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1 **Title Page**

2 **Interactions between emotions and eating behaviors: Main issues, neuroimaging**
3 **contributions, and innovative preventive or corrective strategies**

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17 **Abstract**

18 Emotional eating is commonly defined as the tendency to (over)eat in response to emotion.

19 Insofar as it involves the (over)consumption of high-calorie palatable foods, emotional eating

20 is a maladaptive behavior that can lead to eating disorders, and ultimately to metabolic

21 disorders and obesity. Emotional eating is associated with eating disorder subtypes and with

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

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

36

38 **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
dIPFC	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
vIPFC	Ventrolateral prefrontal cortex
vmPFC	Ventromedial prefrontal cortex
VTA	Ventral tegmental area

39

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59

60

61 **1. Introduction**

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

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

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

105 **2. Emotion processing and the cognitive-emotional brain**

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

109 of interactions among subjective and objective factors, and encompasses both neural and
110 hormonal systems. These systems integrate affective experiences, generate cognitive
111 processes, activate physiological adjustments, and lead to a behavior that is generally goal-
112 directed and adaptive [17]. The function of emotion is to decouple a stimulus from the
113 behavioral response, thus allowing for flexible adaptation to environmental contingencies
114 [18]. *Emotion generation* takes the form of a situation-attention-appraisal-response sequence
115 [19], and commonly begins with the perception of a stimulus within a context. The emotional
116 importance of this stimulus is appraised by the individual, triggering an affective,
117 physiological and behavioral response [18]. *Emotion regulation* refers to the process engaged
118 to modify the experience or expression of this emotion, and can be studied at both neural and
119 behavioral levels [20]. From a philosophical to a clinical perspective, advances in cognitive
120 neurosciences, through the use of neuroimaging, have shed light on the major brain regions
121 involved in emotion processing, be it for emotion generation or regulation. To provide an
122 overview of the brain regions involved in these two aspects, we deal separately with the
123 neural systems involved in generation or regulation in this review, even though they partially
124 overlap (Fig. 1a) [21–23].

125 *Emotion generation* entails the activity of core limbic structures such as the amygdala,
126 which is involved in the perception and encoding of stimuli that are relevant to current or
127 chronic affective goals, and the nucleus accumbens (NAcc), a major component of the ventral
128 striatum that is involved in learning which cues predict rewarding or reinforcing outcomes
129 [21,24]. Other brain regions besides the limbic system also play a role in emotion generation:
130 the periaqueductal gray, which is involved in the coordination of behavioral and physiological
131 emotional responses [21,22,25], and cortical regions such as the ventromedial prefrontal
132 cortex (vmPFC), which is involved in the integration of the affective valence of a specific
133 stimulus elaborated within the amygdala and ventral striatum [24]. Moreover, because of its

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

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

159 goals [35]. Thus, consistent involvement of the PFC (left dlPFC, bilateral vlPFC, and
160 dmPFC), bilateral parietal, left temporal brain regions and motor areas (supplementary motor
161 area; SMA) has been reported in several emotion regulation meta-analyses [22,31,36]. In
162 addition, generating an emotional response brings into play the ACC, NAcc, and insula [32].
163 The dorsal ACC is reported to mediate feelings of negative emotions such as aversion, fear
164 and anxiety, in relation with the vmPFC [24,25,34], while the ventral ACC is involved in fear
165 extinction [25,34]. Overall, emotion processing is driven by brain regions involved in
166 perception (sensory), integration, valuation and cognition, encompassing all the stages of
167 emotion generation and regulation. We can thus distinguish the brain regions involved in
168 triggering affective responses (e.g., amygdala, ventral striatum, insula) from those involved in
169 modulating affect (e.g., ACC, dlPFC, vlPFC, dmPFC, vmPFC) [26]. Nevertheless, the
170 mechanism by which these brain areas interact, depending on the emotion regulation strategy,
171 remains an active research topic [25]. One study found that activity of the amygdala and
172 anterior insula decreased during reappraisal and increased during suppression, whereas
173 activity of the PFC increased with both strategies [37]. Furthermore, downregulation of the
174 amygdala and striatal activity during emotion regulation are probably brought about by
175 increased activity of prefrontal areas [22,24,32,38]. Conversely, reduced recruitment of the
176 dlPFC and vlPFC, as well as greater amygdala activity, have been observed during reappraisal
177 in mood and other mental disorders [39]. This suggests that unsuccessful emotion regulation
178 can be explained by insufficient recruitment of the prefrontal networks involved in top-down
179 regulation, resulting in decreased reactivity of limbic structures. In line with this hypothesis,
180 in an fMRI-based meta-analysis related to cognitive reappraisal, patients with mood or
181 anxiety disorders exhibited reduced recruitment of the frontoparietal network (posterior
182 cingulate cortex, PCC, dmPFC, angular gyrus and vlPFC), compared with healthy controls
183 [40]. In addition to the task-based fMRI approach, resting-state functional connectivity (rs-

