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Neurophysiological Basis of Food Craving

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1. Introduction

Craving is defined as an irresistible urge to consume a substance and its study was initiated in the field of drugs, considering that it constituted an important base for maintaining addictions (Tiffany, 1990, 1995). From a psychophysiological point of view it would be a motivational state that encourages consumption of both, drugs or food (Cepeda-Benito & Gleaves, 2001).

Psychological explanations based on learning theories, being appropriate, are insufficient to explain the irresistible desire for food. That food craving seems to share the neurophysiological basis with the craving for drugs.

The addictive substances share some ability to induce lasting structural changes in the central nervous system, specifically in regions implicated in reinforcement-motivation. Situational elements associated with the intake of these substances become attractive or outgoing incentives. In short, sensitization maintains the addictive behaviour, beyond or independently of other motivational elements (e.g., the rewarding effect of substances) or aversive properties specific to the situation of abstinence. This model of Robinson and Berridge (2003) would be different from the proposed theories of incentive or homeostatic theories.

Craving for drugs and food craving have differences, which seem to lie in the ability of the drug to sensitize, more intensely, the dopaminergic systems, although the process, in both cases is similar, sharing the same brain structures. In craving for drugs, incentive properties of substances (which tend to increase gradually) and the subjective pleasurable effects (which usually decrease) are usually differentiated. In order to understand the phenomenon of food craving it must be distinguished between what one likes and what one wants. Usually one wants what one likes and one likes what one wants, but both (wanting and liking) do not always go together. It seems that the neural substrates are different in each case. The taste, pleasure or enjoyment of food is determined by the opioid system and the

system of neurotransmitters gamma-amino-butyric acid/ benzodiazepines, GABA/BZD), anatomically located in the ventral pallidum and primary gustatory areas of the brainstem. On the other hand, the desire for food (appetitive aspect, incentive) is determined by the mesencephalic dopaminergic system anatomically located in the nucleus accumbens and amygdala.

Neurotransmitter	Effect
Dopamine decrease	Dysphoria *
Serotonin decrease	Dysphoria
γ -Aminobutyric acid decrease	Anxiety **
Neuropeptide Y decrease	Anti-stress
Dynorphin increase	Dysphoria
Corticotropin-releasing factor increase	Stress
Norepinephrine increase	Stress

Table 1. Aversive emotional effects caused by neurotransmitter changes in abstinence of substances (From Koob & Le Moal, 2008)

* Any unpleasant or uncomfortable mood (dissatisfaction, irritableness)

** A combination of edgy symptoms, difficulty in concentration, muscular tension, sleep disturbances, etc.

Taste and desire for food may occur outside of subjective consciousness. As a result, it may be difficult for humans to distinguish between what they like (pleasure) and what they want (craving). Pelchat et al., (2004) identified a specific brain activation in subjects with food craving, located in the hippocampus, insula and caudate. The activation of such structures has been shown in experimental induction studies on the desire for food or drugs. It has been suggested that hippocampus and insula evoke the memory of craving precipitators reinforcing stimuli, whereas the dopamine released in the caudate nucleus is related to the incentive to these stimuli. The desire, as craving, liking or both, has been linked to the parahippocampal and fusiform gyrus, putamen, anterior cingulate cortex, amygdala and orbitofrontal cortex. These last two structures seem to be a key for the motivational control of eating behaviour. What is the role of those extrinsic determinants of the desire for food (learned) that are capable of arousing the desire for it without the homeostatic deficit related with hunger? It seems that the amygdala would be a meeting point of the value of the food given by hunger with the hedonic properties (learning) of that food. We also know that hunger is able to modulate orbitofrontal activity related to the information of the food (sensory, affective value, previous experience) to guide the subsequent behaviour.

The prefrontal cortex mediates complex executive functions (e.g., self-control). It is known that orbitofrontal damage causes behavioural disinhibition and perseveration, with failure in the assessment of the consequences of one's own actions. In addition, dorsolateral lesions cause cognitive deficits such as reduced ability to relate stimuli, less capacity for abstraction and rigidity of thought. Finally, a global damage at the level of medial frontal cortex and anterior cingulate is associated with apathy and lack of future planning.

If craving is associated with brain changes induced by substances, such changes, in turn, will cause a psychological change. Thus, a dysfunction of the cortical systems, that govern decision-making and behavioural inhibition, leads to emotional and cognitive deregulation. A reduced prefrontal activity may increase the activity of subcortical dopamine systems by raising the appetite awareness. In summary, dopaminergic hyperactivity may cause a low activity of prefrontal cortex related to impulse control deficits.

