



REVIEW

Psychological risk factors, cognitive-contextual approaches and neural correlates in eating disorders: an integrative review

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Abstract

Eating disorders (ED) are a significant source of physical and psychosocial impairment, mainly amongst women in adolescence and early adulthood. The development and progression of ED are influenced by a large set of environmental and social factors, but it has also been recognized that ED have important biological and psychological determinants.

This article reviews the literature on ED concerning defensive emotion and psychological processes involved in the development and maintenance of ED, but also their neuroimaging/electrophysiological correlates. Furthermore, it also reviews treatment approaches that focused on the improvement of ED patients' symptomatology.

An extensive literature exists regarding neuroimaging/electrophysiological studies associated with clinical features of ED patients in terms of body image and food processing and reward/inhibition processes. However, studies addressing the neural correlates of psychological processes, such as rumination, experiential avoidance, cognitive fusion and self-criticism, and the defensive emotion of shame, which are key components for the development and maintenance of ED, are still scarce if not lacking. In the context of this cognitive set, cognitive-contextual approaches for ED patients, such as mindfulness, acceptance- and compassion-based interventions are promising to change the way individuals relate to their aversive experiences, but still requires a deeper understanding of the mechanisms by which it operates and further testing of its therapeutic efficacy.

This review highlights the lack of neuroimaging/electrophysiological studies in ED patients in which concerns shame and other relevant psychological processes, but also the scarce evidence in terms of brain modifications associated with therapeutic efficacy of cognitive-contextual approaches that help to change the way individuals relate to their body, weight and food concerns, reduce maladaptive emotion regulation strategies and improve well-being in patients suffering from ED.

Keywords: Eating disorders, Neural correlates, Psychological processes, Cognitive-contextual approaches.

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Eating Disorders (ED)

Anorexia Nervosa (AN), Bulimia Nervosa (BN) and Binge Eating Disorder (BED) are included in the Feeding and Eating Disorders section in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) [1] and have increasingly become the focus of research studies. According to the DSM-5, the 12-month prevalence of AN is 0.4% and of BN is 1% to 1.5%. BED was recently added as a diagnostic category in DSM-5, with a reported 12-month prevalence among U.S. adult females of 1.6% while in males it is 0.8%. In the last decades, ED prevalence has increased and it was estimated that about 30 million people worldwide have a clinical diagnosis of this nature [2].

All these disorders have in common the anomalous eating patterns and diet rules, but also persistent thoughts about food, weight, shape and body self-image [3]. In particular, the patients with an ED have a persistent desire to achieve a thin body ideal, a pathologic fear of gaining weight, a distorted body image, mood changes, which are frequently associated with temperament and idiosyncratic personality traits [4]. It is known that AN and BN patients present opposite extremes of inhibition self-control. BN patients reveal changes in impulse control, namely poor control over initiation and termination of eating, food choice and adaptation/sensitization to reward stimulus [3,5]. Conversely, AN patients tend to present a more rigid and perfectionist attitude, with a certain tendency to obsessive-compulsive behaviour. Furthermore, they are able to sustain self-denial of food, lessening anxiety and negative affect through dietary restriction and reduced daily caloric intake, being even able to delay reward [4,6,7]. Alternatively, BED individuals are characterized by the consumption of large amounts of food in small periods of time, with perceived loss of control (binge episode). Other symptoms include feeling uncomfortably full, eating rapidly even when not hungry, as well as feeling disgusted after the binge episode. However, contrary to BN, compensatory behaviour (e.g. vomiting or the use of laxatives) is not present during these episodes of excessive eating [8]. Therefore, it seems that ED individuals have difficulties in the regulation of their emotional state and negative affect and use ineffective eating behaviours to modulate emotion.

ED patients usually present high levels of comorbidity with other psychological disorders, which poses significant health risks due to high rates of suicide attempts and mortality [9]. Individuals with ED suffer from significant interference in daily functioning and for some patients, these disorders have a chronic course [10]. ED are much more common in females and usually begin during adolescence or young adulthood [11]. Those affected with ED often require long-term expensive treatments, which can be challenging for both the patients and their families [12], and this condition entails significant economic burden for societies as well [13]. Research has revealed the

complex nature of the multifactorial aetiology of ED, involving the interaction of psychological, neurobiological and social factors [14].

