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Overweight obesity: updating perspectives in the area

Abstract

Overweight and obesity have reached epidemic proportions affecting people of all ages of all social groups in the Region of the Americas and the world, increasing mortality and morbidity. The classification of obesity unites anthropometric and clinical descriptors, approaching the concept of "chronic disease" with important clinical consequences, as a multifactorial disease due to genetic, environmental and lifestyle factors, important changes in diet, increased caloric intake, pattern of physical activity, sociocultural factors. The causes include the association of intestinal microbiota, energy extraction from food, fatty acid metabolism, synthesis of intestinal hormones, and regulation of body fat deposits, the alteration in GH secretion in obesity is parallel to alterations in body composition and increase in visceral fat, decrease in lean mass and bone mineral density, as well as genetic factors linked to this pathology. Obesity is linked to morbidities such as cardiovascular disease (CVD), type 2 diabetes, hypertension, certain cancers and sleep apnea/sleep disordered breathing. Health care utilization and medical costs associated with obesity and related diseases have increased dramatically and are expected to continue to rise. Standardized guidelines exist for the clinical-nutritional diagnosis and multifactorial treatment of obesity with emphasis on promoting healthy lifestyles that include balanced nutrition, increased physical activity, and decreased sedentary lifestyles.

Keywords: overweight, obesity, multifactorial disease, healthy lifestyles

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Introduction

Overweight and obesity are major public health problems, and this global epidemic has had farreaching consequences for individuals, society and the economy. Obesity is an established risk factor for numerous health conditions, including hypertension, high cholesterol, diabetes, cardiovascular disease, respiratory problems, musculoskeletal diseases and some forms of cancer. Mortality reports a progressive increase once the overweight threshold is crossed. Therefore, obesity and overweight shorten life expectancy, increase healthcare costs, decrease worker productivity and reduce the GDP of countries.[1] Unhealthy lifestyle habits, such as low physical activity and consumption of high-calorie, nutrient-poor foods and beverages, are considered risk factors for overweight and obesity. Although in recent years genetic factors have been identified that explain the greater susceptibility of some individuals to obesity, the abrupt increase in obesity that has occurred in recent decades and its widespread occurrence are mainly due to major changes in the population's diet, physical activity pattern and other sociocultural factors.[2]

Current figures

Obesity and overweight have reached epidemic proportions. Obesity rates have almost tripled since 1975 and have increased almost fivefold in children and adolescents, affecting people of all ages in all social groups in the Region of the Americas and worldwide. The Region of the Americas has the highest prevalence of all World Health Organization regions, with 62.5% of adults being overweight or obese (64.1% of men and 60.9% of women). Looking at obesity alone, it affects an estimated 28% of the adult population (26% of men and 31% of women).[3] These statistical approximations determined for the year 2016, highlighted by that time that 39% of men and women were overweight, and 11% of men and 15% of women were obese. (OECD, 2019) This means that almost 2 billion adults worldwide were overweight, and of these, more than 500 million were obese. Forty-one million children under the age of five were overweight or obese in 2016; while more than 340 million children and adolescents aged 5-19 were overweight or obese. Both overweight and obese status have shown a marked increase over the past four decades.[4] According to the OECD 2019 report, two Latin American countries, among them Mexico and Chile led the ranking with the highest percentage of adults with obesity or overweight, in first and second position, respectively. The podium is completed by another nation also from the American continent, the United States, with 73% of inhabitants affected by this health problem. Obesity remains one of the most urgent health problems and increasingly affects not only rich countries, but also those with lower incomes. In 2020, there were already more than 2.6 billion overweight people in the world and this figure could increase by more than 50% by 2035. A similar pattern is repeated in the case of the figures for obesity, which could double over the same period. According to the World Obesity Atlas 2023,[5] a study by the World Obesity Federation, by 2035 more than half of the world's population (51%) will have one of these two conditions, if measures such as taxes and limits on the promotion of unhealthy foods are not taken. According to this study, the problem is expected to increase from 38% of the world population in 2020 to more than 50% in 2035 (the figures exclude children under 5 years of age).

While preventing and treating obesity requires a financial investment, the cost of not preventing and treating obesity will be much higher. Estimates presented in this Atlas suggest that, on current trends, overweight and obesity will cost the global economy more than $4 trillion in potential revenue in 2035, nearly 3% of current global gross domestic product (GDP). Globally, overweight and obesity are linked to a higher number of deaths than underweight. Overall, more people are obese than underweight. This is true in all

regions except parts of sub Saharan Africa and Asia. A report by the Lancet Commission on The Global Syndemic on Obesity, Under-nutrition and Climate Change 2019.[6] It highlighted the coexistence of the epidemics of obesity, under-nutrition and climate change, noting that climate change increases food insecurity. While extreme food insecurity has been found to lead to under-nutrition, mild to moderate food insecurity is associated with obesity. With respect to climate change, high ambient temperatures may specifically affect people with higher BMI due to their lower ability to dissipate heat.[7] With the increased frequency of heat waves, the need for hospitalization for heat stroke and heat-related respiratory, cardiovascular, and renal treatment may increase more severely for these individuals. The economic impact is not easy to predict at present, but weather patterns show trends that imply that heat waves may be experienced in many regions of the world over the next decade.

In the wake of the current pandemic, there was a change in the lifestyle of the world's population. Millions of people opted for the home office as a way to continue with their work in a safe way. The drawback is that this modality encourages a sedentary lifestyle and ultimately impacts on health. In 2021, the consulting firm Ipsos conducted the international research Actions & Interventions for weight loss. The work included more than 22 thousand interviews with people from 30 different countries. It also has a section in which the weight of people before and after the health emergency was compared.[8] As a result, Mexicans are the ones who gained the most weight during the last year. The report shows an average increase of 8.5 kilos per adult. In second place is Saudi Arabia (8), Argentina (7.9), Peru (7.7) and Chile (7.5). Undoubtedly, the confinement and quarantine due to the COVID-19 pandemic were an important factor in the alteration of healthy eating habits. On the other hand, physical activity was reduced and the difficulty in accessing the health system also played a role. All these causes led to an increase in the prevalence of overweight and obesity. Data recently published by the Food and Agriculture Organization of the United Nations[9] (FAO) highlighted the seriousness of the nutritional situation on the planet. According to the agency, the amount of calories consumed per person worldwide increased by 9% over the last measurements, this finding is in line with a trend showing that people in all regions of the world have been eating more calories (kcal) since 2000, with the highest peak in Asian countries in 2021. According to this work, the regions of Europe and North America consumed the most calories per day this year: 3,540 calories per person. In the African continent, about 2,600 were consumed; while the Oceania count "was the closest to that of the United States and Europe, with about 3,150 kcal per capita". In Latin America and the Caribbean, this figure reached 3,035 calories per day, while in Asia the figure was 2,922 kcal.

