

Blunted orosensory perception of lipids during obesity: myth or reality?

Arnaud Bernard^{1,2,3,*}  and Philippe Besnard¹ 

¹ UMR Lipides, Nutrition, Cancer, 1231 Inserm, Université de Bourgogne, AgroSup Dijon, Dijon, France

² Laboratoires Phytocea, Paris, France

³ ABioladvice, Noiron-Sous-Gevrey, France

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Abstract – Obesity is now considered as a disease requiring treatment and prevention efforts by reason of severe associated co-morbidities and its growing prevalence in worldwide population. Although origin of this “epidemic” situation is clearly multifactorial, recent changes in our life-style especially about our food supply with an easy access to low-cost energy-dense foods seem to play a significant causal role in this phenomenon. Studies also report that obesity is frequently associated with a preferential consumption of high palatable foods rich in fat and sugar. Since these foods are pleasant and have high hedonic quality, this obesogenic eating behavior change raises the possibility of an alteration of orosensory perception system as a consequence of obesity. This hypothesis has led to a recent literature on the relationship between obesity and sensory sensitivity, known to play a significant role in the food choice. The purpose of this mini-review, focused on the orosensory perception of dietary lipids (*i.e.* taste of fat), is to provide a short overview of what is observed in food-induced obese rodents as compared to what is found in patients with obesity. It tries to answer to the following basic question: is obesity associated with a loss of orosensory sensitivity to dietary lipids that leads to obesogenic food choices?

Keywords: Taste of fat / obesity / food choices / taste perception

Résumé – L’obésité est désormais considérée comme une maladie en tant que telle nécessitant un traitement et une prévention spécifiques en raison des comorbidités sévères qui lui sont associées et de sa prévalence croissante dans la population mondiale. Bien que l’origine de cette situation « épidémique » soit clairement multifactorielle, les changements récents de notre mode de vie, notamment de notre approvisionnement alimentaire avec un accès aisé à des aliments peu chers mais à forte densité énergétique, semblent jouer un rôle causal important dans ce phénomène. Des études rapportent également que l’obésité est fréquemment associée à une consommation préférentielle d’aliments hautement palatables, riches en graisses et en sucres. Comme ces aliments sont agréables en bouche, ce comportement alimentaire obésogène suggère l’existence d’une altération du système de perception orosensorielle des aliments liée à l’état d’obésité. Cette hypothèse est à l’origine d’études récentes sur la relation entre l’obésité et la gustation, connue pour jouer un rôle important dans le choix alimentaire. L’objectif de cette mini-revue, centrée sur la perception orosensorielle des lipides alimentaires (goût du gras), est de fournir un bref aperçu de ce qui est connu chez les rongeurs rendus obèses par voie alimentaire et ce qui est observé chez les patients souffrant d’une obésité. Elle tentera de répondre à une question basique : l’obésité est-elle associée à une perte de sensibilité orosensorielle aux lipides alimentaires conduisant à des choix alimentaires obésogènes ?

Mots-clés : Goût du gras / obésité / choix alimentaires / perception gustative

*Corresponding author: abioladvice@gmail.com; arnaud@phytocea.com

Highlights

- In laboratory rodents, the overconsumption of highly palatable foods (fatty and/or sweet, high-energy foods) is both the cause and consequence of obesity.
- There is an inverse relationship between taste sensitivity and body fat. The loss of sensitivity is reversible as body fat decreases.
- Various players have been identified in this system: satiety-regulatory hormones, certain metabolites of the kynurenine pathway, and inflammatory factors.
- In humans, these observations are not necessarily true, and no relationship has been observed between lipid sensitivity and BMI.