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

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

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

227 **3. Eating behavior and main implications of emotion processing**

228 **3.1. Similar brain regions involved in eating behavior and emotion processing**

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

233 structures and networks that process both environmental and cognitive factors [51,52]. There
234 is strong evidence that these loops are interconnected and capable of influencing each other
235 [53]. However, the nonhomeostatic neural pathways are less well understood, and studies
236 involving paradigms with ingestive behavior have yielded contradictory results in extra-
237 hypothalamic brain regions [52]. The nonhomeostatic loop involves (Fig. 1b) the amygdala
238 for behavioral salience and stress responses, the hippocampus and PCC for their role in
239 memory and learning in the context of eating behavior, and the dlPFC for its function in goal-
240 directed behavior [54]. In addition, the insula plays a role in interoception (perception of
241 sensations from inside the body), homeostasis, and the integration of sensory signals across
242 modalities, while the OFC is regarded as a secondary gustatory cortex [55]. The insula (taste-
243 responsive neurons) sends projections to the OFC and is involved in the perceived
244 pleasantness of taste [52]. The vmPFC also plays a key role in the valuation system, and
245 therefore in food decision-making [56], as well as in conditioned motivation to eat [57]. Both
246 the OFC and vmPFC are involved in assigning incentive motivational values to food stimuli
247 [54]. The striatum is activated during exposure to food cues, with striatal subregions being
248 differently involved: the NAcc mediates reward prediction, while the caudate nucleus is
249 involved in feedback processing, and the putamen in the mediation of habitual behavior [54].
250 Moreover, the ventral tegmental area (VTA) is the starting point of dopaminergic neurons that
251 project toward the reward system and activate it. This catalogue of brain regions highlights
252 the fact that food perception and eating share brain regions involved in perception, cognitive
253 control, reward, and more especially emotion processing (Figs 1a and 1b).

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

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

269 **3.2. Nonhomeostatic factors, food reward and emotional eating**

270 The regulation of eating behavior results from a balance between hunger and satiety,
271 and is governed by both metabolic and nonmetabolic factors, such as exposure to food cues,
272 cognitive and emotional state, and personal and cultural beliefs. These factors can also
273 indirectly influence the homeostatic regulation of food intake, especially through reward and
274 cognitive processes. These processes mainly take place in corticolimbic structures such as the
275 PFC, amygdala and ventral striatum, and can trigger food intake through an executive cortical
276 decision, independently of physiological needs [15]. Here, we focus on the nonmetabolic
277 factors influencing eating behavior that are involved in top-down processes. Prefrontal areas
278 are involved not only in emotion regulation, but also in executive functioning (including
279 inhibitory and attentional control, as well as cognitive flexibility) and the cognitive control of
280 eating behavior [62], which it does by modulating appetitive regions (i.e., OFC, ventral
281 striatum, insula, and amygdala/hippocampus complex) [51]. Furthermore, there is an interplay
282 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 ¹¹C 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