Different substances and food are not the only factors that may sensitize dopaminergic mesocortical system resulting in an "isolation" of the prefrontal cortex to devote itself to less rational behaviours. Daily environmental stressors causing anxiety may sensitize chronically subcortical areas (nucleus accumbens, amygdala and striatum), which are the basis of impulse or acquired appetite manifested as craving (for drugs or food). The mesocortical dopaminergic system hyperactivity (caused by drugs, food or anxiety) increases sensitivity to craving (with relapse, in the case of food, in form of binge eating). The experience of craving is irrational, and there is a deficit of frontal inhibitory control over subcortical systems that mediate incentive appetitive responses and automated and unconscious behaviours.

But the irrational overwhelming desire (craving) is often accompanied by an attempt of rational avoidance. Thus, the first pre-attentive attraction for food (craving) is often accompanied by attempts to avoid its use (restriction), thus emerging an approach-avoidance motivational conflict. The approach would be automatic, pre-attentive, involuntary, emotional, impulsive and irrational (craving) with a subcortical base, and avoidance would be aware, attentive, voluntary, cognitive, planned and rational (control) with a cortical base.

To some extent, it would be correct to say that an aberrant functioning of a body homeostatic system occurs. This system would have the hypothalamus as its brain structure, which receives hormonal signals of hunger and satiety (e.g., leptin released by adipocytes during satiety or ghrelin secreted in the stomach during hunger). The system seems to be perfect to respond to signals of hunger and satiety, when to eat and when to stop eating.

However, paradoxically, human beings can eat while satiated and, therefore, they have other reasons (pleasure) to eat beyond hunger. That brings to mind a second system involved in food motivation, the motivational or reward system. This reward system seems to be constituted by a neural network of cortical and mesolimbic structures with a core role of the nucleus accumbens of the striatum. This reward system has been evolutionarily modified so that pleasurable stimuli (essential for survival such as food, sex and other natural rewards) are attended, desired and wanted, while aversive stimuli (predators, poisons) are also attended but as a result avoided and unwanted. Therefore, the system responds to motivationally relevant cues.

Regarding the food, the reward system regulates the experience or desire to eat (craving) and hedonic responses to food (liking). The desire or craving is associated with dopamine release while liking is also modulated by the release of endogenous opioids such as endorphins.

Eating behaviour results from the interaction of both systems, assuming that motivational-hedonic mechanisms might nullify purely homeostatic mechanisms. Thus, the mere presence of food-related stimuli would become more powerful than the usual satiety signals facing the intake of food.

The role of the motivational system is of interest in understanding both the normal and pathological eating behaviour. Based on animal experiments it is known that rats make less effort to obtain food and eat less when their dopaminergic activity is eliminated. Similarly, human neuroimaging research has shown that dopamine is directly involved in food craving. Using positron emission tomography (PET) an increased release of dopamine has been observed in the striatum in hungry participants compared with satiated participants, both exposed to food stimuli. Furthermore, the amount of dopamine correlated positively with the subjective experience of food craving. On the other hand, it is well known that antipsychotic drugs (blockers of the dopamine D2-receptor binding) increase appetite and often lead to weight gain (sometimes important), while amphetamines (which increase dopamine activity in the brain) reduce appetite.

Wang et al., (2004) found by means of PET, a significant reduction in the density of dopamine-D2 receptors in the striatum of obese patients compared with individuals of normal weight. The number of dopamine-D2 receptors negatively correlated with BMI in obese participants, so that the higher the degree of obesity the lower the number of these receptors. Previously, other authors have found a higher prevalence of the Taq1-A1 allele in obese patients. This allele is also associated with fewer dopamine-D2 receptors. More recent studies have confirmed the relationship between overweight/obesity and a depression of the dopaminergic reward system.

Returning to the beginning, it should be noted that the finding of a lower density of these receptors in obesity has also been found in drug addiction. The question arises whether this reduction in D2-dopamine receptors density is a cause or a consequence of both obesity and substance dependencies. Some authors state that it would be a down-regulation caused by overstimulation of the reward system due to the chronic use of a substance or a sustained overeating. For others, the reduction in receptors density would be an indicator of an innate vulnerability to reach an addiction. Blum et al. (2000) speak of a "reward deficiency syndrome" in which people with fewer dopamine receptors lack the ability to enjoy the simple and routine rewards of everyday life (for lack of adequate dopamine release in response to these stimuli). Therefore, these people are driven to seek more potent stimuli of reward, like food or drugs.

At this point it is worth mentioning some differences with respect to hunger, appetite and craving. Hunger is the basic, very physical need for food. It happens around three to four hours after eating the last meal once the stomach has emptied. The muscular walls of the stomach begin to contract and grind, sending neurohormonal messages to the brain, indicating that it is time to eat again. Meanwhile, dipping levels of blood sugar send similar signals to the brain.

