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Abstract

Emotional eating is commonly defined as the tendency to (over)eat in response to emotion. Insofar as it involves the (over)consumption of high-calorie palatable foods, emotional eating is a maladaptive behavior that can lead to eating disorders, and ultimately to metabolic disorders and obesity. Emotional eating is associated with eating disorder subtypes and with

abnormalities in emotion processing at a behavioral level. However, not enough is known about the neural pathways involved in both emotion processing and food intake. In this review, we provide an overview of recent neuroimaging studies, highlighting the brain correlates between emotions and eating behavior that may be involved in emotional eating. Interaction between neural and neuro-endocrine pathways (HPA axis) may be involved. In addition to behavioral interventions, there is a need for a holistic approach encompassing both neural and physiological levels to prevent emotional eating. Based on recent imaging, this review indicates that more attention should be paid to prefrontal areas, the insular and orbitofrontal cortices, and reward pathways, in addition to regions that play a major role in both the cognitive control of emotions and eating behavior. Identifying these brain regions could allow for neuromodulation interventions, including neurofeedback training, which deserves further investigation.

Keywords: emotional eating, emotion regulation, neuroimaging, therapeutic intervention strategies, stress, gut-brain axis

List of abbreviations

Abbreviation	Meaning
5-HT	Tryptophan
ACC	Anterior cingulate cortex
BED	Binge eating disorder
BLA	Basolateral amygdala
BMI	Body mass index
BOLD	Blood-oxygen-level-dependent
CRH	Corticotrophin-releasing hormone
DA	Dopamine
DEBQ	Dutch Eating Behavior Questionnaire
dlPFC	Dorsolateral prefrontal cortex
dmPFC	Dorsomedial prefrontal cortex
ED	Eating disorder
EEG	Electroencephalography
ENS	Enteric nervous system
fMRI	Functional magnetic resonance imaging
HPA	Hypothalamic-pituitary-adrenal
IFG	Inferior frontal gyrus
LPP	Late positive potential
NAcc	Nucleus accumbens
NE	Norepinephrine
NF	Neurofeedback

OFC	Orbitofrontal cortex
PCC	Posterior cingulate cortex
PET	Positron emission tomography
PFC	Prefrontal cortex
rs-FC	Resting-state functional connectivity
SMA	Supplementary motor area
tDCS	transcranial direct-current stimulation
TFEQ	Three-Factor Eating Questionnaire
vlPFC	Ventrolateral prefrontal cortex
vmPFC	Ventromedial prefrontal cortex
VTA	Ventral tegmental area

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1. Introduction

There has been a great deal of research in the field of eating behavior, as dietary decisions can deeply influence our health. Excessive sugar intake (refined carbohydrates) increases the risk of coronary heart disease [1] and is positively associated with increased risk of breast cancer [2]. In addition, excessive sodium intake, and diets lacking in fruit and whole grains are the leading dietary factors for death, with 11 million deaths (22% of all deaths among adults) across 195 countries [3]. The main causes of these diet-related deaths are cardiovascular disease, cancer, and type-2 diabetes. Unhealthy diet is thus a major health issue, justifying the need for improved dietary decisions. Over the past 20 years, a plethora of research studies have focused on how eating behavior is controlled by conscious but also nonconscious mechanisms. Many factors are involved, both intrinsic (e.g., biological and physiological signals) and extrinsic (external food cues, social and educational habits, etc.). Internal state (i.e., biological and psychological factors) plays an important role in the regulation of food intake, and is consequently involved in eating behavior and disorders (EDs). Individuals are not always rational in their daily decisions, and this also applies to eating behavior [4]. Additionally, emotions are significantly involved in decision-making processes [5] and can influence food choices [6]. *Emotional eating* refers to a behavioral response to the feeling of an emotion or an emotional state. This response affects eating behavior and can lead to increased or suppressed food intake [7]. *Emotional eaters* are defined as people who increase their food intake in response to negative (but also positive) emotions, in order to deal with a specific emotional state (i.e., to decrease a negative state or potentiate a positive one), rather than to fulfil a genuine physiological need for food [8]. Emotional eating includes emotional overeating and emotional binge eating [9,10]. An *overeating* episode refers to the consumption of a large amount of food within a short period

of time, while a *binge-eating* episode is characterized by overeating associated with a sense of loss of control [11]. Eating in response to emotions can thus become a habitual response to exposure to negative emotions and lead to pathological issues such as EDs (e.g. binge eating disorder, BED) [12], weight gain and obesity [13], as well as all the related comorbidities (e.g., cardiovascular disease, some forms of cancer). There is therefore a need to better understand the mechanisms involved in emotional eating, in order to prevent or attenuate this maladaptive behavior using interventional strategies.

Although many neurobiological models of eating behavior have been developed, incorporating homeostatic, cognitive and emotional factors [14–16], little is known about the *neural* mechanisms behind the emotions that influence eating behavior. Neuroimaging can provide insight into the brain mechanisms linked to emotional eating. In particular, electroencephalography (EEG) can provide high temporal resolution, and functional magnetic resonance imaging (fMRI) high spatial resolution and brain coverage, including deep brain regions. These two modalities therefore complement each other. The aim of the present review was to summarize the literature on the brain networks involved in emotional eating behavior, including recent studies that have coupled neuroimaging (MRI and EEG) investigations of emotion regulation with food tasks or assessments of emotional eating. Our goal was to provide a comprehensive understanding of emotional eating, with a broad and integrated overview of current knowledge of the neural pathways and mechanisms involved in the cognitive-emotional brain and a discussion of innovative prevention strategies.

2. Emotion processing and the cognitive-emotional brain

Emotion has been given various definitions in the many theories developed to explain emotion processing. A consensus has emerged, acknowledging its physiological, cognitive and behavioral dimensions. According to this consensus, an emotion consists of a complex set

of interactions among subjective and objective factors, and encompasses both neural and hormonal systems. These systems integrate affective experiences, generate cognitive processes, activate physiological adjustments, and lead to a behavior that is generally goal-directed and adaptive [17]. The function of emotion is to decouple a stimulus from the behavioral response, thus allowing for flexible adaptation to environmental contingencies [18]. *Emotion generation* takes the form of a situation-attention-appraisal-response sequence [19], and commonly begins with the perception of a stimulus within a context. The emotional importance of this stimulus is appraised by the individual, triggering an affective, physiological and behavioral response [18]. *Emotion regulation* refers to the process engaged to modify the experience or expression of this emotion, and can be studied at both neural and behavioral levels [20]. From a philosophical to a clinical perspective, advances in cognitive neurosciences, through the use of neuroimaging, have shed light on the major brain regions involved in emotion processing, be it for emotion generation or regulation. To provide an overview of the brain regions involved in these two aspects, we deal separately with the neural systems involved in generation or regulation in this review, even though they partially overlap (Fig. 1a) [21–23].

Emotion generation entails the activity of core limbic structures such as the amygdala, which is involved in the perception and encoding of stimuli that are relevant to current or chronic affective goals, and the nucleus accumbens (NAcc), a major component of the ventral striatum that is involved in learning which cues predict rewarding or reinforcing outcomes [21,24]. Other brain regions besides the limbic system also play a role in emotion generation: the periaqueductal gray, which is involved in the coordination of behavioral and physiological emotional responses [21,22,25], and cortical regions such as the ventromedial prefrontal cortex (vmPFC), which is involved in the integration of the affective valence of a specific stimulus elaborated within the amygdala and ventral striatum [24]. Moreover, because of its

role in the integration of somatosensory information such as ascending viscerosensory inputs from the body, the insula is also crucial for generating emotions [26,27].

Emotion regulation, also called *modulation of affect*, refers to the cognitive processes that allow individuals to cope with daily-life situations, depending on which emotions they feel, and when and how these are experienced [28]. Emotion regulation encompasses both *automatic* (also called *nonconscious* or *implicit*) and *controlled* (*explicit*) modulation of emotion activation, by affecting one or more aspects of the emotional sequence (situation, attention, appraisal, or response [29]). Regulation strategies have been divided into five categories: situation selection (e.g., behavioral disengagement), situation modification (e.g., problem solving), attentional control (e.g., distraction, concentration), cognitive change (e.g., reappraisal, acceptance), and response modulation (e.g., suppression, substance use, exercising and food preoccupation), leading to behavioral and physiological changes [28,30]. *Cognitive reappraisal* consists of the explicit modification of the self-relevant meaning of an emotion-inducing stimulus [25], and is the most commonly studied adaptive strategy [24]. Various brain regions are elicited by the different regulation strategies [31]. According to the model of the cognitive control of emotion [24], emotion regulation elicits the prefrontal cortex (PFC), particularly the dorsolateral prefrontal cortex (dlPFC) and posterior prefrontal cortex, as well as inferior parietal regions involved in cognitive processes such as selective attention and working memory. Emotion regulation also activates the ventrolateral prefrontal cortex (vlPFC), which contributes to the selection of goal-appropriate emotional responses and information from semantic memory in order to engage in a new stimulus-appropriate reappraisal [24,32]. The dorsomedial prefrontal cortex (dmPFC) plays a role in attributing and evaluating mental states (e.g., intentions) [20,24,33]. In addition, the anterior cingulate cortex (ACC) is involved in performance and conflict monitoring [26,34], and the orbitofrontal cortex (OFC) contributes to the evaluation of sensory stimuli based on individual needs and

goals [35]. Thus, consistent involvement of the PFC (left dlPFC, bilateral vlPFC, and dmPFC), bilateral parietal, left temporal brain regions and motor areas (supplementary motor area; SMA) has been reported in several emotion regulation meta-analyses [22,31,36]. In addition, generating an emotional response brings into play the ACC, NAcc, and insula [32]. The dorsal ACC is reported to mediate feelings of negative emotions such as aversion, fear and anxiety, in relation with the vmPFC [24,25,34], while the ventral ACC is involved in fear extinction [25,34]. Overall, emotion processing is driven by brain regions involved in perception (sensory), integration, valuation and cognition, encompassing all the stages of emotion generation and regulation. We can thus distinguish the brain regions involved in triggering affective responses (e.g., amygdala, ventral striatum, insula) from those involved in modulating affect (e.g., ACC, dlPFC, vlPFC, dmPFC, vmPFC) [26]. Nevertheless, the mechanism by which these brain areas interact, depending on the emotion regulation strategy, remains an active research topic [25]. One study found that activity of the amygdala and anterior insula decreased during reappraisal and increased during suppression, whereas activity of the PFC increased with both strategies [37]. Furthermore, downregulation of the amygdala and striatal activity during emotion regulation are probably brought about by increased activity of prefrontal areas [22,24,32,38]. Conversely, reduced recruitment of the dlPFC and vlPFC, as well as greater amygdala activity, have been observed during reappraisal in mood and other mental disorders [39]. This suggests that unsuccessful emotion regulation can be explained by insufficient recruitment of the prefrontal networks involved in top-down regulation, resulting in decreased reactivity of limbic structures. In line with this hypothesis, in an fMRI-based meta-analysis related to cognitive reappraisal, patients with mood or anxiety disorders exhibited reduced recruitment of the frontoparietal network (posterior cingulate cortex, PCC, dmPFC, angular gyrus and vlPFC), compared with healthy controls [40]. In addition to the task-based fMRI approach, resting-state functional connectivity (rs-

FC) can provide insights into the brain networks that underlie emotion processing, by measuring the temporal correlations between spontaneous blood-oxygen-level-dependent (BOLD) signals and spatially distributed brain regions. Accordingly, effective regulation of negative emotions, as well as increased self-control, have been shown to be correlated with enhanced functional connectivity between the dlPFC and amygdala [41]. Regarding the dispositional use of regulatory strategies in healthy individuals, rs-FC between the left basolateral amygdala (BLA) and left anterior insula, as well as between the right BLA and SMA, have been found to be inversely correlated with the reappraisal scores of the Emotion Regulation Questionnaire [42,43]. In the same study, suppression scores were positively correlated with rs-FC between the right BLA and dACC, and negatively correlated with rs-FC between the left centromedial amygdala and BLA [43]. In another study, successful reappraisal (corresponding to reduced negative affect ratings after emotion regulation) was negatively correlated with rs-FC between the right amygdala and brain clusters in the medial PFC and PCC. This successful reappraisal was also negatively correlated with rs-FC between the bilateral dlPFC and posterior regions of the occipital cortex and ACC [44], suggesting that reduced bottom-up connectivity may also facilitate emotion regulation. In patients diagnosed with major depressive disorder, a meta-analysis of rs-FC studies highlighted hypoconnectivity between the frontoparietal network (especially the dlPFC) and the right PCC, as well as between the affective network (especially the amygdala and NAcc) and the medial PFC, compared with healthy individuals [45]. In obese women, reduced vmPFC activity (BOLD response) was observed during reappraisal, and vmPFC activity was negatively correlated with self-reported emotion regulation difficulties [46]. These patients also displayed decreased rs-FC between the vmPFC and left temporal pole [46]. Although the exact mechanism of emotion regulation between higher level cognitive structures (PFC, temporal poles) and subcortical limbic structures (e.g., amygdala) has not been yet identified, efficient

recruitment of brain regions involved in emotion regulation seems key to adaptive emotion processing and emotional response in daily life. Disruption of those pathways may affect the ability to cope with negative emotions, especially in patients diagnosed with mood disorders. Therefore, maladaptive emotion regulation appears to be a key component of a wide range of affective disorders and disordered eating behaviors.

Neuroimaging research on the mechanisms involved in the cognitive control of emotions has highlighted the interplay between emotion and cognition, thus undermining the traditional view of a clear separation between these two components [47–50]. Emotions can influence perception, attention, working memory and other cognitive aspects (e.g., cognitive performance) [18]. Conversely, cognitive processing is needed to elicit emotional responses, depending on the type of regulation strategy [48]. This relation can be transposed to decision-making, which also involves cognitive processing and emotional response [5]. All the cognitive processes mentioned above are also involved in eating behavior, as attention, working memory, cognitive control and decision-making all affect the nonhomeostatic regulation of food intake. Neuroimaging can help investigate the extent to which the interaction between emotion and cognition is involved in eating behavior and how it may influence food intake and preferences. To address this question, we discuss the role of emotion in the control of food intake in the following section.

