Physiology & Behavior 167 (2016) 60–70

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/phb

Review

The anterior medial temporal lobes: Their role in food intake and body weight regulation

Géraldine Coppin *

The John B. Pierce Laboratory, School of Medicine, Yale University, 290 Congress Avenue, New Haven, CT 06519, USA
Department of Psychiatry, School of Medicine, Yale University, 300 George Street, Suite 901, New Haven, CT 06511, USA

HIGHLIGHTS

- The amygdala and hippocampus play a major role in food intake and weight regulation.
- These functions are far less known to cognitive and affective scientists.
- This review uniquely expands on the interactions between these two brain structures.

ARTICLE INFO

Article history:
Received 26 April 2016
Received in revised form 22 July 2016
Accepted 29 August 2016
Available online 31 August 2016

Keywords:
Amygdala
Hippocampus
Food intake
Body weight
Learning
Memory

ABSTRACT

The anterior medial temporal lobes are one of the most studied parts of the brain. Classically, their two main structures – the amygdalae and the hippocampi – have been linked to key cognitive and affective functions, related in particular to learning and memory. Based on abundant evidence, we will argue for an alternative but complementary point of view: they may also play a major role in food intake and body weight regulation. First, an overview is given of early clinical evidence in this line of thought. Subsequently, empirical evidence is presented on how food intake, including in the extreme case of obesity, may relate to amygdalian and hippocampal functioning. The focus is on the amygdala's role in processing the relevance of food stimuli, cue-induced feeding, and stress-induced eating and on the hippocampus' involvement in the use of interoceptive signals of hunger and satiety, as well as memory and inhibitory processes related to food intake. Additionally, an elaboration takes place on possible reciprocal links between food intake, body weight, and amygdala and hippocampus functioning. Finally, issues that seemed particularly critical for future research in the field are discussed.

Contents

* Correspondence at: University of Geneva, Department of Psychology, Boulevard du pont d'Arve 40, 1205 Geneva, Switzerland.
E-mail address: geraldine.coppin@unige.ch.

http://dx.doi.org/10.1016/j.physbeh.2016.08.028
0031-9384/

1. The anterior medial temporal lobes – classical role

The anterior medial temporal lobes are composed of two major structures – the amygdalae and the hippocampi. Both of them belong to the most widely studied areas of the brain, notably because of their involvement in key cognitive and affective functions.

It is classical neuroscience textbook knowledge that the amygdala is involved in emotion processing (e.g., [72,175]). In particular, its role in fear conditioning paradigms has often been underlined [89,114]. Recently, the idea that the amygdala acts as a "relevance detector", rather than only being involved in the processing of fear-related stimuli, appears to have gained traction in the cognitive and affective sciences literature (see [119,125,140]). According to this idea, the amygdala is involved in the detection of any stimulus or event that can "significantly influence (positively or negatively) the attainment of [one's] goals, the satisfaction of [one's] needs, the maintenance of [one's] own well-being, and the well-being of [one's] species" [140]. This notion has become central in the study of emotion (see e.g., [6]).

As for the hippocampus, it has been related to various cognitive functions (see [108] for a review). It is mainly well-known for its contribution to spatial learning and memory (both in rats, e.g., [75], and in humans, e.g., [107]), in particular, explicit memory (e.g., [152]). Its role in inhibitory processes has also been emphasized (e.g., [28]).

However, based on abundant evidence, the anterior medial temporal lobes play a major role in functions that have not received sufficient attention in the literature and are far less known to cognitive and affective scientists: food intake and body weight regulation. In that light, an assumption of the selfish brain theory (according to this theory, the brain gives priority to its own energy needs; see [120]) will be shared in the present review, namely that the limbic system plays "a central role in the pathogenesis of diseases such as anorexia nervosa and obesity" (p. 143). In this framework, the limbic system's two core regions are defined as the amygdala and hippocampus. In the course of this review, we will endeavor to show that the limbic system's involvement in these functions is not incompatible with the aforementioned ideas and that it is rather complementary to these ideas.

It is worth noting that similar topics have been the object of several recent reviews (e.g., [5,56,78,98,109,117,139]). They include descriptions of studies linking food intake regulation to the amygdala [5] and the hippocampus Kanoski & Grill [78]; [117]) as well as studies showing the influence of memory on food reward processing [56] and obesity [98], and the impact of other factors such as environment and stress on obesity [109,139]. However, rather than reiterating the material covered by these scholarly works, the present review aims to integrate the literature on the functions of both the amygdala and the hippocampus in food intake and body weight regulation in two unique ways. First, this review thoroughly expands on the interactions between these two brain structures and on how important they are for food intake and body weight regulation, thereby filling a gap in the literature. Second, through the presentation of lesion studies in humans, the present work is uniquely ingrained in a neuropsychological and affective sciences perspective. Consequently, this review may benefit researchers in physiology, but also in psychology, cognitive and affective sciences, who may not be as familiar with this literature.

First, early clinical evidence pertaining to the involvement of the anterior medial temporal lobes in food intake will be presented, focusing on Klüver-Bucy syndrome, epileptic human patients, and amnesic patients who have a bilateral lesion of the medial temporal lobes. In a second part, experimental evidence will be discussed regarding the role of the amygdala in food intake, both in healthy-weight individuals as well as overweight and obese individuals. The third part of this review will be dedicated to the same purpose, but for the hippocampus. Subsequently, the reciprocal and dynamic links between food intake, affective and cognitive functioning, as well as amygdala and hippocampus functioning will be discussed. Finally, some of the current outstanding questions in this literature will be presented.

2. The involvement of the anterior temporal lobes in food intake regulation: early evidence

2.1. Klüver-Bucy syndrome

Klüver and Bucy [86] reported a syndrome that follows bitemporal dysfunction. Among other things, this syndrome is characterized by a hyperorality, bulimia, and the ingestion of non-food items (such as tea bags, feces or even shoe polish). These symptoms were reported both in rhesus monkeys and in humans (see for instance [91]). Thus, it appears that the dysfunction of both temporal lobes can lead to excessive intake of food as well as non-food items.

2.2. Psychomotor epileptic patients

Gastaut [42] has reported two types of hunger in patients with psychomotor epilepsy (i.e., epilepsy originating in the temporal lobe). The first of these is called "faim-valle", which translates to "very intense hunger". In patients with faim-valle, epileptic crises are often preceded by a violent hunger, and this rare but reliable sign of an impending epileptic crisis cannot be reduced by food intake. This often co-occurs with other symptoms such as olfactory and gustatory sensations, as well as chewing. The second type of hunger, called "faim postcritique", or post-critical hunger, appears after the crisis and is much more common. It is even present in patients who have had a substantial meal 1 h before the crisis, however, this form of hunger can be reduced by food intake. In both cases, patients do not remember these episodes. Importantly, between epileptic crises, these patients do not display pathologic hunger. Thus, the dysfunction of the temporal lobes can also be associated with hunger dysregulation.

2.3. Amnesic patients with bilateral lesion of the medial temporal lobes

Patient H.M. had a bilateral resection of the medial temporal lobes (including the hippocampus, parahippocampal gyrus and amygdala). He is famously known for his memory deficit. This deficit was, however, not his only impairment – patient H.M. was also unable to access his internal states. Almost never mentioning hunger unprompted, patient H.M. had a unique pattern of response when asked to rate his internal state, both before and after consuming a meal, using a 100-point scale anchored at extreme hunger on one end and at extreme fullness on the other. H.M. initially rated his internal state at 50. However, his rating did not move toward the fullness end of the continuum after a meal. Moreover, when experimenters offered him a second meal 15 min after he was done with the first one, he would eat it. However, he never rated his internal state as extremely full after the second meal. Finally, Hebben et al. noted that when H.M. stopped eating, he never reported that he was "full", only that he was "finished" eating. Interestingly, "his impairment is not attributable to his well-documented memory deficit. Instead, it is believed that the bilateral resection of the amygdala accounts for H.M.'s poor appreciation of his internal states" [52]. It should be noted that the misinterpretation of internal states is a mechanism that has been suggested to play a key role in obesity [143]. More specifically, one could argue that because H.M.'s rating of fullness only changed a little after eating, compared to before eating, he may have had difficulty detecting or utilizing inhibitory signals of satiety rather than excitatory hunger cues. A more recent report, which included two amnesic patients presenting extensive damage to both the amygdala and the hippocampus [136], replicated the results. Additionally, they extended these results by offering the patients a third meal (although for obvious ethical reasons, this could not be tested with a fourth, fifth, or even sixth meal), though the authors attributed this to a lack of conscious memory of previously eaten foods, rather than an impaired perception of internal states. Rozin et al. [136] suggested that this deficit was due to hippocampal damage rather than amygdala damage. In line with the idea that these deficits could be observed with an undamaged amygdala, Davidson & Jarrad [27] showed that rats with selective neurotoxic lesions of the hippocampus (the histology revealed no amygdala damage) were impaired relative to controls in their ability to solve a discrimination problem based on using cues related to their internal states (i.e., cues associated with food deprivation). However, the same neurologically damaged rats were *not* impaired at solving a similar discrimination problem based on external cues (i.e., auditory stimuli).

2.4. Summary of early clinical evidence

In a variety of conditions (Klüver-Bucy syndrome, psychomotor epilepsy, or bilateral damage or resection of the medial temporal lobes in amnesic patients), malfunctioning of the anterior medial temporal lobes has been reported to go hand in hand with dysregulated feeding behaviors and hunger states. The exact mechanism underlying these conditions is, however, unclear.

Moving forward from these early indications of the anterior medial temporal lobes's involvement in food intake, we will now present experimental evidence that has directly implicated the amygdala as one of the major centers involved in food intake and body weight regulation.

3. Amygdala: empirical evidence of its role in food intake and body weight regulation

3.1. Rationale for the interest in the amygdala in relation to food intake

If the amygdala is involved in relevance processing in general, it follows that it should also be involved in assessing the (more specific) meaningfulness of food-related stimuli (e.g., [21]), because of their high relevance for homeostasis. Furthermore, the same argument can be applied to cues predicting food intake. In this article, we will consequently develop the hypothesis that the amygdala is involved (1) in the relevance processing of food stimuli, including (2) in the detection of cues linked to food intake. Additionally, we will present its role in (3) stress-induced eating, and briefly mention (4) its uncertain role in satiety signals.

3.2. Amygdala and relevance of food stimuli

Several pieces of empirical evidence suggest that the amygdala is involved in the relevance processing of food stimuli. For instance, LaBar et al. [88] have shown that amygdala activation was higher when participants were watching food pictures while hungry (i.e. when they were relevant) rather than while sated. No such increase was observed for control pictures of tools. Hinton et al. [59] showed that when participants were hungry, amygdala activity was also increased when participants imagined themselves in a restaurant, selecting their favorite item on a menu. Besides hunger-induced relevance, Arana et al. [4] has shown that amygdala activity was higher when sated participants were reading high-incentive restaurant menus (i.e. displaying the participants' most preferred foods), in comparison to less high-incentive menus (i.e., displaying foods that participants were happy to eat but were not their favorite ones). Moreover, in a task in which participants made purchase decisions about food, amygdala activation was related linearly to the value assigned to a given food [73]. Rudenga & Small [137] provide evidence that the amygdala's response to sucrose ingestion is negatively correlated with the use of artificial sweetener. Thus, it seems that the extensive use of artificial sweetener, by blurring the relationship between sweet taste and its post-ingestive consequences, rendered sucrose ingestion less relevant. Finally, Blechert et al. [10] showed that amygdala response is increased when food is available, in comparison to when it is not.