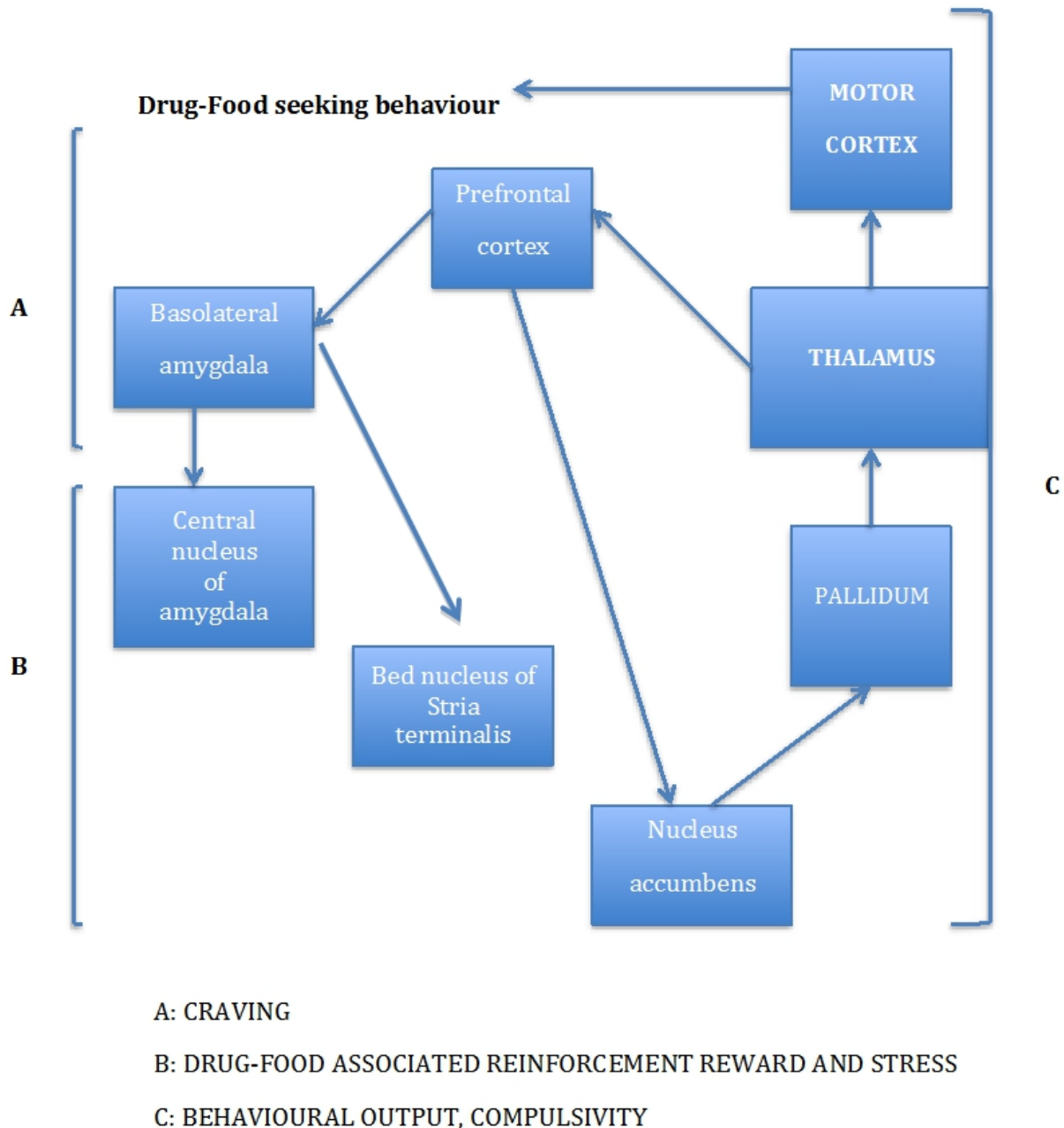


Figure 1. Neural circuits involved in addiction. Modified from Le Moal and Koob (2007)

Appetite is all about the desire for food triggered by anything from the thought, smell and sight of it. One can have an appetite for something even when physically full. The list of things that stimulate, tempt and perpetuate appetite are highly personal and almost endless, and it's easy to see how one can mix them up with genuine cues of physical hunger.

In addition to hunger and appetite, many people experience cravings, a powerful longing for one particular type of food. In practice, cravings are usually emotionally based or simply down to habit.