308 food intake in response to emotion than obese individuals [7,71], possibly depending on
309 emotional features [7]. A recent study showed that normal-weight individuals can increase
310 food intake in response to negative emotions and that emotional overeating is negatively
311 correlated with alcohol consumption, suggesting that different strategies can be adopted to
312 cope with negative emotions [74]. Eating in response to negative emotions is problematic in
313 terms of public health, as the chosen foods are often characterized as unhealthy. Commonly
314 acknowledged to affect food intake and to be involved in eating behavior [14,15,63], emotion
315 processing has been studied in the context of EDs [51,62,75] such as obesity and/or BED
316 [12,76–78], but has been poorly investigated in the context of normal eating behavior.
317 Research has shown that patients with EDs are more predisposed to experience alexithymia
318 (i.e., difficulty identifying and describing feelings) [79], maladaptive emotion regulation
319 [62,75,78], and deficits in emotion differentiation, which is defined as the tendency to
320 experience vague affective states rather than well-defined emotions [80]. They are also more
321 predisposed to dysfunctional processing of emotions related to food information [81]. In this
322 study, reduced control over emotions and reduced attraction to food pictures were mediated
323 by negative affect, supporting the predictive role of negative affect in ED symptomatology
324 [81]. The use of inappropriate emotion regulation strategies in response to the situational
325 demand is a feature of various psychiatric disorders [82], suggesting that emotional eating
326 may reflect a dysregulation of emotion processing in patients with EDs. As emotion
327 dysregulation can be involved in disordered eating behavior, there is growing interest in the
328 assessment of emotion regulation in the context of eating control. Recent studies have focused
329 on *food craving*, which refers to the urge to eat. According to Giuliani and colleagues (2015),
330 food craving can be regarded as an affective state and can be modulated through regulatory
331 strategies such as cognitive reappraisal [83]. Over the past decade, the number of fMRI
332 neuroimaging studies related to reappraisal strategies in the context of food craving has

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

338 **3.3. Toward a more integrated view of emotion, HPA axis, gut microbiota,** 339 **and neuroimmune interactions**

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

358 including one in which sucrose-fed rats were found to secrete less corticosterone after acute
359 stress [90–93]. The BLA is clearly essential for dampening stress, as sucrose-fed rats with
360 bilateral lesions in the BLA had normal corticosterone levels in response to stress [90],
361 whereas nonlesioned sucrose-fed rats exhibited increased structural plasticity (as
362 demonstrated by the increased expression of genes related to structural plasticity) in this
363 limbic region [90]. These results suggest that stress relief can be partly mediated through the
364 reward system, indirectly promoting synaptic remodeling [16,90]. However, it is unclear
365 whether eating comfort foods reduces stress by blunting HPA axis activation, stimulating the
366 dopaminergic reward system, or both [16]. In any event, this phenomenon has important
367 implications for weight issues. Overweight and obesity have been shown to be associated with
368 emotion and stress-related eating [94], suggesting that overeating to cope with stress is a facet
369 of emotional eating, just like overeating to cope with emotions. As emotional eaters are more
370 likely to experience emotion recognition and management difficulties, eating may serve as a
371 fallback. In addition, emotional eating has been shown to mediate the association between
372 depression and obesity in young adults [95]. A similar observation was reported in a
373 prospective cohort study, where emotional eating predicted a greater increase in body mass
374 index (BMI) associated with shorter sleep duration, which itself is considered to be a stressor
375 [96]. Regarding cortisol reactivity, female students with high emotional eating scores and
376 blunted cortisol reactivity were found to consume more kilocalories after a stress induction
377 task [97].