3.3. Amygdala and cue-induced feeding

We have described that in patients suffering from Klüver-Bucy syndrome, a dysfunction of the anterior temporal lobes can lead to disrupted food intake behaviors in the presence of a cue, even if it is loosely related to food (such as a teabag). More recent empirical data have indicated that the amygdala plays a key role in the detection of *cues* associated with food. Using Pavlovian conditioning, Petrovich [122,123] has extensively studied the role of the amygdala in *cue-induced feeding* and *cue-induced inhibition of feeding*. Through Pavlovian conditioning, an initially neutral stimulus acquires an affective value by being associated with a punishing vs. a rewarding experience. Applied to the particular case of food intake, such a neutral stimulus, labeled the *cue*, may signal either a favorable or an unfavorable context regarding food intake. [122,123] has called the effects of the first type of cue cue-induced feeding, and the second type cue-induced inhibition of feeding.

In the case of the second type of conditioning, a cue previously associated with danger can prevent feeding, even in a food-deprived state. To the contrary, sensitivity to cue-induced feeding has been proposed to be a mechanism at play in obesity [143]. Although contradictory findings exist [106,134], it was suggested that while lean individuals mainly eat when their internal physiological state is off balance, food intake in obese individuals is mostly related to their environmental situation. In most current Western countries, many food cues are present in the environment.[1] Taken together, in obese individuals, cues previously associated with food intake may consequently easily and frequently stimulate feeding, even when sated (e.g., [132]). This does not mean that cue-induced feeding necessarily promotes an excessive weight gain. Reppucci and Petrovich [132] did not find significant differences in

[1] "Easy and inexpensive access to energy dense foods and lack of safe spaces for physical activity" defines an obesogenic environment ([151], p. 274).

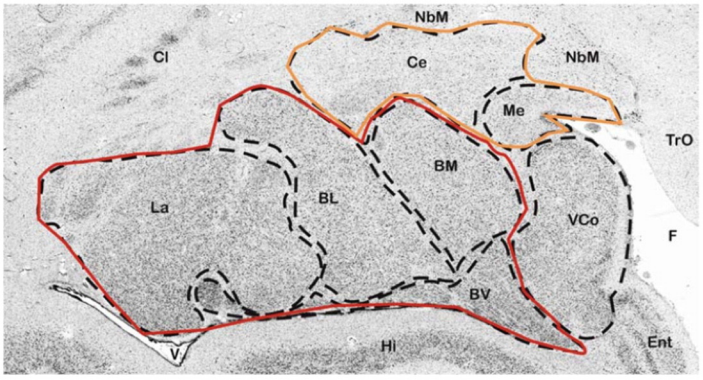

Fig. 1 Cytoarchitecture of the amygdala and neighboring cortical and subcortical structures in a coronal section of a human postmortem brain. The centromedial nucleus is labelled by an *orange line* and the basolateral complex by a *red line*. The *VCo* belongs to the superficially located part of the amygdala. *BL* basolateral nucleus, *BM* basomedial nucleus, *BV* basoventral nucleus, *Ce* central nucleus, *La* lateral nucleus, *Me* medial nucleus, *VCo* (ventral) cortical nucleus. Neighbouring structures: *Cl* Claustrum, *Ent* entorhinal cortex, *F* endorhinal sulcus, *Hi* hippocampus, *NbM* Nucleus basalis of Meynert, *TrO* Tractus opticus, *V* lateral ventricle

Fig. 1. Representations of the different amygdala nuclei. Figure from Amunts et al. [3].

weight between rats trained to feed upon the perception of certain cues (i.e., rats in a cue-induced feeding condition) and control rats. Moreover, Berthoud [9], who has reviewed the literature on this topic, failed to identify an animal or human study establishing a direct link between exposure to conditioned food cues (even in the long-term) and obesity. Lesion studies in rats have shown that food intake elicited by the presence of cue-induced feeding is mediated by the amygdala (more precisely, according to [122], the basolateral area, i.e. the basolateral, basomedial and lateral nuclei, see Fig. 1).

Moreover, empirical evidence using functional Magnetic Resonance Imaging (fMRI) in humans has shown that the activity of the amygdala is more pronounced when a food odor predicts the arrival of its associated food, compared to when it does not [148]. Thus, the amygdala responds to food cues (e.g., [32,166]). Additionally, it has been shown that the amygdala's response to food cues are modulated by hunger [47,88,169] and its response in the absence of hunger could predict weight gain susceptibility [159].

Interestingly, it appears that the amygdala's sensitivity to food cues is related to overeating and Body Mass Index (BMI[2]). Increased amygdala responses to food cues in obese compared to healthy-weight individuals is one of the most replicated findings in the neuroimaging of obesity (e.g., [111,113]; see [16] for a review), including in children (e.g., [14]). External food sensitivity, i.e. a personality trait associated with overeating and obesity,[3] predicts how the amygdala responds to appetizing food pictures [118]. Additionally, self-directedness (i.e., the ability to set and pursue meaningful goals, which is typically reported as altered in eating disorders, e.g., [158]) is negatively correlated with the amygdala response to appetizing food pictures [48]. Moreover, in healthy-weight patients, the orbitofrontal cortex and nucleus accumbens, which are both involved in feeding, modulate the amygdala response to food cues. In fact, this modulation is impaired in obese individuals, regardless of the calorie content (low vs. high) suggested by the cues [156]. Such abnormal amygdala responses to food cues might trigger "non-homeostatic feeding" (also called "hedonic feeding"), i.e. food intake behaviors in states of positive energy, which have the potential to contribute to the development of obesity. Interestingly, the opposite pattern is observed in anorexia nervosa patients. In these patients, the amygdala is hypoactive when patients are presented with high-calorie foods, independently of whether these patients were currently underweight or weight-restored [63].

However, the picture has become slightly complicated, given recent results showing that obese participants have an impaired performance in an implicit conditioned food preference test, which relies on amygdala functioning [74]. In this task, three cues were respectively associated with a 10%, 50% or 90% possibility of receiving food. Obese individuals preferred the cue associated with a 10% possibility of receiving food ([20], Experiment 1). This result might be explained by a difficulty of learning to avoid negative outcomes in obese individuals [39], as this result was not specific to food reward but also replicated with monetary reward ([20]; Experiment 2).

3.4. Amygdala and unhealthy food intake in acute and chronic stress

The amygdala plays a key role in stress-evoked responses (e.g., [45]). In the case of food intake, the amygdala may be the brain structure mediating the excessive pursuit of highly palatable food during stress. Thus, acute stress [138] as well as chronic stress [167] potentiates the amygdala response to food. Tryon et al. [167] also showed that in participants under high chronic stress, in response to high calorie food pictures relative to low calorie ones, the connectivity between the amygdala and regions involved in habit formation, reward, and decision making (bilateral thalamus, left inferior parietal lobe, and left putamen) was increased compared to participants under low chronic stress. By enhancing these connections, high chronic stress may lead to a shift from goal-directed to habitual behaviors. In contrast, in participants under low chronic stress, in response to high calorie food pictures relative to low calorie ones, the connectivity between the amygdala and regions involved in executive functions (e.g., anterior cingulate cortex, dorsolateral prefrontal cortex) was increased compared to participants under high chronic stress. Taken together, these results could explain why stress is associated with unhealthy food intake and poor eating habits (e.g., [129]), which could eventually lead to obesity.

Interestingly, this research topic is right at the intersection between affective sciences and food intake research. For instance, Pool et al. [128] have recently shown that stress affects one of the components of reward but not others. More specifically, stress increases "wanting" (i.e., the motivation to obtain a reward), but not "liking" (i.e., the hedonic pleasure during the reward consumption) for a sweet reward.

[2] Body Mass Index (BMI) is a measure used to estimate an individual's body fat content. It is computed by dividing an individuals' weight by the square of his/her height. BMI is used to classify individuals into underweight (BMI < 18.5), healthy-weight (18.5 < BMI < 25), overweight (25 < BMI < 30), or obese (BMI > 30) categories. Although not without issues (see e.g., [41]), BMI is currently the most commonly used measure in obesity research.

[3] This personality trait can be assessed by the Dutch Eating Behavior Questionnaire [170].

3.5. Additional results

One might wonder which aspects of food intake the amygdala might further be involved in. Wang et al. [173] have demonstrated the involvement of the amygdala in the processing of internal signals of fullness using dynamic gastric balloon distension. In healthy-weight and overweight participants, the activity of the left amygdala was negatively correlated with self-reported fullness. The activity of the right amygdala was negatively correlated with the participants' BMI (see also [44]). The integrity of the amygdala has however been reported as irrelevant in using internal signals of hunger and thirst [133]. More specifically, Rhodes et al. [133] used a task in which rhesus monkeys had to utilize internal contexts (i.e., hunger, thirst) to guide their choices of objects (i.e., objects associated with food, objects associated with water). They showed that learning as well as performance in this task were not disrupted by amygdala lesions. However, there was a slight damage of the hippocampus in three quarters of the monkeys tested in Rhodes et al.' study. As the hippocampus is known to be important in this type of learning based on internal context (see e.g., [82]), it is unclear whether the results reported by Rhodes et al. were caused by lesions of the amygdala, the hippocampus, or both (especially given the important interactions between both structures, see section "Interactions between the amygdala and the hippocampus"). Thus, although, the amygdala seems to be associated with gastric distension, the exact role of the amygdala in fullness signals is still poorly understood [44].

3.6. Summary and conclusion

The amygdala is involved in food intake and body weight regulation, in particular in the relevance processing of food stimuli and the detection of cues linked to food intake. Heightened amygdala responses to food cues, for instance in stressful contexts, may account for excessive levels of caloric intake in overweight and obese individuals.

In a recent review on how deep brain stimulation might be beneficial to obesity treatment, Taghva et al. [165] have emphasized the amygdala as one of the key targets in the neuromodulation of obesity. The results of such clinical trials will probably bring new insights into the involvement of the amygdala in food intake and body weight regulation.

We will now present how the second key area of the anterior medial temporal lobes – the hippocampus – might relate to food intake and body weight regulation.

4. Hippocampus: empirical evidence of its role in food intake and body weight regulation

4.1. Rationale for the interest in hippocampus in relation to food intake

To suggest a potential role of the hippocampus in food intake and body weight regulation may at first strike cognitive and affective researchers in human neuroscience as a somewhat unconventional idea. Although highly connected to the amygdala [127], it has been claimed that the "removal of the hippocampus does not affect olfaction or taste" ([11], p. 857). However, "evidence exists for input to the hippocampus from the amygdaloid complex, implicating it in the processing of emotional information and thus indirectly in interoceptive processes" ([18], p. 120). Moreover, there is accumulating evidence that "dietary factors are associated with the emergence of hippocampal pathology and that hippocampal pathology is associated with the emergence of increased food intake and body weight gain" ([77], p. 60; see also [78,117]). Three reviews [28,78,117] on the hippocampus' role in food intake have also highlighted the fact that its function in this context is more well-known than the amygdala's (see also the last outstanding question).

We will argue that the integrity of the hippocampus is necessary for at least three functions related to food intake, namely (1) the use of interoceptive signals of hunger and satiety, (2) memory of prior food intake, and (3) inhibitory processes in the particular case of feeding.

