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Changes in eating behavior, taste and food preferences and the effects of gastrointestinal hormones

Alessandro Laviano [a, *], Luca Di Lazzaro [a], Angela Koverech [b]

[a] Department of Clinical Medicine, Sapienza University, Rome, Italy
[b] Department of Clinical and Molecular Medicine, Sapienza University, Rome, Italy

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SUMMARY

Eating behavior is a complex response to different internal and external factors and whose aim is to preserve the homeostasis of energy intake, the stability of body weight and ultimately health. Although under physiological conditions, energy intake is relatively stable over the long period, many stimuli (i.e., mechanical, metabolic, environmental, etc.) may acutely influence energy intake. To offset or minimize the effects of such stimuli on energy homeostasis, humans are equipped with neuronal complex mechanisms integrating peripheral and environmental signals. In particular, eating behavior is determined by homeostatic feeding and hedonic feeding. In the presence of changes in taste or smell, these mechanisms interact with peripheral effectors, including gastrointestinal peptides, to preserve energy intake and ultimately body weight. Aging is associated with a progressive inability of these systems to protect net food intake. Also, changes of eating behavior during disease appear to be related to the activation of a specific neuronal emergency circuit, which promotes anorexia. The persistence during evolution of the emergency pathway suggests that still unidentified component of anorexia and fasting metabolism could be exploited to enhance recovery of patients with acute and possibly chronic diseases.

* Corresponding author. Department of Clinical Medicine, Sapienza University of Rome, viale dell'Università 37, 00185 Rome, Italy. Fax +39 064440806.
E-mail address: alessandro.laviano@uniroma1.it (A. Laviano).

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1. Introduction

Food intake is a priority for humans since the ability to sustain long periods of starvation is limited and almost abolished during disease. However, measuring energy and protein intake describes only a fraction of the complexity of the decision process leading to eating and stop eating. Therefore, the term eating behavior more precisely characterizes the process of eating in humans, since it encompasses the complex mechanisms behind the decision to eat and to stop eating. Indeed, the number of meals and their sizes, as well the selection of different foods, are under the variable influence of many internal and external factors, including taste changes, gut motility, psychological distress, and pain among others. Consequently, prevention and treatment of primary and disease-related changes of eating behavior should be based on a thorough assessment of the different influencing factors [1].

2. Homeostatic control of eating behavior

Under physiological condition, the intake of energy, proteins and nutrients with the diet is stable over the long period, although significant variations may occur on a day-to-day basis. An example is provided by observing the food intake of female rats during their estrous cycle, whose food intake is relatively stable despite cyclically and reciprocally recurring changes in meal number and meal size (i.e., proxies for satiety and satiation, respectively), which are synchronized with the estrous cycle [2]. Therefore, under physiological conditions, net food intake is a dynamic process and the homeostasis of energy intake in response to external as well as internal stimuli is maintained via the modulation of satiety and satiation.

The homeostatic control of eating behavior is a complex mechanism mainly integrated in the brain, at the hypothalamic level. Robust evidence point to hypothalamic nuclei as the site of integration of metabolic, hedonic and mechanical signals arising from peripheral and central areas [3]. In particular, activation/inhibition of NPY neurons and/or melanocortin neurons increase/reduce the drive to eating. Of interest, these two sets of neurons are reciprocally innervated, and therefore the prophagic drive activated by the stimulation of NPY neurons is simultaneously reinforced by the concurrent inhibition of melanocortin neurons [4].

As previously mentioned, different factors contribute to modulate the concerted activity of hypothalamic nuclei involved in the homeostatic regulation of eating behavior. Among them, mechanical stimuli from gastrointestinal tract have been extensively studied. Experimental imaging studies revealed that distention of the stomach with an intragastric balloon resulted in significant reduction in food intake [5]. More importantly, gastric distention increased functional activity, as revealed by BOLD fMRI, within homeostatic regions such as the hypothalamus, as well as non homeostatic regions including the hippocampus, amygdala, thalamus, cerebellum and the cortex [5]. These results indicate that gastric distention increases neuronal activity in both homeostatic and non homeostatic brain circuits which regulate food intake. Similarly to mechanical signals, metabolic signals also influence homeostatic eating behavior. To preserve body weight, it is assumed that changes in metabolic rate modulate appetite and food intake. To address this issue, Mc Neil et al. studied the relationship between body composition and resting metabolic rate with acute (1 meal) and daily energy intake [6]. They observed that fat-free mass is the best predictor of acute energy intake whereas resting metabolic rate is a good predictor of daily energy intake in weight stable individuals [6]. However, greater error variance in acute and daily energy intake with increasing resting metabolic rate values was observed, suggesting that the influence of energy metabolism may not be linear across the spectrum of its possible changes.

3. Hedonic control of eating behavior

Food is a potent inducer of metabolic response but also triggers specific rewarding neural circuits. Consequently, the hedonic properties of food contribute to modeling individual eating behavior and may also override the precise balance of the homeostatic control of eating. In fact, many neural circuits that are thought to orchestrate feeding behavior overlap with the brain's reward circuitry both

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