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REVIEW ARTICLE

Multisensory influence on eating behavior: Hedonic consumption☆

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Abstract Research in obesity has traditionally focused on prevention strategies and treatments aimed at changing lifestyle habits. However, recent research suggests that eating behavior is a habit regulated not only by homeostatic mechanisms, but also by the hedonic pathway that controls appetite and satiety processes. Cognitive, emotional, social, economic, and cultural factors, as well as organoleptic properties of food, are basic aspects to consider in order to understand eating behavior and its impact on health. This review presents a multisensory integrative view of food at both the homeostatic and non-homeostatic levels. This information will be of scientific interest to determine behavior drivers leading to overeating and, thus, to propose effective measures, at both the individual and population levels, for the prevention of obesity and associated metabolic diseases.
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PALABRAS CLAVE
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Influencia multisensorial sobre la conducta alimentaria: ingesta hedónica

Resumen Las investigaciones sobre obesidad se centran fundamentalmente en buscar estrategias de prevención y tratamientos encaminados a los cambios de hábitos de estilos de vida. Sin embargo, con nuevas investigaciones, empieza a asumirse que el comportamiento alimentario es una conducta regulada no solo por mecanismos homeostáticos, sino que también es necesario valorar la vía hedónica que regula los procesos de apetito y saciedad. Los factores cognitivos, emocionales, sociales, económicos y culturales y las propiedades organolépticas de los

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alimentos son aspectos básicos a valorar para comprender la conducta alimentaria y su impacto sobre la salud. Esta revisión realiza una integración multisensorial en referencia a la percepción de los alimentos, tanto a nivel homeostático como no homeostático, y de esta manera poder interpretar científicamente las conductas que conducen a una sobrealimentación y a proponer medidas eficaces tanto a nivel individual como poblacional en la obesidad y enfermedades metabólicas asociadas.
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Introduction

Obesity is a multifactorial chronic disease characterized by an increase in body fat that poses a risk to the health of the individual.[1] Few chronic disorders have progressed in such an alarming way over the last few decades as obesity. According to the World Health Organization (WHO), the presence of obesity throughout the world has more than doubled since the year 1980. In 2014, over 1900 million adults were overweight, and of these more than 600 million were obese.[2] These worrisome data are reason enough for adopting measures and implementing prevention and treatment strategies targeted at both the general population and the population at risk, with a view to achieving effective weight loss and securing its subsequent maintenance.[3]

In this regard, research in recent years has made it possible to create and develop different methods, programs and treatments fundamentally focused on favoring changes in lifestyle (diet and physical activity).[3] However, it is increasingly apparent that other types of factors must also be taken into account in relation to this public health problem. Recent studies have demonstrated that apart from lifestyle changes and underlying homeostatic mechanisms, obesity is also markedly influenced by cognitive, social, emotional, economic and even religious factors (Fig. 1).[4,5]

Hunger, satiety and the energy balance are regulated by a neuroendocrine system located within the hypothalamus.[6] This system, based on a network of neurohormonal circuits, also includes molecular signals of peripheral and central origin (known as the *homeostatic system*), as well as other factors of a sensory, mechanical and cognitive nature.[7] This system is also referred to as the *hedonic system*, and is associated with activation of the neuronal reward system in response to any highly palatable food, i.e., any food which independently of its nutritional value produces a pleasurable sensation.[8]

The factors that regulate hedonic consumption include the senses, which detect flavors, smells, textures and even sounds, and play a decisive role in causing an individual to choose one food or another.[9] Considering that this sensory component is very important in terms of energy intake, and therefore relevant to the development of obesity and its associated diseases, the present article offers a review of the different associations detected to date regarding the senses and eating behavior.

Firstly, in order to place the subject in context, a brief description will be provided of the homeostatic component implicated in the regulation of eating behavior.

Homeostatic regulation of eating behavior

Knowledge of the homeostatic regulation of eating behavior has improved greatly in the last 20 years thanks to brain imaging techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). These techniques have afforded improved understanding of the way in which different brain regions respond to food and control the host homeostatic and hedonic responses.[10] The hypothalamus is the main brain center containing a complex network of neuronal mechanisms in charge of regulating hunger, satiety and energy balance.[6]

The hypothalamus is composed of different nuclei (Fig. 2) in charge of regulating a series of body functions such as, for example, food intake. These food consumption-regulating nuclei include the ventromedial nucleus, which when damaged results in increased appetite and obesity; the lateral hypothalamic area, which when damaged induces a decrease in food intake and weight loss; the paraventricular nucleus, which is in charge of receiving information regarding food intake from other brain nuclei; and lastly the arcuate nucleus, which integrates the main appetite-regulating peptide secretory neurons.[11] Recent studies indicate that this latter nucleus is essential for the regulation of appetite, and damage to the arcuate nucleus in mice has been shown to produce hyperphagia and obesity.[7]

All these nuclei are interconnected, and in turn receive information from the central nervous system (e.g., the vagus nerve), hormone stimuli (insulin, leptin, cholecystokinin and glucocorticoids), as well as signals from the digestive system such as ghrelin and peptide YY[14] (Table 1).

The different signals that regulate this neuroendocrine system can be of central origin (originating in the central nervous system) or peripheral origin (originating in peripheral tissues and organs). In terms of the duration of their action, these signals may be short-acting, i.e., activity is limited to during and just after a meal (e.g., cholecystokinin) or long-acting (e.g., leptin).

The anabolic (orexigenic) system is in charge of regulating the maintenance or increase in body weight through the stimulation of food intake; the hunger- and appetite-inducing mechanisms; and activation of the mechanisms that inhibit energy expenditure. By contrast, the catabolic (anorexigenic) system regulates the maintenance or lowering of body weight, stimulating mechanisms that increase energy expenditure and reduce food intake.[15] Both systems are integrated within the hypothalamus, though their activity is complemented with those of other brain centers, as

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