord* **2024**, *57*, 761-779, doi:10.1002/eat.23989.
98. Olivo, G.; Zhukovsky, C.; Salonen-Ros, H.; Larsson, E.M.; Brooks, S.; Schioth, H.B. Functional connectivity underlying hedonic response to food in female adolescents with atypical AN: the role of somatosensory and salience networks. *Transl Psychiatry* **2019**, *9*, 276, doi:10.1038/s41398-019-0617-0.
99. Olivo, G.; Solstrand Dahlberg, L.; Wiemerslage, L.; Swenne, I.; Zhukovsky, C.; Salonen-Ros, H.; Larsson, E.M.; Gaudio, S.; Brooks, S.J.; Schioth, H.B. Atypical anorexia nervosa is not related to brain structural changes in newly diagnosed adolescent patients. *Int J Eat Disord* **2018**, *51*, 39-45, doi:10.1002/eat.22805.

100. Seitz, J.; Herpertz-Dahlmann, B.; Konrad, K. Brain morphological changes in adolescent and adult patients with anorexia nervosa. *J Neural Transm (Vienna)* **2016**, *123*, 949-959, doi:10.1007/s00702-016-1567-9.
101. Davis, H.A.; Smith, G.T.; Keel, P.K. An examination of negative urgency and other impulsigenic traits in purging disorder. *Eat Behav* **2020**, *36*, 101365, doi:10.1016/j.eatbeh.2020.101365.
102. Krug, I.; Giles, S.E.; Granero, R.; Aguera, Z.; Sanchez, I.; Sanchez-Gonzalez, J.; Jimenez-Murcia, S.; Fernandez-Aranda, F. Where does purging disorder lie on the symptomatologic and personality continuum when compared to other eating disorder subtypes? Implications for the DSM. *Eur Eat Disord Rev* **2022**, *30*, 36-49, doi:10.1002/erv.2872.
103. Cleator, J.; Abbott, J.; Judd, P.; Sutton, C.; Wilding, J.P. Night eating syndrome: implications for severe obesity. *Nutr Diabetes* **2012**, *2*, e44, doi:10.1038/nutd.2012.16.
104. Kaur, J.; Dang, A.B.; Gan, J.; An, Z.; Krug, I. Night Eating Syndrome in Patients With Obesity and Binge Eating Disorder: A Systematic Review. *Front Psychol* **2021**, *12*, 766827, doi:10.3389/fpsyg.2021.766827.
105. Lavery, M.E.; Frum-Vassallo, D. An Updated Review of Night Eating Syndrome: An Under-Represented Eating Disorder. *Curr Obes Rep* **2022**, *11*, 395-404, doi:10.1007/s13679-022-00487-9.
106. Bargagna, M.C., M. . Night Eating Syndrome: A Review of Etiology, Assessment, and Suggestions for Clinical Treatment. . *Psychiatry Int* **2024**, *5*, 289-304, doi:https://doi.org/10.3390/psychiatryint5020020.
107. Darcy, A.M.; Fitzpatrick, K.K.; Colborn, D.; Manasse, S.; Datta, N.; Aspen, V.; Shields, C.S.; Le Grange, D.; Lock, J. Set-shifting among adolescents with bulimic spectrum eating disorders. *Psychosom Med* **2012**, *74*, 869-872, doi:10.1097/PSY.0b013e31826af636.
108. Manasse, S.M.; Forman, E.M.; Ruocco, A.C.; Butryn, M.L.; Juarascio, A.S.; Fitzpatrick, K.K. Do executive functioning deficits underpin binge eating disorder? A comparison of overweight women with and without binge eating pathology. *Int J Eat Disord* **2015**, *48*, 677-683, doi:10.1002/eat.22383.
109. Oliva, R.; Morys, F.; Horstmann, A.; Castiello, U.; Begliomini, C. The impulsive brain: Neural underpinnings of binge eating behavior in normal-weight adults. *Appetite* **2019**, *136*, 33-49, doi:10.1016/j.appet.2018.12.043.
110. Mattavelli, G.; Gorrino, I.; Tornaghi, D.; Canessa, N. Cognitive and motor impulsivity in the healthy brain, and implications for eating disorders and obesity: A coordinate-based meta-analysis and systematic review. *Cortex* **2024**, *171*, 90-112, doi:10.1016/j.cortex.2023.10.008.