1.1. Summarizing

- From a psychophysiological point of view, food craving can be defined as a motivational state that encourages the consumption of food.
- Although it appears that in drug craving, drugs sensitize more intensely the dopaminergic systems, the craving for drugs and food share brain structures.
- Taste, pleasure or enjoyment of food is determined by the opioid system and by the system GABA/BZD neurotransmitters. On the other hand, the desire for food is determined by the mesencephalic dopaminergic system.
- The amygdala would be the meeting point between the value of food caused by hunger and the hedonic properties of food.
- Craving is associated with brain changes produced by food, and such changes in turn cause psychological changes.
- Everyday stressors that cause anxiety can chronically sensitize the subcortical areas, which are the base of the impulse or acquired appetite (craving).
- Humans can eat being satiated, so there may be other motives (pleasure) to eat beyond hunger. This suggests a second system involved in food motivation, namely motivational or reward system.
- The “reward deficiency syndrome” (Blum et al., 2000) assumes that people with fewer dopamine receptors do not properly enjoy the regular and simple rewards of everyday life (due to a lack of an adequate release of dopamine after these stimuli), which would lead them to search for more powerful stimuli of reward as, for example, food.

2. Obesity and addiction

Once the link between craving for substances or food is analysed, it is easy to assume that it could be a link between intake and addiction. In fact, there are some similarities between obesity and substance dependencies. In both cases there is a problematic central behaviour: the intake of something repeatedly, excessively, uncontrollably, causing an immediate strengthening effect but which has long-term dire consequences on both physical and psychosocial health. In treatment programs for both obesity and substance dependence, relapses are frequent, the usual relapse trigger being an intense craving that leads to loss of control. It seems that in both cases there is an excessive response to stimuli associated with the substance (drug or food).

In addictions, the responses to signals of substances (craving) are essential for the persistence of dependence. The release of dopamine in the reward system is associated with cognitive reactivity (e.g., attention bias), physiological (e.g., tachycardia), subjective (craving) and behavioural (e.g., approach behaviour) to the perception of signals related to substances. To explain the relationship between the perception of stimuli and substance use different models have been proposed as, for example, the model of Robinson and Berridge (1993), which considers that due to the sensitization of the dopaminergic system of reward (for the repeated use of a substance) certain qualities (incentives) are attributed not only to the substance but to the entire set of stimuli associated with it (by a process of classical

conditioning). Thus, the mere perception of such stimuli would induce a classically conditioned dopamine release in the mesolimbic reward system. The consequence is that such stimuli catch the attention, cause craving and lead to the search for the substance. The model of Robinson and Berridge (1993) was modified by Franken (2003) who added the idea that attention bias and craving have mutually excitatory interactions.

These neurocognitive models related with substances may be applied to eating behaviour, especially in overweight/obesity. The first models of addiction applied to eating behaviour focused on physiological reactivity of food signals (brain response), while more recent models focus on attention bias to stimuli associated with food. The basis of these models is that in obese people there is an increased reactivity to stimuli associated with food compared with people at normal weight.

Our relationship with food presents a clear difference with the relationship with drugs: we need food and we cannot do without it, as we can do with drugs. Therefore, our reward system responds to the food as something attractive, attention grabber and desired. In this sense all persons may be "addicted" to food. However, due to sensitization and hyperresponsiveness (innate or acquired) of the reward system obese people have a greater attention bias than people at normal weight.

This attention bias toward food-related stimuli would be greater in obese people in situations of hunger and satiety. This seems to be contrary to the internality-externality theory of Schachter (1968, 1971), which assumes that obese people would be insensitive to internal cues of hunger and satiety.

Attention bias to stimuli associated with food is related to energy intake and this energy intake is also related with food craving. The reciprocal stimulation between the attention to food and food craving leads people to seek food. This behavioural response is enhanced as it may be seen in obese people compared with persons at normal weight.

People with an intake based on external stimuli usually show greater reactivity to food-related signals. This reactivity is expressed as attention bias, food craving and energy intake.

As obesity, it has been suggested that binge eating disorder has common aspects with addictions. The experience of bingeing is accompanied by the feeling of loss of control and other negative feelings, and often occurs after a previous period of more or less restriction. In patients with binge eating disorder some attention biases have been found (e.g., with the Stroop test) and comparing obese women with binge eating disorder with obese women without that disorder, the first group report more craving related with food stimuli. Therefore it seems that there would be a relationship between the presence of binge eating and the responsiveness to food-related stimuli.

Negative affects have also been analysed in relation to overeating. For example, it was found that patients with overweight/obesity without associated eating disorders and with high negative affect show a tendency to binge in response to negative mood induction and food exposure, while patients with overweight/obesity without associated eating disorders

and with low negative affect and normal weight participants usually eat a similar amount of food under the same experimental conditions. The conclusion is that obese or overweight patients with high negative affectivity present extra difficulties to resist the temptation to eat.