In this review, we aim to analyse neuroimaging/electrophysiological correlates of ED patients' symptomatology, defensive emotions and psychological processes involved in the development and maintenance of ED. Moreover, we are interested to provide a review of contextual cognitive-behavioural approaches that can help ED patients to deal with their aversive internal experiences (e.g. thoughts about food, weight, shape and body self-image, negative affect) that derive from ineffective attempts (e.g. caloric restriction, binge eating, and compensatory behaviours) of self-regulation.

Neuroimaging studies in ED

In the last few years, several neuroimaging techniques have been used to study the brain structure and function in patients with ED, in the hope of shedding some light in the mechanisms underlying ED pathophysiology. Magnetic resonance imaging (MRI) has been used to study differences in gray and white matter volumes, as well as cortical thickness and surface area. Furthermore, functional magnetic resonance imaging (fMRI) is also frequently used, by measuring changes in local blood flow and deoxyhemoglobin levels during brain activation. Other techniques like positron emission tomography (PET) and single photon emission computed tomography (SPECT) are also used, providing information about regional cerebral glucose metabolism or the distribution of neurotransmitter receptors [15].

Several studies have revealed consistent changes in patients with ED, namely reductions in brain volume, modification of neurotransmitter levels, reduced cerebral brain flow and metabolism and lower cognitive test results. Nevertheless, the association between cerebral changes and clinical symptoms is not easy to establish, since some of the dysfunctions may be linked to malnutrition, emotion deregulation or impulse control, which appear to be dysfunctional in these patients [4].

Neurocircuitry of cognitive and emotional processing of visual presentation

Several paradigms have been used to investigate the pathophysiological basis of ED, allowing studying the brain regions involved in the regulation of eating behaviour. Given the importance of body perception in these patients, the majority of these paradigms consist on showing images of body images and food.

One study using fMRI have found that AN patients showed an absence of activation when observing their own body. This finding suggests a possible suppression of the cognitive, perceptual and emotional processing of self-images in these patients, which may be linked to a distorted perception of their own body figure [16]. Vocks and

colleagues have also found that AN patients revealed enhanced limbic activity (amygdala) when looking to other women's body, which may be related to stronger emotional affective reactions, probably due to unfavourable social comparison processes [17]. One study using participants' distorted body images as stimuli have shown that when AN patients see themselves as oversized, there is a higher activation in regions such as the extrastriate body area, superior and inferior parietal lobule and prefrontal areas, which may be the result of higher affective value given to oversized body image [18].

Uher and colleagues presented food and aversive emotional images while undergoing an fMRI task. ED patients considered the food stimuli as threatening and disgusting. In addition, a medial prefrontal response to symptom-provoking stimuli was identified as a common feature both in AN and BN. This abnormal response in medial prefrontal circuits is frequently observed in obsessive-compulsive and addictive disorders, being probably related to the compulsive behaviour associated with these disorders [19].

Overall, neuroimaging studies in ED indicate the existence of differences in the perception and neural processing of both bodies and food stimuli in regions associated with cognitive control, reward and somatosensory input.

Neurocircuitry of reward and inhibition

Subjects with AN and BN present opposite extremes of inhibitory self-control. Wierenga and colleagues raised the hypothesis that there is a disturbance in the valuation of reward, with abnormal reward appraisal being associated with emotion dysregulation, which was manifested by exaggerated anxiety and sensitivity to punishment. Conversely, according to the type of ED, inhibitory behaviour may vary. It is suggested that while AN patients use exaggerated cognitive control to compensate dysfunctional reward processing, BN individuals seem to present deficient cognitive control, being more unstable in the response to food stimuli [3].

In the study of Uher and colleagues [19], using visual food cues, ED patients revealed higher activation in the medial prefrontal cortex (PFC) and inhibition of the lateral PFC. Specifically, individuals with AN presented decreased responses in the orbitofrontal cortex (OFC) and insula, which may indicate that they tend not to respond to the hedonic aspects of food. BN patients, on the other hand, presented increased activations in the insula, suggesting enhanced reward processing in these patients [20].