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

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

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

414 In addition, the microbiota has attracted a great deal of interest over the past decade,
415 with many studies highlighting its role in the gut-brain axis and the association between
416 microbiota changes and behavioral and physiological modifications [111]. With the use of
417 antibiotics and probiotics in animal models and the use of germ-free animal models, many
418 studies have shown that behavioral traits and mental illnesses (e.g., depression) may partly
419 depend on microbiota diversity and metabolic activity [112]. The microbiota appears to
420 interact closely with the immune system, through immune modulation [112,113]. In addition,
421 in neuropsychiatric disorders, alterations in immune homeostasis due to host–microbiota
422 interactions may involve changes in HPA axis activity that could be activated by pro-
423 inflammatory cytokines [114]. Moreover, the microbiota can affect neurotransmitter content
424 [114] through direct synthesis, in particular serotonin from tryptophan (5-HT) [110], or
425 catecholamine (norepinephrine and DA [115]) bioavailability. These monoaminergic
426 neurotransmitters (5-HT and DA) play a major role in brain circuits involved in the regulation
427 of mood, reward and food intake [110,116] and their dysregulation is involved in
428 neuropsychiatric pathologies (e.g., depressive disorders). It is therefore not surprising that gut
429 microbiota dysbiosis may be involved in mood disturbance and behavioral changes, through
430 changes in both immune response and neurotransmission.

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

433 [117]. Therefore, emotional eating, which may trigger intake of comfort foods, may
434 potentially affect gut microbiota, owing to harmful eating habits involving the consumption of
435 palatable food with a high fat and/or sugar content and low fiber content. The Western diet,
436 characterized by a low fiber content, results in reduced short-chain fatty acid synthesis from
437 microbiota fermentation, which is known to modulate inflammation [118]. This type of diet is
438 also associated with alteration of the gut microbiota ecosystem [119]. Stress can also affect
439 the gut microbiome [120]. As illustrated in Panduro et al. (2017)'s review, negative emotions
440 arising from alterations in the energy balance can induce chronic activation of the HPA axis,
441 resulting in higher cortisol levels [121]. The latter may induce dysbiosis, leading to altered
442 intestinal membrane permeability and activation of pro-inflammatory processes [121].
443 Additionally, chronic inflammation can lead to depressive-like behavior in mice [122]. These
444 data suggest that an unhealthy diet can induce dysbiosis and inflammation, which in turn
445 reinforce the onset of negative emotions, as altered gut microbiota can influence mood,
446 thereby reinforcing the emotional eating behavior. Although an increasing number of studies
447 have been investigating the relationship between EDs and microbiota homeostasis [123],
448 specific gut microbiota profiles have not yet been described in emotional eaters. Moreover, it
449 is important to bear in mind that there is a direct bidirectional interaction between the enteric
450 nervous system and the brain. Appositely referred to as a *gut feeling*, this relationship engages
451 various effector pathways that play an important role in feeling states and executive
452 functioning [124]. These results suggest that the way stress interacts with emotion control and
453 emotion regulation processes depends on the type of emotion regulation strategy used, the
454 nature of the stressor (e.g., acute, delayed, or chronic), HPA activation, and individual health
455 status (e.g., immune status, microbiota diversity, affective disorders), not forgetting hormonal
456 effects (sex/gender) and age. The complex interaction between gut and brain in this context

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

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

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

469 **4.1. Neural correlates of emotional eating**

470 Emotional eating is commonly assessed with questionnaires, such as the Dutch Eating
471 Behavior Questionnaire (DEBQ) [125], which evaluates cognitive restriction, externality and
472 emotion in the context of eating behavior, and the Three-Factor Eating Questionnaire (TFEQ)
473 [126]. The Weight-Related Eating Questionnaire combines items from the DEBQ and TFEQ
474 to measure routine and compensatory restraint, external eating, and emotional eating related
475 to weight loss [127]. The emotional eating items in these questionnaires assess the tendency
476 to eat in response to negative emotions. Other items assess the level of emotions, the
477 frequency and amount of food intake [128], or the intensity of the desire to eat [8].
478 Neuroimaging studies investigating eating behavior, including emotional eating behavior, are
479 scarce, but have nonetheless brought new insights into the neural correlates of emotional
480 eating. Some have not reported any link between brain structures [129] or brain activation