Introduction

The human specie has evolved in an environment where food scarcity was prevalent (Teaford and Ungar, 2000). This selection pressure has contributed to the gradual establishment of an effective and complex sensory system allowing the selection of food sources high in energy, easily digested, absorbed, metabolized and pleasant. Paradoxically, this evolutionary advantage gradually became problematic when around the middle of the twentieth century a large part of the world's population had gained free access to low-cost and highly palatable (*i.e.* appealing to senses) foods. Often engineered by the agri-food industry to have a high hedonic quality, these foods are generally energy dense because they are rich in fats and sugars. This rapid change in the way of eating with an easy availability of hypercaloric foods is believed to play a significant role in the worldwide epidemic of obesity (Drewnowski, 2003). Due to its rapid progression and associated co-morbidities (type 2 diabetes, vascular disorders, hypertension, cancers, neurodegenerative diseases), obesity is now one of the major public health challenges of the 21st century.

Surprisingly, a lot of patients suffering from obesity also tends to over-consume high palatable foods (Stewart *et al.*, 2011; Ettinger *et al.*, 2012; Proserpio *et al.*, 2016). This obesogenic behavior suggests that an alteration of the orosensory perception might also be the consequence of obesity. Consistent with this assumption it has been reported that the sensory sensitivity can be modified during obesity (Liem and Russell, 2019). The sensory perception of food is critical to provide information about the food eaten and thus is a major contributor to the food choices. Fungiform, circumvallate and foliate papillae mainly found upon the lingual epithelium lining the tongue dorsum are responsible for the chemo-detection of primary tastants (sweet, salty, bitter, sour and umami). Once detected by specific receptors or ionic channels, gustatory signals generated by the taste bud cells are transferred to specific brain areas involved in nutrient sensing and food reward (Carleton *et al.*, 2010).

Compelling evidence suggests that the gustatory system is also involved in the oral fat detection in addition to olfactory and textural cues (Laugerette *et al.*, 2005; Simons *et al.*, 2011, Running *et al.*, 2015; Besnard *et al.*, 2016). According to this finding, it has been hypothesized that the unhealthy food

choices usually observed during obesity might be due to a change in the oral fat sensitivity. The aim of this mini-review is to provide a short overview of our present knowledge in this field of research both in laboratory rodents and in humans.

2 Taste of fat, as a primary taste modality

Laboratory rodents and humans exhibit a spontaneous appeal to foods rich in fats (Drewnowski and Greenwood, 1983; Finlayson, 2017). It has long been considered that this attraction was only based on textural, olfactory and post-ingestive signals. However, it has been demonstrated for a few years now that the sense of taste also plays a role in the orosensory detection of dietary lipids suggesting the existence of a sixth taste modality (Mattes, 2011). In rodents, the spontaneous attraction for dietary fats (Tsuruta *et al.*, 1999) relies on the oral detection of lipids by specific fatty acid receptors identified in the taste bud cells. Functional implication of these receptors (CD36 and FFAR4) in the food sensing has been highlighted using transgenic mouse models. Indeed, when CD36 or FFAR4 are missing, animals become unable to properly detect lipids (*i.e.* long-chain fatty acids, LCFA) during behavioral tests (Laugerette *et al.*, 2005; Cartoni *et al.*, 2010). It has also been shown in mice that the docking of LCFA on CD36 leads to a signaling cascade leading to the generation of a "lipid message" carried through the cranial nerves VII and IX and the nucleus tractus solitarius towards various brain areas implicated in the gustatory and reward pathways (Gaillard *et al.*, 2008). It is noteworthy that the amount of CD36 protein present in the apical side of the mouse taste bud cells is down-regulated following the lipid intake (Martin *et al.*, 2011). Therefore, the orosensory sensitivity to lipids high before the meal, progressively decreases with food intake and reduces the motivation to eat fat-rich food. From a physiological point of view, this dynamic regulation makes sense by adapting the food intake to the metabolic needs. Consistent with this observation, it has been shown in the mouse the existence of a circadian rhythm in taste buds with a coordinated oscillation of key genes involved in circadian clock and lipid detection/signaling (Bernard *et al.*, 2020). It is likely that this phenomenon participates to the modulation of fat taste perception, sensitivity being strong during hunger before meals and then decreasing progressively during food intake.