One study reported ventral striatal activity in AN patients when observing images of underweight females [20]. These results show that this type of stimuli reflect, in part, a reward value for these patients, since the ideal and unrealistic female body is the one they expect to achieve. In accordance with these data, other studies analysed the neural response of AN patients when comparing themselves with idealized female bodies. Regions associated with emotional and rewarding responses were engaged, namely the insula,

putamen and dorsolateral prefrontal cortex (DLPFC). Alternatively, when presenting oversized body-self-images, it was shown enhanced emotional involvement and prefrontal activations, particularly in the amygdala and DLPFC. Furthermore, while the highest activity in the mesolimbic reward network took place in AN patients, the highest activity in healthy controls happened when observing normal weight individuals. Overall, it seems that while increased activity in the insula and striatum may be linked to motivational salience towards cues (e.g. thinness), the higher activations presented by the amygdala and PFC to overweight body images may occur due to 'top-down' cognitive mechanisms to help reducing the effects of stimuli considered to be threatening by these patients. This is consistent with previous studies reporting prefrontal activity in response to oversized body images [18].

Overall, behavioural, cognitive and neuroimaging data seem to indicate that ED patients present an altered balance between reward and inhibition. While AN patients tend to show excessive inhibition and diminished valuation of reward, BN individuals present a dysregulated inhibitory control and excessive reward sensitivity leading to a pattern of under and overconsumption, respectively [3].

Connectivity studies in ED

There are several brain networks functionally connected that drive behaviour, including the default mode network (DMN), salience network and the sensory and sensorimotor network. Several studies, investigating these networks, corroborate the existence of changes in sensorimotor and visual networks in ED patients either in terms of functional connectivity [15,21], which looks at correlations between brain regions [22], or in effective connectivity [23], which establishes causal effects between neural systems.

A study comparing AN, BN and controls revealed that effective connectivity patterns between the insula and frontal regions distinguished groups while performing a visual food cue task [24]. Furthermore, other studies, using effective connectivity analyses, revealed different networks between AN and healthy controls during visual processing of human bodies, suggesting alterations in the networks that perform body processing in women with AN [23]. A study performed with AN patients also presented reduced effective connectivity within the frontal cortex, the brain cognitive control system, and increased effective connectivity between regions involved in salience processing [25]. In line with these results, another study found that, compared to controls, AN patients and recovered AN patients presented lower activity in the ventral visual network, but increased somatosensory network activity in AN [26]. Additionally, a more recent study showed that despite DMN functional connectivity did not differ between AN and controls, AN patients displayed reduced functional connectivity between the sensorimotor and visual networks, in comparison to healthy control individuals [27].

All these findings suggest the existence of changes in the connectivity of sensorimotor and visual networks of ED individuals, which may be related to differences in visuospatial processing, taking into account the distorted perceptions of body image experienced by these patients. Moreover, they suggest the existence of dysfunctional causal connections within the frontal cortex, which may connect to maladaptive control strategies observed in these patients.

Defensive emotion and psychological processes

A large body of research shows that body image concerns, disordered eating, and some personality features are among the risk factors for the development and maintenance of ED [28]. However, although psychological mechanisms that underlie the emergence and maintenance of eating pathology are beginning to be studied, much still needs to be revealed. Rumination, experiential avoidance, cognitive fusion, and self-criticism are some of the transdiagnostic maladaptive emotion regulation strategies engaged by individuals suffering from ED. Also, the emotion of shame has been considered a key feature in the onset and maintenance of ED.

Rumination

Rumination is a repetitive non-productive cognitive process focused on symptoms of distress, and its possible causes and consequences [29]. There are two types of rumination: while brooding rumination, which relates to a passive comparison of one's current situation to desired standards and is more consistently linked with negative clinical outcomes, reflective rumination pertains a purposeful inward focus aiming to solve distressing symptoms [30].

Rumination has been extensively linked with depression, and it has been also pointed out as a risk factor for ED [31]. Studies have found that brooding rumination is an important cognitive process associated with eating psychopathology [32,33]. Patients with disordered eating engage in this maladaptive emotion regulation strategy focusing on food, dietary restraint, weight, and body shape to cope with their negative emotions. Although individuals with ED might perceive rumination as useful to deal with aversive emotions, this process is, in fact, an avoidance strategy thus reinforcing eating disordered behaviours [34]. ED patients are caught in a vicious cycle, perpetuating suffering and dysfunctional strategies associated with eating psychopathology.