4.2. Hippocampus and the use of interoceptive signals of hunger and satiety

The hippocampus expresses receptors for many peptide hormones related to appetite regulation. For instance, Hsu et al. [66] have shown that ghrelin signaling in the hippocampus can stimulate conditioned appetite. Additionally, glucagon-like peptide-I (GLP-I) receptors in the ventral hippocampus also influence food intake and willingness to work to obtain food (i.e., "wanting"; [65,81]). Furthermore, there is solid evidence showing that hippocampal neurons form a memory of a meal and inhibit meal onset after a meal has been consumed [53, 115,117]. These findings show that the hippocampus is a very likely target of the modulation exerted by interoceptive signal of hunger and satiety. Indeed, this hypothesis has found empirical support, the hippocampus' response to taste stimuli is modulated by internal states of hunger [50]. It has been shown that after hippocampal lesions, rats were unable to use hunger and satiety cues to perform an internal state-conditional task (i.e., a task where a food-deprived state needs to be used as a contextual cue) [27,61]. These rats were unable to use their food-deprived state as a signal to consume food. However, this deficit did not extend to exteroceptive cues. Similarly, Hirsh et al. [60] and Kennedy & Shapiro [82] have shown that hippocampal lesions impair the ability to use internal signals of hunger or thirst in rats. In a later experiment, Kennedy and Shapiro demonstrated that the hippocampus codes the relationship between an internal deprivation state of food/thirst, the memory of the external environment and the selection of a behavior leading to a decrease of the deprivation state [83]. Just like H.M. consistently rated his internal states as 50, the Western diet[4] reduces the sensitivity to internals signals of hunger, and especially of fullness ratings in humans [38]. In accordance with an early suggestion from Jean Anthelme Brillat-Savarin [15], maintaining that "gobbing without appetite is one of the most important causes of obesity" (p. 256), the inhibition of food intake by internal signals may be particularly problematic in the development/maintenance of obesity [31]. We will further discuss the importance of inhibitory processes in the third sub-part of this review dedicated to the hippocampus.

4.3. Hippocampus and memory for food intake

As alluded in the introduction, the hippocampus is well known for its role in declarative memory. This type of memory has been shown to influence food intake (e.g., [78]). Thus, the recall of previously eaten food leads to a decrease in subsequent food intake [54]. In contrast, the disruption of the encoding of the last meal leads to an increase in subsequent food intake [55].

But it is not just our memory that influences food intake – both the quantity and the quality of what we eat influence our memory. Evidence of a link between food intake and impaired memory performance is accumulating (for a review, see [23]). First, for the case of an overly rich diet, such as the Western diet, it has been shown that feeding rats a high-fat diet alters working memory, even after only 3 [77] or 9 days [110]. This type of memory is impaired both in the short and long-term [77]. Furthermore, a Western diet leads to an impaired spatial memory in mice and rats [26,96,168]. In human participants with a healthy BMI, the Western diet affects the ability to recall previously eaten food [38]. More specifically, the quality rather than the quantity of fat ingested matters: a saturated fatty acids diet leads to an impairment of hippocampal activity [142], whereas this does not occur with an isocaloric monounsaturated fatty acids diet. This does not mean that quantity does not matter. For instance, impairments in declarative memory have been associated with a higher BMI (e.g., [19]), where one might expect a higher consumption of saturated fatty acids. Second, for an overly poor diet, impaired learning and memory skills have been

[4] Western diet refers to a diet rich in saturated fatty acids and refined carbohydrates (e.g., [22]). Its consumption has been linked with the current epidemics of obesity (e.g., [67]), as well as cognitive impairments (e.g., [77,112]).

reported in prisoners of war with a history of malnutrition [160,161]. It should be noted that a very similar result has been reported in patients suffering from anorexia nervosa, which can be considered another form of malnutrition: visuo-spatial performance – typically considered to rely on the hippocampus – was impaired [99]. Third, the Western diet has been associated with smaller hippocampal volume [71]. Finally, memory deficits associated with the consumption of a Western diet may also impact the timing of meals (i.e., the postprandial intermeal interval, see [117]), leading to increased meal frequency and size.

The relationship between food intake and memory performance is thought to be mediated by a dysregulation of hippocampal functioning (e.g., [58]). Based on Francis & Stevenson's [38] work, it appears that the impairment of the hippocampus functioning could occur without correlating with weight gain, at least in the early stages of obesity. One potential mechanism underlying this result may be the disruption of the hippocampal neurogenesis (see [92]). As indicated above, the hippocampus expresses receptors for many peptide hormones related to appetite regulation, such as insulin. Interestingly, insulin could improve hippocampal-dependent memory function [103].

Similarly, the explicit memory of recently eaten food's impact on food intake amount is thought to be mediated by the hippocampus. Congruent with this argument, other types of food memories, such as sensory-specific satiety, are not impaired in amnesic patients [57]. Sensory-specific satiety can be defined as the implicit memory for consumed food items, characterized by a decrease in pleasure derived from recently consumed food items while other unconsumed food items are still pleasurable. Sensory-specific satiety is mediated by the orbitofrontal cortex [25] and not by the hippocampus.

4.4. Hippocampus and inhibitory processes related to food intake

The reciprocal links between hippocampal functioning, food intake, and body weight regulation have notably been investigated by Davidson and colleagues (e.g., [28,29,77,98,139]), who focused on the role of the hippocampus in inhibitory learning and memory. Kanoski & Davidson [77] highlighted how body weight regulation depends on the ability to solve a so-called "serial feature negative discrimination problem", in which "interoceptive satiety cues (X) signal that environmental food-related target cues (A) will not be followed by an appetitive postingestive outcome" (p. 66). In other words, to maintain a constant body weight, it is necessary to suppress associations that are irrelevant in a given context, such as inhibiting the response evoked by cues previously associated with food in the absence of hunger signals. For instance, after a rich meal (i.e., in the absence of hunger signals), and in the presence of an appetizing food item, it is necessary to suppress the association between the consumption of this food item and positive postingestive consequences (in this instance, food consumption would likely cause the feeling of being uncomfortably full).

This ability is assumed to rely on the hippocampus. First, following hippocampal lesions, this ability is impaired (e.g., [62]). Second, Sweeney & Yang [164] have identified a likely brain substrate (i.e., glutamatergic projections from the ventral hippocampus to the lateral septum) for this type of inhibitory control. Coincidentally, the ventral hippocampus is a key substrate for inhibitory control of approach tendencies within approach-avoidance paradigms [145]. One may conjecture that similar mechanisms are involved in the inhibition of approach tendencies under satiation. Third, the consumption of a Western diet leads to a decrease in GABA levels in the hippocampus, which may disturb inhibitory processes involved in food intake [141].

Kanoski & Davidson [77] took this argument further in suggesting a "vicious circle" where the more the Western diet is consumed, the more impaired the hippocampus functioning is, and consequently, the more inhibition deficits occur, leading to even more consumption of Western-diet food (see also [51]). Impaired inhibition in overweight and obese individuals is hypothesized to affect food intake (e.g., [49]). In line with this point of view, gastric stimulation in obese individuals activates primarily the right hippocampus, which correlates with a decrease in emotional and uncontrolled eating [172]. In other words, activation of the hippocampus in obese individuals is linked to inhibitory processes related to food intake.

4.5. Summary and conclusion

We have presented evidence that the hippocampus is involved in the use of interoceptive signals of hunger and thirst, memory for previously eaten food and inhibition processes. Because of both its role in the use of interoceptive signals of hunger and thirst as well as in inhibition, an impairment of hippocampal activity may lead to a self-reinforcing vicious circle of unhealthy eating. And because of its role in memory and inhibition, the hippocampus may also be an important structure for other food intake behaviors, such as guiding food decisions (see notably [177]).

We will now discuss the potential reciprocal links between food intake, body weight regulation and the interactions between amygdala and hippocampus functioning, and other parts of the brain that are involved in food intake.

5. Reciprocal links between food intake, body weight regulation, and amygdala and hippocampus functioning

5.1. Interactions between the amygdala and the hippocampus

As previously mentioned, the selfish brain theory (e.g., [121]) assumes that the brain (approximately 2% of the body's mass) gives priority to its energy needs (approximately 50% of the total body glucose utilization is related to its metabolism). The hippocampus/amygdala system is key in this process – it is assumed to be involved in energy homeostasis and metabolic processes (i.e., glucose fluxes; [162]). Accordingly, the selfish brain theory assumes that obesity is "the sequelae of a dysfunction in the hippocampus/amygdala system" ([35], p.138). More specifically, obesity is purportedly associated with a change in the hippocampal set-point (i.e., the point where the energy needs of the brain are fulfilled and the energy stored in the rest of the body is stable).

But more specifically, how do the amygdala and hippocampus interact? Neuroanatomical and tract tracing studies from rodents have shown direct connectivity between these two brain regions. For instance, anterograde and retrograde studies have given a detailed map of the interconnections between the amygdala and the hippocampus (see [127], in particular Fig. 7). Different regions of the hippocampus (in particular the temporal end of the hippocampus) project to the amygdala in parallel in several of its different nuclei. Similarly, the amygdala's different nuclei project to different regions of the hippocampus in parallel [7,94].

As parallel information flowing between the amygdala and the hippocampus is high, the deficits caused by a circumscribed lesion could be partially circumvented [127]. Several studies have however examined the limits of this parallel information flow – namely, how selective amygdala or hippocampus damage/lesions in rodents can affect food intake and body weight. As previously described, rats with selective neurotoxic lesions of the hippocampus (with no amygdala damage) were impaired at using food deprivation cues as signals for shock, while their use of external cues to solve this discrimination was unimpaired [27]. Additionally, Kesner & Williams [84] gave amygdala, hippocampal, or control lesions to rats after performing a discrimination task between two objects; one associated with a food reward and the other associated with no food reward. Only amygdala-lesioned rats displayed deficits in this task. While hippocampal-lesioned rats were able to transfer this initial learning to other food rewards containing different amounts of sugar, the amygdala-lesioned rats were not able to perform this transfer. In contrast, Wheeler, Chang, & Holland [176] have investigated the impact of amygdala and hippocampal lesions on mediated learning, namely, learning about a stimulus while it is not present, based on mental representations. They showed that amygdala lesions did not alter this type of learning, while hippocampal lesions did.

The amygdala and hippocampus interact at several levels, the following examples illustrate some of these significant interactions. First, the amygdala and the hippocampus are associated with two different memory systems, which interact in significant ways, particularly in emotional situations (e.g., [124]). For instance, the connectivity between the amygdala and the hippocampus is increased while individuals encode (e.g., [34]) and retrieve (e.g., [149]) emotional material in comparison to neutral material. Second, glucose is known to have memory-enhancing effects, which are mediated via changes in hippocampal (e.g., [105]) and amygdala (e.g., [144]) activities. Interestingly, glucose also increases the functional connectivity between the amygdala and the hippocampus [116]. Third, cued conditioning relies on the amygdala, but in the case of a conditioning relying on context, the hippocampus would additionally be recruited [97]. Fourth, a lot of studies have shown that a stimulation of the amygdala enhances hippocampal-dependent memory and modulates the synaptic plasticity in the hippocampus (e.g., [1,2,40,69,93,100,102,104]). Fifth, learning to suppress conditioned responses requires interactions between the hippocampus and the (basolateral) amygdala. More specifically, these interactions underlie latent inhibition, i.e. a slower acquisition of a conditioned response caused by repeated non-reinforced presentations of a conditioned stimulus, before the conditioning procedure actually starts (e.g., [24]). Sixth, interactions between the hippocampus and the amygdala are purportedly important for contextual conditioning driven by food reward, although both structures could be complementarily and competitively involved in learning and memory tasks [70].