Overweight and obesity: what are they and what are their differences?

Obesity is perhaps the oldest known metabolic and nutritional disorder in human history. This is evidenced by Egyptian mummies and Greek sculptures. In the Middle Ages. The artists of the time who considered it a tangible sign of well-being the obese body. Obesity and overweight occur gradually, i.e., weight gain usually occurs progressively, and may be largely due to excessive consumption of foods rich in sugars and fats, such as soft drinks (carbonated beverages), alcoholic beverages, snacks, flour, processed foods with excess fat, dressings and fried foods, among others. In addition, epidemiological-nutritional and traditional industrial food transitions explain important changes in food culture. Other factors that influence the problem are the adoption of unhealthy lifestyles and the accelerated processes of urbanization in recent years.[10] According to the World Health Organization, obesity is a chronic disease, characterized by an increase in body fat, associated with increased health risk. Excess weight in relation to height above what is expected, according to the reference population used, is considered overweight, while obesity consists of an abnormally high percentage of body fat, which can be general or localized.[11]

According to WHO, to differentiate between the two concepts, BMI provides the most useful measure, as it is the same for both sexes and for adults of all ages. The current classification of obesity proposed by the WHO is based on the Body Mass Index (BMI), which corresponds to the ratio between weight expressed in kilograms and the square of height, expressed in meters. However, it should be considered as an approximate value because it may not correspond to the same level of fat distribution in different people. The advantages of using BMI are based on the fact that there is a good population correlation (0.7-0.8) with body fat content, and because a positive correlation has been demonstrated with the relative risk of mortality (general and cardiovascular), independent of sex. It is this correlation that has determined the cut-off points for the diagnosis of obesity.[12] In some cases, such as bodybuilders, BMI is not considered a determinant of obesity, because muscle weighs more than fat and although they may have a high BMI, this does not indicate that they should lose weight. To determine whether a person is obese or simply has excess weight due to increased muscle mass, anthropometric techniques and reference standards are used to quantify body weight and fat and to establish the distribution of fat in the different body segments.

Overweight or pre-obesity

Overweight is a premorbid state of obesity and like obesity is characterized by an increase in body weight above normal weight by 10-20% and is accompanied by an accumulation of fat in the body, this is caused by an imbalance between the amount of calories consumed in the diet and the amount of energy (in the form of calories) expended during physical activities. Overweight can also occur due to excess muscle, bone or water. All those with a BMI between 25 and 29.9 kg/m2 are considered to belong to this category. They are considered to be in a static phase when they maintain a stable weight with a neutral energy balance. On the contrary, they are considered to be in a dynamic phase of weight gain when they are increasing their body weight as a result of a positive energy balance, i.e., caloric intake is higher than energy expenditure.[12]

Obesity

The World Health Organization (WHO) considers obesity to be an epidemic of a chronic non-communicable disease that begins at an early age with a multi-causal origin.[13] It is a disease of multifactorial etiology with a chronic course involving genetic, environmental and lifestyle aspects leading to a metabolic disorder.[14] It is characterized by a positive energy balance, which occurs when calorie intake exceeds energy expenditure causing an increase in body fat deposits and therefore weight gain. In adults, obesity is classified according to the Body Mass Index (BMI), due to the good correlation of this indicator with body fat and health risk at the population level. The strong association between abdominal obesity and cardiovascular disease has allowed the acceptance of indirect indicators of abdominal fat such as waist circumference measurement. In children between 2 and 19 years of age, it occurs when the BMI above the 95th percentile for age and sex,[15] or in the standard deviation tables, overweight is

the BMI for age with more than one standard deviation above the median established in the WHO child growth standards, and obesity is greater than two standard deviations. And in the case of children under 5 years of age: overweight more than two standard deviations above the median established in the WHO child growth standards; and obesity more than three standard deviations above the median.[16] The current classification of obesity proposed by the WHO, based on BMI, considers that those persons whose BMI is equal to or greater than 30 kg/m2 are obese.

Obesity has been defined by the WHO as the abnormal or excessive accumulation of fat that can be detrimental to health. The expansion of adipose tissue volume is secondary to a positive energy balance, maintained over time. Triglycerides synthesized as a consequence of excess energy are deposited in the cytoplasm of a cell specialized to store lipids: the adipocyte. The energy balance is the result of the difference between the energy entering the system and the energy used by the organism. Energy input is given by food intake. Energy expenditure is mainly determined by the maintenance of the basic functions of the organism, included in what is called basal metabolism. Energy is also consumed in thermogenesis induced by food and physical activity. In addition, in special situations, the expenditure determined by growth, pregnancy, lactation or disease must be considered. The distribution of this expenditure will depend primarily on the relative importance of the physical activity component.[17,18]

Types of overweight and obesity

According to body mass index (B.M.I.)

It is the most widely used classification of obesity worldwide. BMI has some limitations as an estimator of body fat. Thus, in individuals with a lot of muscle mass the weight is greater and they could be classified as obese when in fact they do not have an increase in fat; the same would occur in individuals of very short stature or patients with edema.

The WHO has proposed a classification of the degree of obesity using the Body Mass Index as a criterion:

-Normo weight: BMI 18.5 - 24.9 Kg/m^2 -Overweight : BMI 25 -29 Kg/m^2:

-Obesity grade I with BMI 30-34 Kg/m^2

-Obesity grade II with BMI 35-39.9

-Obesity grade III with BMI >= 40

In later dates the SEEDO (2007)[19] in addition to the previous classification adds the:

-Type IV (extreme) obesity with BMI >50

The SEEDO defines excess weight in 6 types according to BMI (kg/m2). As a continuous variable, the higher the BMI, the higher the risk of type 2 diabetes mellitus (DM), coronary heart disease, cerebrovascular disease and death of cardiovascular origin (Table 1).