3. Eating behavior and main implications of emotion processing

3.1. Similar brain regions involved in eating behavior and emotion processing

The usual schematic vision of eating behavior regulation features two control loops: *homeostatic regulation*, in which the hypothalamus, especially the arcuate nucleus, plays a major role in integrating metabolic signals and regulating the hormonal system; and *nonhomeostatic regulation* (also called *hedonic regulation*), involving corticolimbic

structures and networks that process both environmental and cognitive factors [51,52]. There is strong evidence that these loops are interconnected and capable of influencing each other [53]. However, the nonhomeostatic neural pathways are less well understood, and studies involving paradigms with ingestive behavior have yielded contradictory results in extra-hypothalamic brain regions [52]. The nonhomeostatic loop involves (Fig. 1b) the amygdala for behavioral salience and stress responses, the hippocampus and PCC for their role in memory and learning in the context of eating behavior, and the dlPFC for its function in goal-directed behavior [54]. In addition, the insula plays a role in interoception (perception of sensations from inside the body), homeostasis, and the integration of sensory signals across modalities, while the OFC is regarded as a secondary gustatory cortex [55]. The insula (taste-responsive neurons) sends projections to the OFC and is involved in the perceived pleasantness of taste [52]. The vmPFC also plays a key role in the valuation system, and therefore in food decision-making [56], as well as in conditioned motivation to eat [57]. Both the OFC and vmPFC are involved in assigning incentive motivational values to food stimuli [54]. The striatum is activated during exposure to food cues, with striatal subregions being differently involved: the NAcc mediates reward prediction, while the caudate nucleus is involved in feedback processing, and the putamen in the mediation of habitual behavior [54]. Moreover, the ventral tegmental area (VTA) is the starting point of dopaminergic neurons that project toward the reward system and activate it. This catalogue of brain regions highlights the fact that food perception and eating share brain regions involved in perception, cognitive control, reward, and more especially emotion processing (Figs 1a and 1b).

Given that similar brain structures are involved in the regulation of emotion and eating behavior, it is important to investigate these underpinnings using relevant food-related tasks in different emotional conditions. A combination of physiological, behavioral and psychological measures is required to grasp the multiple dimensions of the participants'

reactions. Examination of the literature on the neurobiology of food intake control shows that emotions are not always considered, and when they are, they are usually attributed to the amygdala. As mentioned above, emotion and cognitive processes closely interact to regulate behavior, and these circuits encompass many extralimbic structures, not just the amygdala. This raises the question of how these cognitive processes are involved in emotion processing in the context of eating behavior, and how this cognitive-emotional brain influences food intake, in both normal and pathological contexts. As indicated above, impaired emotion regulation can lead to maladaptive behavior, and this also applies to eating behavior. For example, compared with healthy controls, individuals with obesity [58], a condition that has high comorbidity with affective disorders such as depression, have been found to have reduced dlPFC activity in response to a meal [59–61].

3.2. Nonhomeostatic factors, food reward and emotional eating

The regulation of eating behavior results from a balance between hunger and satiety, and is governed by both metabolic and nonmetabolic factors, such as exposure to food cues, cognitive and emotional state, and personal and cultural beliefs. These factors can also indirectly influence the homeostatic regulation of food intake, especially through reward and cognitive processes. These processes mainly take place in corticolimbic structures such as the PFC, amygdala and ventral striatum, and can trigger food intake through an executive cortical decision, independently of physiological needs [15]. Here, we focus on the nonmetabolic factors influencing eating behavior that are involved in top-down processes. Prefrontal areas are involved not only in emotion regulation, but also in executive functioning (including inhibitory and attentional control, as well as cognitive flexibility) and the cognitive control of eating behavior [62], which it does by modulating appetitive regions (i.e., OFC, ventral striatum, insula, and amygdala/hippocampus complex) [51]. Furthermore, there is an interplay between emotion and food intake: emotions can influence food choices and, conversely, food

283 intake can influence emotional state, owing to the impact of nutrients on food choices and
284 mood [63–65].

285 In this review, we chose to focus on the role of emotions in eating behavior, rather
286 than the role of specific foods in emotions per se. Given the increased accessibility of highly
287 palatable and energy-dense foods, nonhomeostatic regulation may overwhelm the homeostatic
288 regulation of food intake. Palatability and pleasantness are powerful determinants of food
289 intake [66]. The latter is mediated by the mesocorticolimbic pathway, including the VTA,
290 which sends projections to limbic areas (including the NAcc) and the PFC. Reciprocally, the
291 PFC sends projections back to the NAcc and VTA [67]. The reward system is a central
292 component of the nonhomeostatic regulation of eating behavior, and is mediated through
293 dopamine (DA) release. A concentration-dependent increase in DA has been reported in the
294 NAcc during oral sucrose stimulation in rats [68] and in correlation with meal pleasantness in
295 the dorsal striatum of healthy individuals, determined with ^{11}C raclopride positron emission
296 tomography (PET) imaging [69]. Enhanced striatal DA release has also demonstrated in BED,
297 using a food stimulation paradigm in [70]. Taken together, these data support the reinforcing
298 effect of food through DA release, and suggest a role in disordered eating behaviors.

299 The reward system may be particularly involved in emotional eating as defined above,
300 where eating may reduce anxiety [71], and eating *comfort foods* (see following section) may
301 blunt the response to acute stress [16]. This is supported by Macht (2008)'s theory, based on
302 hedonistic mechanisms and distraction conveyed by eating palatable foods, in which the
303 immediate positive affective reactions can diminish the impact of stress. This phenomenon
304 may be involved in all individuals' regulation of daily emotions [7]. However, the association
305 between affect and eating comes from research on obesity [72,73], where emotional eating to
306 reduce anxiety is believed to drive compulsive overeating and thus promote obesity [71].
307 Healthy normal-weight individuals may actually be more prone to either increase or suppress

food intake in response to emotion than obese individuals [7,71], possibly depending on emotional features [7]. A recent study showed that normal-weight individuals can increase food intake in response to negative emotions and that emotional overeating is negatively correlated with alcohol consumption, suggesting that different strategies can be adopted to cope with negative emotions [74]. Eating in response to negative emotions is problematic in terms of public health, as the chosen foods are often characterized as unhealthy. Commonly acknowledged to affect food intake and to be involved in eating behavior [14,15,63], emotion processing has been studied in the context of EDs [51,62,75] such as obesity and/or BED [12,76–78], but has been poorly investigated in the context of normal eating behavior. Research has shown that patients with EDs are more predisposed to experience alexithymia (i.e., difficulty identifying and describing feelings) [79], maladaptive emotion regulation [62,75,78], and deficits in emotion differentiation, which is defined as the tendency to experience vague affective states rather than well-defined emotions [80]. They are also more predisposed to dysfunctional processing of emotions related to food information [81]. In this study, reduced control over emotions and reduced attraction to food pictures were mediated by negative affect, supporting the predictive role of negative affect in ED symptomatology [81]. The use of inappropriate emotion regulation strategies in response to the situational demand is a feature of various psychiatric disorders [82], suggesting that emotional eating may reflect a dysregulation of emotion processing in patients with EDs. As emotion dysregulation can be involved in disordered eating behavior, there is growing interest in the assessment of emotion regulation in the context of eating control. Recent studies have focused on *food craving*, which refers to the urge to eat. According to Giuliani and colleagues (2015), food craving can be regarded as an affective state and can be modulated through regulatory strategies such as cognitive reappraisal [83]. Over the past decade, the number of fMRI neuroimaging studies related to reappraisal strategies in the context of food craving has

increased, providing information about the neural substrates involved [84,85]. In patients with EDs, emotion regulation strategies have also been investigated with fMRI [86]. Despite this growing body of literature, it is still difficult to predict whether emotions will trigger or suppress food intake, and a proper experimental setup needs to be implemented to answer this question.

3.3. Toward a more integrated view of emotion, HPA axis, gut microbiota, and neuroimmune interactions

Physiological and behavioral interactions between stress and food intake have been extensively studied in animals. *Stress* can be defined as a cascade of physiological events that translate an initial stressor into a behavioral response. It entails the activation of the neuroendocrine hypothalamic-pituitary-adrenal (HPA) axis, which in turn leads to the increased glucocorticoid synthesis and glucose availability needed to fuel the metabolic demands of stress responses [16]. The neuroendocrine neurons in the hypothalamus are involved in this process: the corticotrophin-releasing hormone neurons of the paraventricular nucleus stimulate the secretion of the adrenocorticotrophic hormone by the anterior pituitary gland, which then stimulates cortisol secretion by the adrenal gland [87]. Stress can affect food intake through different interactions with the central nervous system and energy homeostasis. While acute stress can have either anorexigenic or orexigenic effects, depending on the individual, chronic stress is liable to induce weight gain through metabolic changes, independently of diet [88]. However, the question of whether stress increases or reduces food intake depends on various parameters [89], including sex/gender, duration of the stressor, food accessibility, and macronutrient quality [16]. Eating under stress has behavioral and psychological effects that are usually expected by the individual, such as stress relief through a reward signaling effect. The selected food is often characterized as *comfort food*, referring to highly palatable food items. *Stress eating* has been investigated in several animal studies,

including one in which sucrose-fed rats were found to secrete less corticosterone after acute stress [90–93]. The BLA is clearly essential for dampening stress, as sucrose-fed rats with bilateral lesions in the BLA had normal corticosterone levels in response to stress [90], whereas nonlesioned sucrose-fed rats exhibited increased structural plasticity (as demonstrated by the increased expression of genes related to structural plasticity) in this limbic region [90]. These results suggest that stress relief can be partly mediated through the reward system, indirectly promoting synaptic remodeling [16,90]. However, it is unclear whether eating comfort foods reduces stress by blunting HPA axis activation, stimulating the dopaminergic reward system, or both [16]. In any event, this phenomenon has important implications for weight issues. Overweight and obesity have been shown to be associated with emotion and stress-related eating [94], suggesting that overeating to cope with stress is a facet of emotional eating, just like overeating to cope with emotions. As emotional eaters are more likely to experience emotion recognition and management difficulties, eating may serve as a fallback. In addition, emotional eating has been shown to mediate the association between depression and obesity in young adults [95]. A similar observation was reported in a prospective cohort study, where emotional eating predicted a greater increase in body mass index (BMI) associated with shorter sleep duration, which itself is considered to be a stressor [96]. Regarding cortisol reactivity, female students with high emotional eating scores and blunted cortisol reactivity were found to consume more kilocalories after a stress induction task [97].

At the cellular and behavioral level, the stress response comes in two phases (acute and delayed), allowing the individual to react and adapt fully to the stressor. This requires increased attention, as well as enhanced activity of brain regions involved in both emotion processing and simple behavioral strategies [87]. However, repeated stress exposure may disrupt the balance between these phases and lead to maladaptive behavior in response to

stress events [98]. Adaptive habituation to repeated stress exposure may depend on the emotion regulation strategy used. In the context of repeated stress, a greater decrease in the cortisol level (reflecting HPA axis habituation) has been associated with the habitual use of cognitive reappraisal, but not emotion suppression, suggesting that habitual reappraisal plays an important role in the adaptation of the HPA axis to stress [99]. Increased cortisol may also promote effective emotion regulation in healthy individuals, either by facilitating attentional shifting [100] or by enhancing vlPFC activity and decreasing right amygdala activity [101]. By contrast, in depression or bipolar disorder, cortisol reactivity has been found to predict reduced recruitment of frontoparietal and striatal brain regions during emotion processing, these regions being involved in emotional salience and cognitive control [102]. In addition, higher low-grade inflammation measured from a blood sample has been associated with lower rs-FC within the emotion regulation network [103]. This network encompasses the inferior frontal gyrus (IFG), middle temporal gyrus, and precentral gyrus, as well as the central executive network, which connects the dlPFC and posterior parietal cortex areas [103]. Taken together, these results highlight the relationships between stress, systemic inflammation, metabolic and affective disorders, and obesity. The inability to adapt to repeated stress is associated with systemic inflammation and metabolic disorders such as diabetes and cardiovascular diseases [87,104]. Additionally, HPA axis overstimulation is known to be involved in visceral adipose tissue deposition and weight-related concerns. One example is Cushing's syndrome [105], which interestingly is closely associated with major depressive disorder [106]. Obesity and mood disorders share similar pathophysiological mechanisms, such as altered HPA axis activity and modulation of chronic low-grade inflammatory response [107,108]. The HPA axis regulates various processes, including modulation of immune functions and emotion processing, through glucocorticoid synthesis [109]. Moreover, there is a bidirectional relationship between the gut and the brain, involving endogenous

communication through microbiota factors, cytokines and hormones, and neural communication through the sympathetic, parasympathetic and enteric nervous systems, vagus nerve, and dorsal root ganglia [110], illustrating the complex gut-brain interaction. These results therefore highlight the possible interaction between neural and neuro-endocrine pathways (HPA axis) that may affect emotion processing, and should therefore be considered in the context of emotional eating.

In addition, the microbiota has attracted a great deal of interest over the past decade, with many studies highlighting its role in the gut-brain axis and the association between microbiota changes and behavioral and physiological modifications [111]. With the use of antibiotics and probiotics in animal models and the use of germ-free animal models, many studies have shown that behavioral traits and mental illnesses (e.g., depression) may partly depend on microbiota diversity and metabolic activity [112]. The microbiota appears to interact closely with the immune system, through immune modulation [112,113]. In addition, in neuropsychiatric disorders, alterations in immune homeostasis due to host–microbiota interactions may involve changes in HPA axis activity that could be activated by pro-inflammatory cytokines [114]. Moreover, the microbiota can affect neurotransmitter content [114] through direct synthesis, in particular serotonin from tryptophan (5-HT) [110], or catecholamine (norepinephrine and DA [115]) bioavailability. These monoaminergic neurotransmitters (5-HT and DA) play a major role in brain circuits involved in the regulation of mood, reward and food intake [110,116] and their dysregulation is involved in neuropsychiatric pathologies (e.g., depressive disorders). It is therefore not surprising that gut microbiota dysbiosis may be involved in mood disturbance and behavioral changes, through changes in both immune response and neurotransmission.