481 (BOLD response) [130] and emotional eating scores, but others have. In one study featuring a
482 Go/NoGo paradigm with high- and low-calorie food pictures, the emotional eating score was
483 positively correlated with left dlPFC and left insula activity in the High Go / Low NoGo
484 condition, and with left dlPFC activity in the Low Go / High NoGo condition [131]. As the
485 dlPFC is involved in self-control, as well as in goal-directed behavior, it is hardly surprising
486 that its activity was correlated with emotional eating in a paradigm requiring inhibitory
487 control. This suggests that eating and emotional processing share a common cognitive
488 process. In a second study, the emotional eating score was correlated with the activity of (i)
489 the left insula in lean participants, (ii) the amygdala and OFC in patients with type-2 diabetes,
490 and (iii) the right insula in obese individuals [132]. In line with this observation, the
491 presentation of high- versus low-calorie food pictures highlighted a positive correlation
492 between emotional eating and left insula activity in healthy individuals [132]. These results
493 suggest that in both healthy and obese individuals with or without metabolic disorders, higher
494 emotional eating scores are associated with increased responses in appetite and reward brain
495 regions that are also involved in emotion processing, specifically the OFC and insula. In a
496 third study, participants' emotional eating score when consuming a milkshake was negatively
497 correlated with the activity of the bilateral putamen and caudate, as well as the left insula and
498 OFC [133]. Taken together, these results show that the same brain regions are differentially
499 correlated with emotional eating, depending on the sensory demands (watching or tasting).

500 Although these studies should be interpreted with caution, owing to many differences in terms
501 of experimental paradigms, the OFC and insula appear to be relevant structures. The OFC is
502 closely involved in the representation of pleasantness of reward and the attribution of
503 affective value to food, and also plays a role in emotion, as food smell and taste can elicit
504 emotional states [55]. In the specific context of emotions, the role of the OFC is to represent
505 the reward value of goal-directed behavior through the learning of the stimulus-reinforcer

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

523 As demonstrated by Herwig et al. (2016), food pictures can induce appetitive emotions
524 [136]. Thus, correlations between the passive viewing of food pictures and emotional eating
525 may be related to the emotional response to food information processing. This highlights the
526 need to devise paradigms that induce an emotional state in order to mimic *ecological*
527 situations. Moreover, the questionnaires used may not accurately measure emotional eating
528 behavior per se [137,138]. It has been suggested that emotional eating is a behavioral trait,
529 consisting of a combination of behavioral characteristics that are individually predictable and

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

532 **4.2. Emotion induction during food information processing**

533 In a study featuring negative versus neutral mood induction conditions, emotional (vs.
534 nonemotional) eaters were found to have heightened activity in the (i) left parahippocampal
535 gyrus in response to the anticipated receipt of a milkshake, and (ii) right pallidum in response
536 to the actual receipt of a milkshake while in a negative mood. Across mood conditions, they
537 were found to have greater activity in the left caudate nucleus and pallidum during milkshake
538 consumption [139]. Overall, emotional eaters had greater activity in brain regions involved in
539 food cue processing and reward, supporting the idea that food plays a rewarding role and can
540 therefore alleviate or attenuate negative mood states. In a study assessing the neural
541 processing of food stimuli in different emotional contexts, researchers observed greater
542 activity in the lateral OFC and occipital lobe after negative emotional priming [140]. In a
543 positive priming condition, there was also increased activity in the lateral OFC and occipital
544 pole, as well as in the insula and amygdala. Amygdala activity was greater in the neutral and
545 positive conditions than in the negative condition, possibly reflecting decreased food salience
546 when individuals are in a negative state [140]. Two other studies used EEG recordings to
547 assess emotional eating while viewing food pictures in an emotional paradigm [141,142]. The
548 first of these demonstrated higher amplitude of the late positive potential (LPP) in
549 parietal-occipital brain regions in the high emotional eating group, independently of mood
550 condition, reflecting the high motivational relevance of food [141]. The second study
551 investigated event-related potential responses in a food choice paradigm in the context of
552 emotional eating [142]. While there was no effect of emotional eating on the parietal-occipital
553 P300 amplitude reflecting motivated attention to foods, unrestrained participants with high
554 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