According to SEEDO, BMI does not discriminate the distribution of body fat, does not differentiate between lean mass (LM) and BM, and may inappropriately categorize subjects as short, older, muscular, with fluid retention or pregnant.[20] The accuracy of BMI as an indicator of body fat also appears to be greater in individuals with higher levels of BMI and body fat. While a person with a very high BMI (e.g., 35 kg/m2) is very likely to have high body fat, a relatively high BMI may be the result of high body fat or high lean body mass (muscle and bone).[21]

Table 1 SEEDO criteria to define Obesity in degrees according to BMI in Adults

Category	BMI limit values (kg/m^2)
Under weight	<18,5
Grade I overweight	25,0 – 26,9
Type I obesity	30,0 – 34,9
Type II obesity	35,3 – 39,9
Type III obesity	40,0 – 49,9
Type IV obesity	≥ 50,0

According to the topographic distribution of fat, four phenotypes are recognized

Android or central or abdominal obesity (apple-shaped): excess fat is preferentially located on the face, thorax and abdomen. It is associated with an increased risk of dyslipidemia, diabetes, cardiovascular disease and mortality in general. Ginoid or peripheral obesity (pear-shaped): fat accumulates mainly in the hips and thighs. This type of distribution is mainly related to venous return problems in the lower extremities (varicose veins), biliary lithiasis and knee osteoarthritis (genoarthrosis).

Obesity of homogeneous distribution: it is one in which excess fat does not predominate in any area of the body. It is the predominant phenotype in pre-puberty.

Visceral or abdominal obesity, which may be associated with hyperinsulinemia, resistance (plurimetabolic syndrome, often linked to hypertension).

Following Hirsch's work, some authors classify obesity as hypertrophic and hyperplastic.[22]

Hypertrophic obesity: This is typical of adults and is characterized by a large amount of fat in the adipocytes without an increase in the number of fat cells. These individuals tend to be thin or maintain their average weight until 30 or 40 years of age, when weight gain begins. Weight gain may be associated with an imbalance between caloric intake and utilization. People with hypertrophic obesity tend to have a central fat distribution, this problem tends to be more easily treated.

Hyperplastic obesity: This corresponds to a long-lasting clinical form in which the number of adipocytes is greater, as well as the amount of fat they contain. These individuals tend to be obese since childhood and to have a significant weight gain during adolescence. After this age, the number of adipocytes is maintained throughout life. In this form of obesity the fat distribution is central and peripheral. Treatment is considerably more difficult.

Morbid obesity: The term morbid obesity qualifies people with more than 100% of their ideal weight. It is so called because it is often associated with dangerous and serious life-threatening conditions such as hypertension, diabetes mellitus and arteriosclerosis.

According to its etiology or origin

Exogenous, primary, idiopathic obesity: it is the most frequent type of obesity. In this case the relationship between energy expenditure and intake is altered, its etiopathogenesis may be multifactorial.

Endogenous or secondary obesity: it is associated with some endocrinological or neurological disease, either a hormonal alteration (cushing's syndrome, hypothyroidism, hypothalamic alterations) or obesity associated with genetic diseases (Prader Willi syndrome, Carpenter's syndrome, etc.). and secondary due to the intake of some drugs, which can trigger or aggravate obesity, such as antidepressants, steroids, insulin, oral contraceptives, etc. According to the American College of Endocrinology / American Association of Clinical Endocrinology:[23] The classification of obesity unites anthropometric and clinical descriptors, approaching the concept of "chronic disease with important clinical consequences".

Difference between overweight and obesity

According to WHO, to differentiate between the two concepts, BMI provides the most useful measure of overweight and obesity in the population, as it is the same for both sexes and for adults of all ages. However, it should be considered as an approximate value because it may not correspond to the same level of girth in different people. A BMI value of less than 18.5 is considered to be underweight or below the recommended and healthy weight, which can cause serious damage to health. Between 18.5 and 25 would be the BMIs considered as normal weight, with a healthy proportion between weight and height. From values higher than 25, we would be observing body masses above the healthy range.

One of the differences between obesity and overweight is that, in terms of BMI, between 25 and 30 the person in question would be considered to be overweight and from a BMI of 30 onwards we would be talking about a case of obesity. Another of the main differences between overweight and obesity, and in fact the most important one, is the risk that maintaining these levels of body fat poses to the health of the person who suffers from them. Obesity has been shown to be an important risk factor for the appearance of different pathologies. The most common and well known are heart disease and arteriosclerosis (with the consequent increased risk of vascular and cerebrovascular events such as stroke and stroke). Also arterial hypertension, bone problems, type 2 diabetes, respiratory problems such as bronchitis, liver and kidney problems, sleep apnea or low back pain, sexual dysfunction and even fetal malformations in the case of pregnant women. Likewise, surgical interventions and the effects of anesthesia are more dangerous, there are more sleep problems and a greater tendency to anxiety and depression (Table 2 & 3).

Table 2 The AACE advanced framework and levels of treatment and prevention for chronic diseases

Diagnosis	Anthropometric component	Clinical component	Prevention/Treatment
Normal Weight	BMI < 25 kg/m^2		Primary
Over weight	BMI ≥ 25 – 29.9 kg/m2	No obesity-related complications	Secondary
Obesity	BMI ≥ 30 kg/m^2	No obesity-related complications	
Obesity Stage I	BMI ≥ 25 kg/m^2	Presence of one or more mild-to-moderate obesity related complications	Tertiary
Obesity Stage 2	BMI ≥ 25 kg/m^2	Presence of one or more severe obesity related complications	

Table 3 Differences between Overweight and Obesity

	Overweight	Obesity
What is?	It is the condition in which a person weighs more than what is considered normal for their height, age and sex.	Obesity is a condition in which there is an excessive and generalized deposit and accumulation of body fat.
BMI (Body Mass Index)	A person is considered overweight when their BMI is between 25 and 29.9.	A person is considered Obese when their BMI is 30 or more.
Risks	Depression, Hypertension and Cardiovascular Diseases	In the same way, it is likely that the person will present Depression, Hypertension and Cardiovascular Diseases.
Causes	Overweight is related to the consumption of more calories than the individual's body needs, although other factors such as genetics, stress and others must also be considered.	Likewise, it can be due to the consumption of more calories than the individual's body needs, stress, genetics, depression, hormonal imbalances.
Figures	It is estimated that nearly one billion people around the world live overweight.	Of the billion people worldwide, approximately 300 million are obese.
Geographic Distribution	Its distribution is relatively the same throughout the world.	Obesity is not exclusive to one country either, but there are regions where there are more people with obesity, such as the United States, Mexico and England.
Child Population	The latest figures show that in the world there are currently around 25 million children under 5 years old who are overweight.	5% of the 22 million overweight children are Clinically Obese