Many factors influence gut microbiota diversity, such as medication and poor-quality diet. Mental illnesses such as depression are associated with poor-quality diet and obesity

[117]. Therefore, emotional eating, which may trigger intake of comfort foods, may potentially affect gut microbiota, owing to harmful eating habits involving the consumption of palatable food with a high fat and/or sugar content and low fiber content. The Western diet, characterized by a low fiber content, results in reduced short-chain fatty acid synthesis from microbiota fermentation, which is known to modulate inflammation [118]. This type of diet is also associated with alteration of the gut microbiota ecosystem [119]. Stress can also affect the gut microbiome [120]. As illustrated in Panduro et al. (2017)'s review, negative emotions arising from alterations in the energy balance can induce chronic activation of the HPA axis, resulting in higher cortisol levels [121]. The latter may induce dysbiosis, leading to altered intestinal membrane permeability and activation of pro-inflammatory processes [121]. Additionally, chronic inflammation can lead to depressive-like behavior in mice [122]. These data suggest that an unhealthy diet can induce dysbiosis and inflammation, which in turn reinforce the onset of negative emotions, as altered gut microbiota can influence mood, thereby reinforcing the emotional eating behavior. Although an increasing number of studies have been investigating the relationship between EDs and microbiota homeostasis [123], specific gut microbiota profiles have not yet been described in emotional eaters. Moreover, it is important to bear in mind that there is a direct bidirectional interaction between the enteric nervous system and the brain. Appositely referred to as a *gut feeling*, this relationship engages various effector pathways that play an important role in feeling states and executive functioning [124]. These results suggest that the way stress interacts with emotion control and emotion regulation processes depends on the type of emotion regulation strategy used, the nature of the stressor (e.g., acute, delayed, or chronic), HPA activation, and individual health status (e.g., immune status, microbiota diversity, affective disorders), not forgetting hormonal effects (sex/gender) and age. The complex interaction between gut and brain in this context

justifies merging physiological (e.g., neuroendocrine) and psychological approaches into a holistic approach to investigating and preventing emotional eating.

4. Brain imaging studies in the context of eating behavior and emotions

Structural MRI, fMRI, resting-state MRI and EEG have all been used to investigate eating behavior in brain imaging studies. These are described in the following sections according to their experimental design. In the first section, brain imaging studies assessing emotional eating through questionnaires are reviewed, and the resulting brain correlates are discussed. The second section deals with studies coupling an emotion-induction task with food information processing. The third section focuses on brain imaging studies of stress and the link between emotional eating and coping with stress. The fourth and last section focuses on emotion regulation tasks in the context of food craving. Specific details of each study (sample, paradigm/task used, major findings, etc.) are reported in Tables 1 and 2.

4.1. Neural correlates of emotional eating

Emotional eating is commonly assessed with questionnaires, such as the Dutch Eating Behavior Questionnaire (DEBQ) [125], which evaluates cognitive restriction, externality and emotion in the context of eating behavior, and the Three-Factor Eating Questionnaire (TFEQ) [126]. The Weight-Related Eating Questionnaire combines items from the DEBQ and TFEQ to measure routine and compensatory restraint, external eating, and emotional eating related to weight loss [127]. The emotional eating items in these questionnaires assess the tendency to eat in response to negative emotions. Other items assess the level of emotions, the frequency and amount of food intake [128], or the intensity of the desire to eat [8]. Neuroimaging studies investigating eating behavior, including emotional eating behavior, are scarce, but have nonetheless brought new insights into the neural correlates of emotional eating. Some have not reported any link between brain structures [129] or brain activation

(BOLD response) [130] and emotional eating scores, but others have. In one study featuring a Go/NoGo paradigm with high- and low-calorie food pictures, the emotional eating score was positively correlated with left dlPFC and left insula activity in the High Go / Low NoGo condition, and with left dlPFC activity in the Low Go / High NoGo condition [131]. As the dlPFC is involved in self-control, as well as in goal-directed behavior, it is hardly surprising that its activity was correlated with emotional eating in a paradigm requiring inhibitory control. This suggests that eating and emotional processing share a common cognitive process. In a second study, the emotional eating score was correlated with the activity of (i) the left insula in lean participants, (ii) the amygdala and OFC in patients with type-2 diabetes, and (iii) the right insula in obese individuals [132]. In line with this observation, the presentation of high- versus low-calorie food pictures highlighted a positive correlation between emotional eating and left insula activity in healthy individuals [132]. These results suggest that in both healthy and obese individuals with or without metabolic disorders, higher emotional eating scores are associated with increased responses in appetite and reward brain regions that are also involved in emotion processing, specifically the OFC and insula. In a third study, participants' emotional eating score when consuming a milkshake was negatively correlated with the activity of the bilateral putamen and caudate, as well as the left insula and OFC [133]. Taken together, these results show that the same brain regions are differentially correlated with emotional eating, depending on the sensory demands (watching or tasting). Although these studies should be interpreted with caution, owing to many differences in terms of experimental paradigms, the OFC and insula appear to be relevant structures. The OFC is closely involved in the representation of pleasantness of reward and the attribution of affective value to food, and also plays a role in emotion, as food smell and taste can elicit emotional states [55]. In the specific context of emotions, the role of the OFC is to represent the reward value of goal-directed behavior through the learning of the stimulus-reinforcer

association [35]. According to Rolls (2019), OFC activation is correlated with the subjective emotional experience of affective stimuli, and OFC lesions have been shown to alter emotion-related learning, emotional behavior, and subjective affective state in macaques [35]. In addition, according to the model of cognitive control of emotion [20], the OFC and vmPFC are central to the evaluation of emotional value according to the context. This evaluation guides the selection of the most appropriate action in response to the stimuli. The functions of the insula (i.e., interoception, taste processing as primary gustatory cortex, and integration of multisensory signals) are central to food perception [54]. Owing to its bidirectional connections with many other regions (frontal lobe, subcortical regions, parietal and temporal cortices), the insula is also involved in emotion generation and modulation [26]. Impaired visceral interoceptive activity in the dorsal mid-insula has been observed in patients with anorexia nervosa [134], with heightened insula activity during anxious rumination. It has been suggested that impaired insular activity plays a key role in ED physiopathology, on account of its role in taste interoception, taste processing, the cognitive control network, emotion regulation, and body-image distortion [135]. Consequently, both the OFC and insula are liable to play an important role in emotional eating, and deserve particular consideration in eating behavior studies.

As demonstrated by Herwig et al. (2016), food pictures can induce appetitive emotions [136]. Thus, correlations between the passive viewing of food pictures and emotional eating may be related to the emotional response to food information processing. This highlights the need to devise paradigms that induce an emotional state in order to mimic *ecological* situations. Moreover, the questionnaires used may not accurately measure emotional eating behavior per se [137,138]. It has been suggested that emotional eating is a behavioral trait, consisting of a combination of behavioral characteristics that are individually predictable and

stable across time and situations, possibly forming part of a more general concept of low self-control and high motivation to eat [137].

4.2. Emotion induction during food information processing

In a study featuring negative versus neutral mood induction conditions, emotional (vs. nonemotional) eaters were found to have heightened activity in the (i) left parahippocampal gyrus in response to the anticipated receipt of a milkshake, and (ii) right pallidum in response to the actual receipt of a milkshake while in a negative mood. Across mood conditions, they were found to have greater activity in the left caudate nucleus and pallidum during milkshake consumption [139]. Overall, emotional eaters had greater activity in brain regions involved in food cue processing and reward, supporting the idea that food plays a rewarding role and can therefore alleviate or attenuate negative mood states. In a study assessing the neural processing of food stimuli in different emotional contexts, researchers observed greater activity in the lateral OFC and occipital lobe after negative emotional priming [140]. In a positive priming condition, there was also increased activity in the lateral OFC and occipital pole, as well as in the insula and amygdala. Amygdala activity was greater in the neutral and positive conditions than in the negative condition, possibly reflecting decreased food salience when individuals are in a negative state [140]. Two other studies used EEG recordings to assess emotional eating while viewing food pictures in an emotional paradigm [141,142]. The first of these demonstrated higher amplitude of the late positive potential (LPP) in parietal-occipital brain regions in the high emotional eating group, independently of mood condition, reflecting the high motivational relevance of food [141]. The second study investigated event-related potential responses in a food choice paradigm in the context of emotional eating [142]. While there was no effect of emotional eating on the parietal-occipital P300 amplitude reflecting motivated attention to foods, unrestrained participants with high reactivity had increased P300 amplitudes in response to food images in the negative

555 condition. By contrast, highly restrained participants with low reactivity had decreased food-
556 specific P300 amplitudes in the negative condition. These results are consistent with current
557 theories of emotional eating, according to which weak emotional states are likely to trigger
558 compensatory appetitive attentional mechanisms, whereas highly intense emotional states
559 reverse this pattern [142]. Emotional reactivity may therefore be an independent moderator of
560 emotional overeating.

561 **4.3. Stress and emotional eating**

562 Stress can affect food intake by reducing or increasing eating desire and/or hunger.
563 Few studies have examined the relationships between biological markers of stress and
564 emotional eating, and the neural correlates are still unknown. To date, only one study (in
565 adolescents with different BMIs) has combined the investigation of emotional eating and
566 stress with fMRI [143]. In this study, positive associations were reported between salivary
567 cortisol levels and the functional connectivity of mesolimbic brain regions, as well as between
568 the lateral hypothalamus and the NAcc, and between the lateral hypothalamus and midbrain,
569 in the excess-weight group. There was also a positive association between the emotional
570 eating score and connectivity in the lateral hypothalamic-midbrain network, but again only in
571 the excess-weight group [143]. The hypothalamus is a core area for the homeostatic control of
572 eating behavior and is closely connected to neural circuits involved in emotional behavior. In
573 particular, regarding the neural mechanisms behind the emotional regulation of homeostatic
574 eating, the lateral hypothalamus has bidirectional connections with several brain regions
575 involved in emotion processing, including the amygdala, VTA and NAcc [144]. This
576 highlights the role of the lateral hypothalamus in the modulation of VTA activity. Disruption
577 of this circuitry is associated with changes in mood and emotions, although the functional
578 interactions between hypothalamic circuitry and the mesolimbic reward pathways have yet to
579 be determined [144]. Taken together, these results suggest that stress may affect specific brain

networks in individuals with weight issues. Whether this is a cause or a consequence of their excess weight has yet to be determined. In a 3-year longitudinal study in adolescents, higher negative affect and stressful events were not correlated with the activity of the brain regions related to food reward at the beginning of the study. However, individuals who gained weight during the study had elevated brain responses in the hippocampus, precuneus, middle occipital gyrus, and vermis [145]. These elevated brain responses were correlated with higher negative affect at baseline or the experience of more stressful events among individuals who reported more severe EDs and restrained eating behavior [145]. These results demonstrate the effect of negative emotional situations on brain responses to food, which may influence eating behavior and food intake, thus predisposing individuals to weight gain. Higher responsiveness in reward-related brain regions may mediate the relationship between stressful events and weight gain over time. These results shed additional light on the link between stress and food choices, especially for comfort foods. When researchers investigated the effect of acute stress on self-control and decision-making, functional connectivity between the vmPFC, amygdala, and striatum was positively correlated with salivary cortisol levels when participants chose tastier foods [146]. By contrast, when individuals had to choose between healthy or tasty foods, higher perceived stress was correlated with a greater decrease in connectivity between the dlPFC and vmPFC [146]. These results suggest either that stress reduces dlPFC-vmPFC connectivity or that individuals with reduced dlPFC-vmPFC connectivity are more vulnerable to stress, and have less self-control. Although the authors' main goal was not to investigate the cognitive control of emotions, we can hypothesize that impaired emotion regulation may lead individuals to choose more palatable foods (i.e., comfort foods). In addition, heightened dlPFC activity has been observed in individuals with low chronic stress during the passive viewing of high-calorie food pictures, whereas reduced activity has been observed in individuals with a diagnosis of chronic stress [147]. Given the dlPFC's role in emotion

processing and inhibitory control, these results suggest that individuals with low chronic stress are more liable to suppress emotional reactions to highly palatable foods. Even though these studies did not include emotional assessments, the dlPFC appears to lie at the intersection of executive functioning, goal-directed behavior, and inhibitory control in the context of both eating behavior and emotion processing. Upregulation of the dlPFC may therefore be a promising strategy for preventing emotional eating in EDs [148]. Nevertheless, the extent to which stress is involved in emotional eating remains unclear [149]. Studies investigating the impact of stress on eating behavior should also investigate emotional eating in order to elucidate this relation.

4.4.Emotional regulation of food craving

As emotions are among the factors that modulate eating behavior, a growing number of studies have sought to achieve cognitive regulation of food craving, regarded as an affective state [83], through reappraisal, acceptance, suppression, distraction or imaginative strategies (for a more comprehensive overview of these different strategies, see [28,30]). The number of brain imaging studies in the context of these paradigms in individuals with [86] and without [6,150–157] EDs has increased over the past decade. Meta-analyses have been carried out of studies with [84,85] or without [158] fMRI. Wolz's meta-analysis showed that the cognitive regulation strategy offering the most effective top-down control of food craving was reappraisal, followed by suppression, with distraction having a more modest effect [158]. Brandl et al. (2019) focused on the cognitive control of craving for hedonic (rewarding) stimuli, showing consistent activation of the pre-SMA, SMA, vlPFC and dlPFC across different types of stimulus (e.g., food craving, cigarette craving, monetary reward). This consistent pattern of brain region activation supported the model of common neurocognitive control of both reward and negative emotions [84]. It also included the anterior insula and angular gyrus, which were activated more during cognitive reward control than during reward

cue exposure without control [84]. These results are in line with a previous meta-analysis that focused on the use of food stimuli to assess the neural correlates of dietary self-control: similar brain regions were involved, including the anterior insula, IFG, vlPFC, dlPFC, bilateral SMA, bilateral mid-cingulate cortex, and temporoparietal junction [85]. In addition, EEG recordings during the regulation of craving while being exposed to pictures of high-calorie food revealed higher positive LPP amplitude when individuals had to think about the long-term consequences of eating the food, compared with the immediate consequences [159]. Additionally, LPP amplitudes were positively correlated with self-reported emotional eating. These results indicate that emotional eaters pay increased attention to food because of its relevance for emotion regulation and behavioral control. However, this higher LPP may also be driven by arousal, regardless of valence, owing to possible negative thoughts related to the long-term effects of eating high-calorie foods. As a higher LPP amplitude was observed in the reappraisal of high-calorie food pictures, but became nonsignificant in the late LPP window (slow wave), the authors concluded that it may have reflected successful regulation of craving [159].