It should be noted that we considered here the amygdala as an entity, although due to the existence of different sub-nuclei of the amygdala, it has been assessed as "*neither a structural nor a functional unit*" ([163], p. 323), and the label of "amygdaloid complex" has been suggested. This more fine-grained view (e.g., [174]) makes sense in terms of the functions we discussed here (e.g., [17]), but also in terms of architecture and connectivity (e.g., [126]). For instance, different sub-nuclei of the amygdala underlie cue-induced feeding behavior and cue-induced inhibition of feeding behavior [122]. Current evidence points to the posterodorsal part of the amygdala as the most crucial for food intake regulation and weight gain in rats [135]. The nature of the interactions between the amygdala and hippocampus may consequently vary in different sub-parts of these structures and underlie different functions. In line with this notion, Huff, Emmons, Narayanan, & LaLumiere [68] have shown that the basolateral amygdala interacts with the ventral hippocampus to modulate specific types of learning (e.g., footshock learning) but not others (here context learning).

This is also suggests that similarly, the hippocampus may not be one entity (e.g., [36,53,85,101]). For instance, while the ventral hippocampus has bi-directional connections to the amygdala, the dorsal hippocampus does not have any [33]. Moreover, as mentioned before, GLP-I receptors in the ventral hippocampus, but not in the dorsal hippocampus, influence food intake and willingness to work to obtain food [65,81]. However, the respective roles of the ventral and dorsal hippocampus in meal onset are not currently known [117]. This functional dissociation between the ventral and dorsal hippocampus goes beyond food intake and body weight regulation contexts. For instance, in anxiety-inducing contexts, the ventral hippocampus fires more synchronously with the amygdala than the dorsal hippocampus [90].

Given the findings presented in this section, a better understanding of the interactions between the amygdala and the hippocampus may bring important insights into the mechanisms underlying food intake and body weight regulation. Moreover, interactions with other brain structures are also important to consider.

5.2. Connectivity between the amygdala and the hippocampus and other areas of the brain

Investigating how the anterior medial temporal lobes functionally relate to other parts of the brain is crucial to better understand the dynamics of cerebral dysfunctions and cognitive deficits which accompany food intake disorders such as obesity. In obese individuals, the connectivity between the amygdala and other key structures involved in food intake regulation deviates from the connectivity in lean individuals [157]. The differential direct and indirect connectivity between the amygdala and other important structures in food intake (e.g., caudate nucleus, hypothalamus, nucleus accumbens, ventromedial prefrontal cortex) may consequently contribute to excessive food intake and weight gain.

Regarding the hippocampus, it is known that its connection with the ventral tegmental area, a key component of the reward circuit, may be crucial for context-reward associations [95]. The connectivity of the hippocampus may also change according to an individual's BMI. Recent work has shown that the hippocampal connectivity is decreased in obese individuals in comparison to healthy-weight individuals [43].

6. Conclusion

In this review, we have argued that the anterior medial temporal lobes – composed of the amygdala and the hippocampus - may play an important role in food intake and body weight regulation. The amygdala is essential to assess the relevance of food stimuli, and to cue-elicited feeding and cue-induced inhibition of feeding. In modern environments, where cues associated with feeding are much more present than cues associated with inhibition of feeding, the abundance of these cues may lead to overfeeding. The hippocampus is involved in tracking internal signals of hunger, memory, and inhibitory processes related to food intake. These functions (i.e., relevance of food stimuli, and to cue-elicited feeding and cue-induced inhibition of feeding for the amygdala and internal signals of hunger, memory, and inhibitory processes related to food intake for the hippocampus) complement the functions of the amygdala as a relevance detector and of the hippocampus as involved in memory and inhibition. A better understanding of the role of these two structures in food intake and body weight regulation may bring insights into the dynamics of cerebral dysfunctions and cognitive deficits that accompany food intake disorders such as obesity. Listed below are some of the issues that appear to be particularly critical for the future of the research in this field.

7. Outstanding questions

Are the anterior medial temporal lobes also crucial for representing *future* food intake? Information related to recent eating is encoded in memory and influences future food intake (e.g., [54,55,58]). The hippocampus encodes information relative not only to past events but also to future ones [153]. The amygdala is involved in representing future positive events [146]. From this evidence the following question follows: Can hippocampus and amygdala dysfunctions relate in difficulties representing future food intake and steady decision-making related to future food intake? This question seems all the more relevant since the links between obesity and decision-making processes are still relatively poorly understood (e.g., [130,178]) and may lead to fruitful therapeutic interventions. Moreover, Vartanian, Chen, Reily, & Castel [171] have recently shown that thinking about *future* food intake suppresses eating, similar to *past* food intake, and that the mechanisms underlying this effect need further investigation.

Is it possible to reverse the effects of "vicious circle" between an excessive Western diet consumption, increased body weight, and cognitive impairment? According to Kanoski and Davidson [77], "if the degree of interference with hippocampal function increases with the duration of exposure or the amount of Western diet consumed, this could lead to a "vicious circle" […] in which continued eating leads to greater impairment of hippocampal-dependent memory function which further weakens the ability to inhibit intake of the Western diet that promoted hippocampal dysfunction in the first place" (p.66). To what extent can this "vicious circle" be reversed? In other words,

can weight gain in individuals suffering from anorexia nervosa and weight loss in obese individuals restore (i) hippocampus functioning? and (ii) cognitive functioning? Inverting the question also seems relevant: Can memory skills training in individuals suffering from anorexia nervosa and in obese individuals restore (i) hippocampus functioning? and (ii) lead to weight gain in individuals suffering from anorexia nervosa and weight loss in obese individuals? (see [150] for a discussion on this topic).

The answer to these questions may open avenues for therapy, which its importance is underscored by the difficulty of long-term weight stabilization in individuals suffering from anorexia nervosa [154] and losing and stabilizing weight in obese individuals and post-obese individuals [87], respectively.

Does the link between the Western diet and memory impairment depend on the integrity of sleep and hippocampal neurogenesis? Sartorius et al. [142] have shown that a diet rich in saturated fatty acids (such as the Western diet) impairs sleep behavior in mice. Among the different functions of sleep, its importance for memory has been underlined [179]. Hippocampal neurogenesis, on which hippocampus-dependent memory relies [180], may notably depend on Rapid Eye Movement (REM) sleep [181]. The following question emerges: By consuming an excessive amount of the Western diet, could a perturbation in REM sleep occur, thus leading to altered hippocampal neurogenesis, which in turn impairs memory function? This question seems to be relevant given the bidirectional links between sleep and obesity [182].

Are there sub-types of obesity for which memory functions are differently affected? Insulin sensitivity is linked to both obesity and performance in a hippocampus-dependent memory task [8,46]. While we have considered obese individuals as a homogenous group thus far, this classification was purely based on BMI, although both the root causes and the associated symptoms may in fact differ between individuals. Different sub-types of obesity exist, in particular with regard to insulin resistance – some obese individuals remain insulin sensitive while others develop insulin resistance [155]. Do these sub-groups perform differently in a hippocampus-dependent memory task, and more generally, have different cognitive functioning? This question has been raised by Stingl et al., but has not been yet addressed.

Does a Western diet impact amygdala functioning in similar ways as hippocampal functioning? This review has offered an overview of what is currently known on the effects of a Western diet on hippocampal functioning. However, we have not discussed how these effects may also apply to amygdala functioning. For instance, hippocampal and amygdala functioning are similarly affected by the amount of time individuals have been exposed to a Western diet. More specifically, it has been shown that a Western diet impacts hippocampal [12]) and amygdala functioning [13] more in the juvenile period than in the adult period. They found similar results for amygdalian functioning. However, while this consumption impairs hippocampal-dependent memory, it may *increase* the amygdala-dependent one [131]. Moreover, the impact of a Western diet is faster on hippocampal functioning than on amygdala functioning, as the hippocampal functioning changes earlier than the amygdalian one [12,13]. It is however not currently known to what extent the hippocampal functioning's change may drive the amygdalian one. Thus, further research investigating similarities and differences caused by a Western diet on hippocampal and amygdalian functioning may lead to important discoveries about the anterior medial temporal lobes.

Acknowledgments

This work was supported by an Early.Postdoc mobility fellowship from the Swiss National Science Foundation to Géraldine Coppin (PBGEP1-139853). The authors thank Vanessa Sennwald, Dr. Alexander Skiles, Prof. Dana M. Small and the anonymous reviewers for their insightful comments on earlier versions of the manuscript.

