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Trends in Functional Food Against Obesity

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1. Introduction

The increasingly accepted notion of the relationship between diet and health has opened new perspectives on the effects of food ingredients on physiological functions and health. Among the nutritional complications, increased incidence of obesity and its associated medical complications is creating a pressure from consumers towards the food industry which may provide an opportunity for the development of functional foods designed for the prevention and/or treatment of these pathologies.

Obesity is a multifactor disease where several factors may influence its onset, which includes the contributions of inherited, metabolic, behavioural, environmental, cultural, and socioeconomic factors as it is shown in Figure 1. Most of these factors may play together in different grades of contribution, which may differ between patients, and may influence treatment objectives in each individual.

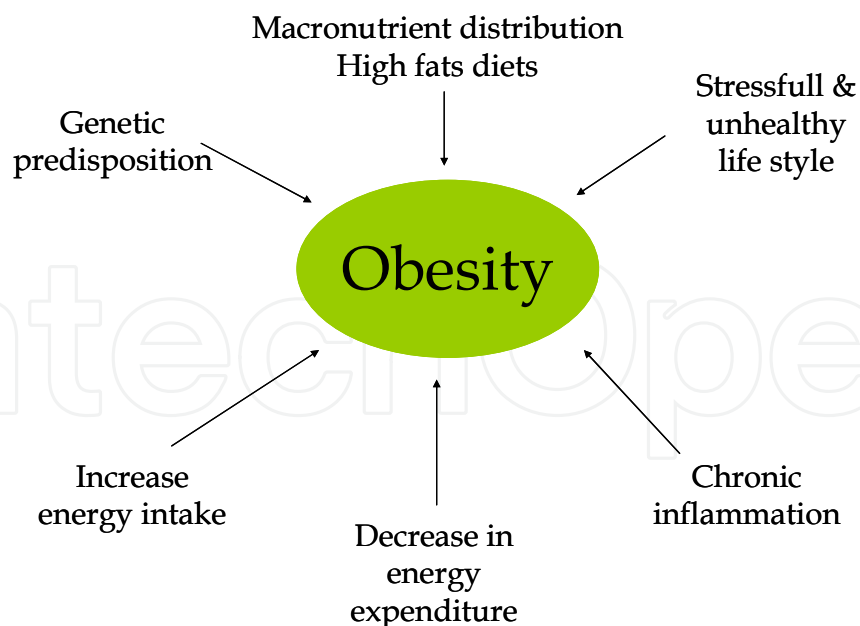


Fig. 1. Obesity, a multifactorial disease.

Moreover, overweight and obesity may raise the risk of other related pathologies like high blood pressure, high blood cholesterol, heart disease, stroke, diabetes, certain types of cancer, arthritis, and breathing problems. As weight increases, so does the prevalence of

health risks. The health outcomes related to these diseases, however, may be improved through weight loss or, at a minimum, no further weight gain. The main goal of any nutritional intervention is to individually determine the principal factors that may contribute with individual obesity predisposition and find specific tools to counteract each factor. Food Industry may play an important role providing enough tools, functional foods, for the prevention and treatment of obesity

In simplified terms, overweight and obesity can be defined as an imbalance where the amount of energy intake exceeds the amount of energy expended. Treatment and prevention of obesity requires changes in one or two of the components of this simplified equation. In this sense, the development of functional foods should be aimed to decrease the amount of energy intake (by lowering the energy density of foods or reducing the food intake) or increasing caloric expenditure through the stimulation of thermogenesis and/or modifying the distribution and use of nutrients as energy fuel between tissues, discouraging fat deposition. A summary of possible strategic features for the development of functional foods against obesity is shown in Figure 2.