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

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

614 **4.4. Emotional regulation of food craving**

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

630 cue exposure without control [84]. These results are in line with a previous meta-analysis that
631 focused on the use of food stimuli to assess the neural correlates of dietary self-control:
632 similar brain regions were involved, including the anterior insula, IFG, vIPFC, dlPFC,
633 bilateral SMA, bilateral mid-cingulate cortex, and temporoparietal junction [85]. In addition,
634 EEG recordings during the regulation of craving while being exposed to pictures of high-
635 calorie food revealed higher positive LPP amplitude when individuals had to think about the
636 long-term consequences of eating the food, compared with the immediate consequences
637 [159]. Additionally, LPP amplitudes were positively correlated with self-reported emotional
638 eating. These results indicate that emotional eaters pay increased attention to food because of
639 its relevance for emotion regulation and behavioral control. However, this higher LPP may
640 also be driven by arousal, regardless of valence, owing to possible negative thoughts related
641 to the long-term effects of eating high-calorie foods. As a higher LPP amplitude was observed
642 in the reappraisal of high-calorie food pictures, but became nonsignificant in the late LPP
643 window (slow wave), the authors concluded that it may have reflected successful regulation
644 of craving [159].

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

655 observed in the *decrease* condition when individuals chose to consume the food item.

656 Following emotion regulation, there was greater activity in the striatum when individuals

657 chose to consume the food item than when they rejected the food. This was more pronounced

658 in the decrease condition. Therefore, reducing emotions significantly modulated activity in

659 the vmPFC and striatum when participants chose to consume foods rather than reject them

660 [6]. Emotion regulation may therefore modulate dietary decisions through both reward and

661 decision-making networks. In addition, cognitive regulation of food craving during choices is

662 associated with differential activity in prefrontal brain regions involved in valuation and

663 decision-making processes, as investigated through a cognitive regulation of craving task

664 [160]. In healthy individuals, valuation of food pictures elicited reduced dlPFC activity after

665 food craving reduction, and higher vmPFC activity after food craving increase [160]. In

666 addition, the posterior parietal cortex and vIPFC exhibited stronger connectivity with the

667 vmPFC during regulation, suggesting that these brain regions are involved in implementing

668 changes in the decision-making circuitry during cognitive regulation [160]. Cognitive

669 regulation therefore appears to affect decision-making through valuation regulation and

670 behavioral control, where chronic stress may affect hedonic valuation but not cognitive

671 regulation [161]. However, individuals with reward and cognitive deficits may be more prone

672 to altered cognitive regulation, requiring increased frontoparietal functional connectivity

673 [162]. Taken together, these studies highlighted overlapping pathways involving emotional

674 processing and decision-making in the context of eating behavior that cannot be regarded as

675 distinct mechanisms. Thus, emotional eating may have two dimensions: 1) a *hedonic*

676 *dimension*, in which food intake is driven by the reward properties of food, and 2) a *cognitive*

677 *dimension*, in which emotion regulation strategies may be impaired, at least when volitional

678 regulation is required. The overlapping brain patterns reported by Brandl et al. (2019) suggest

679 that a combined model of emotional eating is needed. Improving emotion regulation to

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

683 **5. Health and societal issues: New neural targets and innovative strategies for** 684 **prevention and therapy**

685 **5.1. Neuromodulation**

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

701

702 **5.2. Use of neurofeedback in the prevention and treatment of emotional eating**

