

## **Emotion regulation in binge eating disorder**

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### **Summary**

A review of the literature on emotion regulation in binge eating disorder (BED) published both in English and Polish between 1990 and 2020. BED might be considered as an impulsive and compulsive disorder associated with altered reward sensitivity and food-related attentional bias. The growing body of research indicated that there were corticostriatal circuitry alterations in BED, comparable to those observed in substance abuse, including altered function of orbitofrontal, prefrontal and insular cortices with the striatum included. Negative emotions and deficits in their regulation play a significant role in BED. Processing of anger, anxiety and sadness appear to be particularly important in this disorder. Research results identified an increase in negative emotions preceding episodes of binge eating. However, there is still inconsistency when it comes to whether these episodes alleviate negative affect. Individuals with BED more often use non-adaptive emotion regulation strategies, such as rumination and suppression of negative sensations. Whereas adaptive ones, for instance, cognitive reappraisal, are used less often. Clinical implications, besides pharmacology, highlight the high effectiveness of enhanced cognitive behavioral therapy (CBT–E), dialectic-behavioral therapy (DBT) and psychodynamic therapy in the treatment of emotional dysregulation in BED. Further studies, including ecological momentary assessment (EMA), should focus on emotional changes related to the binge cycle and the identification of reinforcing factors of BED.

**Key words:** binge eating disorder, emotion regulation, neuroimaging techniques

### **Introduction**

Binge eating disorder (BED) is the most common among eating disorders [1]. Its prevalence in the general population ranges between 2 and 5%, and it is considerably higher in overweight or obese individuals – 30% [2]. The majority of patients with BED

fulfill the criteria of at least one lifetime comorbid psychiatric disorder (67–79%) [3]. Among the most common are affective disorders and anxiety disorders [4]. The disorder in question affects women three times more often than men [2]. According to the European classification ICD-10 (International Statistical Classification of Diseases and Related Health Problems), BED falls into overeating associated with other psychological disturbances (F50.4), and hence, it is not a separate nosological entity [5]. Then, the American mental classification DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5<sup>th</sup> Edition) isolated BED as a separate entity – 307.51 (F50.8) [4].

Binge eating disorder manifests itself by recurrent uncontrolled episodes of overeating defined as consuming increased amounts of food in a definite period of time (e.g., within two hours) that the majority of people would not eat in similar circumstances and within similar time period. A characteristic feature is the loss of control over the amount and quality of the consumed food, and the difficulty in ceasing to eat despite the sensation of discomfort caused by being overstuffed. Furthermore, a physical feeling of being hungry does not precede episodes of binge eating. Fierce food consumption often takes place in secrecy (due to shame), in private, and the pace is fast. Individuals with BED are aware of losing control over the consumed food, what leads to feeling guilt as well as disgust, and abhorring oneself. Therefore, one of the key parts of BED is the sense of subjective suffering of the ill individual. However, this disorder is not accompanied by compensatory behaviors (such as inducing emesis or abusing laxatives) that are common for bulimia nervosa (BN), and thus, the two disease entities should be differentiated correctly [4].

According to DSM-5, BED can be diagnosed if overeating episodes occur at least once a week for 3 months. The disorder severity is determined by a number of binge eating episodes within a week: mild (1–2 episodes), moderate (4–7 episodes), severe (8–13 episodes), and very severe (14 or more episodes) [4]. BED increases the risk for obesity and somatic consequences associated with it such as heart diseases, hypertension, or type 2 diabetes [6]. Moreover, recurrent episodes of compulsive overeating are the reasons behind self-harm or suicidal attempts [2].

### **Functional mechanisms of BED – neuroimaging studies**

Usage of neuroimaging, especially functional magnetic resonance imaging (fMRI), allowed for evaluation of functional brain changes occurring in individuals with eating disorders [7]. BED may be defined as an impulsive and compulsive disorder associated with altered sensitivity to rewards and attentional bias directed towards food [1]. Studies indicate [1] an occurrence of lesions in corticostriatal loops in individuals with BED, similar to the ones observed in addictions to psychoactive substances, including altered function of the prefrontal cortex (PFC), orbitofrontal cortex (OFC) function, insular (IN) function, and ventral striatum (VS) function.

Results of neuroimaging studies and systemic reviews regarding the altered sensitivity to rewards in BED are inconsistent; most of them suggest hypersensitivity [8–11], while others hyposensitivity [12, 13] of the reward system. For instance, in an fMRI study by Schienle et al. [9], the exposure to pictures of highly caloric food triggered

an increased activation in medial orbitofrontal cortex (mOFC) in women with BED compared to other groups (i.e., healthy individuals, BN). However, there was no difference between the groups with regard to neuronal reactions to images triggering disgust [9]. In another pilot study [10], individuals with BED presented a stronger activation of the ventral striatum (VS) in response to food pictures than the control group. Then, a study conducted by Simon et al. [14] did not show significant differences between patients with bulimic eating disorders (i.e., BED and BN) and the healthy individuals with regard to responses to general reward processing (i.e., monetary rewards). Thus, a question arises whether changes in reward regions are specific for food or reflect general disturbances in reward processing.