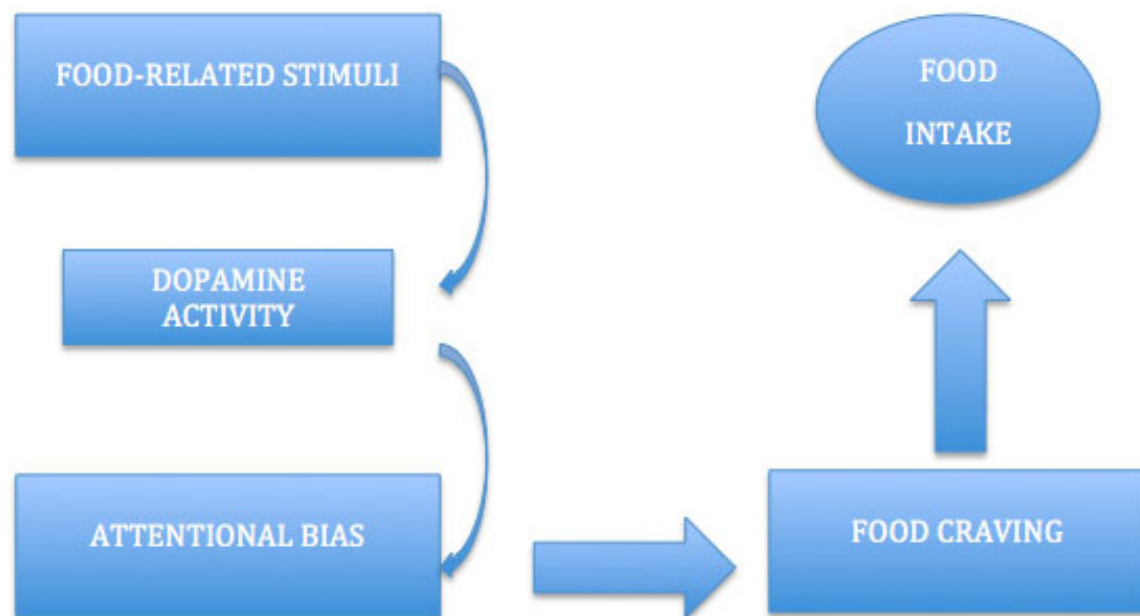


Figure 2. Neurocognitive model derived from Franken (2003).

Something similar occurs with stress, which could increase the vulnerability to eat after being exposed to food signals. Indeed both acute and chronic stresses are related with the maintenance of obesity and with relapse (presence of binge eating) as well as with a greater appetite for hypercaloric foods. It seems that stress signals enter into interaction with the reward system.

2.1. Summarizing

- The neurocognitive models related with substances may be applied to eating behaviour, especially in overweight/obesity.
- The base of these models is that in obese people there is an increased reactivity to stimuli associated with food compared with people at normal weight
- Due to sensitization and hyperresponsiveness (innate or acquired) of the reward system obese people have a greater attention bias than people at normal weight.
- As obesity, it has been suggested that binge eating disorder has common aspects with addictions.
- In patients with binge eating disorder some attention biases have been found and comparing obese women with binge eating disorder with obese women without that disorder, the first group report more craving related with food stimuli.
- Overweight/obese patients without associated eating disorders and with high negative affect show a tendency to binge in response to negative mood induction and food

exposure, while overweight/obese patients without associated eating disorders and with low negative affect and normal weight participants usually eat.

- Both acute and chronic stresses are related with the maintenance of obesity and with relapse (presence of binge eating) as well as with a greater appetite for hypercaloric foods. It seems that stress signals enter into interaction with the reward system.

3. Psychophysiology of craving

As it has been seen before, the current basic model of study focuses on the reactivity to stimuli or signals (cue reactivity). Studies are based on exposing the presence of food and recording the physiological and subjective responses of participants. The response to a substance-related stimuli and the response to the substance itself are different (opposite) for the so called homeostatic theories. However, theories based on the incentive effects of the substances indicate that physiological responses will be consistent with the reinforcing capacities of the substances. Cognitive models also predict different results depending on the different theories of these models. For some authors (Baker et al., 1987) it would be two motivational systems involved in craving, the appetitive and the aversive. According to Baker et al., both can be activated directly by the substances thus contributing to the compulsive consumption of them. Being two reciprocally inhibitory systems, the response to the substance is given by the system that prevails: appetitive response (as indicated by the theories of incentive) or aversive response (as suggested by the homeostatic theories). Tiffany (1990) states that the responses associated with the substance and the contextual stimuli related to their consumption are linked to the consequent behaviour. Faced with substance-related stimuli, it is possible to observe, for example, an increase in the heart rate.