Altogether, these studies strongly suggest that laboratory rodents are able to specifically discriminate LCFA in the oral cavity independently from olfactory, textural, and post-ingestive influences. Interestingly, human investigations lead to a similar conclusion (Mattes, 2011; Besnard *et al.*, 2016). Rodents and humans exhibit distinct food behaviors, particularly in their responses to foods rich in free fatty acids (FFAs). FFA are found in minute quantities in foods rich in fats. In spoiled foods, the concentration of FFA is increased as the fats are broken down and oxidized. Rodents, as scavenger animals, are inclined to consume decaying food, where triglycerides are broken down, enriching the food with FFAs. This type of food, while appealing to rodents, is unpalatable to humans, who find foods high in FFAs unpleasant. This sensation constitutes a warning signal to prevent the ingestion of potentially toxic expired food. However, the orosensory detection of very small amounts of FFA, produced by the hydrolysis of triglycerides by

lingual lipase, might contribute to a pleasant sensation that makes fatty foods palatable.

3 Diet-induced obesity (DIO) alters the orosensory perception of lipids in rodents

Many studies suggest that perception of dietary lipids is blunted by the diet-induced obesity (DIO) in rats and mice. When DIO rodents were subjected to short term behavioral tests, like licking tests, they were found to be unable to properly detect low concentrations of lipids (Shin *et al.*, 2011; Chevrot *et al.*, 2014). Since licking tests were carried out over very short times (from 10 s to 1 min) using experimental conditions minimizing textural cues, this loss of sensitivity is likely due to a dysfunction of the oral lipid detection system and/or of the central perception of the fat taste signal. Interestingly, this sensory defect is associated to change in the eating behavior. Indeed, diet-induced obese (DIO) rats subjected to multiple food choices preferentially consume the diet with a higher lipid content and, therefore, gradually become obese (Gilbertson *et al.*, 1998). This obesogenic eating behavior is likely a compensatory phenomenon required to achieve a hedonic satisfaction. These changes are directly related to the obesity status of animals (Johnson and Kenny, 2010). Indeed, the decrease in both the perception of dietary lipids (evaluated using a gustometer) and the innate preference for lipids (evaluated with a two-bottle double choice paradigm) are negatively correlated with the fat mass of the animals (Chevrot *et al.*, 2013, Dastugue *et al.*, 2022). According to this observation, the question of the potential reversibility of the observed effects was worth asking. Using two complementary strategies inducing a significant loss of the body weight mass, *i.e.* caloric restriction on high fat diet (Chevrot *et al.*, 2013) and bariatric surgery techniques adapted to the small laboratory rodents (Dastugue *et al.*, 2022), it was shown that DIO-induced decrease of fat taste sensitivity is a reversible phenomenon, at least partially.

Mechanisms controlling the orosensory perception of dietary lipids are complex and are still under active research. Obesity-associated endocrine disturbances, for instance the post-prandial reduction of Glucagon-Like Peptide-1 (GLP-1) release, can directly affect the orosensory detection of lipids. Indeed, taste bud cells express the GLP-1 receptor (GLP-1R) of which the gene inactivation increases the lipid detection threshold (= loss of sensitivity) in mice (Martin *et al.*, 2012). At the cellular level, it has been shown that the isolated taste bud cells from DIO mice responds weakly to the stimulation with linoleic acid (LA), using as lipid model, as compared to cells from lean controls. Indeed, the LA-mediated rise of intracellular ionized calcium levels observed in isolated taste cells from lean controls was found dramatically reduced in cells from DIO mice (Ozdener *et al.*, 2014). This blunted response was associated with a disturbance of neurotransmitter release by the taste bud cells and, thus, of the lipid signal transfer to the brain (Ozdener *et al.*, 2014). Diet-induced obesity is not only associated to the reduction of the functional performance of taste bud cells, but also leads to a decrease in the fungiform papillae (Fun) number lining the tongue dorsum from mice (Chan *et al.*, 2013; Kaufman *et al.*, 2018). This phenomenon appears to be elicited by the low-grade systemic