Balodis et al. [12, 13] obtained results different from the above and showed a decreased sensitivity to rewards in BED. A diminished response in the reward anticipation phase appears to be consistent with the concept of reward deficit syndrome, which assumes that individuals with an initially lower level of neuronal activity of reward regions may engage in addictive behaviors or overeat in order to stimulate activity in these regions [13]. However, there is likelihood that exposure to food signals (i.e., stimuli specific to this disorder) instead of monetary ones as described in the above two studies could increase the activity in reward regions [12, 13]. Seemingly contradictory study results regarding the sensitivity of the reward system may result from changes associated with specific stages of the disorder [13]. Namely, initial hypersensitivity of the reward system may be diminished due to episodes of overeating with food that contain large amounts of sugar or fat [13].

A study of Balodis et al. [15], in which participants underwent fMRI during the Stroop test, suggests that obese individuals with BED are characterized by a decreased activity of cortical regions engaged in impulse control and self-regulation (i.e., ventromedial prefrontal cortex, inferior frontal gyrus, insular cortex) as compared to obese individuals without BED. Then, Karhunen et al. [16] used single-photon emission computed tomography (SPECT) and showed that exposure to a real portion of food in obese women with BED was associated with an increase in regional brain blood flow in the left hemisphere, especially in its prefrontal and frontal regions, as compared to obese women and the ones with normal body weight without BED.

The results of the studies indicate that individuals with BED show changes in corticostriatal loops, similar to the ones observed in impulsive, compulsive disorders [1]. The idea of compulsiveness refers to performing repetitive persistent activities that are not associated with the general aim and are maintained despite negative consequences [1]. Available literature proves that individuals with BED are characterized by attentional bias directed towards food [1, 17, 18] and impaired set-shifting characteristic for compulsiveness [1]. The researchers [19] suggest that individuals with an increased sensitivity to reward may perceive food as more satisfying; and then, an increased impulsiveness may make it difficult to resist the temptation to receive this reward. In the environment in which we are surrounded by high-calorie food, altered attentional bias directed towards food may be an important factor for maintaining the disorder [18].

## Emotion regulation in BED

Results of numerous studies prove that deficits in the processes involving regulation of emotions (emotional dysregulation) play a significant role in the occurrence and persistence of eating disorders, including BED [20–23]. According to Gross' definition [24], most often described in psychological literature, emotion regulation involves processes by means of which an individual can influence their own emotional states, and more precisely, emotions that they feel, how they experience them, and how they express them. Then, Linehan [25], an author of a biosocial theory, postulates that emotion dysregulation is one of the most serious problems in psychopathology, and more specifically in borderline personality disorder. She conceptualizes emotion regulation as an ability to: (1) inhibit dysfunctional and impulsive reactions to strong negative and positive emotions; (2) perform coordinated activities in order to maintain an external aim (regardless of the mood, if necessary); (3) self-control of physiological stimuli triggered by strong emotions; (4) regain focused attention while experiencing intense emotions.

It appears that a negative affect plays a key role in the discussed disorder [26]. Numerous empirical studies, applying various methodologies, prove that it directly precedes overeating episodes [27–31]. Having conducted a meta-analysis, Haedt-Matt and Keel [6] noticed that 69–100% of patients with BN and BED reported in retrospection that negative mood caused the episodes of overeating. Moreover, it was observed that BED patients experienced intensified negative affects during the days when the overeating episodes occurred [27, 31, 32]. A negative affect is a broad term that includes various components such as: depressive mood/sadness, anxiety, anger.

It could be believed that emotions other than sadness are also significant in this disorder [3]. Study results regarding anxiety in BED are inconsistent. Some researchers [3, 33] claim that it is less important than other emotions in this disorder, whereas others suggest that it is a stronger predictor for binge eating than depression [26, 32, 34]. While examining the mood that precedes overeating, Arnow et al. [34] obtained the following results with regard to particular variables: anger/frustration (42%), anxiety/excitement (37%), sadness/depression (16%), and grief (5%). Other researchers [e.g., 33] also emphasize the role of anger in BED.

Individuals with BED are less able to tolerate negative mood, when compared to healthy controls [3]. Moreover, a study of Crowther et al. (2001) [as cited in 3] reported that overeating women assessed their daily difficulties as more stressful than the control group that experienced a similar amount of odds. Then, Hilbert and Tuschen-Caffier [35] compared emotion dynamics in patients with BED and patients with bulimia (BN), showing that BED patients experience less negative mood as compared to bulimic patients, and overeat in response to a moderately negative condition.

So far, very few studies have explored the relationship between positive emotions and overeating in BED. Therefore, further research is needed to elaborate on it.

### **Theoretical BED models**

It is assumed that patients with the discussed disorder, who have difficulties with regulating negative emotions, use overeating as means of coping with these emotions and finding a relief [36]. Given that, several theoretical models that explain basic mechanism behind BED were offered. Below there are assumptions for two of the most common models found in literature.