111. Lane, J.M.; Wright, R.O.; Eggers, S. The interconnection between obesity and executive function in adolescence: The role of the gut microbiome. *Neurosci Biobehav Rev* **2023**, *153*, 105337, doi:10.1016/j.neubiorev.2023.105337.
112. Sakib, M.N.; Best, J.R.; Hall, P.A. Bidirectional Associations Between Adiposity and Cognitive Function and Mediation by Brain Morphology in the ABCD Study. *JAMA Netw Open* **2023**, *6*, e2255631, doi:10.1001/jamanetworkopen.2022.55631.
113. Sui, S.X.; Pasco, J.A. Obesity and Brain Function: The Brain-Body Crosstalk. *Medicina (Kaunas)* **2020**, *56*, doi:10.3390/medicina56100499.
114. Yang, Y.; Shields, G.S.; Guo, C.; Liu, Y. Executive function performance in obesity and overweight individuals: A meta-analysis and review. *Neurosci Biobehav Rev* **2018**, *84*, 225-244, doi:10.1016/j.neubiorev.2017.11.020.
115. Song, S.; Li, Q.; Jiang, Y.; Liu, Y.; Xu, A.; Liu, X.; Chen, H. Do Overweight People Have Worse Cognitive Flexibility? Cues-Triggered Food Craving May Have a Greater Impact. *Nutrients* **2022**, *14*, doi:10.3390/nu14020240.

116. Fagundo, A.B.; de la Torre, R.; Jimenez-Murcia, S.; Aguera, Z.; Granero, R.; Tarrega, S.; Botella, C.; Banos, R.; Fernandez-Real, J.M.; Rodriguez, R., et al. Executive functions profile in extreme eating/weight conditions: from anorexia nervosa to obesity. *PLoS One* **2012**, *7*, e43382, doi:10.1371/journal.pone.0043382.
117. Volkow, N.D.; Wang, G.J.; Baler, R.D. Reward, dopamine and the control of food intake: implications for obesity. *Trends Cogn Sci* **2011**, *15*, 37-46, doi:10.1016/j.tics.2010.11.001.
118. Belfort-DeAguiar, R.; Seo, D. Food Cues and Obesity: Overpowering Hormones and Energy Balance Regulation. *Curr Obes Rep* **2018**, *7*, 122-129, doi:10.1007/s13679-018-0303-1.
119. Kass, A.E.; Wildes, J.E.; Coccaro, E.F. Identification and regulation of emotions in adults of varying weight statuses. *J Health Psychol* **2019**, *24*, 941-952, doi:10.1177/1359105316689604.
120. Organization, W.H. *ICD-11: International classification of diseases* 2022; https://icd.who.int/.
121. McGorry, P.; Keshavan, M.; Goldstone, S.; Amminger, P.; Allott, K.; Berk, M.; Lavoie, S.; Pantelis, C.; Yung, A.; Wood, S., et al. Biomarkers and clinical staging in psychiatry. *World Psychiatry* **2014**, *13*, 211-223, doi:10.1002/wps.20144.
122. Harrison, A.; Tchanturia, K.; Treasure, J. Attentional bias, emotion recognition, and emotion regulation in anorexia: state or trait? *Biol Psychiatry* **2010**, *68*, 755-761, doi:10.1016/j.biopsych.2010.04.037.
123. Hanegraaf, L.; Anderson, A.; Neill, E.; Giddens, E.; Boon, E.; Bryant, E.; Calvert, S.; Carroll, B.; Fernandez-Aranda, F.; Ikin, S., et al. Treatment Targets and Strategies for Eating Disorders Recovery: A Delphi Consensus With Lived Experience, Carers, Researchers, and Clinicians. *Int J Eat Disord* **2024**, 10.1002/eat.24304, doi:10.1002/eat.24304.
124. Fonville, L.; Giampietro, V.; Williams, S.C.; Simmons, A.; Tchanturia, K. Alterations in brain structure in adults with anorexia nervosa and the impact of illness duration. *Psychol Med* **2014**, *44*, 1965-1975, doi:10.1017/S0033291713002389.
125. Hagan, K.E.; Christensen, K.A.; Forbush, K.T. A preliminary systematic review and meta-analysis of randomized-controlled trials of cognitive remediation therapy for anorexia nervosa. *Eat Behav* **2020**, *37*, 101391, doi:10.1016/j.eatbeh.2020.101391.