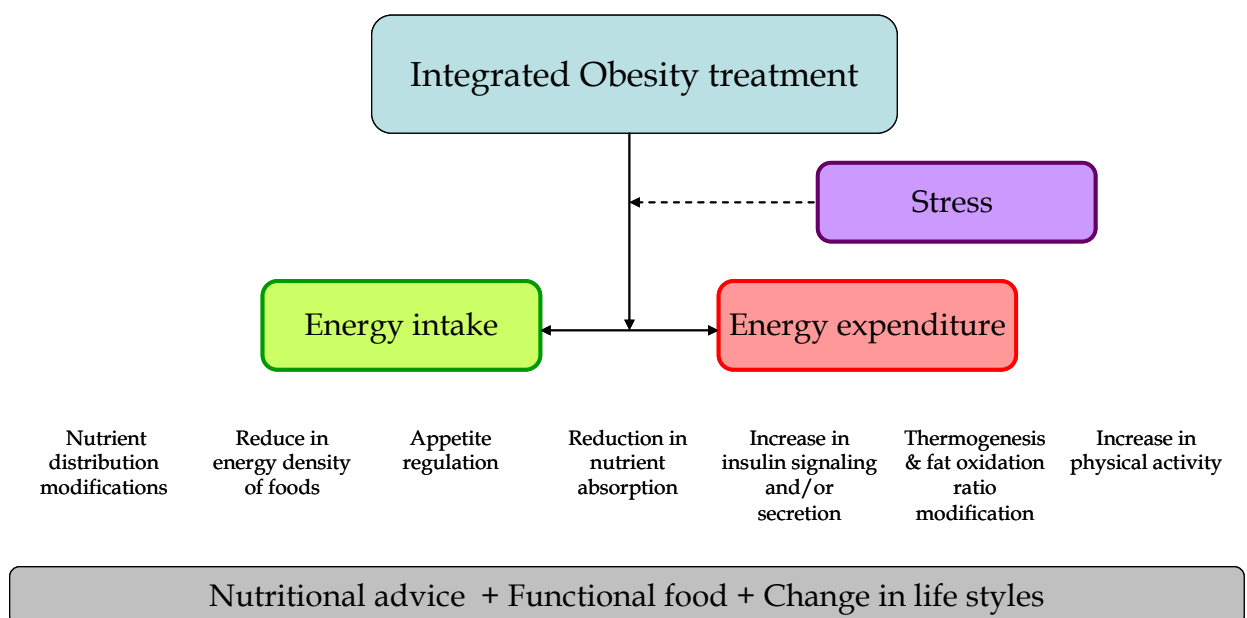


Fig. 2. Main strategic features for the development of functional foods against obesity.

Since several decades ago, in the fight against obesity, food manufactures had offer, a variety of food products named in the beginnings as “dietetic products” based principally in substitutions of sugars and fat by non-nutritive sweeteners and fat replacers respectively. Nowadays, the research and development of new products, should offer the market, food products indicated especially for obese people that besides their low caloric content, can offers the possibility to influence the energy metabolism as well as in the physiological sensation of satiety. Currently, there is a wide variety of products in the market with a low energy density, while the supply of products with bioactive ingredients that decrease appetite, increase caloric expenditure and/or affect the distribution of body fat is scare and in some cases of doubtful effectiveness.

In this context, the new European regulation regarding food labelling, may encourage the food industry to carry out more investment in research and in the determination of the effectiveness of the functional products launched to the market at different levels

(biochemical, molecular, genomic and psychological). In this way, the confidence and scientifically contrasted effectiveness of these products as preventive and therapy tools against obesity, may contribute to reduce the incidence of obesity in the whole population.

The first step in the development of functional foods is the identification of functional factor, condition or compound that produces a specific effect, which is effective as an adjunct in the treatment of obesity. The new European regulation demands that the effectiveness of such functional foods should be properly established with sufficient scientific evidence, including intervention studies in human populations. It is also desirable to establish the possible interactions of the functional ingredient within the body at different levels (genomic, molecular, cellular and psychological). On the other hand, is very important and necessary to investigate the functional ingredients incorporated into food as such, taking into account possible interactions between "functional ingredient" and other food matrix components, its dose, culinary preparation processes and the usual form of consumption. The key points in the evaluation of functional foods would be the safety and efficacy, thereby avoiding misleading advertising to the consumer.

In this sense, it is necessary to establish specific biomarkers (e.g. body mass index, blood cholesterol levels, percentage of body fat) the effectiveness of consumption of functional foods designed against obesity. However, there is still no consensus on the specific relevance and applicability of each of these biomarkers in the context of obesity, so that there is unanimity on tests of functional assessment for all food companies to launch a new functional food to the market.

The objective of this chapter is to describe possible guidelines for the development of functional foods based on the scientific evidence of the actions of several bioactive compounds and nutritional/technological modifications of foods to be used for the prevention and/or treatment of obesity. It describes possible actions that could be undertaken from different levels, starting with the technological modification of food with the aim to produce a satiety feeling towards the incorporation of functional ingredients that may modify energy intake and expenditure.