An Affect Regulation Model, authored by Polivy and Herman, assumes that an increase in negative emotions triggers a binge eating episode, which is supposed to alleviate these emotions by using food as a specific ‘comforter’ and distractor [3]. According to this theory, an overeating episode results in an immediate affect improvement [37]. Overeating as a response to ‘unwanted emotions’ becomes a learnt reaction maintained by negative reinforcement [30].

Then, an Escape from Self-awareness Theory [38] propounds that overeating is motivated by a desire to escape from a negative self-perception (i.e., focus on personal failures; failing to meet high standards) [38]. According to this theory, overeating episodes are attempts to alleviate negative emotions by shifting attention from aversive self-awareness to direct surroundings (e.g., food) [38]. The model assumes alleviation of negative affect during overeating, and then, its subsequent decrease after the overeating episode, when self-awareness is re-established [6].

The above models have two common features: (1) negative emotions cause overeating; (2) overeating aims at alleviating these negative emotions within a short-term perspective (during the overeating episode) or a long-term perspective (after the episode) [3]. Experimental studies verified the assumptions of the above theories and brought only a partial confirmation.

According to our knowledge, there have only been a few experimental studies that would assess the connections between affect and eating behaviors in BED. Moreover, their results are inconsistent. For instance, Agras and Telch [39] and Chua et al. [29] conducted separate experiments in which female participants with BED were randomly ascribed to one of the groups: (1) negative or (2) neutral mood inductions, and then the amount of food consumed by them was assessed. The authors discovered that women from the group in which a negative affect was induced ate more during a taste test and experienced more frequent overeating episodes than the second group. The results of these studies were confirmed by a meta-analysis conducted by Cardi et al. [40], and a systemic review by Leehr [36] that came to a conclusion that a negative affect is a trigger for overeating in the group of individuals with BED as opposed to obese individuals who do not suffer from this disease. However, other studies did not report such a relation [e.g., 41].

### **Application of an ecological momentary assessment (EMA) in emotion regulation in individuals with BED**

A promising research method, adapted to verify the assumptions of the Affect Regulation Model in BED, is ecological momentary assessment (EMA) [6]. This study

method is based on repeated evaluation of emotions, behaviors or experiences made by a studied person on a portable electronic device (e.g., a smartphone) during the day (at random or at specific moments) and in the person's natural habitat [6]. Evaluations repeated in time allow for identifying both emotional states and contextual events preceding overeating episodes and occurring right after them [42]. EMA reduces recall bias because participants evaluate their current state (i.e., mood, behavior), not the state that occurred a few days or weeks ago [6]. In addition, this study method presents with high ecological validity, thereby overcomes the limitations of experimental studies [3].

Prevailing number of EMA studies identified the negative affect as a factor preceding binge eating episodes [20, 27, 28, 30, 31, 35]. The results of a meta-analysis conducted by Haedt-Matt and Keel [6] showed a greater negative affect preceding overeating episodes as compared with an average affect and the affect before normal eating, i.e., without episodes. Munsch et al. [31] obtained some interesting results; according to them, binge eating episodes occurring in BED do not result from an accumulation of aversive affect but from a sudden increase in negative emotions and tension that lead to immediate collapse of emotion and impulse regulation processes. These discoveries indicate a role played by impulsiveness, which is characterized by experiencing fast changing affect states, and is an important factor in BED [31].

According to Dawe and Loxton [19], impulsiveness may be conceptualized as a multidimensional construct including (1) sensitivity to reward and (2) tendencies towards rash-spontaneous impulsiveness. The researchers [8, 19] suggest that BED is a separate phenotype in the obesity spectrum that is characterized by an increased impulsiveness. Results of many studies prove that individuals with BED and non-clinical groups show an increased impulsiveness as compared with healthy individuals [8, 19, 43] that may be the basis for decreased control during the overeating episodes [1].

However, insufficient support was obtained for the second thesis of the Affect Regulation Theory, which says that an overeating episode eliminates the negative affect, what in turn maintains the stodging. Both Stein et al. [27] and Hilbert et al. [35] observed a considerable increase in aversive affect after the episodes of overeating in individuals with BED, and it was supported by a meta-analysis authored by Haedt-Matt and Keel [6]. Hence, these results did not confirm the Affect Regulation Model in BED and did not exclude the assumptions of the Escape from Self-awareness Theory (improvements in negative affect only while eating). Then, other researchers [30, 31] obtained results contradictory to the above. The study of Munsch et al. [31] showed a gradual and permanent mood improvement within several hours after the overeating episode. Similarly, Berg et al. [30] noticed a significant decrease in negative affect while they were examining emotions four hours after the uncontrolled eating.

It should be highlighted that some of the mentioned studies assessed the affect immediately after an overeating episode, whereas others – after a period of time. Thus, it is possible that discrepancies in the above results are associated with this fact. Moreover, it seems possible that overeating does not aim at regulating general negative affect but specific emotions [6]. Kenardy et al. [44] suggest that compulsive eating may decrease some of the components of negative affect (e.g., anger), while simultaneously increasing others (e.g., the feeling of guilt). This idea lines with a trade-off theory by Kenardy

et al. [44], which assumes that a specific negative emotional state is substituted with another one – less aversive. Therefore, it is necessary to conduct further research that would focus on affect changes associated with an overeating cycle, and identification of factors enhancing uncontrolled eating.