References

[1] K. Abe, T. Fujimoto, T. Akaishi, M. Misawa, Basolateral amygdala D1- and D2-dopaminergic system promotes the formation of long-term potentiation in the dentate gyrus of anesthetized rats, Prog. Neuro-Psychopharmacol. Biol. Psychiatry 33 (2009) 552–556, http://dx.doi.org/10.1016/j.pnpbp.2009.02.011.
[2] I. Akirav, G. Richter-Levin, Factors that determine the non-linear amygdala influence on hippocampus-dependent memory, Dose Response Int. J. 4 (2006) 22–37, http://dx.doi.org/10.2203/dose-response.004.01.003.Akirav.
[3] K. Amunts, O. Kedo, M. Kindler, P. Pieperhoff, H. Mohlberg, N.J. Shah, U. Habel, F. Schneider, K. Zilles, Cytoarchitectonic mapping of the human amygdala, hippocampal region and entorhinal cortex: intersubject variability and probability maps, Anat. Embryol. 210 (2005) 343–352, http://dx.doi.org/10.1007/s00429-005-0025-5.
[4] F.S. Arana, J.A. Parkinson, E. Hinton, A.J. Holland, A.M. Owen, A.C. Roberts, Dissociable contributions of the human amygdala and orbitofrontal cortex to incentive motivation and goal selection, J. Neurosci. 23 (2003) 9632–9638.
[5] M.F.C. Areias, P.O. Prada, Mechanisms of insulin resistance in the amygdala: influences on food intake, Behav. Brain Res. 282 (2015) 209–217, http://dx.doi.org/10.1016/j.bbr.2015.01.003.
[6] J. Armony, P. Vuilleumier, The Cambridge Handbook of Human Affective neuroscience, Cambridge University Press, Cambridge, 2013.
[7] M. Bazelot, M. Bocchio, Y. Kasugai, D. Fischer, P.D. Dodson, F. Ferraguti, M. Capogna, Hippocampal theta input to the amygdala shapes feedforward inhibition to gate heterosynaptic plasticity, Neuron 87 (2015) 1290–1303, http://dx.doi.org/10.1016/j.neuron.2015.08.024.
[8] C. Benedict, W.H. Frey II, H.B. Schiöth, B. Schultes, J. Born, M. Hallschmid, Intranasal insulin as a therapeutic option in the treatment of cognitive impairments, Exp. Gerontol. 46 (2011) 112–115, http://dx.doi.org/10.1016/j.exger.2010.08.026.
[9] H.R. Berthoud, The neurobiology of food intake in an obesogenic environment, Proc. Nutr. Soc. 71 (2012) 478–487, http://dx.doi.org/10.1017/S0029665112000602.
[10] J. Blechert, J. Klackl, S.F. Miedl, F.H. Wilhelm, To eat or not to eat: effects of food availability on reward system during food picture viewing, Appetite 99 (2016) 254–261, http://dx.doi.org/10.1016/j.appet.2016.01.006.
[11] I.N. Bogolepova, Structure and development of the human hippocampus in prenatal ontogenesis, Zh. Nevrol. Psikhiatr. Im. S S Korsakova 70 (1970) 857–863.
[12] C. Boitard, N. Etchamendy, J. Sauvant, A. Aubert, S. Tronel, A. Marighetto, S. Layé, G. Ferreira, Juvenile, but not adult exposure to high-fat diet impairs relational memory and hippocampal neurogenesis in mice, Hippocampus 22 (2012) 2095–2100, http://dx.doi.org/10.1002/hipo.22032.
[13] C. Boitard, M. Maroun, F. Tantot, A. Cavaroc, J. Sauvant, A. Marchand, S. Layé, L. Capuron, M. Darnaudery, N. Castanon, E. Coutureau, R.M. Vouimba, G. Ferreira, Juvenile obesity enhances emotional memory and amygdala plasticity through glucocorticoids, J. Neurosci. 35 (2015) 4092–4103, http://dx.doi.org/10.1523/JNEUROSCI.3122-14.2015.
[14] K.N. Boutelle, C.E. Wierenga, A. Bischoff-Grethe, A.J. Melrose, E. Grenesko-Stevens, M.P. Paulus, W.H. Kaye, Increased brain response to appetite tastes in the insula and amygdala in obese compared with healthy weight children when sated, Int. J. Obes. 39 (2015) 620–628, http://dx.doi.org/10.1038/ijo.2014.026.
[15] J.A. Brillat-Savarin, The Physiology of Taste or Meditations on Transcendent Gastronomy, Translated From the Last Paris Edition by F. Robinson, 1825.
[16] K.S. Burger, G.E. Shearrer, A.J. Sanders, Brain-based etiology of weight regulation, Curr. Diab. Rep. 15 (2015) 100, http://dx.doi.org/10.1007/s11892-015-0667-5.
[17] L. Calandreau, A. Desmedt, L. Decorte, R. Jaffard, A different recruitment of the lateral and basolateral amygdala promotes contextual or elemental conditioned association in Pavolvian fear conditioning, Learn. Mem. 12 (2005) 383–388, http://dx.doi.org/10.1101/lm.92305.
[18] O.G. Cameron, Visceral Sensory Neuroscience: Interoception, Oxford University Press, New York, NY, 2002.
[19] L.G. Cheke, J.S. Simons, N.S. Clayton, Higher body mass index is associated with episodic memory deficits in young adults, Q. J. Exp. Psychol. (2016), http://dx.doi.org/10.1080/17470218.2015.1099163.
[20] G. Coppin, S. Nolan-Poulart, M. Jones-Gotman, D.M. Small, Working memory and reward association learning impairments in obesity, Neuropsychologia 65 (2014) 146–155, http://dx.doi.org/10.1016/j.neuropsychologia.2014.10.004.
[21] G. Coppin, D. Sander, B. Schaal, C. Ferdenzi, O. Wathelet, Neuropsychologie affective et Olfaction: Etudier la sensibilité de l'amygdale aux odeurs pour tester les théories de l'émotion [Affective neuropsychology and olfaction: investigate amygdala sensitivity to smells to test theories of emotion], Odeurs et émotions. Le nez a ses raisons… [Odors and Emotions. The Nose has its Reasons…], Presses Universitaires de Dijon, Dijon 2014, pp. 383–398.
[22] L. Cordain, S.B. Eaton, A. Sebastian, N. Mann, S. Lindeberg, B.A. Watkins, J.H. O'Keefe, J. Brand-Miller, Origins and evolution of the Western diet: health implications for the 21st century, Am. J. Clin. Nutr. 81 (2005) 341–354.
[23] Z.A. Cordner, K.L.K. Tamashiro, Effects of high-fat diet exposure on learning & memory, Physiol. Behav. 152 (2015) 363–371, http://dx.doi.org/10.1016/j.physbeh.2015.06.008.
[24] E. Coutureau, P.J. Blundell, S. Killcross, Basolateral amygdala lesions disrupt latent inhibition rats, Brain Res. Bull. 56 (2001) 49–53, http://dx.doi.org/10.1016/S0361-9230(01)00592-5.
[25] H.D. Critchley, E.T. Rolls, Hunger and satiety modify the responses of olfactory and visual neurons in the primate orbitofrontal cortex, J. Neurophysiol. 75 (1996) 1673–1686.
[26] J.N. Darling, A.P. Ross, T.J. Bartness, M.B. Parent, Predicting the effects of a high-energy diet on fatty liver and hippocampal-dependent memory in male rats, Obesity 21 (2013) 910–917, http://dx.doi.org/10.1002/oby.20167.