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

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

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

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

754 outcomes [177]. More recently, Kohl et al. (2019) found that the upregulation of the dlPFC
755 with fMRI-based NF during palatable food picture exposure improved self-control during
756 food choices and reduced the preference for high-calorie items [178]. They also found a
757 decrease in anxiety and depression ratings, suggesting that dlPFC upregulation can improve
758 emotional state and food choices, even if no change in calorie intake was observed [178]. All
759 these studies highlight the potential effectiveness of NF for improving eating behavior (for a
760 descriptive overview of these eating-related NF studies, see [185]). It should be noted that
761 behavioral outcomes are still inconsistent in NF studies [177,186,187], and further research is
762 needed to confirm the potential of NF training as an emerging interventional technique.
763 Moreover, most of these studies lacked a control group with sham NF, and included only a
764 small number of participants, thus preventing the generalization of the results. These
765 limitations increase the risk of experimental bias and insufficient statistical power [185].
766 Experimental paradigms need to be improved, in order to overcome these issues. For this
767 purpose, we recommend standardizing components of the NF protocol, such as the number
768 and duration of the NF sessions. Nevertheless, the above-mentioned research has brought
769 promising outcomes, such as a reduction in food craving [175,176,180], enhanced self-control
770 during food choice [178], and reduced frequency of overeating and binge-eating episodes
771 [179,180]. In addition, some studies have shown that NF can enhance the functional
772 connectivity of the emotion regulation network [170–173], thereby improving emotion
773 regulation. These results highlight the potential effectiveness of NF for emotional eaters. The
774 target brain region should be involved in both emotion regulation and eating behavior. Given
775 its role in both emotional processing [24,26] and the inhibitory control of eating behavior
776 [148], the dlPFC therefore seems to be a suitable candidate for improving emotion regulation
777 and the control of food intake in emotional eaters [10,183].

778 **6. Perspectives and conclusion**

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

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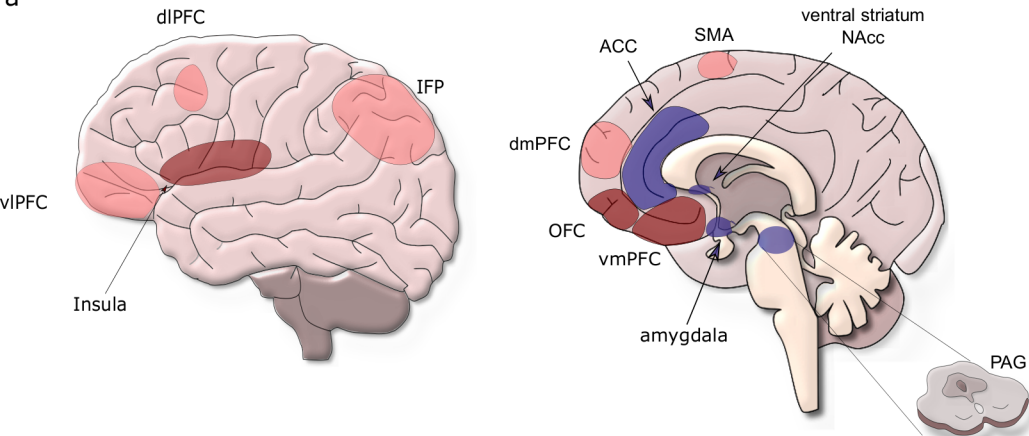
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1375 Legend Fig1.eps

1376 **Figure 1. a. Major brain areas involved in emotional processing, including emotion**
1377 **generation and emotion regulation.** Purple boxes represent areas more related to emotion
1378 generation (Periaqueductal Grey, PAG; amygdala; Nucleus Accumbens, NAcc; striatum
1379 ventral). Pink boxes represent brain areas more specific to affect modulation and emotion
1380 regulation (ventrolateral, dorsolateral, dorsomedial prefrontal cortices, vlPFC; dlPFC; dmPFC;
1381 InFerior Parietal region IFP; supplementary motor area, SMA). Red boxes represent brain areas
1382 that can be involved in both processes (orbitofrontal cortex, OFC/vmPFC; insula; anterior
1383 cingulate cortex, ACC). **b. Simplified scheme of common brain regions related to both**
1384 **eating behavior and emotional processing sharing similar functions.** Red boxes concern
1385 affect modulation whereas blue boxes are related to emotion initiation. Scheme designed with
1386 Inkscape (Inkscape Project. (2020). Inkscape. Retrieved from <https://inkscape.org>).