126. Tchanturia, K.; Doris, E.; Mountford, V.; Fleming, C. Cognitive Remediation and Emotion Skills Training (CREST) for anorexia nervosa in individual format: self-reported outcomes. *BMC Psychiatry* **2015**, *15*, 53, doi:10.1186/s12888-015-0434-9.
127. Beck, A.T.; Dozois, D.J. Cognitive therapy: current status and future directions. *Annu Rev Med* **2011**, *62*, 397-409, doi:10.1146/annurev-med-052209-100032.
128. Brown, T.A.; Wisniewski, L.; Anderson, L.K. Dialectical Behavior Therapy for Eating Disorders: State of the Research and New Directions. *Eat Disord* **2020**, *28*, 97-100, doi:10.1080/10640266.2020.1728204.
129. Sala, M.; Shankar Ram, S.; Vanzhula, I.A.; Levinson, C.A. Mindfulness and eating disorder psychopathology: A meta-analysis. *Int J Eat Disord* **2020**, *53*, 834-851, doi:10.1002/eat.23247.
130. Mattingley, S.; Youssef, G.J.; Manning, V.; Graeme, L.; Hall, K. Distress tolerance across substance use, eating, and borderline personality disorders: A meta-analysis. *J Affect Disord* **2022**, *300*, 492-504, doi:10.1016/j.jad.2021.12.126.
131. Thorsrud, T.; Bang, M.A.; Dahlgren, C.L.; Nordfjaern, T.; Weider, S. Cognitive remediation therapy for patients with eating disorders: a qualitative study. *J Eat Disord* **2024**, *12*, 142, doi:10.1186/s40337-024-01101-0.
132. Koskina, A.; Campbell, I.C.; Schmidt, U. Exposure therapy in eating disorders revisited. *Neurosci Biobehav Rev* **2013**, *37*, 193-208, doi:10.1016/j.neubiorev.2012.11.010.

133. Telch, C.F.; Agras, W.S.; Linehan, M.M. Dialectical behavior therapy for binge eating disorder. *J Consult Clin Psychol* **2001**, *69*, 1061-1065, doi:10.1037//0022-006x.69.6.1061.
134. Keck-Kester, T.; Huerta-Saenz, L.; Spotts, R.; Duda, L.; Raja-Khan, N. Do Mindfulness Interventions Improve Obesity Rates in Children and Adolescents: A Review of the Evidence. *Diabetes Metab Syndr Obes* **2021**, *14*, 4621-4629, doi:10.2147/DMSO.S220671.
135. Braden, A.; Redondo, R.; Ferrell, E.; Anderson, L.; Weinandy, J.G.; Watford, T.; Barnhart, W.R. An Open Trial Examining Dialectical Behavior Therapy Skills and Behavioral Weight Loss for Adults With Emotional Eating and Overweight/Obesity. *Behav Ther* **2022**, *53*, 614-627, doi:10.1016/j.beth.2022.01.008.
136. Chew, H.S.J.; Chng, S.; Rajasegaran, N.N.; Choy, K.H.; Chong, Y.Y. Effectiveness of acceptance and commitment therapy on weight, eating behaviours and psychological outcomes: a systematic review and meta-analysis. *Eat Weight Disord* **2023**, *28*, 6, doi:10.1007/s40519-023-01535-6.
137. Di Natale, A.F.; Pizzoli, S.F.M.; Brizzi, G.; Di Lernia, D.; Frisone, F.; Gaggioli, A.; Rabarbari, E.; Oasi, O.; Repetto, C.; Rossi, C., et al. Harnessing Immersive Virtual Reality: A Comprehensive Scoping Review of its Applications in Assessing, Understanding, and Treating Eating Disorders. *Curr Psychiatry Rep* **2024**, *26*, 470-486, doi:10.1007/s11920-024-01523-2.
138. Riva, G.; Malighetti, C.; Serino, S. Virtual reality in the treatment of eating disorders. *Clin Psychol Psychother* **2021**, *28*, 477-488, doi:10.1002/cpp.2622.
139. Montana, J.I.; Matamala-Gomez, M.; Maisto, M.; Mavrodiev, P.A.; Cavalera, C.M.; Diana, B.; Mantovani, F.; Realdon, O. The Benefits of emotion Regulation Interventions in Virtual Reality for the Improvement of Wellbeing in Adults and Older Adults: A Systematic Review. *J Clin Med* **2020**, *9*, doi:10.3390/jcm9020500.