2. Energy balance, factors that influence energy intake & expenditure

As mentioned before, the strategy for functional food development should be based in the reduction of energy intake and/or in the increase in energy expenditure. The reduction in energy intake can be obtained by increasing satiety feeling either by the activation of satiety centres or by the modification of hunger feeling delaying it's onset. Optimally, a good satiety functional food must be satisfying and have a reduce energy content. The increase in energy expenditure can be regulated by the modification in the metabolic rate via an increase in thermogenesis or by the control in hormonal energetic metabolisms like insulin sensitivity. Additionally it should be also taken into consideration other factors like inflammation, psychological and physiological stress and life styles that may influence the response and adaptation to energy intake and expenditure.

This section includes a brief description of physiological mechanisms that may influence energy balance and possible strategies for counteracting the effect of each mechanism in obesity, as a tool for the manufacture of functional foods.

2.1 Satiety control, a tool for energy intake reduction

Human behaviour towards food can be defined as a physiological and psychological process that may be influenced by genetic and environmental factors in which the individual is

involved. The physiological regulation of the act of eating (hunger and satiety sensations) is a complex interaction between peripheral signals and central nervous system interpretation of these signals, to which must be added physio-psychological variables, such as differences in taste perception and the strictly psychological variables likely influenced by the individual's surrounding environment.

From a physiological point of view the satiety and hunger regulation has been described using two paradigms: the glucostatic hypothesis (Mayer & Thomas, 1967) and the lipostatic model (Kennedy, 1953). The glucostatic hypothesis is based on the assumption that small changes in plasma glucose levels induced signal initiation and termination of eating. However, this model does not take into account how the body regulates the long-term storage and use of energy. The lipostatic model hypothesizes that there are peripheral signals that gives information about the amount of fat or stored energy and therefore the amount of energy needed to maintain a good energy balance. This hypothesis has been supported by the discovery of leptin, an adipokine that is released by adipose tissue in proportion to its fat content. However, since there are no significant fluctuations throughout the day on the composition of body fat and thus leptin, this model may not explain the dynamic behaviour and varying feelings of satiety-hungry induced throughout the day.

The interpretation of these signals is done by the central nervous systems. Recently it has been reported that short- and long-term satiety and hunger feelings may be regulated by several neural circuits at the ventromedial, dorsomedial and paraventricular hypothalamic nuclei for satiety sensations and at the lateral hypothalamus for hunger sensations. Although the hypothalamus is an important centre in the energy balance regulation, there are other brain regions such as the medulla oblongata and cortical and striatal structures, essential for the eating behaviour modulation. For example, some neural circuits of the medulla oblongata seem to have an important role in autonomic eating regulation, limiting the quantity of ingested food through the satiety responses regulation. Whereas, other parts of the brain, like the nucleus accumbens and ventral tegmental area, where dopamine, opioids and cannabinoids signals are integrated, regulated the motivation to eat, the rewards and the acts before eating. In this context, although hunger is connected to the biological needs, there are also psychological factors involved in the food intake regulation. Learning and emotions play a powerful role in determining what to eat, when to eat, and even how much to eat. In this context, the psychological desire of eating and its complicated mechanisms of influence in satiety interpretation difficult the design of functional foods for this purpose.

Although, there are many peripheral signals that can contribute to feeding behaviour and body weight regulation and can be modified by food and food ingredients. It is important to recognize that short-term and long-term food intake and energy balance are regulated through distinct, but interacting, mechanisms. Figure 3, shows a brief review of nowadays known possible satiety signals that may influence eating behaviour which included, beside short and long-term signals, individual social behaviour and other metabolism compounds that may influence satiety feeling.

Short-term regulation of food intake results from an integrated response from neural and humoral signals that originate mainly at the brain, gastrointestinal tract and adipose tissue. Ingested food evokes satiety in the gastrointestinal tract primarily by two distinct ways, i.e. by mechanical stimulation and therefore stimulation of the nerve endings; and by the release of satiety peptides. The scheme is more complicated as both ways seems to be intimately related, since many of the intestinal peptides released may inhibit also gastric emptying thus enhancing gastric mechanoreceptor stimulation.