Even though such interventions are intended to reduce food cravings, the ultimate goal is to improve food choices in favor of healthier foods. Decision-making and emotion regulation brain networks share common prefrontal areas, including the vlPFC, medial PFC (i.e., medial OFC, frontal pole, rostral ACC, and subgenual PFC), dmPFC and dlPFC [5]. The connections between the two vary according to emotional state. Behavior is modulated by reinforcers (reward or punishment) that are assessed by emotion, and top-down control of emotion should have an effect on decision-making [5]. Regarding the impact of incidental negative emotions on food decisions after an emotion regulation task, associations have been found between reduced emotions and increased activity in the PFC, SMA and supramarginal gyrus [6], in line with meta-analyses described above. Reduced activity in the vmPFC was

observed in the *decrease* condition when individuals chose to consume the food item. Following emotion regulation, there was greater activity in the striatum when individuals chose to consume the food item than when they rejected the food. This was more pronounced in the decrease condition. Therefore, reducing emotions significantly modulated activity in the vmPFC and striatum when participants chose to consume foods rather than reject them [6]. Emotion regulation may therefore modulate dietary decisions through both reward and decision-making networks. In addition, cognitive regulation of food craving during choices is associated with differential activity in prefrontal brain regions involved in valuation and decision-making processes, as investigated through a cognitive regulation of craving task [160]. In healthy individuals, valuation of food pictures elicited reduced dlPFC activity after food craving reduction, and higher vmPFC activity after food craving increase [160]. In addition, the posterior parietal cortex and vlPFC exhibited stronger connectivity with the vmPFC during regulation, suggesting that these brain regions are involved in implementing changes in the decision-making circuitry during cognitive regulation [160]. Cognitive regulation therefore appears to affect decision-making through valuation regulation and behavioral control, where chronic stress may affect hedonic valuation but not cognitive regulation [161]. However, individuals with reward and cognitive deficits may be more prone to altered cognitive regulation, requiring increased frontoparietal functional connectivity [162]. Taken together, these studies highlighted overlapping pathways involving emotional processing and decision-making in the context of eating behavior that cannot be regarded as distinct mechanisms. Thus, emotional eating may have two dimensions: 1) *a hedonic dimension*, in which food intake is driven by the reward properties of food, and 2) *a cognitive dimension*, in which emotion regulation strategies may be impaired, at least when volitional regulation is required. The overlapping brain patterns reported by Brandl et al. (2019) suggest that a combined model of emotional eating is needed. Improving emotion regulation to

counteract food craving may have positive results regarding the control of hedonic food intake (e.g., comfort foods), therefore acting on the two assumed dimensions of emotional eating.

5. Health and societal issues: New neural targets and innovative strategies for prevention and therapy

5.1. Neuromodulation

Neuromodulation consists in modifying neural activity or excitability, using either invasive or noninvasive techniques. Noninvasive neuromodulation techniques, such as transcranial magnetic stimulation and transcranial direct-current stimulation (tDCS), are being increasingly used in interventional studies of disordered eating behavior [148,163]. To demonstrate the causal implication of the brain regions described above, there is a need for interventional studies aimed at modulating these brain regions. Interestingly, in ED subtypes, tDCS-based modulation of the dlPFC has had positive effects on food craving, improved mood, and reduced daily calorie intake [164]. Current knowledge of neuromodulation is based on case-report studies or heterogeneous volunteer samples [163,164], and its long-term efficiency remains unexplored. It nevertheless seems well suited for the treatment of disordered eating behaviors [163]. Given that overeating may be driven by emotional states, it could be useful to apply noninvasive neuromodulation techniques to prevent emotional eating, by targeting brain areas that are involved in both the cognitive control of emotion and eating behavior. To date, no intervention study featuring a neuromodulation approach has been conducted in the context of emotional eating.

5.2. Use of neurofeedback in the prevention and treatment of emotional eating

Neurofeedback (NF) appears to be a promising indirect brain stimulation strategy. It is

a form of biofeedback in which individuals are trained to voluntarily up- or downregulate their own brain activity while receiving realtime feedback [165,166]. Several neuroimaging techniques can be used to provide NF, such as EEG, fMRI and, more recently, functional near-infrared spectroscopy (fNIRS). Most studies have been conducted with EEG, given the high temporal resolution this modality can offer. An increasing number of studies are being performed with fMRI, given the higher spatial resolution and whole-brain coverage. A combination of these modalities, allowing both fine spatial and accurate temporal resolution to be achieved [167], has already been applied in the context of emotion [168,169]. NF can target one or more brain areas, as well as the functional connectivity between brain regions. As mentioned above, this functional connectivity can be altered in neuropsychiatric diseases, and associated therefore with disordered mood or behavior [166]. NF can be used either as an intervention per se, or as a way of assessing or enhancing conventional treatments, or even of improving knowledge in fundamental neuroscience [165]. NF has not yet been applied in the context of emotional eating, but has shown promising results for regulating emotions in healthy individuals [170–173] or patients with mood disorders [169,174], as well as the eating behavior of healthy individuals with normal weight or overweight [175], overweight/obese individuals with [176] or without food addiction [177,178], restrained eaters with overeating/binge eating episodes [179,180], and patients with EDs such as anorexia [181].

Most of the studies investigating the effect of fMRI-based NF in emotion regulation in healthy individuals have targeted the amygdala [170,171,173], because of its main role in emotion processing [182]. Downregulation of the amygdala in healthy individuals during exposure to aversive pictures is related to increased functional connectivity between the amygdala and the dmPFC, dlPFC, vlPFC, ACC [170] and vmPFC [171]. Upregulation of the right dlPFC with fNIRS-based NF has been associated with improved individual emotion regulation abilities [172]. Moreover, functional connectivity within the emotion regulation

network, including structures such as the dlPFC, vlPFC and SMA, and between this network and the amygdala, has been found to increase after repeated NF sessions [172]. This shows that NF can enhance the cognitive processes involved in emotion regulation, which could be very valuable in the context of emotional eating [183]. Most NF studies of eating behavior have used EEG [175,176,179–181]. EEG-based NF training in healthy individuals intended to increase theta/alpha ratio, has been associated with fewer food craving events directly after the intervention, as well as 4 months later [175]. In addition, upregulation of the infraslow frequency of the PCC has been associated with reduced food craving in obese individuals with food addiction [176]. Regarding overeating episodes, after downregulation of high beta activity (EEG) during a food cue exposure, authors observed a decrease in the frequency of overeating episodes [179], binge eating episodes and food craving events, as well as improvements in perceived stress among female restrained eaters [180]. However, improvements in perceived stress and food craving were also noticed in a group who performed mental imagery (imagining pleasant and vivid images), compared with a waitlist group [180]. Consequently, the authors investigated the presence and role of physiological (changes in brain activity) and/or psychological (increased self-efficacy and subjective control) learning mechanisms in NF. They found that the group who received NF used both physiological and psychological learning mechanisms, as measured by EGG activity or changes in somatic self-efficacy, with a greater effect of the physiological learning mechanisms, whereas the group who performed mental imagery only used psychological learning mechanisms [184]. These results indicate that only the NF training was associated with physiological changes, and that it promoted different learning mechanisms compared with the mental imagery strategy. The upregulation of the anterior insula cortex with fMRI-based NF in the context of eating behavior shows that both obese and lean individuals are able to upregulate the activity of this brain region, despite the absence of behavioral or mood

outcomes [177]. More recently, Kohl et al. (2019) found that the upregulation of the dlPFC with fMRI-based NF during palatable food picture exposure improved self-control during food choices and reduced the preference for high-calorie items [178]. They also found a decrease in anxiety and depression ratings, suggesting that dlPFC upregulation can improve emotional state and food choices, even if no change in calorie intake was observed [178]. All these studies highlight the potential effectiveness of NF for improving eating behavior (for a descriptive overview of these eating-related NF studies, see [185]). It should be noted that behavioral outcomes are still inconsistent in NF studies [177,186,187], and further research is needed to confirm the potential of NF training as an emerging interventional technique. Moreover, most of these studies lacked a control group with sham NF, and included only a small number of participants, thus preventing the generalization of the results. These limitations increase the risk of experimental bias and insufficient statistical power [185]. Experimental paradigms need to be improved, in order to overcome these issues. For this purpose, we recommend standardizing components of the NF protocol, such as the number and duration of the NF sessions. Nevertheless, the above-mentioned research has brought promising outcomes, such as a reduction in food craving [175,176,180], enhanced self-control during food choice [178], and reduced frequency of overeating and binge-eating episodes [179,180]. In addition, some studies have shown that NF can enhance the functional connectivity of the emotion regulation network [170–173], thereby improving emotion regulation. These results highlight the potential effectiveness of NF for emotional eaters. The target brain region should be involved in both emotion regulation and eating behavior. Given its role in both emotional processing [24,26] and the inhibitory control of eating behavior [148], the dlPFC therefore seems to be a suitable candidate for improving emotion regulation and the control of food intake in emotional eaters [10,183].

6. Perspectives and conclusion

Although neuroimaging studies of eating behavior in pathological or healthy conditions have been somewhat heterogeneous in terms of number of participants, sex, and experimental paradigm, they have shown which brain regions and networks are involved. These regions and networks could be used as targets for neuromodulation strategies (e.g., NF) designed to counteract maladaptive eating behaviors related to emotion. Emotional eating involves not just the amygdala, but overlapping mechanisms at the intersection between reward circuitry, cognitive control (executive functioning, decision-making) and emotion regulation, all three of which are intrinsically connected. In line with previous resting-state fMRI studies [188], more attention should be paid to the vmPFC and OFC on account of their involvement in valuation and decision-making, the insula for interoception and affect modulation, the dlPFC for cognitive control, and the VTA and striatum (NAcc, caudate and putamen) for reward processing. As it has been suggested that emotional eating is a predictor of chronic BED, elucidating the brain mechanisms involved could help identify early vulnerability factors in emotional eaters and thus yield crucial information for preventive care.

References

1. Temple NJ. Fat, Sugar, Whole Grains and Heart Disease: 50 Years of Confusion. Nutrients. 2018; https://doi.org/10.3390/nu10010039

2. Debras C, Chazelas E, Srour B, Kesse-Guyot E, Julia C, Zelek L, et al. Total and added sugar intakes, sugar types, and cancer risk: Results from the prospective NutriNet-Santé cohort. Am J Clin Nutr. 2020; http://doi.org/10.1093/ajcn/nqaa246

3. Afshin A, Sur PJ, Fay KA, Cornaby L, Ferrara G, Salama JS, et al. Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2019; http://dx.doi.org/10.1016/ S0140-6736(19)30041-8

4. Hofmann W, Friese M, Wiers RW. Impulsive versus reflective influences on health behavior: a theoretical framework and empirical review. Health Psychol Rev. 2008; http://dx.doi.org/10.1080/17437190802617668

5. Mitchell DGV. The nexus between decision making and emotion regulation: A review of convergent neurocognitive substrates. Behav Brain Res. 2011; http://dx.doi.org/10.1016/j.bbr.2010.10.030

6. Morawetz C, Steyrl D, Berboth S, Heekeren HR, Bode S. Emotion Regulation Modulates Dietary Decision-Making via Activity in the Prefrontal–Striatal Valuation System. Cereb Cortex. 2020; http://dx.doi.org/10.1093/cercor/bhaa147

7. Macht M. How emotions affect eating: A five-way model. Appetite. 2008; http://doi.org/10.1016/j.appet.2007.07.002

8. Arnow B, Kenardy J, Agras WS. The emotional eating scale: The development of a measure to assess coping with negative affect by eating. Int J Eat Disord. 1995; https://onlinelibrary.wiley.com/doi/10.1002/1098-108X(199507)18:1%3C79::AID-

EAT2260180109%3E3.0.CO;2-V

9. Evers C, Dingemans A, Junghans AF, Boevé A. Feeling bad or feeling good, does emotion affect your consumption of food? A meta-analysis of the experimental evidence. Neurosci Biobehav Rev. 2018; https://doi.org/10.1016/j.neubiorev.2018.05.028

10. Macht M, Simons G. Emotional Eating. In: Nyklíček I, Vingerhoets A, Zeelenberg M, editors. Emotion Regulation and Well-Being. New York, NY: Springer New York; 2011. p. 281–95. http://link.springer.com/10.1007/978-1-4419-6953-8_17

11. Sultson H, Kukk K, Akkermann K. Positive and negative emotional eating have different associations with overeating and binge eating: Construction and validation of the Positive-Negative Emotional Eating Scale. Appetite. 2017; http://dx.doi.org/10.1016/j.appet.2017.05.035

12. Zeeck A, Stelzer N, Linster HW, Joos A, Hartmann A. Emotion and eating in binge eating disorder and obesity. Eur Eat Disord Rev. 2011; http://doi.org/10.1002/erv.1066

13. Koenders PG, Van Strien T. Emotional eating, rather than lifestyle behavior, drives weight gain in a prospective study in 1562 employees. J Occup Environ Med. 2011; http://doi.org/10.1097/JOM.0b013e31823078a2

14. Berridge KC, Robinson TE. Liking, wanting, and the incentive-sensitization theory of addiction. Am Psychol. 2016; http://doi.org/10.1037/amp0000059

15. Berthoud H-R. Metabolic and hedonic drives in the neural control of appetite: who is the boss? Curr Opin Neurobiol. 2011; http://doi.org/10.1016/j.conb.2011.09.004

16. Ulrich-Lai YM, Fulton S, Wilson M, Petrovich G, Rinaman L. Stress exposure, food intake and emotional state. Stress. 2015; http://doi.org/10.3109/10253890.2015.1062981

17. Kleinginna PR, Kleinginna AM. A categorized list of motivation definitions, with a suggestion for a consensual definition. Motiv Emot. 1981; http://doi.org/10.1007/BF00993889

18. Brosch T, Scherer K, Grandjean D, Sander D. The impact of emotion on perception, attention, memory, and decision-making. Swiss Med Wkly. 2013; http://doi.org/10.4414/smw.2013.13786

19. Gross JJ, Sheppes G, Urry HL. Cognition and Emotion Lecture at the 2010 SPSP Emotion Preconference. Cogn Emot. 2011; https://doi.org/10.1080/02699931.2011.555753

20. Ochnser K, Gross J. The cognitive control of emotion. Trends Cogn Sci. 2005; https://doi.org/10.1016/j.tics.2005.03.010

21. Kober H, Barrett LF, Joseph J, Bliss-Moreau E, Lindquist K, Wager TD. Functional grouping and cortical–subcortical interactions in emotion: A meta-analysis of neuroimaging studies. Neuroimage. 2008; http://doi.org/10.1016/j.neuroimage.2008.03.059

22. Buhle JT, Silvers JA, Wager TD, Lopez R, Onyemekwu C, Kober H, et al. Cognitive Reappraisal of Emotion: A Meta-Analysis of Human Neuroimaging Studies. Cereb Cortex. 2014; http://doi.org/10.1093/cercor/bht154

23. Gross JJ, Feldman Barrett L. Emotion Generation and Emotion Regulation: One or Two Depends on Your Point of View. Emot Rev. 2011; http://doi.org/10.1177/1754073910380974