[27] T.L. Davidson, L.E. Jarrad, A role for hippocampus in the utilization of hunger signals, Behav. Neural Biol. 59 (1993) 167–171.
[28] T.L. Davidson, S.E. Kanoski, E.K. Walls, L.E. Jarrad, Memory inhibition and energy inhibition, Physiol. Behav. 86 (2005) 731–746, http://dx.doi.org/10.1016/j.physbeh.2005.09.004.
[29] T.L. Davidson, S.E. Kanoski, L.A. Schier, D.J. Clegg, S.C. Benoit, A potential role for the hippocampus in energy intake and body weight regulation, Curr. Opin. Pharmacol. 7 (2007) 613–616, http://dx.doi.org/10.1016/j.coph.2007.10.008.
[31] T.L. Davidson, S.E. Kanoski, K. Chan, D.J. Clegg, S.C. Benoit, L.E. Jarrad, Hippocampal lesions impair retention of discriminative responding based on energy state cues, Behav. Neurosci. 124 (2010) 97–105, http://dx.doi.org/10.1037/a0018402.
[32] A. Dietrich, M. Hollmann, D. Mathar, A. Villringer, A. Horstmann, Brain regulation of food craving: relationships with weight status and eating behavior, Int. J. Obes. (2016), http://dx.doi.org/10.1038/ijo.2016.28.
[33] M.S. Fanselow, H.W. Dong, Are the dorsal and ventral hippocampus functionally distinct structures? Neuron 65 (2010) 7–19, http://dx.doi.org/10.1016/j.neuron.2009.11.031.
[34] M. Fastenrath, D. Coynel, K. Spaiek, A. Milnik, L. Gschwind, B. Roozendaal, A. Papassotiropoulos, D.J.F. de Quervain, Dynamic modulation of amygdala-hippocampal connectivity by emotional arousal, J. Neurosci. 34 (2014) 13935–13947, http://dx.doi.org/10.1523/JNEUROSCI.0786-14.2014.
[35] H.L. Fehm, W. Kern, A. Peters, The selfish brain: competition for energy resources, Prog. Brain Res. 153 (2006) 129–140, http://dx.doi.org/10.1016/S0079-6123(06)53007-9.
[36] J. Ferbinteanu, R.J. McDonald, Dorsal and ventral hippocampus: same or different? Psychobiology 3 (2000) 314–324.
[38] H.M. Francis, R.J. Stevenson, Higher reported saturated fat and refined sugar intake is associated with reduced hippocampal-dependent memory and sensitivity to interoceptive signals, Behav. Neurosci. 125 (2011) 943–955, http://dx.doi.org/10.1037/a0025998.
[39] M.J. Frank, R.C. O'Reilly, A mechanistic account of striatal dopamine function in human cognition: psychopharmacological studies with cabergoline and haloperidol, Behav. Neurosci. 120 (2006) 497–517.
[40] S. Frey, J. Bergado-Rosado, T. Seidenbecher, H.C. Pape, J.U. Frey, Reinforcement of early long-term potentiation (Early-LTP) in dentate gyrus by stimulation of the basolateral amygdala: heterosynaptic induction mechanisms of late-LTP, J. Neurosci. 21 (2001) 3697–3703.
[41] S.M. Garn, W.R. Leonard, V.M. Hawthorne, Three limitations of the body mass index, Am. J. Clin. Nutr. 44 (1986) 996–997.
[42] M.H. Gastaut, Les troubles du comportement alimentaire chez les épileptiques psychomoteurs [Eating behavior disorders in psychomotor epileptics], Rev. Neurol. 92 (1955) 55–62.
[43] P. Geha, G. Cecchi, P. Constable, C. Abdallah, D.M. Small, Reorganization of Brain Connectivity in Obesity, 2016 Manuscript submitted for publication.
[44] A. Geliebter, Neuroimaging of gastric distension and gastric bypass surgery, Appetite 71 (2013) 459–465, http://dx.doi.org/10.1016/j.appet.2013.07.002.
[45] P.J. Gianaros, L.K. Sheu, K.A. Matthews, R. Jennings, S.B. Manuck, A.R. Hariri, Individual differences in stressor-evoked blood pressure reactivity vary with activation, volume, and functional connectivity of the amygdala, J. Neurosci. 28 (2004) 990–999, http://dx.doi.org/10.1523/JNEUROSCI.3606-07.2008.
[46] M.M. Gonzales, T. Tarumi, S.C. Miles, H. Tanaka, F. Shah, A.P. Haley, Insulin sensitivity as a mediator of the relationship between BMI and working memory-related brain activation, Obesity 18 (2010) 2131–2137, http://dx.doi.org/10.1038/oby.2010.183.
[47] J.A. Gottfried, J. O'Doherty, R.J. Dolan, Encoding predictive reward value in human amygdala and orbitofrontal cortex, Science 301 (2003), http://dx.doi.org/10.1126/science.1087919 1104–1007.
[48] O. Grimm, M.J. Jacob, N.B. Kroemer, L. Krebs, S. Vollstädt-Klein, A. Kobiella, U. Wolfensteller, M.N. Smolka, The personality trait self-directedness predicts the amygdala's reaction to appetizing cues in fMRI, Appetite 58 (2012) 1023–1029, http://dx.doi.org/10.1016/j.appet.2012.02.007.
[49] R. Guerrieri, C. Nederkoorn, A. Jansen, The interaction between impulsivity and a varied food environment: its influence on food intake and overweight, Int. J. Obes. 32 (2008) 708–714, http://dx.doi.org/10.1038/sj.ijo.0803770.
[50] L. Haase, B. Cerf-Ducastel, C. Murphy, Cortical activation in response to pure taste stimuli during the physiological states of hunger and satiety, NeuroImage 44 (2011) 1008–1021, http://dx.doi.org/10.1016/j.neuroimage.2008.09.044.
[51] S.L. Hargrave, S. Jones, T.L. Davidson, The outward spiral: a vicious cycle model of obesity and cognitive dysfunction, Curr. Opin. Behav. Sci. 9 (2016) 40–46, http://dx.doi.org/10.1016/j.cobeha.2015.12.001.
[52] N. Hebben, S. Corkin, H. Eichenbaum, K. Shedlack, Diminished ability to interpret and report internal states after bilateral medial temporal resection: case H.M, Behav. Neurosci. 99 (1985) 1031–1039, http://dx.doi.org/10.1037/0735-7044.99.6.1031.
[53] Y.O. Henderson, G.P. Smith, M.B. Parent, Hippocampal neurons inhibit meal onset, Hippocampus 23 (2013) 100–107, http://dx.doi.org/10.1002/hipo.22062.
[54] S. Higgs, Memory and its role in appetite regulation, Physiol. Behav. 85 (2005) 67–72, http://dx.doi.org/10.1016/j.physbeh.2005.04.003.
[55] S. Higgs, Cognitive influences on food intake: the effects of manipulating memory for recent items, Physiol. Behav. 94 (2008) 734–739, http://dx.doi.org/10.1016/j.physbeh.2008.04.012.
[56] S. Higgs, Cognitive processing of food rewards, Appetite (2016), http://dx.doi.org/10.1016/j.appet.2015.10.003.
[57] S. Higgs, A.C. Williamson, P. Rotshein, G.W. Humphreys, Sensory-specific satiety is intact in amnesics who eat multiple meals, Psychol. Sci. 19 (2008) 623–628, http://dx.doi.org/10.1111/j.1467-9280.2008.02132.x.
[58] S. Higgs, E. Robinson, M. Lee, Learning and memory processes and their role in eating: implications for limiting food intake in overeaters, Curr. Obes. Rep. 1 (2012) 91–98, http://dx.doi.org/10.1007/s13679-012-0008-9.
[59] E.C. Hinton, J.A. Parkinson, A.J. Holland, F.S. Arana, A.C. Roberts, A.M. Owen, Neural contributions to the motivational control of appetite in humans, Eur. J. Neurosci. 20 (2004) 1411–1418, http://dx.doi.org/10.1111/j.1460/9568.2004.03589.x.
[60] R. Hirsh, B. Leber, K. Gillman, Fornix fibers and motivational states as controllers of behavior: a study stimulated by the contextual retrieval theory, Behav. Biol. 22 (1978) 463–478, http://dx.doi.org/10.1016/S0091-6773(78)92583-X.
[61] B.J. Hock, M.D. Bunsey, Differential effects of dorsal and ventral hippocampal lesions, J. Neurosci. 18 (1998) 7027–7032.
[62] P.C. Holland, J.A. Lamoureux, J.S. Han, M. Gallagher, Hippocampal lesions interfere with Pavlovian negative occasion setting, Hippocampus 9 (1999) 143–157, http://dx.doi.org/10.1002/(SICI)1098-1063(1999)9:2<143::AID-HIPO6>3.0.CO;2-Z.
[63] L.M. Holsen, E.A. Lawson, J. Blum, E. Ko, N. Makris, P.K. Fazeli, A. Klibanski, J.M. Goldstein, Food motivation circuitry hypoactivation related to hedonic and nonhedonic aspects of hunger and satiety in women with active anorexia nervosa and weight-restored women with anorexia nervosa, J. Psychiatry Neurosci. 5 (2012) 322–332, http://dx.doi.org/10.1503/jpn.110156.
[65] T.M. Hsu, J.D. Hahn, V.R. Konanur, A. Lam, S.E. Kanoski, Hippocampal GLP-I receptors influence food intake, meal size, and effort-based responding for food through volume transmission, Neuropsychopharmacology 40 (2015) 327–337, http://dx.doi.org/10.1038/npp.2014.175.
[66] T.M. Hsu, J.D. Hahn, V.R. Konanur, E.E. Noble, A.N. Suarez, J. Thai, E.M. Nakamoto, S.E. Kanoski, Hippocampus ghrelin signaling mediates appetite through lateral hypothalamic orexin pathways, eLIFE 4 (2015), e11190, http://dx.doi.org/10.7554/eLIFE.11190.
[67] F.B. Hu, R.M. van Dam, S. Liu, Diet and risk of type II diabetes: the role of types of fat and carbohydrate, Diabetologia 44 (2001) 805–817.
[68] M.L. Huff, E.B. Emmons, N.S. Narayanan, R.T. LaLumiere, Basolateral amygdala projections to ventral hippocampus modulate the consolidation of footshock, but not contextual, learning in rats, Learn. Mem. 23 (2016) 51–60, http://dx.doi.org/10.1101/lm.039909.115.
[69] Y. Ikegaya, H. Saito, K. Abe, High-frequency stimulation of the basolateral amygdala facilitates the induction of long-term potentiation in the dentate gyrus in vivo, Neurosci. Res. 22 (1995) 203–207, http://dx.doi.org/10.1016/0168-0102(95)00894-7.
[70] R. Ito, T.W. Robbins, B.L. McNaughton, B.J. Everitt, Selective excitotoxic lesions of the hippocampus and basolateral amygdala have dissociable effects on appetitive cue and place conditioning based on path integration in a novel Y-maze procedure, Eur. J. Neurosci. 23 (2006) 3071–3080, http://dx.doi.org/10.1111/j.1460-9568.2006.04883.x.
[71] F.N. Jacka, N. Cherbuin, K.J. Anstey, P. Sachdev, P. Butterworth, Western diet is associated with a smaller hippocampus: a longitudinal investigation, BMC Med. 13 (2015) 215, http://dx.doi.org/10.1186/s12916-015-0461-x.
[72] P.H. Janak, K.M. Tye, From circuits to behavior in the amygdala, Nature 517 (2015) 284–292, http://dx.doi.org/10.1038/nature14188.
[73] R.L. Jenison, A. Rangel, H. Oya, H. Kawasaki, A. Howard, Value encoding in single neurons in the human amygdala during decision making, J. Neurosci. 31 (2011) 331–338, http://dx.doi.org/10.1523/JNEUROSCI.4461-10.2011.
[74] I.S. Johnsrude, A.M. Owen, N.M. White, W.V. Zhao, V. Bohbot, Impaired preference conditioning after anterior temporal lobe resection in humans, J. Neurosci. 20 (2000) 2649–2656.
[75] B.R. Kaada, E.W. Rasmussen, O. Kveim, Effects of hippocampal lesions on maze learning and retention in rats, Exp. Neurol. 3 (1961) 333–355, http://dx.doi.org/10.1016/0014-4886(61)900009-7.
[77] S.E. Kanoski, T.L. Davidson, Western diet consumption and cognitive impairment: links to hippocampal dysfunction and obesity, Physiol. Behav. 103 (2011) 59–68, http://dx.doi.org/10.1016/j.physbeh.2010.12.003.
[78] S.E. Kanoski, H.J. Grill, Hippocampus contributions to food intake control: mnemonic, neuroanatomical, and endocrine mechanisms, Biol. Psychiatry (2015), http://dx.doi.org/10.1016/j.biopsych.2015.09.011 (pii: S0006-3223(15)00771-4).
[81] S.E. Kanoski, S.M. Fortin, K.M. Ricks, H.J. Grill, Ghrelin signaling in the ventral hippocampus stimulates learned and motivational aspects of feeding via PI3K-Akt signaling, Biol. Psychiatry 73 (2013) 915–923, http://dx.doi.org/10.1016/j.bipsych.2012.07.002.
[82] P.J. Kennedy, M.L. Shapiro, Retrieving memories via internal context requires the hippocampus, J. Neurosci. 24 (2004) 6979–6985, http://dx.doi.org/10.1523/JNEUROSCI.1388-04-2004.
[83] P.J. Kennedy, M.L. Shapiro, Motivational states activate distinct hippocampal representations to guide goal-directed behaviors, Proc. Natl. Acad. Sci. 106 (2009) 10805–10810, http://dx.doi.org/10.1073/pnas.0903259106.
[84] R.P. Kesner, J.M. Williams, Memory for magnitude of reinforcement: dissociation between the amygdala and the hippocampus, Neurobiol. Learn. Mem. 64 (1995) 237–244, http://dx.doi.org/10.1006/nlme.1995.0006.
[85] K.G. Kjelstrup, F.A. Tuvnes, H.A. Steffenach, R. Murison, E.I. Moser, M.B. Moser, Reduced fear expression after lesions of the ventral hippocampus, Proc. Natl. Acad. Sci. 16 (2002) 10825–10830, http://dx.doi.org/10.1073/pnas.152112399.
[86] H. Klüver, P.C. Bucy, Preliminary analysis of functions of the temporal lobes in monkeys, Arch. Neurol. Psychiatr. 42 (1939) 979–1000.
[87] F.M. Kramer, R.W. Jeffery, J.L. Forster, M.K. Snell, Long-term follow-up of behavioral treatment for obesity: patterns of weight regain among men and women, Int. J. Obes. 13 (1989) 123–136.
[88] K.S. LaBar, D.R. Gitelman, T.B. Parrish, Y.H. Kim, A.C. Nobre, M.M. Mesulam, Hunger selectivity modulates corticolimbic activation to food stimuli in humans, Behav. Neurosci. 115 (2001) 493–500, http://dx.doi.org/10.1037//0735-7044.115.2.493.
[89] J. LeDoux, The Emotional Brain, Simon and Schuster, New York, 1996.

[90] E. Likhtik, J.M. Stujenske, M.A. Topiwala, A.Z. Harris, J.A. Gordon, Prefrontal entrainment of amygdala activity signals safety in learned fear and innate anxiety, Nat. Neurosci. 17 (2014) 106–113, http://dx.doi.org/10.1038/nn.35982.

[91] R. Lilly, J.L. Cummings, F. Benson, M. Frankel, The human Klüver-Bucy syndrome, Neurology 33 (1983) 1141–1145, http://dx.doi.org/10.1212/WNL.33.9.1141.

[92] A. Lindqvist, P. Mohapel, B. Bouter, H. Frielingsdorf, D. Pizzo, P. Brundlin, C. Erlanson-Albertsson, High-fat diet impairs hippocampal neurogenesis in male rats, Eur. J. Neurol. 13 (2006) 1385–1388, http://dx.doi.org/10.1111/j.1468-1331.2006.01500.x.

[93] E.S. Lovitz, L.T. Thompson, Memory-enhancing intra-basolateral amygdala clenbuterol infusion reduces post-burst after hyperpolarizations in hippocampal CA1 pyramidal neurons following inhibitory avoidance learning, Neurobiol. Learn. Mem. 119 (2015) 34–41, http://dx.doi.org/10.1016/j.nlm.2014.12.004.

[94] R. Lübkemann, J. Eberhardt, F.W. Röhl, K. Janitzky, S. Nullmeier, O. Stork, H. Schwegler, R. Linke, Identification and characterization of GABAergic projection neurons from ventral hippocampus to amygdala, Brain Sci. 5 (2015) 299–317, http://dx.doi.org/10.3390/brainsci5030299.

[95] A.H. Luo, P. Tahsili-Fahadan, R.A. Wise, C.R. Lupica, G. Aston-Jones, Linking context with reward: a functional circuit from hippocampal CA3 to ventral tegmental area, Science 333 (2011) 353–356, http://dx.doi.org/10.1126/science.1204622.

[96] K.R. Magnusson, L. Hauck, B.M. Jeffrey, V. Elias, A. Humphrey, R. Nath, A. Perrone, L.E. Bermudez, Relationships between diet-related changes in the gut microbiome and cognitive flexibility, Neuroscience 300 (2015) 128–140, http://dx.doi.org/10.1016/j.neuroscience.2015.05.016.

[97] A. Marschner, R. Kalisch, B. Vervliet, D. Vansteenwegen, C. Büchel, Dissociable roles for the hippocampus and the amygdale in human cued versus context fear conditioning, J. Neurosci. 28 (2008) 9030–9036, http://dx.doi.org/10.1523/JNEUROSCI.1651-08.2008.