Causes of overweight and obesity

In the vast majority of cases, obesity is caused by an increase in caloric intake, often associated with sedentary activity. Current knowledge on genetics and molecular biology allows us to consider the etiopathogenesis of obesity as a complex phenomenon, highlighting obesity as a very heterogeneous disorder in its origin, involving a variety of factors, both genetic and nutritional.[24–26] The body weight of a person is determined by the relationship of several factors, among which are: environmental, cultural, genetic, social and energy expenditure factors, where the level of physical activity of individuals plays a determining role. A bibliographic review was carried out in scientific documents and databases Web of Science (WOS), Pubmed, Scopus, refining the search results according to language, English and Spanish and date of publication, last 13 years.[27] Different factors favor the development of this pathology. In relation to economic factors, there is a higher prevalence among the lower social classes in developed countries as opposed to the higher social

classes in developing countries. It should be added to this situation that poor people buy and consume cheaper foods, usually fats, sugars and flours, as well as adopting consumption patterns of industrialized foods and beverages.[28] Social factors include working in lower-skilled jobs, being widowed or having children at an early age. It is important to highlight the obesogenic environment that can be favored at home, where parental figures can reinforce incorrect habits such as overeating and sedentary lifestyles. Among the environmental factors, the increase in age stands out, and in women the hormonal changes produced as a result of menopause favor both an increase in intake and a decrease in energy consumption.

However, one of the most important factors in the development of obesity is the individual's lifestyle. This will be favored in the presence of a diet defined by frequent consumption of energydense foods, consumption in excess of needs, habits related to portion size or the number of intakes throughout the day. For example, the absence or incomplete breakfast, at early ages, has been related to the presence of obesity. The scientific evidence of the FESNAD-SEEDO Consensus, 2010, highlights that energy-dense eating patterns can lead to weight gain in adults (level of evidence 1+). Regular fast food consumption (more than once a week) may contribute to increased energy intake and weight gain and obesity (level of evidence 1+). Frequent consumption of sugar-sweetened beverages is associated with higher BMIs (level of evidence 2+).[29] Other alterations in eating behavior such as fast, binge eating, binge eating or snacking between meals are also related to the presence of overweight and obesity. Perinatal characteristics related to the pregnant woman or the subject itself can also contribute to the development of obesity and subsequent metabolic alterations. Examples are a Body Mass Index (BMI) ≥25kg/m2 before or during the conception period, the presence of several births, Diabetes Mellitus (DM) or smoking during gestation, insufficient caloric intake in the first 2 trimesters of pregnancy, absence of breastfeeding, high birth weight, presence of excess adiposity prior to age 5 years or early menarche, prior to age 11 years.[30]

Other factors

Studies have shown that people who sleep little (less than 7 hours a day), reduce the hormone leptin (appetite suppressant) and increase the hormone ghrelin and therefore significantly increase appetite which results in increased intake, choosing less healthy foods, resulting in an increase in BMI, which would contribute to the development of obesity in the long term in situations of sleep restriction.[31] Other research highlights that eating dinner less than three hours before bedtime and skipping breakfast is significantly related to obesity.[32] Research in recent years has shown that there is a relationship between the intestinal microbiota and the development of different diseases, including diabetes and obesity. This association has been established by the identification of differences between the germs that colonize the intestine of obese people and people of normal weight. Researchers have attributed the association between the intestinal microbiota and obesity to the fact that alterations in the microbiota have an impact on energy extraction from food, fatty acid metabolism, the synthesis of intestinal hormones involved in energy balance, and the regulation of body deposits of adipose tissue.[33] It has been identified through different studies that in people with excess weight there are changes in the composition of their intestinal flora, with a considerable decrease in the amounts of bifidobacteria and lactobacilli, while other germs increase, mainly large negative bacteria that have lipopolysaccharides in their membranes as a major component that can stimulate inflammation. These germs are also attributed the property of making food energy more profitable, so they are able to obtain more energy from the same daily caloric intake.[34] Evidence suggests that the microbiota of overweight individuals has a greater capacity to degrade non-digestible carbohydrates from vegetables and therefore favors energy absorption and thus excess weight.[35] Obesity is accompanied by changes in the plasma levels of certain hormones and changes in their secretion and/or clearance patterns. Some of these alterations are secondary to obesity while others could play a role in its pathogenesis.

The onset of obesity may also be due to a disease of endocrine origin. The main glands of the endocrine system are the hypothalamus, pituitary gland, thyroid gland, parathyroid glands, pancreas, adrenal glands and gonads (testes in men and ovaries in women). In obesity, the clearest alteration in the hypothalamic-pituitary system is related to growth hormone (GH). It is known that the alteration in GH secretion in obesity is parallel to alterations in body composition such as increased visceral fat, decreased lean mass and bone mineral density. Finally, genetic factors, where a single gene or several genes may be involved, should be mentioned. Among the genes related to obesity, it is necessary to highlight the FTO gene and MC4R. The rs9939609 polymorphism of the FTO gene is related to a higher BMI, waist circumference or levels of insulin, triglycerides and adiponectin. A relationship has also been found between variations in this gene and eating behavior habits such as frequency of consumption, intake, hunger or satiety.[36,37] Similarly, variations in the MC4R gene are related to eating behavior that favors the development of this disease as well as higher values of fat mass or BMI.[38,39] Other factors that have been related to diversity are alterations in basal energy expenditure and total energy expenditure, some hormonal alterations, inflammation, oxidative stress and even some infections.