24. Ochsner KN, Silvers JA, Buhle JT. Functional imaging studies of emotion regulation: a synthetic review and evolving model of the cognitive control of emotion. Ann N Y Acad Sci. 2012; http://doi.org/10.1111/j.1749-6632.2012.06751.x

866 25. Etkin A, Büchel C, Gross JJ. The neural bases of emotion regulation. Nat Rev
867 Neurosci. 2015; http://doi.org/10.1038/nrn4044
868 26. Helion C, Krueger SM, Ochsner KN. Emotion regulation across the life span [Internet].
869 1st ed. Vol. 163, Handbook of Clinical Neurology. Elsevier B.V.; 2019. 257–280 p.
870 http://dx.doi.org/10.1016/B978-0-12-804281-6.00014-8
871 27. Craig AD. How do you feel — now? The anterior insula and human awareness. Nat
872 Rev Neurosci. 2009; http://doi.org/10.1038/nrn2555
873 28. Gross JJ. The Emerging Field of Emotion Regulation: An Integrative Review. Rev Gen
874 Psychol. 1998; http://journals.sagepub.com/doi/10.1037/1089-2680.2.3.271
875 29. Gross JJ. Supplemental Material for Emotion Regulation. Emotion. 2020;
876 http://doi.org/10.1037/emo0000703.supp
877 30. Peña-Sarrionandia A, Mikolajczak M, Gross JJ. Integrating emotion regulation and
878 emotional intelligence traditions: a meta-analysis. Front Psychol. 2015;
879 http://doi.org/10.3389/fpsyg.2015.00160
880 31. Morawetz C, Bode S, Derntl B, Heekeren HR. The effect of strategies, goals and
881 stimulus material on the neural mechanisms of emotion regulation: A meta-analysis of
882 fMRI studies. Neurosci Biobehav Rev. 2017;
883 http://dx.doi.org/10.1016/j.neubiorev.2016.11.014
884 32. Park C, Rosenblat JD, Lee Y, Pan Z, Cao B, Iacobucci M, et al. The neural systems of
885 emotion regulation and abnormalities in major depressive disorder. Behav Brain Res.
886 2019; https://doi.org/10.1016/j.bbr.2019.04.002
887 33. Dixon ML, Thiruchselvam R, Todd R, Christoff K. Emotion and the prefrontal cortex:
888 An integrative review. Psychol Bull. 2017; http://dx.doi.org/10.1037/bul0000096
889 34. Etkin A, Egner T, Kalisch R. Emotional processing in anterior cingulate and medial

prefrontal cortex. Trends Cogn Sci. 2011; http://doi.prg/10.1016/j.tics.2010.11.004

35. Rolls ET. The orbitofrontal cortex and emotion in health and disease, including depression. Neuropsychologia. 2019; https://doi.org/10.1016/j.neuropsychologia.2017.09.021

36. Kohn N, Eickhoff SB, Scheller M, Laird AR, Fox PT, Habel U. Neural network of cognitive emotion regulation — An ALE meta-analysis and MACM analysis. Neuroimage. 2014; http://dx.doi.org/10.1016/j.neuroimage.2013.11.001

37. Goldin PR, McRae K, Ramel W, Gross JJ. The Neural Bases of Emotion Regulation: Reappraisal and Suppression of Negative Emotion. Biol Psychiatry. 2008; http://doi.org/10.1016/j.biopsych.2007.05.031

38. Diekhof EK, Geier K, Falkai P, Gruber O. Fear is only as deep as the mind allows. Neuroimage. 2011; http://dx.doi.org/10.1016/j.neuroimage.2011.05.073

39. Zilverstand A, Parvaz MA, Goldstein RZ. Neuroimaging cognitive reappraisal in clinical populations to define neural targets for enhancing emotion regulation. A systematic review. Neuroimage. 2017; http://dx.doi.org/10.1016/j.neuroimage.2016.06.009

40. Picó-Pérez M, Radua J, Steward T, Menchón JM, Soriano-Mas C. Emotion regulation in mood and anxiety disorders: A meta-analysis of fMRI cognitive reappraisal studies. Prog Neuro-Psychopharmacology Biol Psychiatry. 2017; http://dx.doi.org/10.1016/j.pnpbp.2017.06.001

41. Paschke LM, Dörfel D, Steimke R, Trempler I, Magrabi A, Ludwig VU, et al. Individual differences in self-reported self-control predict successful emotion regulation. Soc Cogn Affect Neurosci. 2016; http://doi.org/10.1093/scan/nsw036

42. Gross JJ, John OP. Individual differences in two emotion regulation processes:

implications for affect, relationships, and well-being. J Pers Soc Psychol. 2003; https://doi.org/10.1037/0022-3514.85.2.348

43. Picó-Pérez M, Alonso P, Contreras-Rodríguez O, Martínez-Zalacaín I, López-Solà C, Jiménez-Murcia S, et al. Dispositional use of emotion regulation strategies and resting-state cortico-limbic functional connectivity. Brain Imaging Behav. 2018; http://link.springer.com/10.1007/s11682-017-9762-3

44. Uchida M, Biederman J, Gabrieli JDE, Micco J, de Los Angeles C, Brown A, et al. Emotion regulation ability varies in relation to intrinsic functional brain architecture. Soc Cogn Affect Neurosci. 2015; http://doi.org/10.1093/scan/nsv059

45. Kaiser RH, Andrews-Hanna JR, Wager TD, Pizzagalli DA. Large-Scale Network Dysfunction in Major Depressive Disorder. JAMA Psychiatry. 2015; http://doi.org/10.1001/jamapsychiatry.2015.0071

46. Steward T, Picó-Pérez M, Mestre-Bach G, Martínez-Zalacaín I, Suñol M, Jiménez-Murcia S, et al. A multimodal MRI study of the neural mechanisms of emotion regulation impairment in women with obesity. Transl Psychiatry. 2019; http://dx.doi.org/10.1038/s41398-019-0533-3

47. Duncan S, Barrett LF. Affect is a form of cognition: A neurobiological analysis. Cogn Emot. 2007; http://doi.org/10.1080/02699930701437931

48. Okon-Singer H, Hendler T, Pessoa L, Shackman AJ. The neurobiology of emotion-cognition interactions: fundamental questions and strategies for future research. Front Hum Neurosci. 2015; http://doi.org/10.3389/fnhum.2015.00058

49. Pessoa L. A Network Model of the Emotional Brain. Trends Cogn Sci. 2017; http://dx.doi.org/10.1016/j.tics.2017.03.002

50. Ochsner KN, Gross JJ. Cognitive Emotion Regulation. Curr Dir Psychol Sci. 2008;

http://doi.org/10.1111/j.1467-8721.2008.00566.x

51. Mishra A, Anand M, Umesh S. Neurobiology of eating disorders - an overview. Asian J Psychiatr. 2017; http://dx.doi.org/10.1016/j.ajp.2016.10.009

52. Shin AC, Zheng H, Berthoud H-R. An expanded view of energy homeostasis: Neural integration of metabolic, cognitive, and emotional drives to eat. Physiol Behav. 2009; http://dx.doi.org/10.1016/j.physbeh.2009.02.010

53. Liu CM, Kanoski SE. Homeostatic and non-homeostatic controls of feeding behavior: Distinct vs. common neural systems. Physiol Behav. 2018; http://doi.org/10.1016/j.physbeh.2018.02.011

54. Hollmann M, Pleger B, Villringer A, Horstmann A. Brain imaging in the context of food perception and eating. Curr Opin Lipidol. 2013; http://doi.org/10.1097/MOL.0b013e32835b61a4

55. Rolls E. Functions of the orbitofrontal and pregenual cingulate cortex in taste, olfaction, appetite and emotion. Acta Physiol Hung. 2008; http://doi.org/10.1556/APhysiol.95.2008.2.1

56. Schmidt L, Tusche A, Manoharan N, Hutcherson C, Hare T, Plassmann H. Neuroanatomy of the vmPFC and dlPFC Predicts Individual Differences in Cognitive Regulation During Dietary Self-Control Across Regulation Strategies. J Neurosci. 2018; http://doi.org/10.1523/JNEUROSCI.3402-17.2018

57. Petrovich GD, Ross CA, Holland PC, Gallagher M. Medial Prefrontal Cortex Is Necessary for an Appetitive Contextual Conditioned Stimulus to Promote Eating in Sated Rats. J Neurosci. 2007; http://doi.org/10.1523/JNEUROSCI.5001-06.2007

58. Le DSNT, Pannacciulli N, Chen K, Del Parigi A, Salbe AD, Reiman EM, et al. Less activation of the left dorsolateral prefrontal cortex in response to a meal: a feature of

962 obesity. Am J Clin Nutr. 2006; http://doi.org/10.1093/ajcn/84.4.725

963 59. Chan KL, Cathomas F, Russo SJ. Central and Peripheral Inflammation Link Metabolic
964 Syndrome and Major Depressive Disorder. Physiology. 2019;
965 http://doi.org/10.1152/physiol.00047.2018

966 60. Luppino FS, de Wit LM, Bouvy PF, Stijnen T, Cuijpers P, Penninx BWJH, et al.
967 Overweight, Obesity, and Depression. Arch Gen Psychiatry. 2010;
968 http://doi.org/10.1001/archgenpsychiatry.2010.2?doi=10.1001/archgenpsychiatry.2010.
969 2

970 61. Milaneschi Y, Simmons WK, van Rossum EFC, Penninx BW. Depression and obesity:
971 evidence of shared biological mechanisms. Mol Psychiatry. 2019;
972 http://dx.doi.org/10.1038/s41380-018-0017-5

973 62. Donofry SD, Roecklein KA, Wildes JE, Miller MA, Erickson KI. Alterations in
974 emotion generation and regulation neurocircuitry in depression and eating disorders: A
975 comparative review of structural and functional neuroimaging studies. Neurosci
976 Biobehav Rev. 2016; http://dx.doi.org/10.1016/j.neubiorev.2016.07.011

977 63. Singh M. Mood, food, and obesity. Front Psychol. 2014;
978 http://doi.org/10.3389/fpsyg.2014.00925

979 64. Flaskerud JH. Mood and Food. Issues Ment Health Nurs. 2015;
980 http://doi.org/10.3109/01612840.2014.962677

981 65. Spencer SJ, Korosi A, Layé S, Shukitt-Hale B, Barrientos RM. Food for thought: how
982 nutrition impacts cognition and emotion. npj Sci Food. 2017;
983 http://dx.doi.org/10.1038/s41538-017-0008-y

984 66. Berthoud H-R. Homeostatic and non-homeostatic pathways involved in the control of
985 food intake and energy balance. Obesity (Silver Spring). 2006;

http://doi.wiley.com/10.1038/oby.2006.308

67. White FJ. Synaptic regulation of mesocorticolimbic dopamine neurons. Annu Rev Neurosci. 1996; http://doiq.org/10.1146/annurev.ne.19.030196.002201

68. Hajnal A, Smith GP, Norgren R. Oral sucrose stimulation increases accumbens dopamine in the rat. Am J Physiol Integr Comp Physiol. 2004; http://doi.org/10.1152/ajpregu.00282.2003

69. Small DM, Jones-Gotman M, Dagher A. Feeding-induced dopamine release in dorsal striatum correlates with meal pleasantness ratings in healthy human volunteers. Neuroimage. 2003; http://doi.org/10.1016/S1053-8119(03)00253-2

70. Wang G-J, Geliebter A, Volkow ND, Telang FW, Logan J, Jayne MC, et al. Enhanced striatal dopamine release during food stimulation in binge eating disorder. Obesity (Silver Spring). 2011; http://dx.doi.org/10.1038/oby.2011.27/nature06264

71. Canetti L, Bachar E, Berry EM. Food and emotion. Behav Processes. 2002; http://doi.org/10.1016/S0376-6357(02)00082-7

72. Bruch H. Obesity and anorexia nervosa: psychosocial aspects. Aust N Z J Psychiatry. 1975; http://doi.org/10.3109/00048677509159842

73. Ganley RM. Emotion and eating in obesity: A review of the literature. Int J Eat Disord. 1989; http://doi.org/10.1002/1098-108X(198905)8:3%3C343::AID-EAT2260080310%3E3.0.CO;2-C

74. Constant A, Gautier Y, Coquery N, Thibault R, Moirand R, Val-Laillet D. Emotional overeating is common and negatively associated with alcohol use in normal-weight female university students. Appetite. 2018; https://doi.org/10.1016/j.appet.2018.07.012

75. Prefit A-B, Cândea DM, Szentagotai-Tătar A. Emotion regulation across eating pathology: A meta-analysis. Appetite. 2019;

1010 http://doi.org/10.1016/j.appet.2019.104438

1011 76. Kittel R, Brauhardt A, Hilbert A. Cognitive and emotional functioning in binge-eating
1012 disorder: A systematic review. Int J Eat Disord. 2015; http://doi.org/10.1002/eat.22419

1013 77. Micanti F, Iasevoli F, Cucciniello C, Costabile R, Loiarro G, Pecoraro G, et al. The
1014 relationship between emotional regulation and eating behaviour: a multidimensional
1015 analysis of obesity psychopathology. Eat Weight Disord. 2017;
1016 http://doi.org/10.1007/s40519-016-0275-7

1017 78. Svaldi J, Griepenstroh J, Tuschen-Caffier B, Ehring T. Emotion regulation deficits in
1018 eating disorders: a marker of eating pathology or general psychopathology? Psychiatry
1019 Res. 2012; http://doi.org/10.1016/j.psychres.2011.11.009

1020 79. D'Agata F, Caroppo P, Amianto F, Spalatro A, Caglio MM, Bergui M, et al. Brain
1021 correlates of alexithymia in eating disorders: A voxel-based morphometry study.
1022 Psychiatry Clin Neurosci. 2015; http://doi.org/10.1111/pcn.12318

1023 80. Mikhail ME, Keel PK, Burt SA, Neale M, Boker S, Klump KL. Low emotion
1024 differentiation: An affective correlate of binge eating? Int J Eat Disord. 2020;
1025 http://doi.org/10.1002/eat.23207

1026 81. Sierra I, Senín-Calderón C, Roncero M, Perpiñá C. The Role of Negative Affect in
1027 Emotional Processing of Food-Related Images in Eating Disorders and Obesity. Front
1028 Psychol. 2021; http://doi.org/10.3389/fpsyg.2021.723732

1029 82. Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion-regulation strategies across
1030 psychopathology: A meta-analytic review. Clin Psychol Rev. 2010;
1031 http://dx.doi.org/10.1016/j.cpr.2009.11.004