[98] A.A. Martin, T.L. Davidson, Human cognitive function and the obesogenic environment, Physiol. Behav. 136 (2014) 185–193, http://dx.doi.org/10.1016/j.physbeh.2014.02.062.

[99] J.L. Mathias, P.S. Kent, Neuropsychological consequences of extreme weight loss and dietary restriction in patients with anorexia nervosa, J. Clin. Exp. Neuropsychol. 20 (1998) 548–564, http://dx.doi.org/10.1017/jcen.20.4.548.1476.

[100] J.L. McGaugh, L. Cahill, B. Roozendaal, Involvement of the amygdala in memory storage: interaction with other brain systems, Proc. Natl. Acad. Sci. 93 (1996) 13508–13514.

[101] S.B. McHugh, R.M.J. Deacon, J.N.P. Rawlins, M. David, Amygdala and ventral hippocampus contribute differentially to mechanisms of fear and anxiety, Behav. Neurosci. 118 (2004) 63–78, http://dx.doi.org/10.1037/0735-7044.118.1.63.

[102] C.K. McIntyre, J.L. McGaugh, C.L. Williams, Interacting brain systems modulate memory consolidation, Neurosci. Biobehav. Rev. 36 (2012) 1750–1762, http://dx.doi.org/10.1016/j.neubiorev.2011.11.001.

[103] E.C. McNay, Insulin and ghrelin: peripheral hormones modulating memory and hippocampal function, Curr. Opin. Pharmacol. 7 (2007) 628–632, http://dx.doi.org/10.1016/j.coph.2007.10.009.

[104] J.R. McReynolds, K.M. Anderson, K.M. Donowho, C.K. McIntyre, Noradrenergic actions in the basolateral complex of the amygdala modulate Arc expression in hippocampal synapses and consolidation of aversive and non-aversive memory, Neurobiol. Learn. Mem. 115 (2014) 49–57, http://dx.doi.org/10.1016/j.nlm.2014.08.016.

[105] C. Messier, Glucose improvement of memory: a review, Eur. J. Pharmacol. 490 (2004) 33–57, http://dx.doi.org/10.1016/j.ejphar.2004.02.043.

[106] A.W. Meyers, A.J. Stunkard, M. Coll, Food accessibility and food choice: a test of Schachter's externality hypothesis, Arch. Gen. Psychiatry 37 (1980) 1133–1135.

[107] B. Milner, Visually-guided maze learning in man: effects of bilateral hippocampal, bilateral frontal, and unilateral cerebral lesions, Neuropsychologia 3 (1965) 317–338, http://dx.doi.org/10.1016/0028-3932(65)90005-9.

[108] R.G.M. Morris, Theories of hippocampal function, in: P. Andersen, R. Morris, D. Amaral, T. Bliss, J. O'Keefe (Eds.), The Hippocampus Book, New York, Oxford University Press 2006, pp. 581–713.

[109] M.J. Morris, J.E. Beilharz, J. Maniam, A.C. Reichelt, R.F. Westbrook, Why is obesity such a problem in the 21st century? The intersection of palatable food, cues and reward pathways, stress, and cognition, Neurosci. Biobehav. Rev. 58 (2015) 36–45, http://dx.doi.org/10.1016/j.neubiorev.2014.12.002.

[110] A.J. Murray, N.S. Knight, L.E. Cochlin, S. McAleese, R.M. Deacon, J.N. Rawlins, K. Clarke, Deterioration of physical performance and cognitive function in rats with short-term high-fat feeding, FASEB J. 12 (2009) 4353–4360, http://dx.doi.org/10.1096/fj.09-139691.

[111] J. Ng, E. Stice, S. Yokum, C. Bohon, An fMRI study of obesity, food, reward, and perceived caloric density. Does a low-fat label make food less appealing? Appetite 57 (2011) 65–72, http://dx.doi.org/10.1016/j.appet.2011.03.017.

[112] L.G. Nilsson, E. Nilsson, Overweight and cognition, Scand. J. Psychol. 50 (2009) 660–667, http://dx.doi.org/10.1111/j.1467-9450.2009.00777.x.

[113] L. Nummenmaa, J. Hirvonen, J.C. Hannukainen, H. Immonen, M.M. Lindroos, P. Salminen, Dorsal striatum and its limbic connectivity mediate abnormal anticipatory reward processing in obesity, PLoS One 7 (2012), e31089.

[114] A. Öhman, S. Mineka, Fears, phobias, and preparedness: toward an evolve module of fear and fear learning, Psychol. Rev. 108 (2001) 483–522, http://dx.doi.org/10.1037/0033-295X.108.3.483.

[115] M.B. Parent, Cognitive control of meal onset and meal size: role of dorsal hippocampal-dependent memory, Physiol. Behav. (2016), http://dx.doi.org/10.1016/j.physbeh.2016.03.036.

[116] M.B. Parent, D.L. Krebs-Kraft, J.P. Ryan, J.S. Wilson, C. Harenski, S. Hamman, Glucose administration enhances fMRI brain activation and connectivity related to episodic memory encoding for neutral and emotional stimuli, Neuropsychologia 49 (2011) 1052–1066, http://dx.doi.org/10.1016/j.neuropsychologia.2011.02.013.

[117] M.B. Parent, J.N. Darling, Y.O. Henderson, Remembering to eat: hippocampal regulation of meal onset, Am. J. Physiol. Regul. Integr. Comp. Physiol. 15 (2014) R701–R713, http://dx.doi.org/10.1152/ajpregu.00496.2013.

[118] L. Passamonti, J.B. Rowe, C. Schwarzbauer, M.P. Ewbank, E. von dem Hagen, A.J. Calder, Personality predicts the brain's response to viewing appetizing foods: the neural basis of a risk factor for overeating, J. Neurosci. 29 (2009) 43–51, http://dx.doi.org/10.1523/JNEUROSCI.4966-08.2009.

[119] L. Pessoa, Emotion and cognition and the amygdala: from "what is it?" to "what's to be done?", Neuropsychologia 48 (2010) 3416–3429, http://dx.doi.org/10.1016/j.neuropsychologia.2010.06.038.

[120] A. Peters, U. Schweiger, L. Pellerin, C. Hubold, K.M. Oltmanns, M. Conrad, B. Schultes, J. Born, H.L. Fehm, The selfish brain: competition for energy resources, Neurosci. Biobehav. Rev. 28 (2004) 143–180, http://dx.doi.org/10.1016/j.neubiorev.2004.03.002.

[121] A. Peters, B. Kubera, C. Hubold, D. Langemann, The selfish brain: stress and eating behavior, Front. Neurosci. 5 (2011) 74, http://dx.doi.org/10.3389/fnins.2011.00074.

[122] G.D. Petrovich, Forebrain circuits and control of feeding by learned cues, Neurobiol. Learn. Mem. 95 (2011) 152–158, http://dx.doi.org/10.1016/j.nlm.2010.10.003.

[123] G.D. Petrovich, Learning and the motivation to eat: forebrain circuitry, Physiol. Behav. 104 (2011) 585–589, http://dx.doi.org/10.1016/j.physbeh.2011.04.059.

[124] E.A. Phelps, Human emotion and memory: interactions of the amygdala and hippocampal complex, Curr. Opin. Neurobiol. 14 (2004) 198–202, http://dx.doi.org/10.1016/j.conb.2004.03.015.

[125] E.A. Phelps, The human amygdala and the control of fear(2009) in: P.J. Whalen, E.A. Phelps (Eds.), The Human Amygdala, Guilford Press, New York 2009, pp. 204–219.

[126] M. Pikkarainen, A. Pitkänen, Projections from the lateral, basal and accessory basal nuclei of the amygdala to the perirhinal and postrhinal cortices in rat, Cereb. Cortex 11 (2001) 1064–1082, http://dx.doi.org/10.1093/cercor/11.11.1064.

[127] A. Pitkänen, M. Pikkarainen, N. Nurminen, A. Ylinen, Reciprocal connections between the amygdala and the hippocampal formation, perirhinal cortex, and postrhinal cortex in rat — a review, Ann. N. Y. Acad. Sci. 911 (2000) 369–391, http://dx.doi.org/10.1111/j.1749-6632.2000.tb06738.x.

[128] E. Pool, T. Brosch, S. Delplanque, D. Sander, Stress increases cue-triggered "wanting" for sweet reward in humans, J. Exp. Psychol. Anim. Learn. Cogn. 41 (2015) 128–136, http://dx.doi.org/10.1037/xan0000052.

[129] E. Pool, S. Delplanque, G. Coppin, D. Sander, Is comfort food really comforting? Mechanisms underlying stress-induced eating, Food Res. Int. (2015), http://dx.doi.org/10.1016/j.foodres.2014.12.034.

[130] A. Rangel, Regulation of dietary choice by the decision-making circuitry, Nat. Neurosci. 16 (2013) 1717–1724, http://dx.doi.org/10.1038/nn.3561.

[131] A.C. Reichelt, J. Maniam, R.F. Westbrook, M.J. Morris, Dietary-induced obesity disrupts trace fear conditioning and decreases hippocampal reelin expression, Brain Behav. Immun. 43 (2015) 68–75, http://dx.doi.org/10.1016/j.bbi.2014.07.005.

[132] C.J. Reppucci, G.D. Petrovich, Learned food-cue stimulates persistent feeding in sated rats, Appetite 59 (2012) 437–447, http://dx.doi.org/10.1016/j.appet.2012.06.007.

[133] S.E.V. Rhodes, D.P. Charles, E.J. Howland, E.A. Murray, Amygdala lesions in rhesus monkeys fail to disrupt object choices based on internal context, Behav. Neurosci. 126 (2012) 270–278, http://dx.doi.org/10.1037/a0027229.

[134] J. Rodin, Current status of the internal-external hypothesis for obesity: what went wrong? Am. Psychoanal. 36 (1981) 361–372.

[135] B.L. Rollins, B.M. King, Amygdala-lesion obesity: what is the role of the various amygdaloid complex? Am. J. Physiol. Regul. Integr. Comp. Physiol. 279 (2000) R1348–R1356.

[136] P. Rozin, S. Dow, M. Moscovitch, S. Rajaram, What causes humans to begin and end a meal? A role for memory for what has been eaten, as evidenced by a study of multiple meal eating in amnesic patients, Psychol. Sci. 9 (1998) 392–396.

[137] K. Rudenga, D.M. Small, Amygdala response to sucrose consumption is inversely related to artificial sweetener use, Appetite 58 (2012) 504–507, http://dx.doi.org/10.1016/j.appet.2011.12.001.

[138] K.J. Rudenga, R. Sinha, D.M. Small, Acute stress potentiates brain response to milkshake as a function of body weight and chronic stress, Int. J. Obes. 37 (2013) 309–316, http://dx.doi.org/10.1038/ijo.2012.39.

[139] C.H. Sample, A.A. Martin, S. Jones, S.L. Hargrave, T.L. Davidson, Western-style diet impairs stimulus control by food deprivation state cues: implications for obesogenic environments, Appetite 93 (2015) 13–23, http://dx.doi.org/10.1016/j.appet.2015.05.018.

[140] D. Sander, J. Grafman, T. Zalla, The human amygdala: an evolved system for relevance detection, Rev. Neurosci. 14 (2003) 303–316, http://dx.doi.org/10.1515/REVNEURO.2003.14.4.303.