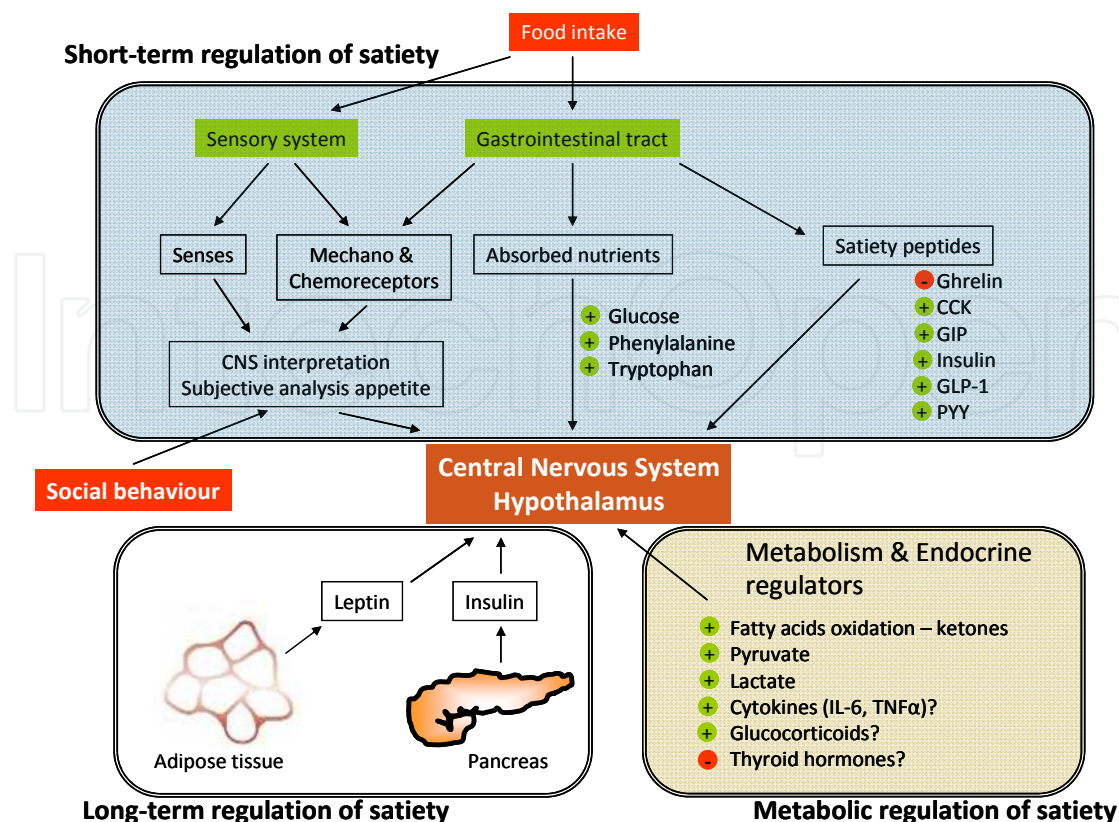


Fig. 3. Short- and long-term regulation of satiety. Principal signals implied in energy regulation and satiety.

The postprandial satiety consequences of food intake are determined both by the specific chemical composition and the characteristics physical properties of the food. Accordingly, different foods, despite their equal energy content, can differ in their capacity to affect postprandial metabolism, especially secretion of gastrointestinal peptides, thereby regulating energy homeostasis. A classical example is fibre where several differences in chemical structures and characteristic physical properties can be observed. For example, bulk/volume, viscosity, water-holding capacity, adsorption/binding, or fermentability may determine the subsequent physiological behaviour of fibre even though it is ingested in the same quantity. Table 1 and below is included a brief description of the principal satiety signals derived from the gastrointestinal tract and its possible effects in food intake behaviour.

Peptide	Organ of synthesis	Receptor related with satiety signals	Effects on food intake
CCK	Proximal intestine I-cells	CCK1R	Decrease
GLP-1	Distal intestine L-cells	GLP1R	Decrease
PYY ₃₋₃₆	Distal intestine L-cells	Y2R	Decrease
PP	Pancreatic F cells	Y4R, Y5R	Decrease
Amylin	Pancreatic β -cells	CTRs, RAMPs	Decrease
Gastric leptin	Stomach P-cells	Leptin receptor	Decrease
Ghrelin	Gastric X/A-cells	Ghrelin receptor	Increase

Table 1. Gastrointestinal satiety peptides that may regulate food intake.