1387

a



b

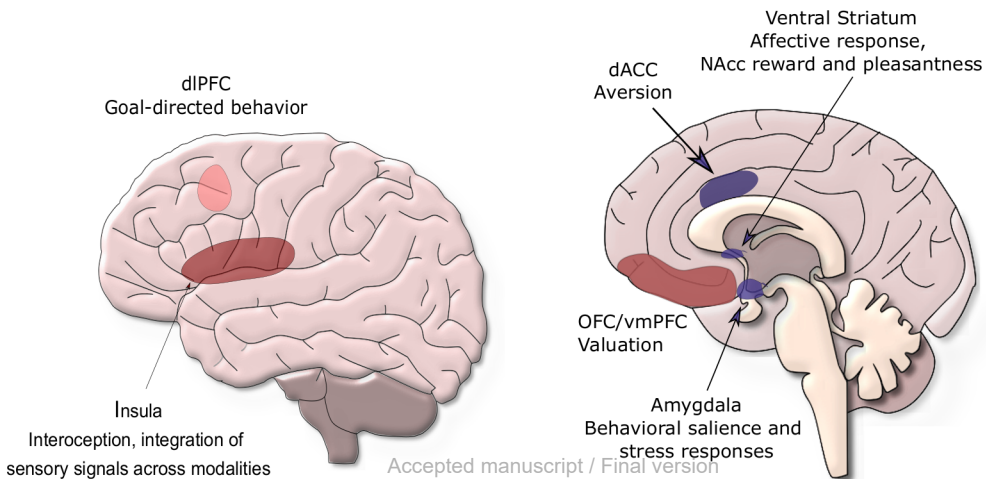


Table 1. Details of MRI studies in which emotional eating has been assessed. Details include population criteria (e.g. age, BMI, health condition), experimental paradigm, type of the questionnaire and major results of interest

References	Population	Age	BMI	Task	Questionnaire	Results of interest
Bohon, 2014	n=162 adolescents (82 females), healthy	15.3 (\pm 1.1)	20.8 (\pm 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 (\pm 1.75), range [18-25]	23.05 (\pm 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 (\pm 1.52), range [17-25]	20.59 (\pm 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 n=16 healthy lean individuals	normoglycemic 57.8 (\pm 1.9)	normoglycemic 23.2 (\pm 0.4)	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 type 2 diabetes obese patients (T2DM)	T2DM	T2DM	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.
	n=16 normoglycemic obese individuals	61.4 (\pm 1.5)	34.0 (\pm 0.9)			
		Obese 58.0 (\pm 2.1) Range [40-70]	Obese 32.6 (\pm 0.7)			
Wood, 2017	n=20 healthy individuals (12 females)	19.8 (\pm 1.0), range [18-22]	22.6 (\pm 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

“ $x \pm x$ ” means “average \pm 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				Interaction between emotional eating and mood (consummatory condition)
			n=11 low emotional eating score				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	<p>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</p> <p>Interaction with the amygdala Higher activity in left amygdala in the contrast after neutral priming relative to negative priming</p>
Maier et al. 2015	fMRI	Stress	n=51 healthy males	21 (± 2)	22.55 (± 2.06)	Stress induction task (SECP) and food choice task	<p>Functional connectivity Greater positive functional connectivity between vmPFC and portions of the amygdala and striatum (stressed > control participants) when choosing the tastier item</p> <p>Greater vmPFC connectivity with the Amygdala, ventral striatum and bilateral insula during tastier choices for the stressed group</p> <p>Stronger correlation of vmPFC connectivity during tastier choices with individual cortisol levels compared to self-reported perceived stress in the striatum and extended amygdala</p>
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	<p>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</p>

			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) Range [17.60-27.80]	Cognitive regulation of food craving task	Positive correlation with emotional eating score and parieto-occipital LPP 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: <u>Increased</u> ACC activity, amygdala, right medial OFC, left putamen and caudate regions, <u>reduced</u> 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: <u>higher</u> activity in the amygdala, caudate, right ACC, left putamen and right medial OFC and <u>reduced</u> 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

“ $x \pm x$ ” means “average \pm 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/m²); 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).