But what does this physiological response mean? It could indicate a preparation for physical action (Obrist et al., 1970), a cognitive effort to process the desire of the substance (Tiffany, 1990) or a negative affect resulting from the frustration of not being able to consume the substance (Drobes et al., 2001). Therefore, faced with a substance, the physiological responses may indicate different aspects to consider. There is a good example of the complexity of the meaning of the responses. Thus, an increase in skin conductance as a response to the smell and the presence of alcohol in alcoholics has been found (Staiger et al., 1999) as well as a response to the presence of chocolate cookies in people with food craving (Wilson & Mercer, 1990). However, the presentation of stimuli-related and not related to alcohol causes a similar skin conductance in alcoholics (Stormak et al., 1993), and considering the psychophysiological responses to food, restrictive and not restrictive people, have similar responses (Overduin et al., 1997).

Other psychophysiological measures such as heart rate and blood pressure have been considered as powerful psychophysiological predictors of eating behaviour. For example, induction of stress (with increased heart rate and blood pressure) may inhibit food intake in non-restrictive females (but not in restrictive). In some diseases, such as bulimia nervosa, an increased attention to pictures of food accompanied by slow heart rate has been observed (Laberg et al., 1991).

The psychophysiological measures of food craving highlights the anticipation of eating (cognitive development), the knowledge of the responses to food signals and the knowledge of affective responses to such stimuli (Cavallo & Pinto, 2001; Lang et al., 1993, Overduin et al., 1997).

The response to food craving in the form of binge eating (not necessary but usual) has led to analyse its triggers, both on the basis of food deprivation as well as postulating negative moods at the origin. The homeostatic model argues that food restriction produces biological effects (e.g., changes in brain neurotransmitters) that cause the uncontrollable desire to eat (craving). Binge eating would be the way to restore the lost balance, in many cases all that happens as well. In patients with eating disorders, the onset of binge eating episodes is frequently preceded by dieting (Green, 2001; Polivy & Herman, 2002; Stice, 2001).

As homeostatic theories, cognitive theories consider that food restriction is a food craving trigger. Thoughts about food, body image and weight are usual explanations that people give about their restrictions or overeating. The restriction involves feelings of psychophysiological deprivation (craving) that lead to loss of behavioural control and possible binge episodes. Moreover if it is likely possible the use of compensatory behaviours, the probability of chaos increases (Gendall & Joyce, 2001; Jansen, 2001). Models based on physiological deprivation (homeostatic) and psychological deprivation (cognitive), as triggers of craving and binge eating, have empirical support. Nevertheless it is well known that only the feeling of hunger does not completely explain the uncontrolled episodes. In addition, the fact that negative emotional states (anxiety, boredom, sadness) may also cause uncontrolled episodes suggests that food deprivation is not a sufficient explanation for the presence of craving and bingeing (Moreno, 2003; Stice & Fairburn, 2003). Considering the distinction between objective and subjective binge eating, it was found that food deprivation with a negative emotional state may raise the former, while negative mood is capable, by itself, to elicit subjective binge eating (Agras & Telch, 1998). The relationship between negative mood and eating behaviour leads to talk about emotional eating, which takes the value of food to alleviate these negative moods. Craving implies a link between emotional states and food intake, although craving does not always lead to the intake (Hetherington & Macdiarmid, 1993). In regards to the relationship between craving and binge eating, the influence of two fundamental variables such as hunger and stress have been suggested. It has even been shown that the craving-binge relationship increases when the sensation of hunger is lower but the feeling of tension is highest. In short, even with less hunger if there is a high tension, the probability that craving will end up in a binge episode increases (Waters, Hill, & Waller, 2001).

3.1. Summarizing

- It would be two motivational systems involved in craving, the appetitive and the aversive, and both can be activated directly by substances/food thus contributing to the compulsive consumption of them.

- Being two reciprocally inhibitory systems, the response to the substance is given by the system that prevails: appetitive response (as indicated by the theories of incentive) or aversive response (as suggested by the homeostatic theories).
- Faced with a substance, the physiological responses may indicate different aspects to consider.
- Induction of stress (with increased heart rate and blood pressure) may inhibit food intake in non-restrictive females (but not in restrictive).
- In some diseases, such as bulimia nervosa, an increased attention to pictures of food accompanied by slow heart rate has been observed.
- The response to food craving in the form of binge eating (not necessary but usual) has led to analyse its triggers, both on the basis of food deprivation as well as postulating negative moods at the origin.
- Models based on physiological deprivation (homeostatic) and psychological deprivation (cognitive), as triggers of craving and binge eating, have empirical support.
- Only the feeling of hunger does not explain completely the uncontrolled episodes. In addition, the fact that negative emotional states (anxiety, boredom, sadness) may also cause uncontrolled episodes suggests that food deprivation is not a sufficient explanation for the presence of craving and bingeing.