### **Strategies for emotion regulation in BED**

Every person, most often unconsciously, uses emotion regulation strategies in order to cope with everyday experiences [3]. Many models assume that individuals with BED use overeating as a way to decrease negative emotions due to the lack of more adaptive strategies of regulating them [45]. It means that patients with this disorder resort to stodging since they do not know another way of coping with their emotions. Researchers list various strategies for emotion regulation, however, for the purpose of this paper, we are going to discuss the most commonly used ones by individuals with BED.

An example of an adaptive strategy for emotion regulation is cognitive reappraisal defined by Gross as “changing a meaning of a situation in order to change emotional reaction to this situation,” or as a re-evaluation of the situation in order to modify its emotional significance [24, p. 195]. It is counted among antecedent-focused strategies since it is ‘triggered’ before an emotional reaction takes its final shape [24]. Learning the skill of cognitive reappraisal is an important part of behavioral therapies [45].

Then, suppression is counted among non-adaptive strategies for emotion regulation [46]. It is an example of a response-focused strategy since it appears when an emotion is fully formed [24]. This strategy may be a good short-term solution, however, it fails in the long-term perspective [3]. Suppression of unwanted thoughts leads to their intensification [47] and causes an increase in physiological stimulation (due to activation of sympathetic nervous system) [24]. Thus, the strategy has an opposite result. Chronic suppression prevents getting used to emotional stimuli, and simultaneously increases susceptibility to various types of psychopathological symptoms [45]. This strategy is listed as a common one among individuals with eating disorders, including BED [45].

Another non-adaptive strategy, strongly associated with psychopathology, is rumination [48]. It is based on constant passive focusing on thoughts on oneself and one’s situation in a self-critical and pessimistic way [49]. It consists of two dimensions: (1) reflexive rumination, and (2) brooding rumination [49]. Rumination is an attempt to understand and solve one’s own problems, however, it fails to generate results in the end [47]. The discussed strategy is commonly used by individuals suffering from depression, anxiety disorders and eating disorders [48].

Svaldi et al. [46] induced sadness in a group of women with BED and the control group by screening three films. Every film came with a different instruction: (1) watch the clip, (2) watch the clip and try to suppress emotions, (3) watch the clip and evaluate your emotional state again. Then, a tendency towards overeating was assessed by using a questionnaire. The results of the study showed that, as opposed to cognitive reappraisal, suppression leads to an increased desire to overeat and a decrease in the parasympathetic activation in women with BED, as compared to the healthy group.

The authors showed that individuals with BED less often use cognitive reappraisal and more often suppress their emotions as compared to the healthy group. Then, in another study [41], in which participants were asked to watch the film freely or suppress their emotions when watching it, no differences between the groups were observed with regard to a later calorie intake during the taste test. Therefore, the studies did not confirm the hypothesis that suppression of negative emotions leads to overeating in BED.

Then, results obtained by Dondzilo et al. [48], Wang et al. [49] and then by Smith et al. [47] indicated that rumination (i.e., passive comparison of current situation with desired standards) was an important process engaged in occurrence and persistence of BED psychopathology. The researchers suggest [48] that preoccupation with food and body shape/weight may be perceived as a type of rumination specific for eating disorders. Moreover, rumination seems to upkeep focus of an individual on failing to meet the desired standards associated with the body, while simultaneously enforcing the negative affect, which in turn leads to binge eating episodes [48].

Results of studies that compare eating disorders (anorexia – AN with BN and BED) with regard to the used strategies for emotion regulation are inconsistent. Both Aldao et al. [45] and Monell et al. [21] did not find significant differences between particular eating disorders in strategies used for emotion regulation. Similarly, the study of Svaldi et al. [23] did not present such differences, although the authors assume that the group with BED may be characterized by a more adaptive pattern of emotional regulation than the two other groups. On the other hand, some study results suggest [e.g., 22] that individuals with BED experience less difficulties with emotion regulation than people with other eating disorders. Nevertheless, there is likelihood that the differences between the groups result from the character of studies, and not the diagnosis [21].

### **Clinical implications of emotion dysregulation in BED**

BED treatment requires interdisciplinary approach and cooperation of many specialists, including a psychiatrist, a psychotherapist, a dietician, and a physician [2]. A psychotherapy is the standard form of treatment for emotion dysregulation in BED, with widely proven effectiveness for enhanced cognitive behavioral therapy (CBT-E) [50] as well as dialectical behavior therapy (DBT), so-called ‘third-wave’ [51]. Another effective form of psychotherapeutic intervention in BED is psychodynamic therapy [2]. According to this approach, the cause of eating disorders may be regarded as displaced traumatic experiences of a person or abnormal course of individuation-separation process [2]. Psychodynamic therapy aims to help the patient to process the ‘unwanted’ emotions and their cause, and to learn how to function in harmony with them [2].