However, regulation of negative affect may have different results across ED. Naumann, Tuschen-Caffier, and Voderholzer [35] showed that rumination after an induced state of sadness led AN patients to feel more desire to abstain from eating, and BN patients feeling more desire to binge.

Some neuroimaging studies in ED patients have underlined this factor, suggesting that altered dorsal anterior cingulate cortex (dACC)—precuneus functional connectivity might be the basis of disorder-specific rumination on eating, weight and body shape in patients with ED [21,36].

While performing resting-state functional connectivity in AN and BN patients, Lee et al. [36] found that both patient groups compared to healthy controls displayed increased synchrony between dorsal dACC and precuneus, with this activity being positively correlated with Body Shape Questionnaire scores. Specific synchronous activity between dACC and retrosplenial cortex was found in AN, whereas between dACC and medial OFC occurred in BN. The dACC is described as a core region in the cognitive control network [37] and the precuneus as a central region in the DMN [38], whereas both the dACC [39] and the precuneus [40] are involved in self-referential processing.

Moreover, it is thought that interoceptive awareness is altered in ED patients, as these patients may be hypersensitive to interoceptive sensations due to its constant monitoring [41]. Dorsal mid-insula activity was found to be increased in AN compared to controls specifically to stomach sensations but not to other interoceptive attention types, with this activation being negatively correlated with measures of trait and state anxiety, and harm avoidance. This region also responded during an anxious induced rumination task. Importantly, during the interoceptive attention tasks, increased activity was found in the precuneus in the healthy controls group compared to the AN patients.

Experiential avoidance

Experiential avoidance relates to the unwillingness to experience aversive private events, such as emotions, thoughts, bodily sensations, and deliberate actions to change, control, avoid, escape, delay the aversive experiences or the contexts that elicit them [42,43]. Although experiential avoidance (that includes thought suppression) can be an effective short-term strategy, when this is rigidly and inflexibly applied, it becomes problematic and interferes with psychosocial functioning and leading to values-incongruent actions [44]. Experiential avoidance is being associated with a higher risk of developing psychopathology and lower psychological well-being [45].

A growing body of research shows that patients with ED present high levels of experiential avoidance [33] and often avoid experiences related to food, eating and body-image exposure. Indeed, eating disordered symptoms are behaviours whose function is to avoid aversive internal experiences, namely thoughts, feelings and body sensations [46].

No studies in ED were found directly addressing the neural correlates of experiential avoidance. However, using electroencephalography (EEG), Cochrane et al. [47] report that individuals in the high experiential avoidance group showed greater negativity in the left hemisphere, which suggested that these individuals engaged in more verbal strategies to regulate their emotional responses.

A study of resting state functional connectivity [48] in a sample of AN-restrictive type in progress for less than six months at the time of scanning found a negative correlation between the executive cortical network functional connec-

tivity maps and the anterior cingulate cortex (ACC) [48]. The authors suggest that these early onset alterations in ACC may be directly connected with the pathophysiology of AN, and could lead to the cognitive inflexibility and ruminative thoughts regarding body image and weight of AN patients.

Thought suppression involves continuous inhibition and, in healthy controls, seems to be related to activity in the DLPFC [49], with extension to the right lateral frontoparietal network [50], suggesting a coordinated engagement of the executive control and dorsal attention networks. Moreover, increased fluctuation during suppression was found in this frontoparietal network [50], with re-emergence of intrusive thoughts being associated with activation of the ACC [49]. These findings corroborate the difficulty of intrusive thoughts management through suppression maintenance. No studies underlying brain function or structure related with thought suppression were found using ED patients.

Cognitive fusion

Cognitive fusion refers to the perception of contents of thoughts as literal truths, rather than experiencing thoughts as transient and subjective internal states. When a behaviour is determined by these verbal stimuli in an unhelpful way, psychological distress may arise [51].

Individuals suffering from ED are usually fused with disturbing thoughts about body image, food, and social comparison. Thus, they try to regulate those aversive internal products by engaging in pernicious behaviours (dieting, binge eating, and purging). Indeed, a recent research showed that cognitive fusion regarding body image mediates the relationship between unfavourable social comparisons and eating psychopathology [52].

Cognitive fusion seems to be interrelated with experiential avoidance since both are based on natural language process [53]. For example, ED patients often try to get rid of thoughts with which they are fused, such as "I am fat" or "I must lose weight". To avoid those painful thoughts and feelings they engage in eating disordered behaviours.