Physiopathology of obesity

Scientific evidence shows the relationship between obesity and other factors such as subclinical inflammation, neuro hormonal activation with increased sympathetic tone, elevated insulin and leptin levels, increased free fatty acid exchange and the localization of fat in certain parts of the body such as the intra-abdominal or subepicardial level.[40] Adipose tissue is a specialized connective tissue consisting of lipid-rich cells called adipocytes. As it comprises about 20-25% of the total body weight in healthy individuals, the main function of adipose tissue is to store energy in the form of lipids (fat).Adipose tissue is the storage organ for energy in the form of fat. In obese individuals, adipocytes are larger in size and number. Under these conditions, fatty acids that cannot be deposited in adipose tissue tend to accumulate ectopically in other organs, producing lipotoxicity. Thus, reactive forms of fatty acids accumulate in muscle, liver, heart, pancreatic beta cells producing insulin resistance, fatty liver, cardiotoxicity and decreased insulin secretion respectively. Adiposopathy is described as the anatomical and functional alteration of adipose cells and tissue promoted by positive caloric balance in genetically and environmentally susceptible individuals.[41] There are two types of adipose tissue (AT) in humans, white and brown. Brown adipose tissue

is specialized for thermogenesis, an adaptation to the cold climate of homeothermic organisms. Brown adipocytes are characterized by multiple triglyceride droplets, which are accessible for rapid hydrolysis and oxidation of their fatty acids. Brown fatty tissue is present in infant humans mainly.[42] White adipose tissue (WAT) constitutes the majority of adipose tissue and is located throughout the body and is generally subdivided into visceral and subcutaneous, with visceral being positively related to the risk of developing insulin resistance.[43,44] The TAB is a metabolically dynamic organ, being the main site of energy storage in the form of triglycerides, although this tissue is now also known as an endocrine organ that releases multiple molecules, known as adipokines, which have pro- and anti-inflammatory activities and also participate in the regulation of energy metabolism and influence feeding behaviors.[45,46] TAB is not uniform throughout the body and fat depots vary in composition, microvasculature, innervation, metabolic characteristics, extracellular matrix composition and in their ability to secrete adipokines. Generally the expression of proinflammatory cytokines (IL-6, IL.8, MCP-1, PAI-1) is higher in visceral AT and that of anti-inflammatory cytokines (leptin and IP-10) is higher in subcutaneous AT.[47] The TAB can be divided into two fractions, the mature adipocyte fraction and the stromal tissue fraction, composed of many cell types such as preadipocytes, immune cells, including macrophages and lymphocytes, endothelial cells and fibroblasts.

The most common metabolic disorder of adipose tissue is obesity. This energy imbalance between higher energy intake and lower long-term energy expenditure leads the adipocyte to present a hyperplasia and hypertrophy response with a consequent increase in adipose tissue. In addition, adipocytes in the obese state present an alteration in their function, particularly their endocrine function.[48,49] The adipocyte can develop by two processes: by hypertrophy (increasing its size) and by hyperplasia (increasing its number from a precursor cell that goes through a series of steps until it differentiates to its final stage, from preadipocyte to mature adipocyte). Traditionally it has been considered that at a certain moment in the growth of an adipocyte, as its fat volume increases (hypertrophy), it will reach a critical size threshold at which a process of hyperplasia will occur, stimulating a precursor cell and thus generating a new adipose cell. Thus, hypertrophy in large adipocytes has been associated with increased release of inflammatory factors or altered insulin sensitivity in both animal and human models.[50,51] During normal caloric balance adipocytes undergo an initial hypertrophy that induces cell signaling for recruitment, proliferation and differentiation of new fat cells. However, if adipogenesis is impaired, it leads to a lack of adipocytes ready for proper proliferation resulting in lipodystrophy. Thus, a positive caloric balance generates new fat cells due to inadequate adipogenesis, which leads to excessive hypertrophy causing a dysfunctional and pathogenic adipocyte, as demonstrated by increased markers of intracellular endoplasmic reticulum stress and mitochondrial dysfunction.[41]

In childhood and adolescence the dominant developmental process is hyperplasia at certain stages, due to the fact that adipogenesis is easier. On the contrary, in adulthood this situation is more difficult, being able to reach a greater size in the adipocyte without stimulating hyperplasia, being the development by hypertrophy the normative mechanism of development in the subcutaneous adipose tissue in weight gain. However, this does not mean that in the face of chronic overeating a child cannot develop by adipocyte hypertrophy and generate the disturbances typical of adults. In fact, in adulthood the number of adipocytes remains practically stable with respect to the total reached during adolescence, and that is why prevention is so important in childhood and adolescence, since a significant weight loss decreases the volume and not the number of adipocytes.[52,53] Initially, in the development of hypertrophy there is a transitory state of inflammation that is considered necessary. The problem arises when this situation is perpetuated, since it would compromise the integrity of the adipocyte, hypertrophied in excess, modifying both its metabolic behavior and generating adaptations in the tissue, and even, ultimately, leading it to apoptosis. At this point there would be an infiltration of immune cells with a pro-inflammatory profile, altering the cellular microenvironment, and generating a state of tissue inflammation known as lipoinflammation.[54,55] This phenomenon discharges inflammatory factors into the circulation that can travel to other tissues, generating in turn alterations in these tissues and giving rise to a low-grade systemic inflammatory condition.[56] Together with the alteration of angiogenesis, there will be a situation of hypoxia and alteration of the extracellular matrix (fibrosis), further aggravating the inflammatory situation of the same.[57,58]

The larger size of the adipocyte, together with a concomitant inflammatory state, conditions its functioning: (a) altering its secretory profile with higher leptin and lower adiponectin production (which inhibits its expression by inflammatory factors such as TNFα), (b) causing lower insulin sensitivity, (c) leading to worse mitochondrial function and higher endoplasmic reticulum stress, (d) producing higher basal lipolysis, (e) altering the cell cytoskeleton, and (f) causing lower de novo lipogenesis.[59] This increase in basal lipolysis is known as the "overflow hypothesis", i.e., the adipocyte has saturated its capacity to deposit triglycerides and these are directed to other tissues and deposited ectopically in them, thus generating lipotoxicity and insulin resistance.[42,60] The increased flow of free fatty acids, together with inflammatory factors, converts a situation of insulin resistance and local inflammation into a state of systemic insulin resistance and chronic low-grade inflammation.[61,62] Due to its limited hyperplastic capacity, development by hypertrophy and inflammatory generation, and to its greater response to catecholamines and lesser inhibitory response of insulin to lipolysis, visceral adipose tissue becomes the first store of triglycerides due to the incompetence of subcutaneous adipose tissue to store excess energy. Its anatomical proximity to the liver, more due to the flow of inflammatory factors when it is hypertrophied than due to excess fatty acids (portal theory), conditions the health of this organ, which in turn conditions the systemic health of the individual.[63] Therefore, increased central fat deposition is considered a risk factor in itself when stratifying a higher incidence of metabolic syndrome, type II diabetes mellitus or cardiovascular disease.[64,65] Therefore, the capacity for proper adipose tissue expansion, hyperplasia versus hypertrophy, is what largely determines the existence of metabolically healthy obese subjects and metabolically diseased lean subjects. Although currently, the metabolically healthy obese phenotype is considered as a transitional state to disease (Table 4).[66]