The prognosis for patients with BED is more favorable than for other eating disorders, [4] whilst 50–80% of individuals who have undergone treatment reach clinical remission. The combination of pharmacological and psychotherapeutic interventions brings the most successful results [2]. Nevertheless, there is still a significant proportion of patients who are not helped by the above-mentioned treatments, which indicates the need for further clinical research with therapeutic implications.



## Conclusions

To conclude, negative emotions and deficits in their regulation play a significant role in BED. Individuals with BED apply non-adaptive strategies for emotion regulation more often than healthy individuals. They also present statistically significantly higher levels of emotion intensity, greater emotional lability with a tendency to depressive states. Interestingly, BED do not differ much in emotion regulation from other groups of eating disorders. Some authors [21, 23, 45] are in favor of the hypothesis that difficulties in emotion regulation are not associated with a specific diagnostic category. There seems to be a trans-diagnostic risk/maintenance factor that underlies various forms of psychopathology.

Table 1. Review of research on emotion regulation in BED

Authors, year of publication	Subjects (n)	Method	Results
Keating et al. (2019)	overeating women = 55	EMA 14 days; the assessment of depressive symptoms, emotion dysregulation, binge eating	Before binge eating episodes: depressive symptoms
Monell et al. (2018)	individuals with ED = 999 HC = 252	DERS, EDE-Q	Emotion dysregulation ED > HC
Brockmeyer et al. (2014)	women with ED = 120, including AN-R, AN-BP, BN, BED HC = 89	DERS, SCID, PHQ, BDI-II	Emotion dysregulation ED > HC; AN-R = AN-BP = BN; BED less severe ER difficulties in some domains than other ED
Aviram-Friedman et al. (2018)	OB women with BED = 13 OB women without BED = 29	Exposure to a food stimulus vs control stimulus fMRI	Food stimulus in BED: ↑ activity in brain areas: insula, anterior cingulate cortex, Brodmann areas 19 & 32, inferior parietal lobule, posterior cingulate cortex
Svaldi et al. (2012)	women with AN = 20, BN = 18, BED = 25, BPD = 15, MDD = 16 HC = 42	ERQ, ICARUS, DERS, AIM	Emotion dysregulation: ED, MDD, BPD > HC; BED slightly more adaptive pattern of ER than AN, BN Adaptive strategies of ER: ED < HC; non-adaptive ED > HC

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Stein et al. (2007)	women with BED = 33	EMA 7 days; the assessment of emotions, hunger, binge status	Negative mood and hunger: prebinge time > nonbinge time Postbinge time: ↑ negative mood
Greeno et al. (2000)	women with BED = 41 HC = 38	EMA 6 days; the examination of mood, appetite, setting of eating episodes	BED, before binge episodes: poor mood, feeling of poor eating control, craving sweets
Chua et al. (2004)	OB women with BED = 40	Experimental manipulation of mood (neutral vs. sad film) prior to a taste test	Sad film: ↓ mood; Negative mood: ↑ food intake
Berg et al. (2015)	OB individuals = 50	EMA 2 weeks; an examination of eating episodes, global negative affect, fear, guilt, hostility, sadness	Global negative affect and guilt: ↑ prior to binge eating episodes, ↓ following binge eating episodes
Munsch et al. (2011)	women with BED = 22	EMA 1 week; the assessment of positive and negative mood, tension	Before the binge eating episode: strong, immediate ↑ negative mood and tension Following binge eating: ↓ negative mood and tension (measured up to 4h after the episode)
Schulz and Laessle (2010)	OB women with BED = 40 OB women without BED = 44	SCID, BDI, STAI, DEBQ, The assessment of naturalistic food intake and mood before and after the episode (food diaries)	Negative mood: binge days > nonbinge days Comorbidity (in particular mood and anxiety disorders, substance-related disorders) BED > HC BED predictors: anxiety, emotional eating BED: ↑ Depressive symptoms: ↑ food intake
Zeeck et al. (2011)	Individuals with BED = 20 OB = 23 NWC = 20	mDES, EES, TAS-20, SCL-27 The assessment of emotions, alexithymia, overall psychopathology and binge eating	↑ Negative emotions, alexithymia BED > HC The emotion most often reported preceding binge eating: anger

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Arnou et al. (1992)	OB women with BED = 19	The interview The assessment of feelings before, during and following the episode; occurrence of restrictive food rules between binge episodes	Factors triggering binge eating: ↑ negative mood, diet violations Before the episode: ↑ anger, ↑ anxiety, ↑ depression After: ↑ guilt
Hilbert and Tuschien-Caffier (2007)	women with BED = 20 BN = 20 HC = 20	EMA The examination of mood, thoughts before, during and after binge eating episodes	Before the episode: ↑ negative mood, BN and BED BN > BED After the episode: ↑ negative mood
Agras and Telch (1998)	OB women with BED = 60	1) 14-hour period of caloric deprivation vs. no deprivation 2) Induction of negative vs. neutral mood before eating	Caloric deprivation and negative mood: ↑ number of binge eating episodes
Dingemans et al. (2009)	women with BED = 66	Induction of negative mood; g1) emotion suppression, g2) no additional instruction Taste task, BDI	↑ Depressive symptoms: ↑ food intake ↑ Negative mood: ↑ food intake Food intake: g1 = g2 After the episode of binge eating: ↑ mood
Schag et al. (2013)	OB individuals with BED = 25 OB without BED = 26 NWC = 25	Visual saccade tracking Food stimuli vs. neutral stimuli Experiment 1) assessment of reward sensitivity Experiment 2) assessment of disinhibition	Experiment 1) BED = OB = NWC initial fixation time BED: gazed longer at food stimuli Experiment 2) BED > OB, NWC difficulties in inhibiting first saccades regardless of the type of stimuli BED > OB, NWC difficulties in inhibiting second saccades toward food stimuli
Hege et al. (2015)	OB individuals with BED = 37 OB without BED = 19	Visual, food-related go/no-go task; fMRI BIS-11	BED: ↑ Attentional impulsiveness: ↓ response inhibition performance and hypoactivity in the prefrontal control network BED: ↓ food-specific inhibition