To our knowledge, no neuroimaging studies were performed in ED patients nor in healthy participants regarding cognitive fusion. Nevertheless, an fMRI study was carried out in which a similar concept of cognitive fusion-immersion – was used. They reported that individuals in the immersion condition showed greater activity in areas associated with self-processing and visceral states [54].

Self-criticism

Self-criticism is an unforgiving form of self-to-self relationship that arises in response to one's errors or negative evaluation of personal attributes (e.g. physical appearance, behaviour, personality and intellectual features) that could lead to social disapproval or rejection. This defensive strategy is activated in an attempt to correct and improve personal attributes or behaviours to protect the self, and thus to achieve a sense of belonging to the social group [55–57].

Gilbert, Clarke, Hempel, Miles, and Irons [56] stated that self-criticism has two forms: inadequate self and hated self. Also, they identified two functions of self-criticism. One function is related to self-improvement, and the other one is focused on harming the self.

A growing body of research has emphasized the association between self-criticism and eating pathology [58–60]. Specifically, Barrow found that the self-improvement function of self-criticism in ED patients was at a similar level to non-clinical samples, but self-harming was significantly higher [61].

Self-critical individuals are believed to be more prone to feeling shame. In fact, ED patients engage in a vicious cycle of self-criticism considering their body and weight, which triggers and intensifies feelings of shame and thus elicit behaviours to change their body and shape. Individuals with AN change their undesirable body and shape by restrictive eating or excessive exercise, which increases pride and temporarily reduces shame (shame-pride cycle). In BN, individuals binge and purge to deal with shame, but the secrecy and the perception of these behaviours as abnormal may lead to more self-critical thoughts (shame-shame cycle) [62].

Studies directly addressing the neural correlates of self-criticism in healthy participants have used mainly verbal material or imagery instructions. Longe and colleagues [63] found self-criticism related activity in PFC and dACC regions. Activation of ACC has been found in relation to body dissatisfaction [64] but also during positive self-appraisal [65], being also reported its connection with non-clinical eating disordered psychopathology [64]. In fact, the exact network involved in positive and negative self-appraisal seems puzzling, as two studies [65,66] found the same network of regions when contrasting between self-critical vs. neutral and positive self-appraisal vs. neutral conditions. Using verbal material, dorsomedial and dorso-lateral PFC [65], mid-frontal regions, insula, precuneus and visual areas [66,67] responded to both positive and negative conditions when contrasted with neutral adjectives. Positive self-appraisal seems to evoke stronger activity in the precuneus, visual areas [65], amygdala and insula [65,67] when directly contrasted with self-critical adjectives, whereas self-critical displayed increased occipital activations [65], but also amygdala activation [67]. Occipital/visual regions such as visual areas and the precuneus seem to activate more during self-criticism conditions [65,66].

Crucially, contrasting self-critical vs. negative non-self-referential adjectives engages activation in the medial posterior cingulate cortex [67], suggesting that this area is specifically relevant during self-related evaluation.

Neuroimaging/electrophysiology studies in ED regarding self-criticism were found only when referring to related concepts, such as body dissatisfaction and self-evaluation/appraisal in AN [68,69] and BN patients [68,70], using event-related potentials (ERP) [68,70] and fMRI [69]. Friederich et al. [69] used pictures of slim idealized

female bodies and images of interior home designs. They found that in the AN patient group, compared to healthy controls, the self-other body-shape comparison resulted in increased activation of the right sensorimotor brain regions (insula, premotor cortex) and less activation of the rostral ACC, suggesting altered interoceptive awareness to body self-comparison and/or altered implicit motivation to thin-idealized body images in AN patients.

Finally, to better understand the nature of self-criticism found in ED, Blechert et al. [68] used verbal material specifically related to shape/weight to evaluate if non-appearance domains of performance and interpersonal related self-evaluation were linked to shape/weight concerns in AN and BN patients. For this, they used shape/weight related initial sentences, ending these either with congruent or incongruent adjectives. They assessed the N400 component of the ERP, which is usually increased if a sentence is completed by a semantically unexpected or affectively incongruent word, reflecting early stages of sentence comprehension. ERP differences between congruent and incongruent ending-sentence adjectives were found in BN but not in AN nor in healthy controls, suggesting that BN is more susceptible to the harmful influence of shape/weight on self-evaluation [71].