4. Conclusions

Theories focused on the psychophysiological mechanisms of food craving (e.g., Robinson & Berridge, 1993, 2003) argue that reinforcement and appetitive motivation that causes food can cause lasting changes in the brain structures involved, as the nucleus accumbens and amygdala. It would be a sensitization of dopaminergic systems that may explain the maintenance of craving regardless of the pleasurable effects of food (as suggested by the theories of incentive) or the aversive effects of food deprivation (as homeostatic theories propose). As noted by Garavan et al. (2000) and Wexler et al. (2001), craving needs the prefrontal and limbic structures involved in cognitive and emotional processes. Therefore, negative emotional states such as anxiety or depression, and those related to food stimuli that cause negative affective reactions can stimulate the mesocortical dopaminergic system and reduce the inhibitory control that the prefrontal and frontal cortex have on the subcortical structures, increasing the vulnerability to food craving. As a result, the sequence represented in Figure 3 would be triggered.

The approach to food can be done as an unconscious, automatic and preattentive level (appetitive motivational system) and avoidance could occur later as an attentional, conscious and controlled level (defence motivational system). Hyperactivation of the amygdala would explain the defensive style and the greater negative affect of people with high food craving and bulimia nervosa.

Overall, the theories that attempt to explain food craving emphasize the role of food deprivation (with the consequent psychological and physical discomfort) or the role of the

relationship between dietary restraint and negative moods (eg, Polivy & Herman, 2002 ; Stice & Fairburn, 2003).

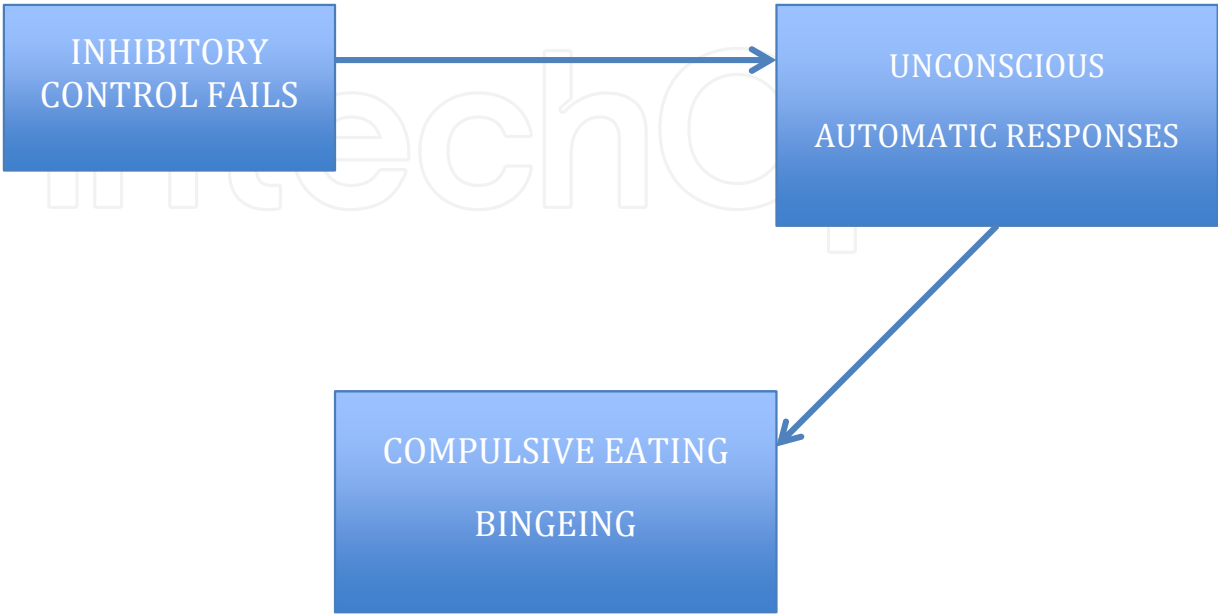


Figure 3. Cortical inhibitory control failure and its consequences.

Certain negative affects reduce the ability to control food intake, resulting in loss of control (binge). Deprivation of food should be accompanied by a negative mood in relation to craving and uncontrolled behaviour. In fact, among patients with bulimia nervosa, the combination of food craving without food deprivation (hunger) and negative affect seems to be the best predictor of binge eating (Moreno, 2003). In summary, the approach-avoidance motivational conflict related to food would be modulated by mood and food deprivation.