Table 4 Different types of adipose tissues and their characteristics

Adipose tissue and origin	Location	Features	Synthesis	Functions
White adipocyte (ab). Mesodermal origin	Distributed throughout the body	Spherical shape, single and voluminous fatty vacuole that constitutes more than 90% of its volume (unicular).[9]	-Most important producer of leptin that promotes the production of proinflammatory factors such as resistin, tumor necrosis factor alpha (fntα) and interleukin 6 (il6).[10,11] -Acylation stimulating protein (asp) that promotes triglyceride storage by inhibiting lipolysis -Adipsin or factor d, complement-related protein that increases its expression in obesity. -Visfatin increased in patients with MS, a marker of atherogenesis.[12] -Omentin, increases insulin sensitivity, inhibits fntα production and has anti-inflammatory and cardioprotective effects.[13]	Main effector of lipogenesis and lipolysis processes.[7] Through leptin they intervene in the regulation of all hormonal axes, from regulation of appetite to regulation of the immune response. Important source of adult stem cells.
Brown adipocyte (ap). Ab-independent mesodermal origin	It is located between the scapulae, in the armpits, on the back of the neck and around the great vessels of the trunk.[5]	It has several small lipid vacuoles (multilocular), a greater number of mitochondria and glycogen granules.	-Uncoupled protein 1 (ucp-1) proteins are more abundant in fetuses and newborns.[13] -Deiodinase type II (d2) of tetraiodothyronine (t4) regulates the basal metabolic rate.[14,15] -Peroxisome proliferator-activated receptor activating protein 1α (pgc1-α) -Adiponectin, a hormone that increases insulin sensitivity in muscles, liver and other organs, in addition to promoting the oxidation of fatty acids. (3,18) -Fibroblast growth factor (fgf) types 16,19 and 21. Fdf21 activates thermogenesis and lipolysis directly and increases diponectin production.[18]	The main function is thermogenesis during the first year of life, it generates about 300 watts per kilogram (w/kg), in adults it decreases with age and BMI increases, dm or sm.[16,17]
Beige adipocyte (abg). Mesodermal origin by transdifferentiation of abs/ cell-derived myogenic factor 5 (Myf-5-).[19]	They develop in the WAT, mainly in the inguinal region.[9]	Multiple lipid vacuoles in smaller quantities than aps (paucilocular).	-Able to express UCP-1 - They produce FGF21, a factor that optimizes oxygen consumption and promotes thermogenesis. - Chimerin, induced by cold in mice and promotes thermogenesis.[20]	Thermogenic and adiponectin producers.
Pink adipocyte (ar). Transdifferentiation of abs.[21]	Exclusive existence in lactating mammary glands of female mammals.[22]	It is a milk-secreting cell, pink color in these organs with common stains.	-producers of leptin, a hormone that promotes the proliferation of the mammary epithelium, and prevents obesity in offspring.[23] - s100b protein, which promotes the maturation of the central nervous system in the neonate, vasodilator and has pro-inflammatory effects. - perilipin b, phosphoprotein that regulates lipolysis in fat vacuoles. Important role in the hydrolysis of dairy lipids.[18,23]	They can store large amounts of fat, producing adipokines. Considered a fatty reservoir that serves as a substrate for milk production.
Hepatic stellate cell (hepatic stellate cell). Probable mesodermal origin.[21]	Located in the space of disse, underlying the endothelial cells.[2,24]	First phenotype: multiple vesicles filled with vitamin A and small organelles 5-8% of all liver cells. Elongated cytoplasm and processes that interact with endothelium, nerve cells and hepatocytes. Second phenotype: highly fibrogenic cell.[25]	-release extracellular mediators - adiponectin expressed by the ceh in the quiescent state. - chemerin, which increases in obese patients.[26]	Those of the first phenotype have well-defined functions in the regulation of the homeostasis of the extracellular matrix.[25,27] The second phenotype in obesity, a state that presents with yperleptinemia, hyperresistinemia and adiponectinemia, can produce steatosis, and subsequently liver fibrosis, as a consequence of the altered regulation exerted by these adipokines on the cehs.[16,27]

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Physiology of appetite and satiety

Eating is a cross-cutting process in the life of the human being, which is mediated by physiological and environmental components. Hunger, appetite, satiety and energy balance are regulated by a redundant neuroendocrine system that is integrated at the level of the hypothalamus. This system consists of a dense and complex network of neurohormonal circuits where molecular signals of both peripheral and central origin, both short and long lasting, intersect with other sensory, mechanical and cognitive factors.[67,68] From the time the food is consciously perceived by the subject until it is ingested, a whole series of sensitive signals are involved (including smell and taste, texture, temperature and even the appearance and presentation of the food); All these signals are transmitted through the cranial nerves to the central nervous system, and cause the initiation of the feeding act by mobilizing the subject to grasp the food, placing it in the oral cavity, tasting it, and finally, chewing and swallowing.[69] The physiological need to ingest food is called hunger, while appetite is the psychological desire to eat as it is associated with sensory experiences, and the stopping of eating when the digestive system stops is considered as satiety.[70]

The sensation of hunger is related to the urgent need to ingest food, also influenced by physiological signals such as hypoglycemia, gastric contractions and increased bowel sounds.[71] Appetite, on the other hand, corresponds to the psychological desire to eat specific foods, more associated with sensory and emotional experiences, and without association with the previous physiological hunger signals. If food consumption has an effect, there is a signal of satisfaction or fullness, known as satiation, experienced during or immediately after food intake, which invites to stop eating; and satiety, which refers to a longer period without hunger or without the need to require food until the next hunger signal.[72,73] Food intake is the beginning of a complex process that seems to occur from brain signals in response to external stimuli, the process of ingestion or feeding is initiated by an incentive to reach the food goal, through the following phases:[74]

1. Onset phase: Causes changes in the cerebral cortex allowing the selection of an optimal motor program for the nutrient procurement phase.
2. Consumption phase: action of saliva secretion, hydrochloric acid, insulin and other regulatory responses.
3. Term phase: Action of the gastric filling and satiety signals.