Ghrelin. Ghrelin is the only mammalian substance that has been shown to increase appetite and food intake when delivered to humans. Circulating ghrelin levels typically rise just before and fall shortly after a meal, thus playing a role in meal time hunger and meal initiation. The postprandial ghrelin response is affected principally by the caloric content of meals. Thus, high energy-rich meals suppress ghrelin more than lower ones. In humans, across the range of intakes of 220 to 1000 kcal, the lowest point of postprandial ghrelin was found to decrease by about 2.4% for every 100 kcal increase of energy intake (Callahan et al., 2004). Recent findings suggest that postprandial suppression of ghrelin is not mediated by nutrients in the stomach or duodenum but rather from post-ingestive increases in lower intestinal osmolarity (via enteric nervous signaling) as well as from insulin surges (Cummings, 2006). In contrast, the short-term parenteral administration of glucose and insulin in physiological doses may not suppress ghrelin levels (Gruendel et al., 2007). Moreover, ghrelin concentration is not affected by stomach distension, since the administration of water did not influence its concentrations (Shiyya et al., 2002). In relation to the possible modification of its plasmatic levels, it is known that increased fibre content of the meal has shown both to decrease postprandial ghrelin concentrations as well as to inhibit the decrease.

Cholecystokinin (CCK). The inhibitory effect of CCK on food intake has been confirmed in numerous species, including humans. It is however short-lived, lasting less than 30 min. Therefore, CCK may inhibit food intake within the meal by reducing meal size and duration but does not affect the onset of a next meal. Thus it may have an important role in the causal chain leading to satiation or meal termination. Gastric distension augments the anorectic effects of CCK in humans. However, other mechanisms including activation of duodenal chemosensitive fibres and activation of CCK receptor 1 in the pyloric sphincter that may slow down gastric emptying may be implicated.

Glucose-dependent insulinotropic polypeptide (GIP). GIP is released in response to the presence of nutrients in the intestinal lumen. The major stimuli for GIP release are dietary fat and carbohydrates. Protein seems to have no effect, although some evidence exists indicating that the intraduodenal administration of aminoacids can stimulate GIP release.

Glucagon-like peptide 1 (GLP-1). GLP-1 is an incretin hormone released in response to food intake. GLP-1 is typically very low in the fasting state, but rise quickly after food intake, especially after carbohydrate intake. The rise of GLP-1 has been correlated with increased satiety and less hunger. GLP-1 is thought to play an important role in the "ileal break", a mechanism that regulates the flow of nutrients from the stomach into the small intestine. It is also suggested that portal GLP-1 might influence the production of ghrelin (Lippl et al., 2004) and the increase in β -cell mass in the pancreas, thus improving the insulin production.

Peptide tyrosine-tyrosine (PYY). PYY is a member of the pancreatic polypeptide fold family including neuropeptide Y (NPY) and pancreatic polypeptide (PP). PYY mediates ileal and colonic breaks, mechanisms that ultimately slow gastric emptying and promote digestive activities to increase nutrient absorption. Plasma concentration of PYY increases after meals consistent with a meal-related signal of energy homeostasis. Nutrients stimulate PYY release within 30 minutes of ingestion, reaching usually a maximum within 60 min. The release is directly proportional to caloric intake; however meal composition may affect postprandial PYY release. In humans, infusion of PYY₃₋₃₆ (active form) comparable to those after a meal result in decreased energy intake at subsequent meals compared with a control group (Batterham et al., 2002)

Amylin. Amylin is co-secreted together with insulin from pancreatic β -cells. It is a 37 amino acid peptide with anorexigenic effects that have shown to reduce meal size as well as the number of meals. The inhibitory effect of amylin on food intake is thought to be due to the inhibition of gastric emptying.

Pancreatic polypeptide (PP). PP is secreted by F-cells on the endocrine pancreas comprising approximately less than 5% of islet volume. The main function of PP is thought to be the inhibition of exocrine pancreas. Its secretion is controlled by the parasympathetic nervous system and shows a biphasic manner in proportion to food intake.

Leptin. Leptin is a peptide hormone which is released from white adipose tissue and acts in the hypothalamus to promote weight loss, both by reducing appetite and food intake and by increasing energy expenditure. Circulating leptin concentrations are highly positively correlated with body mass index. Despite this strong association, leptin levels shows large individual variation for a given degree of adiposity, indicating the likely effect of variables other than adipose mass, such as genetic and environmental factors. Additionally, food consumption stimulates leptin secretion after a meal and high carbohydrate meals results in greater leptin responses. Although leptin does not seem to play an important role in the short-term regulation of food intake, when subjects are in energy balance, plasma leptin is negatively correlated with appetite and food intake when energy balance is disturbed. Leptin therefore seems to have a role in the regulation of food intake when energy stores changes.