140. Navarro-Haro, M.V.; Modrego-Alarcon, M.; Hoffman, H.G.; Lopez-Montoyo, A.; Navarro-Gil, M.; Montero-Marin, J.; Garcia-Palacios, A.; Borao, L.; Garcia-Campayo, J. Evaluation of a Mindfulness-Based Intervention With and Without Virtual Reality Dialectical Behavior Therapy((R)) Mindfulness Skills Training for the Treatment of Generalized Anxiety Disorder in Primary Care: A Pilot Study. *Front Psychol* **2019**, *10*, 55, doi:10.3389/fpsyg.2019.00055.
141. Marcolini, F.; Ravaglia, A.; Tempia Valenta, S.; Bosco, G.; Marconi, G.; De Ronchi, D.; Atti, A.R. Severe enduring anorexia nervosa (SE-AN) treatment options and their effectiveness: a review of literature. *J Eat Disord* **2024**, *12*, 48, doi:10.1186/s40337-024-01006-y.
142. Chmiel, J.; Kurpas, D.; Rybakowski, F.; Leszek, J. The Effectiveness of Transcranial Direct Current Stimulation (tDCS) in Binge Eating Disorder (BED)-Review and Insight into the Mechanisms of Action. *Nutrients* **2024**, *16*, doi:10.3390/nu16101521.
143. Duriez, P.; Bou Khalil, R.; Chamoun, Y.; Maatoug, R.; Strumila, R.; Seneque, M.; Gorwood, P.; Courtet, P.; Guillaume, S. Brain Stimulation in Eating Disorders: State of the Art and Future Perspectives. *J Clin Med* **2020**, *9*, doi:10.3390/jcm9082358.
144. Sato, Y.; Saito, N.; Utsumi, A.; Aizawa, E.; Shoji, T.; Izumiyama, M.; Mushiake, H.; Hongo, M.; Fukudo, S. Neural basis of impaired cognitive flexibility in patients with anorexia nervosa. *PLoS One* **2013**, *8*, e61108, doi:10.1371/journal.pone.0061108.
145. Friederich, H.C.; Walther, S.; Bendszus, M.; Biller, A.; Thomann, P.; Zeigermann, S.; Katus, T.; Brunner, R.; Zastrow, A.; Herzog, W. Grey matter abnormalities within cortico-limbic-striatal circuits in acute and weight-restored anorexia nervosa patients. *Neuroimage* **2012**, *59*, 1106-1113, doi:10.1016/j.neuroimage.2011.09.042.
146. Kaye, W.H.; Fudge, J.L.; Paulus, M. New insights into symptoms and neurocircuit function of anorexia nervosa. *Nat Rev Neurosci* **2009**, *10*, 573-584, doi:10.1038/nrn2682.

147. Lee, D.J.; Elias, G.J.B.; Lozano, A.M. Neuromodulation for the treatment of eating disorders and obesity. *Ther Adv Psychopharmacol* **2018**, *8*, 73-92, doi:10.1177/2045125317743435.
148. Gallop, L.; Flynn, M.; Campbell, I.C.; Schmidt, U. Neuromodulation and Eating Disorders. *Curr Psychiatry Rep* **2022**, *24*, 61-69, doi:10.1007/s11920-022-01321-8.
149. Formolo, D.A.; Gaspar, J.M.; Melo, H.M.; Eichwald, T.; Zepeda, R.J.; Latini, A.; Okun, M.S.; Walz, R. Deep Brain Stimulation for Obesity: A Review and Future Directions. *Front Neurosci* **2019**, *13*, 323, doi:10.3389/fnins.2019.00323.
150. Gouveia, F.V.; Silk, E.; Davidson, B.; Pople, C.B.; Abrahao, A.; Hamilton, J.; Ibrahim, G.M.; Muller, D.J.; Giacobbe, P.; Lipsman, N., et al. A systematic review on neuromodulation therapies for reducing body weight in patients with obesity. *Obes Rev* **2021**, *22*, e13309, doi:10.1111/obr.13309.
151. Smolinska, K.; Szopa, A.; Sobczynski, J.; Serefko, A.; Dobrowolski, P. Nutritional Quality Implications: Exploring the Impact of a Fatty Acid-Rich Diet on Central Nervous System Development. *Nutrients* **2024**, *16*, doi:10.3390/nu16071093.
152. Gall, Z.; Szekely, O. Role of Vitamin D in Cognitive Dysfunction: New Molecular Concepts and Discrepancies between Animal and Human Findings. *Nutrients* **2021**, *13*, doi:10.3390/nu13113672.