Dietary factors

Energy density

Energy density (ED) refers to the amount of energy that is contributed by a given amount of food (kcal/g or kj/g). The ED of a food depends on its water content (adding weight without calories), fiber content (adding volume with limited calories) and macronutrient composition, mainly fat, due to the high energy content per gram. For example, at the beginning of a meal, a food with low ED can be effective in reducing energy intake, since - mainly due to its volume - it exerts a satiating effect.[75]

Diet composition

Although there is a genetic component to obesity, the fact that the incidence of this disease has increased dramatically in the last 25 years reflects the involvement of environmental and behavioral factors. The macronutrients present in the diet (e.g., proteins, fats, carbohydrates, alcohol) are responsible for providing energy to the body, so the type of macronutrients in the diet conditions the energy intake. Specifically, proteins stand out as the most satiating macronutrien[76] High protein intake seems to play an important role in the regulation of body weight, through the following mechanisms: increased satiety related to increased thermogenesis, body composition and decreased energy efficiency. On the other hand, carbohydrates tend to exert a more rapid satiating effect than fats, participating in short-term appetite regulation. On the other hand, although the body seems to generate physiological responses that are activated by the ingestion of fats, scientific evidence has shown that people who consume foods high in fats tend to increase their energy intake and body weight. In addition, since fat-rich foods tend to be highly palatable and contain a high ED, a large amount of energy can be consumed before satiety is induced.

Fiber can be useful in the treatment of obesity, since it generates a lower energy intake. In addition, it can promote satiety by increasing the volume of food and decreasing its energy density, which increases gastric volume and delays its emptying. Therefore, increasing fiber intake has an immediate impact on body weight control.[77] Specifically, soluble fiber, which provides viscosity to food, is the one that most efficiently increases the satiating effect. This type of fiber forms a viscous gel matrix in the intestine that increases gastric volume, leading to a greater feeling of satiety, in addition to the fact that it can absorb glucose in the small intestine and lead to lower postprandial glycemic and insulinemic responses.[78] In contrast, although insoluble fiber has limited effects on gastric emptying and absorption of other nutrients in the small intestine, it can be partially fermented in the large intestine.

Feeding patterns

A dietary pattern includes a description of the general diet, including food groups and nutrients, their combination and variety, as well as the frequency and quantity with which they are usually consumed. Although it is a fact that individual consumption patterns influence an individual's state of health or disease, the habitual omission of breakfast has been found to be associated with a higher body mass index in adults and, on the contrary, the consumption of an adequate breakfast has been found to be associated with both weight loss and weight maintenance. In the same sense, it has been documented that eating a single meal does not promote the maintenance of a healthy weight.[79]

Portion size

For some decades now, there have been warnings about the increase in the portion size of commercially available food and beverages, which has been suggested to promote an increase in energy intake among the population. In this regard, it has been shown that there is a clear relationship between the amount of food served and the amount consumed, even when participants serve themselves.[80] According to data and observations, over the last 30 years plate sizes increased by 36%; probably the increase in the average size of plates used to serve food translates into larger portions and higher caloric intake, which is associated with overweight and obesity.[81]

Sensory cues such as color, texture, odor and food packaging

Sensory experience, linked to aspects such as color, texture and smell, as well as emotional and memory associations with foods,

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meals or preparations also determine the selection and subsequent intake of foods.[82] The sensory characteristics of foods can determine preferences, portion size and satiety, and even facilitate dietary learning. For example, the visualization of a food in close proximity to the individual is the first cue or invitation for consumption, and the appearance of that food or meal can maximize consumption. Likewise, it has been identified that aspects such as environmental temperature, light, sound or the performance of alternative activities such as watching television or working on the computer can influence food intake and responsiveness or satiety perception.[83]

Psychological factors

Food restriction

Dietary restriction refers to the reduction in food consumption in order to control body weight, it can involve from omitting sometime of food, to the null consumption of energy-dense foods, which implies that the person will have to develop a strategy to control caloric intake.[84] Food restriction may be a common behavior among people with obesity, however it is also considered a risk behavior for the development of eating disorders, since it is associated with the loss of control over eating that can result in binge eating,[85] characterized by the compulsive intake of an excessive amount of food in a short period of time.

Emotional state

Emotional state can influence eating behavior, but eating behavior can also be affected by emotional state. In addition, although it has been identified that in people with negative emotional states food consumption increases,[86] in others it decreases.[87] Food cravings, particularly for high-fat foods, are a factor that has been found to strongly influence appetite control and, therefore, body weight.

Stress

Stress is a reality of modern daily life, and the interaction between stress and food is complex. Animal studies have linked acute stress to increased food intake, however, in humans food intake may increase or decrease during or after the period of stress, depending on their food profile, personality and phenotype.[88] When experiencing acute stress, as a threat to personal safety, there is an instantaneous physiological response that reduces intake in the short term. On the other hand, chronic stress elicits a more passive response, driven by the hypothalamic-pituitaryadrenal axis, with increases in cortisol that can induce individuals to consume hedonic, energydense foods and, potentially, unwanted weight gain and obesity. A relationship between stress and increased between-meal consumption of high-fat and high-sugar foods has been reported (Wallis and Hetherington, 2009). In addition, it has been documented that the relationship between food intake and stress may vary according to the sex and physiological state of the individual.[89]

Dream

Several explanations have been put forward to describe the link between sleep and increased food intake, and consequently obesity:[25] Additional time awake, which increases opportunities for eating; Increased ghrelin, with its subsequent stimulation of the appetite center and reduction of satiety-promoting hormones; Altered thermoregulation; and Increased fatigue, resulting in lower levels of physical activity.[90] It has also been suggested that alterations in sleep and meal timing, along with sleep duration, could influence hormone levels related to satiety and food intake,[91] as well as lead to alterations in glycemic control and insulin resistance, ultimately leading to increased hunger and subsequent food intake.

Hedonic role of foods or meals

The term hedonic appetite refers to the concern and desire to consume food for pleasure and in the absence of physical hunger.[92] Thus, eating behavior is not only regulated by homeostatic mechanisms, but also by other emotional, sensory, mechanical and cognitive factors that have been identified and, together, have been called the hedonic system. This system is activated by reward mechanisms and experiences when faced with foods that, regardless of their nutritional value, offer the consumer pleasure and gratification; these foods are usually dense in calories and rich in sugar, salt and fat. The impulse to consume these foods can even override physiological signals, favoring an imbalance in energy balance. Therefore, food reward would be the major driver for food intake, beyond the sensation of appetite or satiety. The palatability of hedonic foods is influenced by organoleptic characteristics such as odor, texture, color, or even the sound emitted.