In the case of bulimia nervosa, an autonomic hyporeactivity, a defensive style and high negative affect have been reported (e.g., Legenbauer, Vögele, & Ruddel, 2004). This hyporesponsiveness is more characteristic of diffuse anxiety than of fear, and it must be taken into account that anxiety and fear have different neural basis. Thus, the central nucleus of the amygdala is responsible for fear and the bed nucleus of the stria terminalis is responsible for anxiety (Lang et al., 2000), both structures having similar efferent connections and being prepared to respond to significant emotional stimuli when well developed information comes from the basolateral nucleus of the amygdala (Davis, 1992). These subcortical structures can take the emotional control when the prefrontal cortex does not properly inhibit emotional stimuli, with consequent automatic and defensive responses.

The main ideas of this chapter would be summarised as indicated in Figure 4.

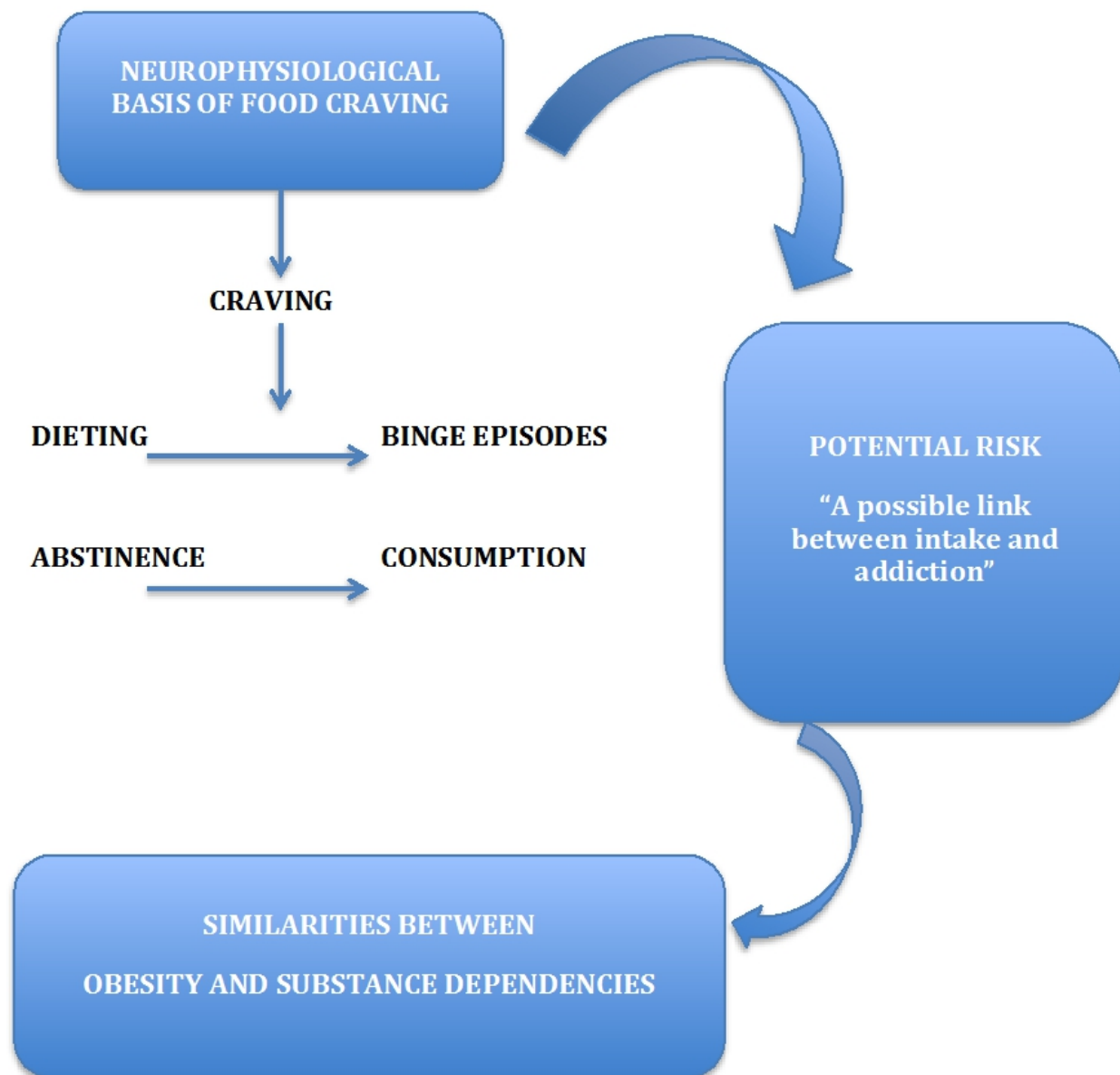


Figure 4. Main relationships with respect to neurophysiological basis of food craving, intake and addiction, and obesity and substance dependencies.

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