1032 83. Giuliani NR, Berkman ET. Craving Is an Affective State and Its Regulation Can Be
1033 Understood in Terms of the Extended Process Model of Emotion Regulation. Psychol

Inq. 2015; http://doi.org/10.1080/1047840X.2015.955072

84. Brandl F, Le Houcq Corbi Z, Mulej Bratec S, Sorg C. Cognitive reward control recruits medial and lateral frontal cortices, which are also involved in cognitive emotion regulation: A coordinate-based meta-analysis of fMRI studies. Neuroimage. 2019; https://doi.org/10.1016/j.neuroimage.2019.07.008

85. Han JE, Boachie N, Garcia-Garcia I, Michaud A, Dagher A. Neural correlates of dietary self-control in healthy adults: A meta-analysis of functional brain imaging studies. Physiol Behav. 2018; https://doi.org/10.1016/j.physbeh.2018.02.037

86. Steward T, Martínez-Zalacaín I, Mestre-Bach G, Sánchez I, Riesco N, Jiménez-Murcia S, et al. Dorsolateral prefrontal cortex and amygdala function during cognitive reappraisal predicts weight restoration and emotion regulation impairment in anorexia nervosa. Psychol Med. 2020; http://doi.org/10.1017/S0033291720002457

87. de Kloet ER, Joëls M, Holsboer F. Stress and the brain: from adaptation to disease. Nat Rev Neurosci. 2005; http://doi.org/10.1038/nrn1683

88. Rabasa C, Dickson SL. Impact of stress on metabolism and energy balance. Curr Opin Behav Sci. 2016; http://dx.doi.org/10.1016/j.cobeha.2016.01.011

89. Dallman MF. Stress-induced obesity and the emotional nervous system. Trends Endocrinol Metab. 2010; http://dx.doi.org/10.1016/j.tem.2009.10.004

90. Ulrich-Lai YM, Christiansen AM, Ostrander MM, Jones AA, Jones KR, Choi DC, et al. Pleasurable behaviors reduce stress via brain reward pathways. Proc Natl Acad Sci. 2010; http://doi.org/10.1073/pnas.1007740107

91. Ulrich-Lai YM, Ostrander MM, Thomas IM, Packard BA, Furay AR, Dolgas CM, et al. Daily limited access to sweetened drink attenuates hypothalamic-pituitary-adrenocortical axis stress responses. Endocrinology. 2007;

https://academic.oup.com/endo/article/148/4/1823/2502010

92. Egan AE, Thompson AMK, Buesing D, Fourman SM, Packard AEB, Terefe T, et al. Palatable Food Affects HPA Axis Responsivity and Forebrain Neurocircuitry in an Estrous Cycle-specific Manner in Female Rats. Neuroscience. 2018; https://doi.org/10.1016/j.neuroscience.2018.05.030

93. Packard AEB, Di S, Egan AE, Fourman SM, Tasker JG, Ulrich-Lai YM. Sucrose-induced plasticity in the basolateral amygdala in a 'comfort' feeding paradigm. Brain Struct Funct. 2017; http://doi.org/10.1007/s00429-017-1454-7

94. Ozier AD, Kendrick OW, Leeper JD, Knol LL, Perko M, Burnham J. Overweight and obesity are associated with emotion- and stress-related eating as measured by the eating and appraisal due to emotions and stress questionnaire. J Am Diet Assoc. 2008; http://doi.org/10.1016/j.jada.2007.10.011

95. Lazarevich I, Irigoyen Camacho ME, Velázquez-Alva M del C, Zepeda Zepeda M. Relationship among obesity, depression, and emotional eating in young adults. Appetite. 2016; http://doi.org/10.1016/j.appet.2016.09.011

96. Konttinen H, van Strien T, Männistö S, Jousilahti P, Haukkala A. Depression, emotional eating and long-term weight changes: a population-based prospective study. Int J Behav Nutr Phys Act. 2019; https://doi.org/10.1186/s12966-019-0791-8

97. van Strien T, Roelofs K, de Weerth C. Cortisol reactivity and distress-induced emotional eating. Psychoneuroendocrinology. 2013; http://doi.org/10.1016/j.psyneuen.2012.08.008

98. Joëls M. Corticosteroids and the brain. J Endocrinol. 2018; http://doi.org/10.1530/JOE-18-0226

99. Roos LG, Janson J, Sturmbauer SC, Bennett JM, Rohleder N. Higher trait reappraisal

predicts stronger HPA axis habituation to repeated stress. Psychoneuroendocrinology. 2019; https://doi.org/10.1016/j.psyneuen.2018.10.018

100. Langer K, Wolf OT, Jentsch VL. Delayed effects of acute stress on cognitive emotion regulation. Psychoneuroendocrinology. 2021; https://doi.org/10.1016/j.psyneuen.2020.105101

101. Jentsch VL, Merz CJ, Wolf OT. Restoring emotional stability: Cortisol effects on the neural network of cognitive emotion regulation. Behav Brain Res. 2019; https://doi.org/10.1016/j.bbr.2019.03.049

102. Peters AT, Van Meter A, Pruitt PJ, Briceño EM, Ryan KA, Hagan M, et al. Acute cortisol reactivity attenuates engagement of fronto-parietal and striatal regions during emotion processing in negative mood disorders. Psychoneuroendocrinology. 2016; http://doi.org/10.1016/j.psyneuen.2016.07.215

103. Nusslock R, Brody GH, Armstrong CC, Carroll AL, Sweet LH, Yu T, et al. Higher Peripheral Inflammatory Signaling Associated With Lower Resting-State Functional Brain Connectivity in Emotion Regulation and Central Executive Networks. Biol Psychiatry. 2019; https://doi.org/10.1016/j.biopsych.2019.03.968

104. Glaser R, Kiecolt-Glaser JK. Stress-induced immune dysfunction: implications for health. Nat Rev Immunol. 2005; http://doi.org/10.1038/nri1571

105. Raff H, Carroll T. Cushing's syndrome: from physiological principles to diagnosis and clinical care. J Physiol. 2015; http://doi.org/10.1113/jphysiol.2014.282871

106. Pivonello R, Simeoli C, De Martino MC, Cozzolino A, De Leo M, Iacuaniello D, et al. Neuropsychiatric disorders in Cushing's syndrome. Front Neurosci. 2015; http://doi.org/10.3389/fnins.2015.00129

107. Castanon N, Luheshi G, Layé S. Role of neuroinflammation in the emotional and

cognitive alterations displayed by animal models of obesity. Front Neurosci. 2015; http://doi.org/10.3389/fnins.2015.00229

108. Castanon N, Lasselin J, Capuron L. Neuropsychiatric Comorbidity in Obesity: Role of Inflammatory Processes. Front Endocrinol (Lausanne). 2014; http://doi.org/10.3389/fendo.2014.00074

109. Tsigos C, Chrousos GP. Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. J Psychosom Res. 2002; http://doi.org/10.1016/s0022-3999(02)00429-4

110. Jenkins TA, Nguyen JCD, Polglaze KE, Bertrand PP. Influence of Tryptophan and Serotonin on Mood and Cognition with a Possible Role of the Gut-Brain Axis. Nutrients. 2016; http://doi.org/10.3390/nu8010056

111. Kelly JR, Borre Y, O' Brien C, Patterson E, El Aidy S, Deane J, et al. Transferring the blues: Depression-associated gut microbiota induces neurobehavioural changes in the rat. J Psychiatr Res. 2016; http://dx.doi.org/10.1016/j.jpsychires.2016.07.019

112. Agustí A, García-Pardo MP, López-Almela I, Campillo I, Maes M, Romaní-Pérez M, et al. Interplay Between the Gut-Brain Axis, Obesity and Cognitive Function. Front Neurosci. 2018; http://doi.org/10.3389/fnins.2018.00155

113. Martins LB, Monteze NM, Calarge C, Ferreira AVM, Teixeira AL. Pathways linking obesity to neuropsychiatric disorders. Nutrition. 2019; http://doi.org/10.1016/j.nut.2019.03.017

114. Fung TC, Olson CA, Hsiao EY. Interactions between the microbiota, immune and nervous systems in health and disease. Nat Neurosci. 2017; http://doi.org/10.1038/nn.4476

115. Asano Y, Hiramoto T, Nishino R, Aiba Y, Kimura T, Yoshihara K, et al. Critical role of gut microbiota in the production of biologically active, free catecholamines in the

gut lumen of mice. Am J Physiol Gastrointest Liver Physiol. 2012; http://doi.org/10.1152/ajpgi.00341.2012

116. Salamone JD, Correa M. The Mysterious Motivational Functions of Mesolimbic Dopamine. Neuron. 2012; http://doi.org/10.1016/j.neuron.2012.10.021

117. Appelhans BM, Whited MC, Schneider KL, Ma Y, Oleski JL, Merriam PA, et al. Depression Severity, Diet Quality, and Physical Activity in Women with Obesity and Depression. J Acad Nutr Diet. 2012; http://doi.org/10.1016/j.jand.2012.02.006

118. Li M, van Esch BCAM, Wagenaar GTM, Garssen J, Folkerts G, Henricks PAJ. Pro- and anti-inflammatory effects of short chain fatty acids on immune and endothelial cells. Eur J Pharmacol. 2018; http://doi.org/10.1016/j.ejphar.2018.05.003

119. Daniel H, Gholami AM, Berry D, Desmarchelier C, Hahne H, Loh G, et al. High-fat diet alters gut microbiota physiology in mice. ISME J. 2014; http://doi.org/10.1038/ismej.2013.155

120. Menneson S, Ménicot S, Ferret-Bernard S, Guérin S, Romé V, Le Normand L, et al. Validation of a Psychosocial Chronic Stress Model in the Pig Using a Multidisciplinary Approach at the Gut-Brain and Behavior Levels. Front Behav Neurosci. 2019; http://doi.org/10.3389/fnbeh.2019.00161

121. Panduro A, Rivera-Iñiguez I, Sepulveda-Villegas M, Roman S. Genes, emotions and gut microbiota: The next frontier for the gastroenterologist. World J Gastroenterol. 2017; http://doi.org/10.3748/wjg.v23.i17.3030

122. Orsal AS, Blois SM, Bermpohl D, Schaefer M, Coquery N. Administration of interferon-alpha in mice provokes peripheral and central modulation of immune cells, accompanied by behavioral effects. Neuropsychobiology. 2008; http://doi.org/10.1159/000201718

123. Carbone EA, D'Amato P, Vicchio G, De Fazio P, Segura-Garcia C. A systematic review on the role of microbiota in the pathogenesis and treatment of eating disorders. Eur Psychiatry. 2020; http://doi.org/10.1192/j.eurpsy.2020.109

124. Mayer EA. Gut feelings: the emerging biology of gut-brain communication. Nat Rev Neurosci. 2011; http://dx.doi.org/10.1038/nrn3071

125. van Strien T, Frijters JER, Bergers GPA, Defares PB. The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. Int J Eat Disord. 1986; https://doi.org/10.1002/1098-108X(198602)5:2%3C295::AID-EAT2260050209%3E3.0.CO;2-T

126. Karlsson J, Persson LO, Sjöström L, Sullivan M. Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. Int J Obes. 2000;

127. Schembre S, Greene G, Melanson K. Development and validation of a weight-related eating questionnaire. Eat Behav. 2009; http://dx.doi.org/10.1016/j.eatbeh.2009.03.006

128. Masheb RM, Grilo CM. Emotional overeating and its associations with eating disorder psychopathology among overweight patients with Binge eating disorder. Int J Eat Disord. 2006; http://doi.org/10.1002/eat.20221

129. Song S, Zhang Y, Qiu J, Li X, Ma K, Chen S, et al. Brain structures associated with eating behaviors in normal-weight young females. Neuropsychologia. 2019; http://doi.org/10.1016/j.neuropsychologia.2019.107171

130. Chen F, He Q, Han Y, Zhang Y, Gao X. Increased BOLD Signals in dlPFC Is Associated With Stronger Self-Control in Food-Related Decision-Making. Front Psychiatry. 2018; http://doi.org/10.3389/fpsyt.2018.00689

131. Wood SMW, Schembre SM, He Q, Engelmann JM, Ames SL, Bechara A. Emotional

eating and routine restraint scores are associated with activity in brain regions involved in urge and self-control. Physiol Behav. 2016; http://doi.org/10.1016/j.physbeh.2016.08.024

132. van Bloemendaal L, Veltman DJ, ten Kulve JS, Drent ML, Barkhof F, Diamant M, et al. Emotional eating is associated with increased brain responses to food-cues and reduced sensitivity to GLP-1 receptor activation. Obesity (Silver Spring). 2015; http://doi.org/10.1002/oby.21200

133. Bohon C. Greater emotional eating scores associated with reduced frontolimbic activation to palatable taste in adolescents. Obesity. 2014; http://doi.org/10.1002/oby.20759

134. Kerr KL, Moseman SE, Avery JA, Bodurka J, Zucker NL, Simmons WK. Altered Insula Activity during Visceral Interoception in Weight-Restored Patients with Anorexia Nervosa. Neuropsychopharmacology. 2016; http://dx.doi.org/10.1038/npp.2015.174

135. Steward T, Menchon JM, Jiménez-Murcia S, Soriano-Mas C, Fernandez-Aranda F. Neural Network Alterations Across Eating Disorders: A Narrative Review of fMRI Studies. Curr Neuropharmacol. 2018; http://doi.org/10.2174/1570159x15666171017111532

136. Herwig U, Dhum M, Hittmeyer A, Opialla S, Scherpiet S, Keller C, et al. Neural Signaling of Food Healthiness Associated with Emotion Processing. Front Aging Neurosci. 2016; http://doi.org/10.3389/fnagi.2016.00016

137. Bongers P, Jansen A. Emotional Eating Is Not What You Think It Is and Emotional Eating Scales Do Not Measure What You Think They Measure. Front Psychol. 2016; http://doi.org/10.3389/fpsyg.2016.01932

138. Bongers P, de Graaff A, Jansen A. “Emotional” does not even start to cover it: Generalization of overeating in emotional eaters. Appetite. 2016; http://dx.doi.org/10.1016/j.appet.2015.11.004

139. Bohon C, Stice E, Spoor S. Female emotional eaters show abnormalities in consummatory and anticipatory food reward: a functional magnetic resonance imaging study. Int J Eat Disord. 2009; http://doi.org/10.1002/eat.20615

140. García-García I, Kube J, Morys F, Schrimpf A, Kanaan AS, Gaebler M, et al. Liking and left amygdala activity during food versus nonfood processing are modulated by emotional context. Cogn Affect Behav Neurosci. 2020; http://doi.org/10.3758/s13415-019-00754-8