Shame

Shame is a self-conscious emotion that arises when an individual has a sense of being defective, inferior, inadequate, and unattractive due to one's characteristics and behaviours. Gilbert distinguished two types of shame: external and internal shame. External shame has an outwards focus and the individual is certain that others perceive the self as flawed, worthless, and inferior. Internal shame, in which the attention focus is directed inward, refers to inner experiences and self-evaluations of being imperfect, powerless, and inferior [72].

Both types of shame co-occur in ED patients and shame has been consistently associated with eating disordered behaviours [59,73]. Troop, Allan, Serpall, and Treasure [74] found that different patterns of internal and external shame are contingent on ED diagnosis. Anorexia symptoms were more highly associated with external shame than bulimic ones, and contrarily, individuals who reported bulimic symptoms reported higher levels of internal shame. Although more research is needed to consolidate this finding, these results demonstrate the important role of shame in the perpetuation of eating disordered psychopathology.

Goss and Gilbert [75] suggest that individuals suffering from ED experience shame when they perceive a negative social threat, and thus may engage in behaviours to achieve a socio-cultural ideal body weight and shape in order to strive for others' approval. Therefore, shame has a protective function and disordered eating behaviours are maladaptive strategies to deal with this emotion.

No studies in ED disentangling the neural correlates of shame were performed so far. In healthy individuals, us-

ing verbal sentences to induce shame and guilt, the shame condition elicited ACC and parahippocampal gyrus activation, whereas the guilt condition in the fusiform gyrus and the middle temporal gyrus. Specific activations were found for shame in the frontal lobe medial and inferior frontal gyrus, and for guilt in the amygdala and insula, when directly contrasting the two emotions [76]. As seen in the previous sections, alterations in brain function concerning the ACC, the insula, the amygdala and the inferior frontal gyrus have been reported in ED individuals and may contribute to the core psychopathology found in ED.

Therapeutic approaches

Several efficacy studies using Cognitive-Behavioural Therapy (CBT) have shown the reduction of eating disordered symptoms [77], and it is the recommended psychological treatment for ED. Nevertheless, some criticism has been raised due to the high number of individuals that do not respond to therapy and to the high relapse rates after CBT. The limitations brought by the traditional treatments for ED gave rise to the study of innovative treatments by the researchers. Given this, contextual cognitive-behavioural approaches, such as mindfulness, acceptance and compassion have gained increased interest in the treatment of ED. These approaches focus on changing the way individuals relate to aversive internal experiences, with awareness and distance, rather than diminishing their frequency or changing their content as in CBT [78,79]. Therefore, contextual cognitive-behavioural approaches may be seen as transdiagnostic approaches. Despite based on different theoretical models, these approaches have been integrated to become more contextual-function interventions. This functional contextual approach addresses psychological processes linked to mindfulness and acceptance, which promotes the flexibility to dealing with difficult experiences, instead of engaging in disordered eating patterns.

There are scarce studies regarding the integration of these contextual cognitive-behavioural approaches for ED. Nevertheless, the existing ones, though primarily focused on obesity, have shown promising results reducing shame, self-criticism, psychological distress and ED symptoms [80,81].

Mindfulness, acceptance and compassion-based approaches have recently been considered as important adaptive emotion regulation strategies to cope with negative emotions and thus diminishing eating pathology and improving well-being [82-84].

Mindfulness

Mindfulness-based interventions entail observing our experiences (e.g. thoughts, sensations, feelings) in the present-moment with non-judgmental awareness, and without changing them [85]. These interventions aim to promote well-being and reduce distress through the practice of mindfulness [86]. Mindfulness skills are effective alterna-

tives to defensive maladaptive strategies, such as rumination and experiential avoidance, to deal with aversive experiences [87,88]. A growing body of research had shown empirical support for mindfulness-based interventions aid not only in the psychological health of patients but also in alleviating physical symptoms [83,89,90].

Mindfulness-based interventions for people with ED encourage nonjudgmental and nonreactive observation and acceptance of bodily sensations, perceptions, cognitions, and emotions, but also allows to observe hunger and satiety cues. The shift to autopilot from increased awareness allows the individual be more willing to accept aversive experiences and to choose adaptive behaviours in stressful circumstances [91].