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Aldao and Nolen-Hoeksema (2010)	Graduates = 252	COPE, ERQ, RRS, WBSI, MASQ-SF, EDE-Q  The assessment of relationship between cognitive emotion regulation strategies and ED, anxiety, and depression	Psychopathology: maladaptive ER strategies > adaptive
Svaldi et al. (2010)	women with BED = 27 HC = 25	Induction of negative mood (sad film clip); g1) no additional instruction; g2) suppression; g3) cognitive reappraisal	Suppression BED > HC Cognitive reappraisal BED < HC BED: suppression: ↑ food craving, ↓ parasympathetic activation
Smith et al. (2019)	graduates = 263	EDDS, MASQ-SF, RRS, WBSI  The examination of association of brooding rumination and thought suppression with ED symptoms	↑ brooding rumination: ↑ binge eating among women
Donzilo et al. (2016)	Women (age: 17–24) = 119	RRS-ED  The examination of association between rumination, eating, weight, and ED symptoms	↑ Brooding rumination: ↑ ED symptoms, including binge eating and dieting
Wang et al. (2017)	OB individuals with BED = 237	SCID-I/P, EDE, RRS, WBIS  The assessment of relationship between rumination and ED psychopathology	Rumination: ↑ ED psychopathology

EMA – ecological momentary assessment; ED – eating disorders; BED – binge eating disorder;

AN-R – anorexia nervosa, restrictive type; AN-BP – anorexia nervosa, binge/purge type; BN – bulimia nervosa; HC(s) – healthy control(s);

ER – emotion regulation; OB – obese; MDD – major depressive disorder; BPD – borderline personality disorder; NWC(s) – healthy normal weight control(s); DERS – Difficulties in Emotion Regulation Scale; EDE-Q – Eating Disorder Examination Questionnaire; SCID – Structured Clinical Interview for DSM-IV; PHQ – Patient Health Questionnaire; BDI-II – Beck Depression Inventory II; ERQ – Emotion Regulation Questionnaire; ICARUS – Inventory of Cognitive Affect Regulation Strategies; AIM – Affect Intensity Measure; PANAS – Positive and Negative Affect Schedule; STAI – State-Trait Anxiety Inventory; DEBQ – Dutch Eating Behavior Questionnaire;

mDES – Modified Differential Emotions Scale; EES – Emotional Eating Scale; TAS-20 – Toronto Alexithymia Scale; SCL-27 – Symptom-Check-List; BIS-11 – Barratt Impulsiveness Scale; RRS – Ruminative Response Scale; WBSI – White Bear Suppression Inventory; MASQ-SF – Mood and Anxiety Symptom Questionnaire – Short Form; EDDS – Eating Disorder Diagnostic Scale; RRS-ED – Ruminative Response Scale for Eating Disorders; EDE – Eating Disorder Examination; WBIS – Weight-Bias Internalization Scale.