[141] C. Sandoval-Salazar, J. Ramírez-Emiliano, A. Trejo-Bahena, C.I. Ovideo-Solís, M.S. Solís-Ortiz, A high-fat diet decreases GABA concentration in the frontal cortex and hippocampus of rats, Biol. Res. 49 (2016) 15, http://dx.doi.org/10.1186/s40659-016-0075-6.

[142] T. Sartorius, C. Ketterer, S. Kullmann, M. Balzer, C. Rotermund, S. Binder, M. Hallschmid, J. Machann, F. Schick, V. Somoza, H. Preissl, A. Fritsche, H.U. Häring, A.M. Hennige, Monounsaturated fatty acids prevent the aversive effects of obesity on locomotion, brain activity, and sleep behavior, Diabetes 61 (2012) 1669–1679, http://dx.doi.org/10.2337/db11-1521.

[143] S. Schachter, Obesity and eating. Internal and external cues differentially affect the eating behavior of obese and normal subjects, Science 161 (1968) 751–756.

[144] J.P. Schroeder, M.G. Packard, Systemic or intra-amygdala injections of glucose facilitate memory consolidation for extinction of drug-induced conditioned reward, Eur. J. Neurosci. 17 (2003) 1482–1488, http://dx.doi.org/10.1046/j.1460-9568.2003.02578.x.

[145] A. Schumacher, E. Vlassov, R. Ito, The ventral hippocampus, but not the dorsal hippocampus is critical for learned approach-avoidance decision making, Hippocampus 26 (2016) 530–542, http://dx.doi.org/10.1002/hipo.22542.
[146] T. Sharot, A.M. Riccardi, C.M. Raio, E.A. Phelps, Neural mechanisms mediating optimism bias, Nature 450 (2007) 102–105, http://dx.doi.org/10.1038/nature06280.
[148] D.M. Small, M.G. Veldhuizen, J. Felsted, Y.E. Mak, F. McGlone, Separable substrates for anticipatory and consummatory food chemosensation, Neuron 57 (2008) 786–797, http://dx.doi.org/10.1016/j.neuron.2008.01.021.
[149] A.P.R. Smith, K.E. Stephan, M.D. Rugg, R.J. Dolan, Task and content modulate amygdala-hippocampal connectivity in emotional retrieval, Neuron 49 (2006) 631–638, http://dx.doi.org/10.1016/j.neuron.2005.12.025.
[150] E. Smith, P. Hay, L. Campbell, J.N. Trollor, A review of the association between obesity and cognitive function across the lifespan: implications for novel approaches to prevention and treatment, Obes. Rev. 12 (2011) 740–755, http://dx.doi.org/10.1111/j.1467-789X.2011.00920.x.
[151] K.E. Speirs, J.T. Hayes, S. Musaad, A. VanBrackle, M. Sigman-Grant, All 4 Kids & Copy, & Obesity Resiliency Research Team, Is family sense of coherence a protective factor against the obesogenic environment? Appetite 99 (2016) 268–276, http://dx.doi.org/10.1016/j.appet.2016.01.025.
[152] L.R. Squire, C.E.L. Stark, R.E. Clark, The medial temporal lobe, Annu. Rev. Neurosci. 27 (2004) 279–306, http://dx.doi.org/10.1146/annurev.neuro.27.070203.144130.
[153] L.R. Squire, A.S. van der Horst, S.G.R. McDuff, J.C. Frascino, R.O. Hopkins, K.N. Mauldin, Role of the hippocampus in remembering the past and imagining the future, Proc. Natl. Acad. Sci. 107 (2010) 19044–19048, http://dx.doi.org/10.1073/pnas.1014391107.
[154] H.C. Steinhausen, The outcome of anorexia nervosa in the 20th century, Am. J. Psychiatr. 159 (2002) 1284–1293, http://dx.doi.org/10.1176/appi.ajp.159.8.1284.
[155] K.T. Stingl, S. Kullmann, C. Ketterer, M. Heni, H.U. Häring, A. Fritsche, H. Preissl, Neuronal correlates of reduced memory performance in overweight subjects, NeuroImage 60 (2012) 362–369, http://dx.doi.org/10.1016/j.neuroimage.2011.12.012.
[156] L.E. Stoeckel, R.E. Weller, E.W. Cook III, D.B. Twieg, R.C. Knowlton, J.E. Cox, Widespread reward-system activation in obese women in response to high-calorie foods, NeuroImage 41 (2008) 636–647, http://dx.doi.org/10.1016/j.neuroimage.2008.02.031.
[157] L.E. Stoeckel, J. Kim, R.E. Weller, J.E. Cox, E.W. Cook III, B. Horwitz, Effective connectivity of a reward network in obese women, Brain Res. Bull. 79 (2009) 388–395, http://dx.doi.org/10.1016/j.brainresbull.2009.05.016.
[158] S. Sullivan, C.R. Cloninger, T.R. Przybeck, S. Klein, Personality characteristics in obesity and Relationship with successful weight loss, Int. J. Obes. 31 (2007) 669–674, http://dx.doi.org/10.1038/sj.ljo.0803464.
[159] X. Sun, N.B. Kroemer, M.G. Veldhuizen, A.E. Babbs, I.E. de Araujo, D.R. Gitelman, R.S. Sherwin, R. Sinha, D.M. Small, Basolateral amygdala response to food cues in the absence of hunger is associated with weight gain susceptibility, J. Neurosci. 20 (2015) 7964–7976, http://dx.doi.org/10.1523/JNEUROSCI.3884/14.2015.
[160] P.B. Sutker, A.N. Allain, J.L. Johnson, N.M. Butters, Memory and learning performances in POW survivors with history of malnutrition and combat veteral controls, Arch. Clin. Neuropsychol. 7 (1992) 431–444.
[161] P.B. Sutker, J.J. Vasterling, K. Brailey, A.N. Allain, Memory, attention, and executive deficits in POW survivors: contributing biological and psychological factors, Neuropsychology 9 (1995) 118–125.
[162] L.W.W. Swanson, Cerebral hemiphere regulation of motivated behavior, Brain Res. 886 (2000) 113–164.
[163] L.W.W. Swanson, G.D. Petrovich, What is the amygdala? Trends Neurosci. 21 (1998) 323–331, http://dx.doi.org/10.1016/S0166-2236(98)01265-X.
[164] P. Sweeney, Y. Yang, An excitatory ventral hippocampus to lateral septum circuit that suppresses feeding, Nat. Commun. 6 (2015) 10188, http://dx.doi.org/10.1038/ncomms10188.
[165] A. Taghva, J.D. Corrigan, A.R. Rezai, Obesity and brain addiction circuitry: implications for deep brain stimulation, Neurosurgery 71 (2012) 224–238, http://dx.doi.org/10.1227/NEU.0b013e31825972ab.
[166] D.W. Tang, L.K. Fellows, D.M. Small, A. Dagher, Food and drug cues activate similar brain regions: a meta-analysis of functional MRI studies, Physiol. Behav. 106 (2012) 317–324, http://dx.doi.org/10.1016/j.physbeh.2012.03.009.
[167] M.S. Tryon, C.S. Carter, R. DeCant, K.D. Laugero, Chronic stress exposure may affect the brain's response to high calorie food cues and predispose to obesogenic eating habits, Physiol. Behav. 120 (2013) 233–242, http://dx.doi.org/10.1016/j.physbeh.2013.08.010.
[168] E.L. Underwood, L.T. Thompson, A high-fat diet causes impairment in hippocampal memory and sex-dependent alterations in peripheral metabolism, Neural Plast. (2016), http://dx.doi.org/10.1155/2016/7385314.
[169] L.N. van der Laan, D.T.D. de Ridder, M.A. Viergever, P.A.M. Smeets, The first taste is always with the eyes: a meta-analysis on the neural correlates of processing visual food cues, NeuroImage 55 (2011) 296–303, http://dx.doi.org/10.1016/j.neuroimage.2010.11.055.
[170] T. van Strien, J.E.R. Frijters, G.P.A. Bergers, P.B. Defares, The Dutch Eating Behaviour Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior, Int. J. Eat. Disord. 5 (1986) 295–315, http://dx.doi.org/10.1002/1098-108X(198602)5:2<295::AID-EAT2260050209>3.0.CO;2-T.
[171] L. Vartanian, W.H. Chen, N.M. Reily, A.D. Castel, The parallel impact of episodic memory and episodic future thinking on food intake, Appetite 101 (2016) 31–36, http://dx.doi.org/10.1016/j.appet.2016.02.149.
[172] G.J. Wang, J. Yang, N.D. Volkow, F. Telang, Y. Ma, W. Zhu, C.T. Wong, D. Tomasi, P.K. Thanos, J.S. Fowler, Gastric stimulation in obese subjects activates the hippocampus and other regions involved in brain reward circuitry, Proc. Natl. Acad. Sci. 103 (2006) 15641–15645, http://dx.doi.org/10.1073/pnas.0601977103.
[173] G.J. Wang, D. Tomasi, W. Backus, R. Wang, F. Telang, A. Geliebter, J. Korner, A. Bauman, J.S. Fowler, P.K. Thanos, N.D. Volkow, Gastric distension activates satiety circuitry in the human brain, NeuroImage 39 (2008) 1824–1831, http://dx.doi.org/10.1016/j.neuroimage.2007.11.008.
[174] K.M. Wassum, A. Izquierdo, The basolateral amygdala in reward learning and addiction, Neurosci. Biobehav. Rev. 57 (2015) 271–283, http://dx.doi.org/10.1016/j.neubiorev.2015.08.017.
[175] P.J. Whalen, E.A. Phelps, The Human Amygdala, Guilford Press, New York, 2009.
[176] D.S. Wheeler, S.E. Chang, P.C. Holland, Odor-mediated taste learning requires dorsal hippocampus, but not basolateral amygdala activity, Neurobiol. Learn. Mem. 101 (2013) 1–7, http://dx.doi.org/10.1016/j.nlm.2012.12.015.
[177] G.E. Wimmer, D. Shohamy, Preference by association: how memory mechanisms in the hippocampus bias decisions, Science 338 (2012) 270–273, http://dx.doi.org/10.1126/science.1223252.
[178] M. Wu, T. Brockmeyer, M. Hartmann, M. Skunde, W. Herzog, H.C. Friederich, Reward-related decision making in eating and weight disorders: a systematic review and meta-analysis of the evidence from neuropsychological studies, Neurosci. Biobehav. Rev. 61 (2016) 177–196, http://dx.doi.org/10.1016/j.neubiorev.2015.11.017.
[179] S. Diekelmann, J. Born, The memory function of sleep, Nat. Rev. Neurosci. 11 (2010) 114–126, http://dx.doi.org/10.1038/nrn2762.
[180] G. Winocur, J.M. Wojtowicz, M. Sekeres, J.S. Snyder, S. Wang, Inhibition of neurogenesis interferes with hippocampus-dependent memory function, Hippocampus 16 (2006) 296–304, http://dx.doi.org/10.1002/hipo.20163.
[181] A.D. Mueller, R.J. Mear, R.E. Mistlberger, Inhibition of hippocampal neurogenesis by sleep deprivation is independent of circadian disruption and melatonin suppression, Neuroscience 193 (2011) 170–181, http://dx.doi.org/10.1016/j.neuroscience.2011.07.019.
[182] X. Chen, M.A. Beydoun, Y. Wang, Is Sleep Duration Associated With Childhood Obesity? A Systematic Review and Meta-analysis, Obesity 16 (2008) 265–274, http://dx.doi.org/10.1038/oby.2007.63.