Lavender and colleagues [92] have shown in a non-clinical sample that mindfulness is associated with ED behaviours. In a systematic review that included eight studies, they found initial evidence supporting the effectiveness of mindfulness-based interventions to the treatment of ED [83]. Particularly for BED, a recent meta-analysis found large or medium-large effects of mindfulness-based interventions (including both approaches focused on mindfulness and acceptance strategies) [93].

Despite the increasing interest in therapeutic interventions with mindfulness training, to date, there are no studies about neural mechanisms associated with mindfulness-based interventions in ED patients. However, a review of several studies about mindfulness meditation in healthy participants showed that mindfulness impacts information processing in the medial cortex and associated DMN, insula and amygdala [94]. Also, structural imaging studies corroborate these findings, adding changes in the hippocampus as a result of mindfulness practice [94]. Boccia, Piccardi, and Guariglia [95] highlight that meditation activates brain areas involved in processing self-relevant information, self-regulation, focused problem-solving, adaptive behaviour and interoception. Besides that, expert meditators present brain changes, concerning areas involved in self-referential processes (e.g. self-awareness and self-regulation).

Acceptance

Acceptance and Commitment Therapy (ACT) is a psychological intervention focused on six psychological processes. ACT aims psychological flexibility, which means being in the present moment with non-judgmental awareness and acceptance, being open to and defuse from distressing inner experiences, and making choices accordingly with personal values and life directions. ACT encourages the acceptance of experience of emotional pain as part of the human life, without attempting to alter or suppress it, and the behavioural change must be suitable with what is meaningful. Mindfulness is an important component of ACT, and together they constitute a countermeasure to experiential avoidance and cognitive fusion [51].

The goal in ACT for ED patients is to promote a value-driven behavioural change through the use of meta-

phors and eyes-closed exercises in order to present more psychological flexibility in response to eating disordered cognitions, emotions, and behaviours. In a non-clinical sample, Masuda, Price, Anderson and Wendell [96] found that the combination of disordered eating-related cognition and psychological inflexibility predicted poor psychological outcomes.

An efficacy study of ACT for ED patients showed that this treatment may be a useful treatment, given its impact in reducing eating pathology [84]. A study comparing BED patients who participated in a 1-day ACT workshop and a wait-list control group showed that the ACT group reported less binge eating behaviour and consequently mediated weight changes [97].

To our knowledge, there are no neuroimaging studies involving acceptance and ED. So far, there is only one neuroimaging study with ACT in opioid addicts with chronic pain [98], but ACT seems to be a promising treatment with changes in brain activity and connectivity.

Self-compassion

Self-compassion can be conceptualised as the desire to alleviate one's suffering, involving a non-judgmental attitude towards our inner experiences, opening to experience feelings of caring and kindness toward oneself, and the recognition of experiences as part of the human condition [99].

Compassion-Focused Therapy (CFT) was developed by Gilbert [100] and is an acceptance-based cognitive-behavioural approach rooted in evolutionary psychology, neuroscience, and Buddhist teachings. CFT acts as an adaptive strategy to regulate shame and self-criticism, which are two aspects that contribute to the development and maintenance of several forms of psychopathology and traditional psychotherapeutic approaches seem to have difficulty treating them.

Gilbert [101] argues the existence of three evolutionary systems responsible for emotion regulation: threat system, which detects and responds to a threat, with predominant emotions of anger, anxiety, fear, disgust, and shame; resource-seeking system, which is responsible to drive individuals to pursuit resources and rewards, and is associated with excitement, joy, enthusiasm and pleasure; and soothing system, which concerns a sense of well-being, nurture and feeling safe, and triggers emotions such as calmness and content.

CFT develop compassionate attributes and skills through the use of techniques focused on attention, imagery, feeling, thinking and behaviour. The practice of CFT techniques encompasses the awareness and tolerance of the present moment and the ability to warmth, kindness and soothing ourselves when facing emotional distress [101,102].

More recently, CFT was adapted to people with ED (CFT-E) [62,103], to integrate aetiological and maintaining factors in ED, such as shame and pride. Also, CFT-E adapted CFT techniques to cultivate the soothing system in order to address eating disordered thoughts, feelings,

and behaviours and normalize eating and weight. CFT-E defends that eating disordered behaviours are an expression of overstimulation of the drive and threat systems [62]. Thus, ED patients have difficulty to access the soothing system for self-warmth or to be soothed by others and tend to regulate their affect through drive and/or pride systems.