141. Blechert J, Goltsche JE, Herbert BM, Wilhelm FH. Eat your troubles away: Electrocortical and experiential correlates of food image processing are related to emotional eating style and emotional state. Biol Psychol. 2014; http://dx.doi.org/10.1016/j.biopsycho.2013.12.007

142. Schnepper R, Georgii C, Eichin K, Arend A-K, Wilhelm FH, Vögele C, et al. Fight, Flight, – Or Grab a Bite! Trait Emotional and Restrained Eating Style Predicts Food Cue Responding Under Negative Emotions. Front Behav Neurosci. 2020; http://doi.org/10.3389/fnbeh.2020.00091

143. Martín-Pérez C, Contreras-Rodríguez O, Vilar-López R, Verdejo-García A. Hypothalamic Networks in Adolescents With Excess Weight: Stress-Related Connectivity and Associations With Emotional Eating. J Am Acad Child Adolesc Psychiatry. 2019; https://doi.org/10.1016/j.jaac.2018.06.039

144. Sweeney P, Yang Y. Neural Circuit Mechanisms Underlying Emotional Regulation of Homeostatic Feeding. Trends Endocrinol Metab. 2017;

http://dx.doi.org/10.1016/j.tem.2017.02.006

145. Yang X, Casement M, Yokum S, Stice E. Negative affect amplifies the relation between appetitive-food-related neural responses and weight gain over three-year follow-up among adolescents. NeuroImage Clin. 2019; https://doi.org/10.1016/j.nicl.2019.102067

146. Maier SU, Makwana AB, Hare TA. Acute Stress Impairs Self-Control in Goal-Directed Choice by Altering Multiple Functional Connections within the Brain's Decision Circuits. Neuron. 2015; http://dx.doi.org/10.1016/j.neuron.2015.07.005

147. Tryon MS, Carter CS, Decant R, Laugero KD. Chronic stress exposure may affect the brain's response to high calorie food cues and predispose to obesogenic eating habits. Physiol Behav. 2013; http://dx.doi.org/10.1016/j.physbeh.2013.08.010

148. Val-Laillet D, Aarts E, Weber B, Ferrari M, Quaresima V, Stoeckel LE, et al. Neuroimaging and neuromodulation approaches to study eating behavior and prevent and treat eating disorders and obesity. NeuroImage Clin. 2015; http://dx.doi.org/10.1016/j.nicl.2015.03.016

149. Reichenberger J, Schnepper R, Arend A-K, Blechert J. Emotional eating in healthy individuals and patients with an eating disorder: evidence from psychometric, experimental and naturalistic studies. Proc Nutr Soc. 2020; http://doi.org/10.1017/S0029665120007004

150. Cosme D, Mobasser A, Zeithamova D, Berkman ET, Pfeifer JH. Choosing to regulate: does choice enhance craving regulation? Soc Cogn Affect Neurosci. 2018; http://doi.org/10.1093/scan/nsy010

151. Giuliani NR, Mann T, Tomiyama AJ, Berkman ET. Neural Systems Underlying the Reappraisal of Personally Craved Foods. J Cogn Neurosci. 2014;

http://doi.org/10.1162/jocn_a_00563

152. Giuliani NR, Cosme D, Merchant JS, Dirks B, Berkman ET. Brain Activity Associated With Regulating Food Cravings Predicts Changes in Self-Reported Food Craving and Consumption Over Time. Front Hum Neurosci. 2020; http://doi.org/10.3389/fnhum.2020.577669

153. Hollmann M, Hellrung L, Pleger B, Schlögl H, Kabisch S, Stumvoll M, et al. Neural correlates of the volitional regulation of the desire for food. Int J Obes (Lond). 2012; http://doi.org/10.1038/ijo.2011.125

154. Siep N, Roefs A, Roebroeck A, Havermans R, Bonte M, Jansen A. Fighting food temptations: the modulating effects of short-term cognitive reappraisal, suppression and up-regulation on mesocorticolimbic activity related to appetitive motivation. Neuroimage. 2012; http://doi.org/10.1073/pnas.1007779107

155. Maier SU, Hare TA. Social Neurobiology of Eating BOLD activity during emotion reappraisal positively correlates with dietary self-control success. Soc Cogn Affect Neurosci. 2020; http://doi.org/10.1093/scan/nsaa097

156. Siep N, Roefs A, Roebroeck A, Havermans R, Bonte M, Jansen A. Fighting food temptations: The modulating effects of short-term cognitive reappraisal, suppression and up-regulation on mesocorticolimbic activity related to appetitive motivation. Neuroimage. 2012; http://dx.doi.org/10.1016/j.neuroimage.2011.12.067

157. Yokum S, Stice E. Cognitive regulation of food craving: effects of three cognitive reappraisal strategies on neural response to palatable foods. Int J Obes (Lond). 2013; http://doi.org/10.1038/ijo.2013.39

158. Wolz I, Nannt J, Svaldi J. Laboratory-based interventions targeting food craving: A systematic review and meta-analysis. Obes Rev. 2020;

http://doi.org/10.1111/obr.12996

159. Meule A, Kübler A, Blechert J. Time course of electrocortical food-cue responses during cognitive regulation of craving. Front Psychol. 2013; http://doi.org/10.3389/fpsyg.2013.00669

160. Hutcherson CA, Plassmann H, Gross JJ, Rangel A. Cognitive Regulation during Decision Making Shifts Behavioral Control between Ventromedial and Dorsolateral Prefrontal Value Systems. J Neurosci. 2012; http://doi.org/10.1523/JNEUROSCI.6387-11.2012

161. Ferreira S, Veiga C, Moreira P, Magalhães R, Coelho A, Marques P, et al. Reduced Hedonic Valuation of Rewards and Unaffected Cognitive Regulation in Chronic Stress. Front Neurosci. 2019; http://doi.org/10.3389/fnins.2019.00724

162. Ferreira S, Moreira P, Magalhães R, Coelho A, Marques P, Portugal-Nunes C, et al. Frontoparietal hyperconnectivity during cognitive regulation in obsessive-compulsive disorder followed by reward valuation inflexibility. J Psychiatr Res. 2021; http://doi.org/10.1016/j.jpsychires.2020.11.008

163. Dalton B, Bartholdy S, Campbell IC, Schmidt U. Neurostimulation in Clinical and Sub-clinical Eating Disorders: A Systematic Update of the Literature. Curr Neuropharmacol. 2018; http://doi.org/10.2174/1570159x16666180108111532

164. Jáuregui-Lobera I, Martínez-Quiñones J V. Neuromodulation in eating disorders and obesity: a promising way of treatment? Neuropsychiatr Dis Treat. 2018; http://doi.org/10.2147/NDT.S180231

165. Bartholdy S, Musiat P, Campbell IC, Schmidt U. The potential of neurofeedback in the treatment of eating disorders: a review of the literature. Eur Eat Disord Rev. 2013; http://doi.org/10.1002/erv.2250

166. Sitaram R, Ros T, Stoeckel L, Haller S, Scharnowski F, Lewis-Peacock J, et al. Closed-loop brain training: the science of neurofeedback. Nat Rev Neurosci. 2017; http://doi.org/10.1038/nrn.2016.164

167. Lioi G, Cury C, Perronnet L, Mano M, Bannier E, Lécuyer A, et al. Simultaneous EEG-fMRI during a neurofeedback task, a brain imaging dataset for multimodal data integration. Sci Data. 2020; http://doi.org/10.1038/s41597-020-0498-3

168. Zotev V, Phillips R, Yuan H, Misaki M, Bodurka J. Self-regulation of human brain activity using simultaneous real-time fMRI and EEG neurofeedback. Neuroimage. 2014; http://dx.doi.org/10.1016/j.neuroimage.2013.04.126

169. Zotev V, Mayeli A, Misaki M, Bodurka J. Emotion self-regulation training in major depressive disorder using simultaneous real-time fMRI and EEG neurofeedback. NeuroImage Clin. 2020; https://doi.org/10.1016/j.nicl.2020.102331

170. Herwig U, Lutz J, Scherpiet S, Scheerer H, Kohlberg J, Opialla S, et al. Training emotion regulation through real-time fMRI neurofeedback of amygdala activity. Neuroimage. 2019; http://doi.org/10.1016/j.neuroimage.2018.09.068

171. Paret C, Ruf M, Gerchen MF, Kluetsch R, Demirakca T, Jungkunz M, et al. fMRI neurofeedback of amygdala response to aversive stimuli enhances prefrontal-limbic brain connectivity. Neuroimage. 2016; http://dx.doi.org/10.1016/j.neuroimage.2015.10.027

172. Yu L, Long Q, Tang Y, Yin S, Chen Z, Zhu C, et al. Improving Emotion Regulation Through Real-Time Neurofeedback Training on the Right Dorsolateral Prefrontal Cortex: Evidence From Behavioral and Brain Network Analyses. Front Hum Neurosci. 2021; http://doi.org/10.3389/fnhum.2021.620342

173. Zotev V, Krueger F, Phillips R, Alvarez RP, Simmons WK, Bellgowan P, et al. Self-

regulation of amygdala activation using real-time FMRI neurofeedback. Domschke K, editor. PLoS One. 2011; http://doi.org/10.1371/journal.pone.0024522

174. Young KD, Siegle GJ, Zotev V, Phillips R, Misaki M, Yuan H, et al. Randomized Clinical Trial of Real-Time fMRI Amygdala Neurofeedback for Major Depressive Disorder: Effects on Symptoms and Autobiographical Memory Recall. Am J Psychiatry. 2017; http://doi.org/10.1176/appi.ajp.2017.16060637

175. Imperatori C, Valenti EM, Della Marca G, Amoroso N, Massullo C, Carbone GA, et al. Coping food craving with neurofeedback. Evaluation of the usefulness of alpha/theta training in a non-clinical sample. Int J Psychophysiol. 2017; http://dx.doi.org/10.1016/j.ijpsycho.2016.11.010

176. Leong SL, Vanneste S, Lim J, Smith M, Manning P, De Ridder D. A randomised, double-blind, placebo-controlled parallel trial of closed-loop infraslow brain training in food addiction. Sci Rep. 2018; http://doi.org/10.1038/s41598-018-30181-7

177. Frank S, Lee S, Preissl H, Schultes B, Birbaumer N, Veit R. The obese brain athlete: self-regulation of the anterior insula in adiposity. Luque RM, editor. PLoS One. 2012; http://doi.org/10.1371/journal.pone.0042570

178. Kohl SH, Veit R, Spetter MS, Günther A, Rina A, Lührs M, et al. Real-time fMRI neurofeedback training to improve eating behavior by self-regulation of the dorsolateral prefrontal cortex: A randomized controlled trial in overweight and obese subjects. Neuroimage. 2019; https://doi.org/10.1016/j.neuroimage.2019.02.033

179. Schmidt J, Martin A. Neurofeedback Reduces Overeating Episodes in Female Restrained Eaters: A Randomized Controlled Pilot-Study. Appl Psychophysiol Biofeedback. 2015; http://doi.org/10.1007/s10484-015-9297-6

180. Schmidt J, Martin A. Neurofeedback Against Binge Eating: A Randomized Controlled

Trial in a Female Subclinical Threshold Sample. Eur Eat Disord Rev. 2016; http://doi.org/10.1002/erv.2453

181. Lackner N, Unterrainer H-F, Skliris D, Shaheen S, Dunitz-Scheer M, Wood G, et al. EEG neurofeedback effects in the treatment of adolescent anorexia nervosa. Eat Disord. 2016; http://doi.org/10.1080/10640266.2016.1160705

182. Barreiros AR, Almeida I, Baía BC, Castelo-Branco M. Amygdala Modulation During Emotion Regulation Training With fMRI-Based Neurofeedback. Front Hum Neurosci. 2019; http://doi.org/10.3389/fnhum.2019.00089

183. Braden A, Musher-Eizenman D, Watford T, Emley E. Eating when depressed, anxious, bored, or happy: Are emotional eating types associated with unique psychological and physical health correlates? Appetite. 2018; https://doi.org/10.1016/j.appet.2018.02.022

184. Schmidt J, Martin A. The Influence of Physiological and Psychological Learning Mechanisms in Neurofeedback vs. Mental Imagery Against Binge Eating. Appl Psychophysiol Biofeedback. 2020; https://doi.org/10.1007/s10484-020-09486-9

185. Imperatori C, Mancini M, Della Marca G, Valenti EM, Farina B. Feedback-Based Treatments for Eating Disorders and Related Symptoms: A Systematic Review of the Literature. Nutrients. 2018; http://doi.org/10.3390/nu10111806

186. Ihssen N, Sokunbi MO, Lawrence AD, Lawrence NS, Linden DEJ. Neurofeedback of visual food cue reactivity: a potential avenue to alter incentive sensitization and craving. Brain Imaging Behav. 2017; http://doi.org/10.1007/s11682-016-9558-x

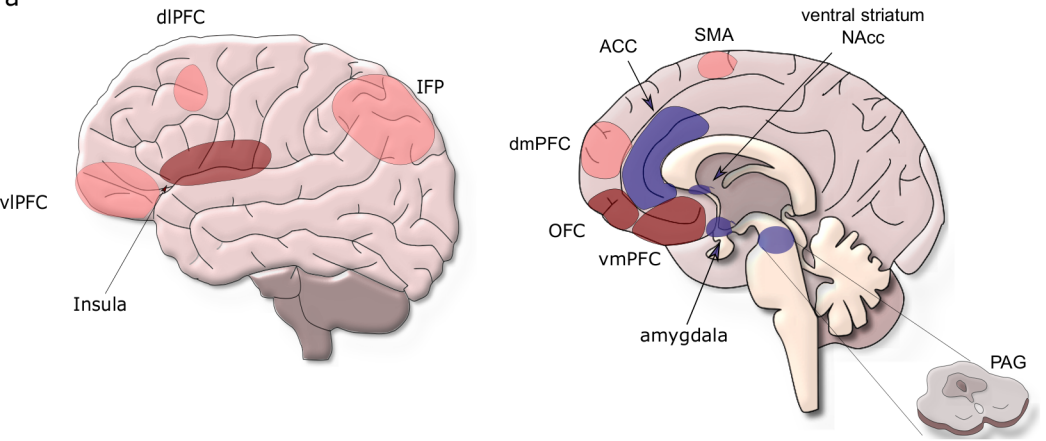
187. Spetter MS, Malekshahi R, Birbaumer N, Lührs M, van der Veer AH, Scheffler K, et al. Volitional regulation of brain responses to food stimuli in overweight and obese subjects: A real-time fMRI feedback study. Appetite. 2017; http://doi.org/10.1016/j.appet.2017.01.032