Insulin. Insulin is the major endocrine and metabolic polypeptide hormone secreted by β -cells of the endocrine pancreas and one of the key adiposity signals in the brain influencing energy homeostasis. Plasma insulin concentrations are in direct proportions to changes in adipose mass. Insulin concentrations are increased at positive energy balance and decreased at the times of negative energy balance. Additionally, plasma insulin concentrations are largely determined by peripheral insulin sensitivity which is related to the amount and distribution of body fat in insulin-resistant patients. Insulin thus provides information to the central nervous system about the size and distribution of the adipose mass to regulate metabolic homeostasis.

Besides the described satiety peptides and hormones, other absorbed food derived compounds, metabolites and hormones may also serve as satiety signals for the central nervous system. For example, aminoacids such as phenylalanine and tryptophan that are precursors to monoamine neurotransmitters suppress food intake in humans. The ratio of plasma tryptophan to other amino acids may influence brain serotonin levels, which are known to have inhibitory influence on food intake. Some authors have suggested that the oral administration of 5-hydroxytryptophan may reduce food intake, as well as a reduction in carbohydrate intake and a higher satiety feeling in obese subjects (Cangiano et al., 1992).

Other metabolisms by-products like ketones (from fatty acids), which are metabolic substrate for the central nervous system, are known to inhibit feeding. Moreover, lactate and pyruvate have been reported to induce satiety effects in animal models (Nagase et al., 1996). And finally, endocrine regulators of food intake like cytokines (IL-6 and $\text{TNF}\alpha$), glucocorticoids and thyroid hormones have also been described to regulate satiety and hunger feelings. However the mechanisms of how they may influence feeding behaviour, possibly related with energy expenditure administration, are not well understood.

Food formulation for the induction of the release of satiety peptides or the decrease of ghrelin could be basically obtained by the incorporation of functional ingredients that may

increase satiety peptides release in the gut, as well as by rheological modifications with the objective of producing sensory stimulation in the first phases of digestion and also by a higher gastric distension and emptying rate time. The possible strategies are described below.

2.1.1 Energy reduced satisfying foods, combined strategy to reduce energy intake

The combination of energy reduced foods with functional ingredients that stimulate satiety peptides release may be an interesting and ambitious approach. It should be taken in consideration that the release of satiety peptides is normally proportional to the energy intake and energy density of food. In this sense, it seems difficult to design energy reduced food with also the ability to stimulate satiety. However, some approximations to this goal can be developed.

There are some evidences that macronutrients may regulate the secretion of satiety peptides. For example, fats and proteins may increase the release of CCK and reduce ghrelin concentrations by its effect in gastric emptying delay. Other authors suggest also some effects in GLP-1, GIP and PYY, however the exact mechanisms of action is still unknown.

In the other hand, carbohydrates, especially viscous and fermentable fibre (with a reduce energy content), have been widely investigated because of its satiety effects in humans. Figure 4 briefly schematises the possible mechanisms of action of fibres in the induction of satiety.