CFT is gaining empirical support for its use in individuals suffering from ED [104–106]. A preliminary investigation showed that low self-compassionate individuals with higher fear of self-compassion presented more shame and ED pathology [107].

Until today, there are no self-compassion neuroimaging studies in ED. However, some studies with non-clinical samples have shown the underlying changes in brain systems through caring and nurturing behaviour or thoughts. Simon-Thomas et al. [108] found that compassion was associated with activation in the midbrain periaqueductal gray, a region that is activated during pain and the perception of others pain, and that has been implicated in parental nurturance behaviours. In fact, compassion training engages the insula, ventral striatum and medial OFC [80], which are brain regions implicated in positive valuation, love, and affiliation and increased individuals positive affect [110]. It seems that after a compassion training, individuals are more altruistic and that was linked to the activation of brain regions, such as the inferior parietal cortex and DLPFC, implicated in understanding the suffering of others, executive and emotional control and reward processing [111].

Conclusions

We sought to review the defensive emotion (shame) and psychological processes involved in the development and maintenance of ED, but also their neuroimaging/electrophysiological correlates. Besides that, contextual cognitive-behavioural approaches that help ED patients to deal with their aversive internal experiences and its efficacy in terms of outcomes in brain changes have been reviewed.

Noninvasive neuroimaging/electrophysiological techniques have been used to study the underlying changes in brain function of ED patients. A growing body of research has shown abnormalities in neural systems underlying decision-making and reward, emotional and body image processing. Structures such as the dACC, insula, precuneus/parietal and cerebellum, as well as frontal regions such as the medial, dorsolateral and ventromedial PFC seem to be involved in the specific neural basis of psychological vulnerability-related features.

Research has shown that ED patients present difficulties in emotion regulation engaging in maladaptive strategies (e.g. self-criticism, rumination, experiential avoidance, cognitive fusion) to deal with negative affect – shame, anxiety, sadness, guilt – that arises from body image disturbance, weight concerns or the perception of being inferior.

The available literature suggests that contextual cognitive-behavioural approaches can interrupt the effects of

maladaptive processes and consequently diminish eating disordered symptoms, improving psychological flexibility, well-being and quality of life. These approaches seem to be effective and useful in other psychiatric disorders, and thus, more research is needed to prove their efficacy in ED.

More studies, both cross-sectional and longitudinal, underpinning the neurophysiology of brain mechanisms and psychiatric symptoms in ED patients are necessary to disentangle the maintenance mechanisms and to clarify the specific processes underlying the different ED subtypes. Therefore, studies using methods such as MRI, fMRI, EEG, PET or SPECT are particularly useful, although still scarce in what respects neural correlates of ED regarding these issues. In fact, this review points to a lack of studies in ED patients directly targeting the neural correlates of cognitive fusion, self-criticism, and shame, as well as the efficacy in terms of brain functional and structural changes when using mindfulness-, acceptance- and compassion-based interventions.

Although many questions remain unanswered, efforts are nevertheless being made to understand the mechanisms involved in the genesis and maintenance of ED and the neural processes that underlie maladaptive emotion regulation strategies. Importantly, there is an increasing interest in contextual cognitive-behavioural approaches, but these approaches require further testing and studies regarding their potential on ED.

Abbreviations

ACC: Anterior cingulate cortex; ACT: Acceptance and commitment therapy; AN: Anorexia nervosa; BED: Binge eating disorder; BN: Bulimia nervosa; CBT: Cognitive-behavioural therapy; CFT: Compassion-focused therapy; CFT-E – Compassion-focused therapy for eating disorders; dACC: dorsal anterior cingulate cortex; DLPFC: Dorsolateral prefrontal cortex; DMN: Default mode network; DSM-5: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; ED: Eating disorders; EEG: Electroencephalography; ERP: Event-related potentials; fMRI: Functional magnetic resonance imaging; MRI: Magnetic resonance imaging; OFC: Orbitofrontal cortex; PET: Positron emission tomography; PFC: Prefrontal cortex; SPECT: Single photon emission computed tomography

Competing interests

The authors declare no conflict of interest.

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