## References

1. Kessler RM, Hutson PH, Herman BK, Potenza MN. *The neurobiological basis of binge-eating disorder*. *Neurosci. Biobehav. Rev.* 2016; 63: 223–238.
2. Kucharska K, Kostecka B, Tylec A, Bonder E, Wilkos E. *Binge eating disorder – Etiopathogenesis, disease and treatment*. *Adv. Psychiatry. Neurol.* 2017; 26(3): 154–164.
3. Dingemans A, Danner U, Parks M. *Emotion Regulation in Binge Eating Disorder: A Review*. *Nutrients.* 2017; 9(11): 1274.
4. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders. 5th ed.*; Arlington: American Psychiatric Association Press; 2013.
5. *Klasyfikacja zaburzeń psychicznych i zaburzeń zachowania w ICD-10. Opisy kliniczne i wskazówki diagnostyczne*. Krakow–Warsaw: University Medical Publishing House “Vesalius”; 2000.
6. Haedt-Matt AA, Keel PK. *Revisiting the affect regulation model of binge eating: A meta analysis of studies using Ecological Momentary Assessment*. *Psychol. Bull.* 2011; 137(4): 660–681.
7. 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.
8. Schag K, Teufel M, Junne F, Preissl H, Hautzinger M, Zipfel S et al. *Impulsivity in binge eating disorder: Food cues elicit increased reward responses and disinhibition*. *PLoS One.* 2013; 8(10): e76542.
9. Schienle A, Schafer A, Hermann A, Vaitl D. *Binge-eating disorder: reward sensitivity and brain activation to images of food*. *Biol. Psychiatry.* 2009; 65: 654–661.
10. Lee JE, Namkoong K, Jung YC. *Impaired prefrontal cognitive control over interference by food images in binge eating disorder and bulimia nervosa*. *Neurosci. Lett.* 2017; 651: 95–101.
11. Bodell LP, Wildes JE, Goldschmidt AB, Lepage R, Keenan KE, Guyer AE et al. *Associations between neural reward processing and binge eating among adolescent girls*. *J. Adolesc. Health.* 2018; 62(1): 107–113.
12. Balodis IM, Grilo CM, Kober H, Worhunsky PD, White MA, Stevens MC et al. *A pilot study linking reduced fronto-striatal recruitment during reward processing to persistent bingeing following treatment for binge-eating disorder*. *Int. Eat. Disord.* 2014; 47(4): 376–84.
13. Balodis IM, Kober H, Worhunsky PD, White MA, Stevens MC, Pearlson GD et al. *Monetary reward processing in obese individuals with and without binge eating disorder*. *Biol. Psychiatry.* 2013; 73(9): 877–886.
14. Simon JJ, Skunde M, Walther S, Bendszus M, Herzog W, Friederich HC. *Neural signature of food reward processing in bulimic-type eating disorders*. *Soc. Cogn. Affect. Neurosci.* 2016; 11(9): 1393–1401.

15. Balodis IM, Molina ND, Kober H, Worhunsky PD, White MA, Sinha R et al. *Divergent neural substrates of inhibitory control in binge eating disorder relative to other manifestations of obesity*. *Obesity* (Silver Spring). 2013a; 21(2): 367–377.
16. Karhunen LJ, Vanninen EJ, Kuikka JT, Lappalainen RI, Tiihonen J, Uusitupa MI. *Regional cerebral blood flow during exposure to food in obese binge eating women*. *Psychiatry Res*. 2000; 99(1): 29–42.
17. Aviram-Friedman R, Astbury N, Ochner CN, Contento I, Geliebter A. *Neurobiological evidence for attention bias to food, emotional dysregulation, disinhibition and deficient somatosensory awareness in obesity with binge eating disorder*. *Physiol. Behav*. 2018; 184: 122–128.
18. Svaldi J, Tuschen-Caffier B, Peyk P, Blechert J. *Information processing of food pictures in binge eating disorder*. *Appetite*. 2010; 55(3): 685–694.
19. Giel KE, Teufel M, Junne F, Zipfel S, Schag K. *Food-related impulsivity in obese and binge eating disorder – a systematic update of the evidence*. *Nutrients*. 2017; 9(11): e1170.
20. Keating L, Mills JS, Rawana JS. *Momentary predictors of binge eating: An attachment perspective*. *Eat. Behav*. 2019; 32: 44–52.
21. Monell E, Clinton D, Birgegard A. *Emotion dysregulation and eating disorders – Associations with diagnostic presentation and key symptoms*. *Int. J. Eat. Disord*. 2018; 51(8): 921–930.
22. Brockmeyer T, Skunde M, Wu M, Bresslein E, Rudofsky G, Herzog W et al. *Difficulties in emotion regulation across the spectrum of eating disorders*. *Compr. Psychiatry*. 2014; 55(3): 565–571.
23. Svaldi J, Griepenstroh J, Tuschen-Caffier B, Ehring T. *Emotion regulation deficits in eating disorders: a marker of eating pathology or general psychopathology?* *Psychiatry Res*. 2012; 197(1–2): 103–111.
24. Szczygieł D. *Regulacja emocji a dobrostan. Konsekwencje wyprzedzającej i korygującej regulacji emocji*. In: Derbis R, Baka Ł, editors. *Oblicza jakości życia*. Czestochowa: Jan Długosz University Publishing House; 2014. p. 191–205.
25. Linehan MM. *Uzasadnienie treningu umiejętności w terapii dialektyczno-behawioralnej*. In: Linehan MM, editor. *Terapia dialektyczno-behawioralna (DBT). Trening umiejętności*. Krakow: Jagiellonian University Press; 2016. p. 32–33.
26. Rosenbaum D, White K. *The relation of anxiety, depression, and stress to binge eating behavior*. *J. Health Psychol*. 2015; 20(6): 887–898.
27. Stein R, Kenardy J, Wiseman CV, Douchis JZ, Arnow BA, Wilfley DE. *What's driving the binge in binge eating disorder? A prospective examination of precursors and consequences*. *Int. J. Eat. Disord*. 2007; 40(3): 195–203.
28. Greeno CG, Wing RR, Shiffman S. *Binge antecedents in obese women with and without binge eating disorder*. *J. Consult. Clin. Psychol*. 2000; 68(1): 95–102.
29. Chua JL, Touyz S, Hill AJ. *Negative mood-induced overeating in obese binge eaters: An experimental study*. *Int. J. Obes. Relat. Metab. Disord*. 2004; 28(4): 606–610.
30. Berg KC, Crosby RD, Cao L, Crow SJ, Engel SG, Wonderlich SA et al. *Negative affect prior to and following overeating-only, loss of control eating-only, and binge eating episodes in obese adults*. *Int. J. Eat. Disord*. 2015; 48(6): 641–653.
31. Munsch S, Meyer AH, Quartier V, Wilhelm FH. *Binge eating in binge eating disorder: A breakdown of emotion regulatory process?* *Psychiatry Res*. 2011; 195(3): 118–124.