inflammation notably linked to the lipopolysaccharides (LPS)-dependent endotoxemia due to the obesity-mediated intestinal dysbiosis (Chan *et al.*, 2013; Kaufman *et al.*, 2018; Bernard *et al.*, 2019). However, this change in the systemic LPS load does not seem to be the only one responsible for the reduction of Fun number since the chronic intraperitoneal diffusion of LPS concentrations similar to what is found during obesity-mediated low-grade inflammation, does not reproduce the same phenotype in mice (Bernard *et al.*, 2019). Therefore, others mechanisms are likely involved in the fat taste desensitization observed in DIO-rodents. Consistent with this assumption, it was recently shown that metabolites from the tryptophan/kynurenine pathway might impact the orosensory perception of dietary lipids (Bernard *et al.*, 2021). Using a metabolomic approach, it has been highlighted a higher plasma quinolinic acid level in DIO mice than in lean controls. Moreover, plasma quinolinic acid level was found to be inversely correlated with the lingual fungiform number and the ability to detect dietary lipids. The fact that quinolinic acid becomes neurotoxic when it is produced in excess raises the possibility that the over-activation of the tryptophan/kynurenine pathway found in DIO mice contributes to the degradation of their fat taste perception. Indeed, the plasma quinolinic acid levels is under the control of the first limiting enzyme of the pathway, the IDO1 (Indoleamine-pyrrole 2,3-dioxygenase 1), known to be over-activated by a proinflammatory environment. Consistent with this hypothesis, the pharmacological inhibition of IDO1 using the antagonist 1-methyl-tryptophan (1MT) increased the Fun number and improved the orosensory capacities to detect dietary lipids in obese mice (Bernard *et al.*, 2021). It is plausible that the intestinal microbiota is an actor of the observed phenomena since it has already been demonstrated that the phenotype induced by an IDO1 inhibition with 1MT is transmissible between animals by means of gut microbiota transfer (Laurans *et al.*, 2018). Finally, it was recently reported that obesity can also disturb the gustatory papillae rhythmicity (Bernard *et al.*, 2020). Indeed, amplitude of clock gene oscillations is decreased in the circumvallate papillae from DIO mice. Interestingly, the down-regulation of the lipid sensing genes (CD36, FFAR4) occurring in the middle of the dark period in lean controls was not found in DIO mice suggesting that their taste sensitivity remains high despite HFD consumption. It is likely that the disruption of this regulatory loop might also play a role in the preferential consumption of energy-dense foods observed in DIO mice (Shin *et al.*, 2011).

In brief, compelling evidence supports that (i) lipid orosensory detection is disturbed in obese mice, (ii) this dysfunction is broadly reversible with a weight loss and (iii) is, at least partially, dependent from functional disruption affecting gustatory papillae.

4 Is the orosensory perception of lipids also disturbed in patients with obesity?

Unlike rodents, the existence of a correlation between the obesity state and the sensitivity of oral lipid detection remains debated in humans since some studies report an association between body mass index (BMI) and the lipid detection threshold (Stewart *et al.*, 2010, 2011; Martinez-Ruiz *et al.*,