188. Donofry SD, Stillman CM, Erickson KI. A review of the relationship between eating behavior, obesity and functional brain network organization. Soc Cogn Affect Neurosci. 2020; https://doi.org/10.1093/scan/nsz085

Legend Fig1.eps

Figure 1. a. Major brain areas involved in emotional processing, including emotion generation and emotion regulation. Purple boxes represent areas more related to emotion generation (Periaqueductal Grey, PAG; amygdala; Nucleus Accumbens, NAcc; striatum ventral). Pink boxes represent brain areas more specific to affect modulation and emotion regulation (ventrolateral, dorsolateral, dorsomedial prefrontal cortices, vlPFC; dlPFC; dmPFC; InFerior Parietal region IFP; supplementary motor area, SMA). Red boxes represent brain areas that can be involved in both processes (orbitofrontal cortex, OFC/vmPFC; insula; anterior cingulate cortex, ACC). **b**. **Simplified scheme of common brain regions related to both eating behavior and emotional processing sharing similar functions.** Red boxes concern affect modulation whereas blue boxes are related to emotion initiation. Scheme designed with Inkscape (Inkscape Project. (2020). Inkscape. Retrieved from https://inkscape.org).

a



b

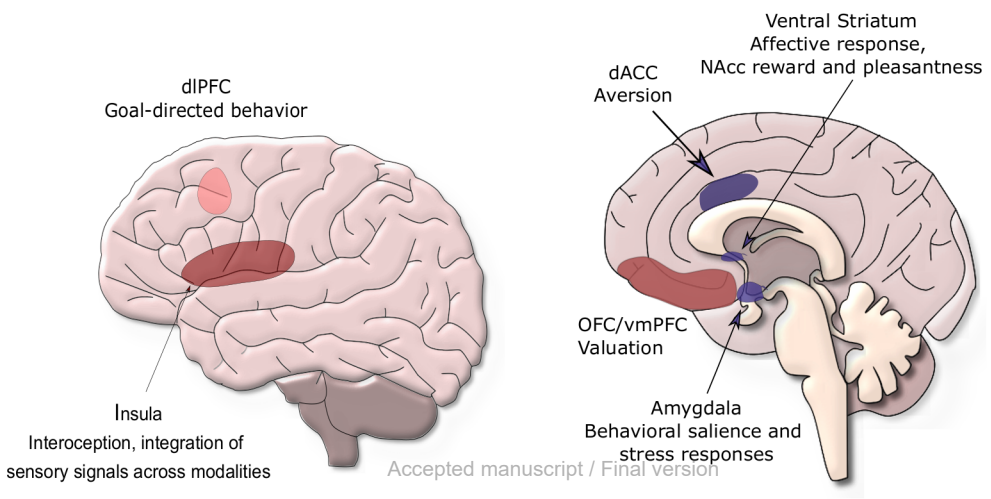


Table 1. Details of MRI studies in which emotional eating has been assessed. Details include population criteria (e.g. age, BMI, health condition), experimental paradigm, type of the questionnaire and major results of interest

References	Population	Age	BMI	Task	Questionnaire	Results of interest
Bohon, 2014	n=162 adolescents (82 females), healthy	15.3 (±1.1)	20.8 (±1.9), range [18-25]	Chocolate milkshake fMRI paradigm	DEBQ	**Anticipation of Taste** No significant relation between emotional eating scores and brain response to anticipation of milkshake receipt **Taste Receipt** Negative correlation between emotional eating scores and activation in the right thalamus, putamen and caudate, and the left caudate, putamen, insula and OFC
Chen et al. 2018	n=43 healthy females	20.47 (±1.75), range [18-25]	23.05 (±4.44), range [15.56-29.32]	Food rating and decision-making task (high and low calorie food pictures)	DEBQ	**Success in self-control** Significant activated BOLD responses in dlPFC **Failure in self-control** Significant activated BOLD responses in MCC **Correlation With DEBQ** No significant correlation with emotional eating and brain analysis
Song et al. 2019	n=158 normal-weight healthy females	19.40 (±1.52), range [17-25]	20.59 (± 1.45), range [18.02-23.98]	Structural MRI scan (voxel-based morphometry)	TFEQ-R18	**Uncontrolled eating scores** Positively associated with the GMV of both sides of the cerebellum, and negatively correlated with the GMV on the left side of the ACC, MCC and SMAs **Restrained eating scores** Positive correlation scores with the GMV of the right side of precuneus **Emotional eating scores** No correlation with any brain structure
Van Bloemendaal et al. 2015	n=48 individuals	normoglycemic	normoglycemic	Passive viewing task of high-calorie, low-calorie food and neutral pictures	DEBQ	**Association between emotional eating and brain responses to food pictures (no administration, placebo condition)** **Food versus nonfood pictures** Positive correlation between emotional eating and right insula activity for obese individuals, bilateral amygdala, bilateral OFC and right insula for T2DM and no correlation for normoglycemic individuals
	n=16 healthy lean individuals	57.8 (±1.9)	23.2 (±0.4)			

	n=16 type 2 diabetes obese patients (T2DM) n=16 normoglycemic obese individuals	T2DM 61.4 (±1.5) Obese 58.0 (±2.1) Range [40-70]	T2DM 34.0 (±0.9) Obese 32.6 (±0.7)	randomized, placebo-controlled, crossover study (GLP-1 receptor agonist)		**High versus low calorie food pictures** Positive correlation of emotional eating and brain activity in left insula for healthy individuals, in right insula for obese individuals and in right inferior OFC for T2DM patients.
Wood, 2017	n=20 healthy individuals (12 females)	19.8 (±1.0), range [18-22]	22.6 (±3.0), range [18.5-31.3]	High-calorie food go and low-calorie food nogo task (HGo/LNogo), Low-calorie food go and high-calorie food nogo task (LGo/HNogo)	WREQ	**HGo vs. LGo contrast** Positive correlation between bilateral dlPFC and left insula activity with emotional eating **HGo vs. HNogo contrast** Positive correlations between emotional eating and routine restraint with left dlPFC activity

" x ± x" means "average ± standard deviation"; Body Mass Index (BMI; kg/m²); functional magnetic resonance imaging (fMRI); Dutch Eating Behavior Questionnaire (DEBQ); Three Eating Factor Questionnaire – Revised 18 items (TFEQ-R18); Weight-Related Eating Questionnaire (WREQ); type 2 diabetes (T2DM); glucagon-like peptide-1 (GLP-1); Blood Oxygen-level dependant (BOLD); grey matter volume (GMV); orbitofrontal cortex (OFC); dorsolateral prefrontal cortex (dlPFC); midcingulate cortex (MCC); anterior cingulate cortex (ACC); supplementary motor area (SMA); high-go and low-nogo (HGo/LNogo); low-go and high-nogo (LGo/HNogo).

Table 2. Details of neuroimaging studies related to section 4.2 (Emotion induction during food processing), 4.3 (Stress and emotional eating) and 4.4 (Emotion regulation of food craving). Details include population criteria (*e.g.* age, BMI, health condition), neuroimaging modality, experimental paradigm of the studies and major results of interest.

References	Modality	Paradigm	Population	Age	BMI	Task	Results of interest
Blechert et al. 2014	EEG	Mood induction	n=45 healthy women	LEE 24.5 (±6.01)	LEE 21.4 (±2.37), range [17.8-27.3]	Passive viewing and rating food pictures task with negative or neutral emotion induction	Higher parieto-occipital LPP amplitude for HEE than LEE, no effect of mood condition
			n=25 HEE n=20 LEE	HEE 22.8 (±2.78)	HEE 22.5 (±4.01) Range [17.1-33.8]		**Contrast negative > neutral mood** higher relative positivity (reduced negativity) LPP
Bohon, 2009	fMRI	Mood induction	n=21 female college students	20.1 (±2.0)	24.4 (±4.5)	Consummatory and anticipatory food reward task in a negative or neutral mood state	**Interaction between emotional eating and mood (anticipatory condition)** Emotional eaters showed greater activation in the left parahippocampal gyrus and left ventral ACC in negative mood compared to neutral mood state
			n=10 high emotional eating score n=11 low emotional eating score				**Interaction between emotional eating and mood (consummatory condition)** Emotional eaters showed greater activation in the right pallidum for milkshake receipt in a negative mood state

Garcia-Garcia et al. 2020	fMRI	Mood induction	n=58 healthy women	n.a. range [20-35]	25.63 (±5.84) range [17.67-46.83]	Emotional priming task with a fMRI event-related food processing task	**Contrast "foods > objects" depending on the emotional priming condition** Negative priming: higher activity in the occipital pole and lateral OFC; Neutral priming: higher activity in the occipital pole, lateral OFC, insula and amygdala; Positive priming: bilateral activity in the lateral OFC, occipital pole, insula and left amygdala **Interaction with the amygdala** Higher activity in left **amygdala** in the contrast after neutral priming relative to negative priming
Maier et al. 2015	fMRI	Stress	n=51 healthy males	21 (±2)	22.55 (±2.06)	Stress induction task (SECP) and food choice task	**Functional connectivity** Greater positive functional connectivity between vmPFC and portions of the amygdala and striatum (stressed > control participants) when choosing the tastier item Greater vmPFC connectivity with the Amygdala, ventral striatum and bilateral insula during tastier choices for the stressed group Stronger correlation of vmPFC connectivity during tastier choices with individual cortisol levels compared to self-reported perceived stress in the striatum and extended amygdala
Martin-Perez et al. 2019	Resting-state functional connectivity (fMRI)	Stress	n=32 NW	n.a. Range [10-19]	BMI percentile between 5th and 85th for NW and at or above 85th for EW	Virtual reality version of the TSST, cortisol salivary measure and DEBQ	**Associations between hypothalamic functional connectivity and stress response** Significant positive association with the connectivity in the lateral hypothalamus (LH)-NAcc network and the LH-midbrain network in OW individuals

			n=22 EW females adolescents				**Associations of emotional eating behavior** No difference of emotional eating score between groups, positive association between functional connectivity in LH-midbrain network and emotional eating in EW individuals, no significant correlation in NW individuals
Meule et al. 2013	EEG	Emotion regulation	n=26 healthy female students	23 (±2.23) range [18-27]	23.12 (±2.80)	Cognitive regulation of food craving task	Positive correlation with emotional eating score and parieto-occipital LPP
					Range [17.60-27.80]		Higher LPP amplitude in the HC-LATER condition than all other conditions
Morawetz et al. 2020	fMRI	Emotion regulation	n=35 healthy participants (29 females)	23.17 (±3.44)	21.26 (±2.38)	Emotion regulation task (look vs decrease) and food choice task (chose or reject)	**Contrasted downregulation condition with the look condition** Increased activity in several regions in the PFC (middle frontal gyrus), SMA, and supramarginal gyrus
							Interaction for choice and emotion regulation Decreasing emotions correlated with less reduced activity in the vmPFC during the choose condition compared with rejection trials, rejected food items associated with a negative signal change in the striatum (for each regulation condition)
Schnepper et al. 2020	EEG	Mood induction	n=69 healthy women	21.9 (±3.77), range [16-50]	22.3 (±3.09), normal weight range	Emotional eating task	No correlation with parieto-occipital P300 activity and emotional eating
							Increased P300 amplitudes to food images under negative emotion in high-reacting and low restrained individuals

Tryon et al. 2013	fMRI	Stress	n=30 women	Tot=39.7 (±2.3)	Tot=25.6 (±0.9), range [18-39]	Stress induction task (TSST) before passive viewing food pictures task	**HiCal vs. control condition** HCS group: Increased ACC activity, amygdala, right medial OFC, left putamen and caudate regions, reduced activity in the contralateral side of the caudate and putamen, left anterior PFC cortex, right ACC, bilateral dlPFC and the left lateral OFC ; LCS group: Increased bilateral dlPFC activity
			n=16 high chronic stressed (HCS)	HCS=46.81 (±1.09)	HCS=26.76 (±1.35)		**HiCal vs LoCal** HCS group: higher activity in the amygdala, caudate, right ACC, left putamen and right medial OFC and reduced activity in the left anterior PFC and left dlPFC ; LCS group : greater activity in the left anterior PFC, caudate regions and the left dlPFC
			n=14 low chronic stress group (LCS)	LCS=31.64 (±3.64)	LCS=24.27 (±1.26)		**Functional connectivity** (HiCal vs LoCal contrast) HCS group: Enhanced functional connectivity with the amygdala in the bilateral thalamus, left inferior parietal lobe and left putamen ; LCS group : enhanced functional connectivity with the amygdala in the right ACC, left anterior PFC, left caudate, left insula and left dlPFC
Yang et al. 2019	fMRI	Stress	n=135 healthy adolescents (73 females)	15.01 (±0.87)	21.16 (±2.25), range [18-25]	Food picture and food receipt paradigm	**Baseline negative affect as a moderator**
						3 years follow-up study of BMI	**Appetizing > unappetizing food images** Higher negative affect was associated with left hippocampus activity and with higher future weight gain

	Milkshake > tasteless solution Positive correlation between high negative affect with vermis activation and future weight gain with LFHS milkshake and positive correlation with high negative affect and high precuneus activation to the LFLS milkshake and greater weight gain
	Stressful events as a moderator
	Milkshake > tasteless Individuals with high stressful events showed a positive relation between right middle occipital gyrus activation and BMI gain, individuals with fewer stressful events showed lower right middle occipital gyrus activity associated with higher weight gain

" x ± x" means "average ± standard deviation"; Electroencephalography (EEG); Low Emotional Eaters (LEE); High Emotional Eaters (HEE); Late Positive Potential (LPP); Anterior Cingulate Cortex (ACC); Orbitofrontal Cortex (OFC); Socially Evaluated Cold Pressor Test (SECPT); ventromedial prefrontal cortex (vmPFC); Trier Social Stress task (TSST); normal-weight (NW), excess weight (EW); lateral hypothalamus (LH); Nucleus accumbens (NAcc); Normal Weight (NW); Excess Weight (EW); Dutch Eating Behavior Questionnaire (DEBQ); high-calorie reappraisal (HC-LATER); Supplementary Motor Area (SMA) ; body mass index (BMI; kg/m^2); high-calorie and low-calorie (HiCal, LoCal); high chronic stressed (HCS); low chronic stressed (LCS); Prefrontal Cortex (PFC); dorsolateral Prefrontal Cortex (dlPFC); Low Fat High Sugar (LFHS); Low Fat Low Sugar (LFLS).