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Mini-review

Is the taste of fat regulated?

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ABSTRACT

Over the last decade, converging data have been accumulated both in rodents and humans, supporting the existence of a sixth taste modality devoted to the perception of dietary lipids. It is well known that the sense of taste is determinant for the food choice and that the overconsumption of highly palatable energy-dense foods contributes to the current obesity epidemic. Thus, an important issue in terms of Public Health is to understand the mechanisms by which the oro-sensory perception of fat is regulated. An overview of our current knowledge in this field of investigations is proposed in this mini-review.

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1. Introduction

Obesity reaches epidemic levels in the world. By reason of deleterious effects of obesity-associated diseases (i.e. type 2 diabetes, vascular disorders, hypertension, cancers), it is one of the major public health challenges of the 21st century. Origin of this epidemic is clearly multifactorial. Nevertheless, availability of highly palatable energy-dense foods rich in fat and sugar seems to play a significant role in this phenomenon by changing our eating habits. Although hedonic reward value of foods is closely linked to the sense of taste which constitutes a driving force leading to preferential consumption of high palatable foodstuffs, putative involvement of gustation in the obesity risk remains largely neglected. Four primary taste modalities (sweet, salty, bitter and sour) were initially described, to which *umami* was recently added. Over the last decade, compelling evidences were accumulated supporting the implication of a taste component in the oro-sensory detection of dietary lipids, in addition to textural and olfactory cues [1,2]. Interestingly, a correlation between sensitivity of the oral lipid detection system and attraction for fatty foods has been recently reported [3]. Therefore, it can be thought that the efficiency of the "taste of fat" might influence the food choice and, thereby, the energy balance. This assumption raises possibility that a dysfunction of this oral lipid sensing system might influence the eating behavior and, thereby, contribute to the obesity risk. The goal of this mini-review is to provide an overview on our current knowledge about the regulation of the system responsible for the oro-sensory detection of lipids by attempting to answer a basic question: Is the fat taste regulated?

2. The fat taste paradigm

Taste perception is mediated by taste receptor cells (TRC), which are clustered in taste buds, a specialized onion-shaped structure. Mammalian taste bud cells are classified into four subsets (type I, II, III and IV) in function of their structural and functional characteristics [4]. In the lingual epithelium, taste buds are located in three types of gustatory papillae (i.e. fungiform, foliate and circumvallate) and establish synaptic contacts with afferent fibers of chorda tympani and glossopharyngeal nerves (VIIth and IXth cranial nerves, respectively). These nerves transfer the gustatory signals to the brain and digestive tract *via* the nucleus of solitary tract (NST) in the brainstem leading to adaptations in eating behavior and digestive function (Fig. 1). The chemoreception of tastants takes place in the apical side of the TRC.

According to the recent proposition of Richard Mattes from Purdue University (USA) concerning the minimal elements required to constitute a primary taste quality (i.e. effective

Abbreviations: AEA, endocannabinoid anandamide; CCK, cholecystokinin; CVP, circumvallate papillae; DIO, diet-induced obesity; GLP-1, glucagon-like peptide-1; *Glp1-r*, GLP-1 receptor; GPCR, G protein-coupled receptors; LCFA, long-chain fatty acids; NPY, neuropeptide Y; NST, nucleus of solitary tract; PPY, pancreatic polypeptide; PYY, peptide YY; TRC, taste receptor cells; VIP, vasoactive intestinal peptide.

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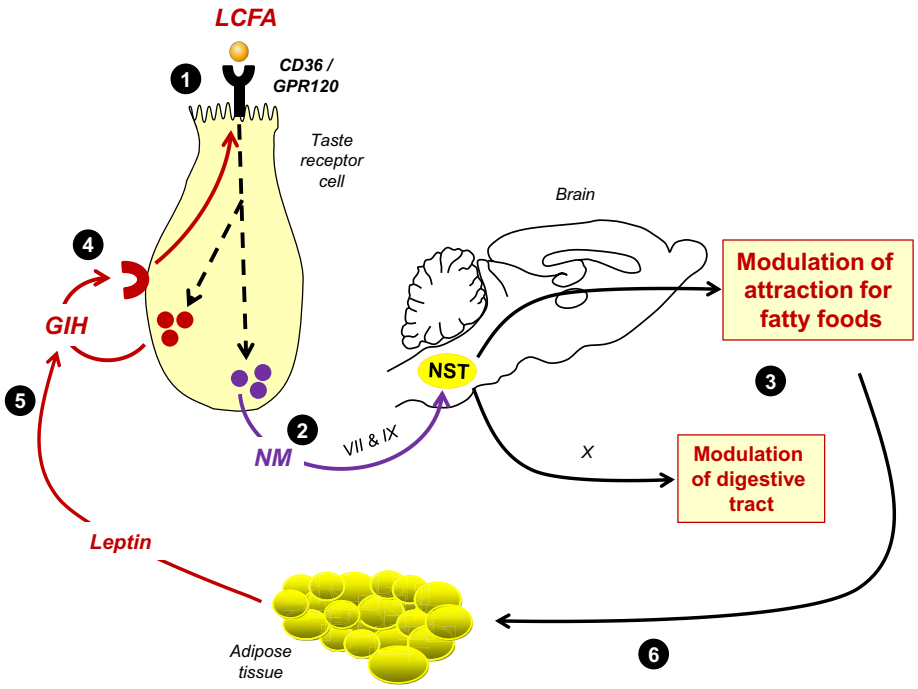