2014; Newman *et al.*, 2016; Karmous *et al.*, 2018) while others find no recurrent correlation between these 2 parameters (Mattes, 2011; Stewart and Keast, 2012; Chevrot *et al.*, 2014; Costanzo *et al.*, 2017; Tucker *et al.*, 2017). This inter-species difference could have its origin not only in the subjects themselves (genetic heterogeneity and specific dietary habits), but also in the variability of methodological approaches used (Choi *et al.*, 2023). Indeed, when assessing the ability to detect dietary fats using lipid models (mainly oleic or linoleic acid), the results observed are inconsistent and vary, depending on the methods, the authors, and the populations. The lipid detection thresholds are generally established in humans by means of triangular tests (3-Alternative Force Choice test or 3-AFC) during which the subject must identify, among three solutions, the one containing the sapid molecule, the concentration of the latter being gradually increased until the moment when the subject becomes able to detect it without error several times in succession. To preferentially target the taste component, the experiments are generally (but not always) carried out under conditions that minimize olfactory (wearing a nose clip), trigeminal (texturing of the control solution and similar particle size between the different emulsions) and post-ingestive (fasted state and absence of ingestion of the solutions) influences. While this method is effective when the taste tested is easily identifiable (*e.g.* sweetness), it is more problematic when the oral sensation cannot be clearly verbalized, which is the case for lipids (Mattes, 2011). Moreover, the BMI only imperfectly reflects the state of adiposity of the subjects. Finally, these psychophysical investigations are generally performed with a limited number of subjects. In brief, the relationship between obesity and orosensory lipid detection acuity is no systematic in human, but appears to be limited to some subjects. Indeed, it was reported using the 3-AFC method that the rare subjects unable to detect the lipid samples, whatever the concentration used, were all obese (BMI > 30 kg/m²) and differed from other participants (lean or obese) by an overconsumption of fatty foods (Chevrot *et al.*, 2014). Moreover, the exploration of the fat taste sensitivity in patients with a morbid obesity before and after a bariatric surgery has revealed the existence of two subgroups: subjects with an improvement of orosensitivity to lipids after intervention and unresponsive ones despite similar body weight and fat mass loss (Bernard *et al.*, 2021). The origin of this dichotomy remains to be fully understood. However, recent studies have highlighted that these “unresponsive” patients were characterized by a higher systemic inflammatory status (Bernard *et al.*, 2021) and displayed a specific pro-inflammatory microbiota in the direct vicinity of gustatory papillae (Bernard *et al.*, 2022). Interestingly, inflammation is known to promote the degradation of tryptophan along the kynurenin pathway, a phenomenon associated with a reduction of the fungiform papillae (fun) density in mice (Bernard *et al.*, 2021). Such a scenario is also plausible in human. Indeed, (i) the plasma kynurenin levels of “unresponsive” patients did not decrease after surgery in contrast to what is found in patients with an improved lipid sensitivity (Bernard *et al.*, 2021), (ii) inflammation arising from obesity reduces the Fun abundance in human (Kaufman *et al.*, 2020) and (iii) Fun number is positively to higher fat related to high fat taste sensitivity in human (Zhou *et al.*, 2021). Further investigations are required to explore this hypothesis.

5 Conclusion

In rodents (rats and mice), diet-induced obesity is associated to a degradation of the fat taste sensitivity with a preferential consumption of high fat diet probably to compensate a hedonic deficit. This sensory dysfunction is inversely correlated to fat mass and is reversible, at least partially, following a weight loss. The great interest of this animal model, easily standardizable and genetically manipulable, is to help to identify the mechanisms linking obesity and fat taste deficit. Obesity-mediated homeostatic imbalance appears to be complex since it acts both at oral (taste detection) and central level (taste perception) and imply notably endocrine, metabolic and inflammatory factors (Besnard *et al.*, 2016). Further investigations are still required to fully decipher how this intricate regulatory pathway work.

In humans, the functional link between obesity, fat taste sensitivity and attraction for high lipid foods is not systematic, but is only found in some subjects and, this independently of the BMI. This observation raises a basic question: why does obesity differentially affect fat taste sensitivity in humans? The answer to this intriguing question could ultimately lead to personalized medical and nutritional care for subjects with obesity in order to avoid weight rebound following weight loss.

Conflicts of Interest

A.B. and P.B. discloses no conflicts.

Author contribution statement

Drafting the manuscript: A.B. ; Review the manuscript: A.B., P.B.

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