Neurophysiological basis of ingestion behavior

What controls dietary intake?

The regulation of food intake is mediated by a complex mechanism involving the hypothalamus, which is responsible for regulating hunger and satiety signals. These signals allow energy homeostasis to be achieved through its structures, such as the lateral nucleus (responsible for the sensation of hunger) and the ventromedial nucleus (responsible for the management and control of the sensation of satiety during the process of food intake).[71] The paraventricular, dorsomedial and arcuate nuclei of the hypothalamus also contribute to the regulation of food intake and influence the production of thyroid, adrenal, insulin and glucagon hormones, as well as ghrelin and leptin. In this way, the regulation of intake and energy balance in the short, medium and long term takes place.[93] From the moment we perceive food until the onset of its ingestion, a whole series of sensitive signals will intervene (food odor, taste, texture, temperature and even the appearance or presentation of the food), signals that will be transmitted through the cranial nerves to the central nervous system (CNS). On the other hand, the onset of insulin secretion also mediated by these sensitive signals seems to be another factor that will trigger nutrient intake. This whole set of signals inducing food intake is known as the cephalic phase of feeding.

At the gastrointestinal level, several peptide molecules have been identified that are involved in the induction of satiety and whose synthesis and secretion will be in proportion to the amount of food ingested. Their mechanisms of action will be variable, and they can act locally or be released into the bloodstream, in which case their level of action becomes endocrine. The information provided by these peptides reaches our brain, specifically the nucleus of the solitary tract (an area located in the brainstem) through afferent vagal fibers from peripheral nerves. From this region, the information will in turn be transmitted to the hypothalamus.[71] The hypothalamus receives signals from the gastrointestinal system through the vagus nerve, which inform about aspects such as gastric fullness. Likewise, chemical signals are sent about the presence of nutrients in the blood (glucose,

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amino acids, fatty acids) that can determine the satiety signal. As for the hunger stimuli or orexigenic stimuli at the peripheral level, in the first instance we recognize ghrelin, a neuropeptide produced in the stomach, in the cells of the gastric fundus; and motilin, a hormone synthesized by the duodenum. Ghrelin has the ability to cross the blood-brain barrier and acts on the arcuate nucleus, where it activates receptors related to feeding behavior such as NPY and Agouti-related peptide (AgRP), which increase food intake and reduce energy metabolism, as well as melanin-concentrating hormone (MCH), produced in the lateral hypothalamus.[94]

Ghrelin, also known as the hunger hormone, stimulates the search for and intake of food in both healthy and obese individuals. Although this is the best known role of ghrelin, it also has functions on the sleep-wake cycle, regulates glucose metabolism, is involved in intestinal motility by accelerating gastric emptying, induces adiposity and is related to energy metabolism.[92] Ghrelin, an octanoylated peptide of 28 amino acids, is secreted into the bloodstream by endocrine cells lining the bottom of the stomach. Ghrelin secretion is stimulated by fasting, increases during the preprandial period, and is suppressed by food intake. Ghrelin is orexigenic; it increases food intake when administered peripherally, and acts in part by directly modulating the activity of agoutirelated neuropeptide Y neurons in the arcuate nucleus of the hypothalamus; for these reasons, it is of great interest, along with leptin, in the study of body weight regulation and obesity.[93] NPY is synthesized directly in the hypothalamus at the level of the arcuate nucleus; its effect is directly at the central level, where it stimulates and potentiates food intake and, in many cases, weight gain. Ghrelin, NPY and AgRP are inhibited by satiety signals derived from leptin, insulin and glucose. Leptin plays the opposite role to ghrelin. This hormone, released by adipose tissue, acts at the hypothalamic level, reducing food intake and increasing energy expenditure.[94]

In normal situations, leptin is released into the bloodstream and signals to the brain the state of the body's energy reserves, which is why leptin is of great interest, as it has an enormous influence on the regulation of body weight. The main afferent signal that allows the brain to detect the magnitude of energy reserves is the hormone leptin. Discovered in 1994, this 167 amino acid cytosine-like protein is released by the adipocyte. Under basal circumstances the circulating serum leptin concentration correlates with fat mass and decreases after weight loss. The decreasing leptin concentration informs the brain of decreased fat storage resulting from a negative energy balance. This results in compensatory effects on appetite and energy expenditure aimed at replenishing stores and restoring energy balance.

Conclusions

A perceived decrease in leptin concentration increases the amount of food consumed and minimizes energy expenditure. Leptin concentration does not change with meals, and leptin does not acutely change meal size. It is important to clarify that leptin not only plays a role in energy regulation, but also that its production varies with food intake, sex, age, exercise and circulating glucose.[95] There are pathophysiological mechanisms in which there is a reduction in tissue sensitivity to leptin, called leptin resistance; this is the case of obese individuals in whom, despite the increase in leptin concentrations, the anorexigenic effect of leptin is not as expected.[96,97] In normal conditions, insulin, released by the pancreas, acts at the level of the arcuate nucleus and paraventricular body in the hypothalamus: it decreases intake and increases energy expenditure. Thus, insulin shares some similarities with leptin. High insulin levels are characteristic of obesity. In addition, insulin can increase food intake, probably by decreasing glucose concentrations, Insulin decreases rapidly after caloric restriction and before significant changes in the amount of body fat occur. As related above, the gut and adipose tissue play a crucial role in signaling the control of hunger and satiety, either by eliciting hunger signals that invite the individual to ingest food or, conversely, by reducing food intake.[20] Neurotransmitters, noradrenaline, serotonin, histamine, gamma-aminobutyric acid and various peptides may be involved in the transmission of information that regulates food intake and nutrient reserves. Serotonin plays an important role in the regulation of food intake and nutrient reserves. Tryptophan and 5-hydroxytryptophan decrease feed intake. Substances that block the effect of serotonin can increase body weight and those that stimulate the release of serotonin or inhibit its uptake at the nerve endings will cause a reduction in the release of body weight. Thus both noradrenaline and serotonin play important but generally reciprocal roles in the regulation of food intake via structures located in the medial and lateral hypothalamus.[98,99]

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Conflicts of interest

The authors declares that there is no conflicts of interest.

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