Fig. 1. Regulation of the taste of fat: working model. (1) Interaction between a long-chain fatty acid (LCFA) and lipid sensors (CD36 and/or GPR120) located at the apical side of the taste bud cell triggers a signaling cascade leading to the release of neuromediators (NM) and gastro-intestinal hormones (GIH). (2) The lipid signal is transmitted to the nucleus of solitary tract (NST) *via* the gustatory nerves (VII = chorda tympani, IX = glossopharyngeal nerve). (3) Integration of the lipid signal by the brain modulates the attraction for fatty foods. A transfer of the lipid signal towards the digestive tract (X = vagus nerve) also occurs *via* a reflex loop (tongue/NST/periphery axis) and generates digestive secretions preparing the body to lipid incoming. (4) Gastro-intestinal hormones (GIH) release of by the taste bud cells stimulated by LCFA can act locally in an autocrine and/or paracrine manner to regulate the function of lipid receptors (CD36/GPR120). (5) Regulation by peripheral mediators: example of the leptin. (6) Obesity, by increasing the plasma leptin levels, leads to a decrease in the sensitivity to fat taste. This event might lead to an overconsumption of lipid-rich foods to reach a hedonic fulfillment inducing a vicious circle promoting obesity.

stimulus, specific reception and signaling, involvement of gustatory pathway, physiological impacts, and identifiable sensation [5]), it appears that "Fat" might be eligible. Indeed, long-chain fatty acids (LCFA) have been shown to be the effective stimuli in both human and rodents [1,2,6,7]. It is noteworthy that LCFA can be efficiently released from triglycerides (TG) by the lingual lipase in these species [8,9]. Chemoreception of free LCFA is elicited by lipid-binding proteins specifically expressed in TRC, such as CD36 and GPR120. The glycoprotein CD36, which belongs to the scavenger receptors family, was the first to be identified in TRC in rodents [10,11] and more recently in humans [12]. In the mouse, the binding of LCFA to lingual CD36 triggers the activation of a specific transduction system leading to the secretion of neurotransmitters previously stored in TRC [13,14] (Fig. 1-1). This event produces a specific signal conveyed by peripheral and central nervous system involved in the gustatory pathway [15] (Fig. 1-2). From a physiological point of view, lingual CD36 was shown to contribute to the preference for fat in mice and human [9,10]. It also participates to the digestive anticipation preparing the body to the fat incoming. This phenomenon, which takes place through a reflex loop (tongue/brainstem/periphery axis) involving the NST, results in early secretion of digestive enzymes (e.g. lipases) and hormones (e.g. cholecystokinin (CKK), pancreatic polypeptide Y (PPY), peptide YY (PYY), insulin) [5,16] known to play a role in feeding behavior (Fig. 1-3). Another LCFA receptor, GPR120 has recently been identified in TRC in mice [17] and humans [18]. This member of the G protein-coupled receptors (GPCR) family also plays a significant role in the generation of fat preference since GPR120-null mice are unable to detect properly an oily source during behavioral test [17]. Nevertheless, the respective roles of CD36 and GPR120 in TRC remain to be elucidated. The implication of the free fatty acid receptor 1 (FFAR1, also termed GPR40) is more questionable. Albeit, it was reported to be involved in the spontaneous preference for fat in the mouse [17] GPR40 mRNA seem lacking in CVP in different species including rat [19], mouse [20] and human [18]. Origin of this discrepancy remains to be elucidated. Finally, albeit LCFA can be discriminated in the oral cavity when non-gustatory cues are eliminated in humans, sensation is non-easily identifiable, a clear lexicon being lacking [5]. "Fat" is similar to *umami*, in this regard. Despite of this limitation, a body of evidences is consistent with the implication of the sense of taste in the oro-sensory perception of lipids.

To these six minimal elements required to define a primary taste quality must be added a seventh: be flexible. Indeed, if this sensory property plays significant physiological functions, it must be tightly regulated.

3. Modulation of fat taste sensitivity

How lingual CD36 might be regulated? To answer this basic question, we postulated that CD36 expressed in taste buds might share similar regulatory pathways with intestinal CD36.

3.1. Lipid-mediated regulation

Consistent with this assumption, an adaptation of the oral lipid sensing system in function of the lipid content of diet was suspected. Indeed, it was previously shown a lipid-mediated down-regulation of CD36 in intestinal cells [21]. As expected, CD36 protein level in circumvallate papillae (CVP) displays a diurnal rhythm with a down-regulation during the dark period corresponding to the food intake. This phenomenon is strictly dependent on the

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