32. Schulz S, Laessle RG. *Associations of negative affect and eating behaviour in obese women with and without binge eating disorder*. *Eat. Weight. Disord.* 2010; 15(4): 287–293.
33. Zeeck A, Stelzer N, Linster H, Joos A, Hartmann A. *Emotion and eating in binge eating disorder and obesity*. *Eur. Eat. Disord. Rev.* 2011; 19(5): 426–437.
34. Arnow B, Kenardy J, Agras WS. *Binge eating among the obese: a descriptive study*. *J. Behav. Med.* 1992; 15(2): 155–170.
35. Hilbert A, Tuschen-Caffier B. *Maintenance of binge eating through negative mood: a naturalistic comparison of binge eating disorder and bulimia nervosa*. *Int. J. Eat. Disord.* 2007; 40(6): 521–530.
36. Leehr EJ, Krohmer K, Schag K, Dresler T, Zipfel S, Giel KE. *Emotion regulation model in binge eating disorder and obesity – A systematic review*. *Neurosci. Biobehav. Rev.* 2015; 49: 125–134.
37. Polivy J, Herman CP. *Etiology of binge eating: Psychological mechanisms*. In: Fairburn CG, Wilson GT, editors. *Binge eating: nature, assessment, and treatment*. New York: The Guilford Press; 1993. p. 173–205.
38. Heatherton TF, Baumeister RF. *Binge eating as escape from self-awareness*. *Psychol. Bull.* 1991; 110(1): 86–108.
39. Agras WS, Telch CF. *Effects of caloric deprivation and negative affect on binge eating in obese binge-eating disordered women*. *Behav. Ther.* 1998; 29(3): 491–503.
40. Cardi V, Leppanen J, Treasure J. *The effects of negative and positive mood induction on eating behaviour: A meta-analysis of laboratory studies in the healthy population and eating and weight disorders*. *Neurosci. Biobehav. Rev.* 2015; 57: 299–309.
41. Dingemans AE, Martijn C, Jansen A, van Furth EF. *The effect of suppressing negative emotions on eating behavior in binge eating disorder*. *Appetite* 2009; 52(1): 51–57.
42. Schatten H, Allen KJ, Arney M. *Assessment of Emotion Dysregulation Using Ecological Momentary Assessment*. In: Beauchaine TP, Crowell SE. *The Oxford Handbook of Emotion Dysregulation*. New York: Oxford University Press; 2020.
43. Hege MA, Stingl KT, Kullmann S, Schag K, Giel KE, Zipfel S et al., *Attentional impulsivity in binge eating disorder modulates response inhibition performance and frontal brain networks*. *Int. J. Obes. (Lond.)* 2015; 39(2): 353–360.
44. Kenardy J, Arnow B, Agras WS. *The aversiveness of specific emotional states associated with binge-eating in obese subjects*. *Aust. N. Z. J. Psychiatry* 1996; 30(6): 839–844.
45. Aldao A, Nolen-Hoeksema S. *Specificity of cognitive emotion regulation strategies: A transdiagnostic examination*. *Behav. Res. Ther.* 2010; 48(10): 974–983.
46. Svaldi J, Caffier D, Tuschen-Caffier B. *Emotion suppression but not reappraisal increases desire to binge in women with binge eating disorder*. *Psychother. Psychosom.* 2010; 79(3): 188–190.
47. Smith KE, Mason TB, Anderson NL, Lavender JM. *Unpacking cognitive emotion regulation in eating disorder psychopathology: The differential relationships between rumination, thought suppression, and eating disorder symptoms among men and women*. *Eat. Behav.* 2019; 32: 95–100.
48. Dondzilo L, Rieger E, Palermo R, Byrne S, Bell J. *Association between rumination factors and eating disorder behaviours in young women*. *Adv. Eat. Disord.* 2016; 4(1): 84–98.
49. Wang SB, Lydecker JA, Grilo CM. *Rumination in patients with binge-eating disorder and obesity: Associations with eating-disorder psychopathology and weight-bias internalization*. *Eur. Eat. Disord. Rev.* 2017; 25(2): 98–103.

50. de Jong M, Schoorl M, Hoek HW. *Enhanced cognitive behavioural therapy for patients with eating disorders: a systematic review*. *Curr. Opin. Psychiatry*. 2018; 31(6): 436–444.
51. Davis LE, Attia E. *Recent advances in therapies for eating disorders* [version 1; peer review: 2 approved]. *F1000Research*. 2019; 8: 1693.

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