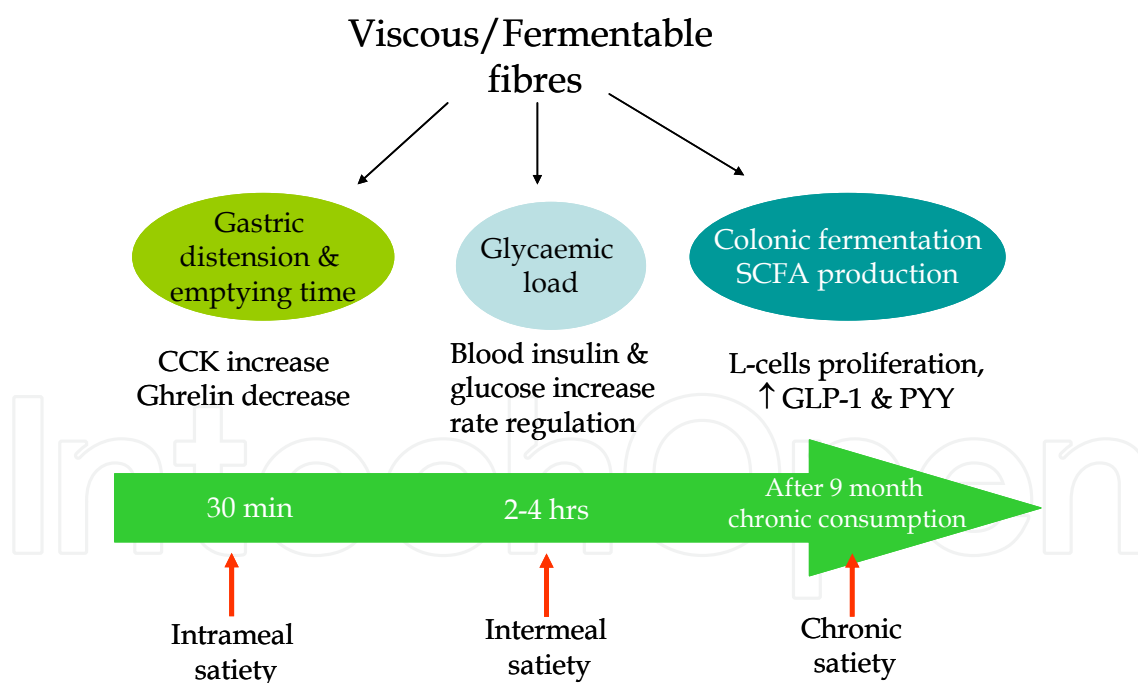


Fig. 4. Mechanisms of action of fibre in satiety induction. SCFA: short-chain fatty acids.

Viscous fibres have been related to satiety because of its effects in gastric distension and emptying rates which may increase CCK and reduce ghrelin secretion. Later, in the small intestine, viscous fibre may regulate bolus transit time, which may affect the total glycemic load, regulating glucose and insulin levels, hence inducing direct satiety stimulation in central nervous system satiety centre. And finally, fibre colonic fermentation by products

like short-chain fatty acids are recognized to induce L-cells proliferation in the colon, which are the main cells where GLP-1 and PYY is produced. Thus, chronic fibre intake may chronically increase the amount of these satiety peptides in plasma. Notwithstanding, several authors suggest that the amount of fibre that may exert satiety feeling in humans should be higher than 8 g/day, which difficult its incorporation in technologically modified foods. Moreover, it should be taken into consideration that viscous fibre may produce acute satiety feeling (intrameal and intermeal satiety); whereas fermentable fibres may produce chronic satiety feeling, however after a chronic consumption higher than 9 months.

Other bioactive compounds like polyphenols have been described also to induce a higher rate of secretion of satiety peptides like GLP-1 possibly because of its effects in the inhibition of glucose uptake by enterocytes (McCarty, 2005). In this context, the addition of antioxidant dietary fibre (fibres with associated antioxidant compounds) to food formulation may help to increase satiety feeling of foods.

In brief, it can be said that glucose and fat are the main inductors of satiety peptides. However, it is widely accepted that the increase in glucose and fat uptake are strongly related with obesity. The next step should be the reduction of energy density of formulated foods.

Currently there are a variety of food ingredients that can be used with the purpose to replace fat and sugar in foods formulations (Table 2 & 3) with the intention to reduce the total calorie intake. For example, the average consumption of simple sugars in the Spanish diet by 2005 (MAPA, 2005) was around 113 g/person/day an estimated 18% of total calories (2424 kcal/person/day), exceeding by 8% the recommended dietary intake of simple sugars. Dairy products, beverages, cakes, pastries, etc., target products for the replacement of simple sugars with non-nutritive sweeteners, contributed to 68% and 62% of the total intake of simple sugars and sucrose in the Spanish diet respectively (Figure 5). Its replacement with non-nutritive sweeteners could result in a reduction in energy intake around 310 kcal/day. However, the abuse in the intake of non-nutritive sweeteners is also criticized by the scientific community, since some of them are banned in other countries, such as the use of cyclamate by the Food and Drug Administration in the United States.

Generic name (commercial name)	Sweetener capacity (compare with sucrose)
Potassium acesulfame (Sunett)	180-200
Alitame	2000
Aspartame (Equal, Nutrasweet)	180
Cyclamates	30-50
Glycyrrhizin	30-50
Lo han guo	30
Neotame	8000-13000
Perillartine	2000
Saccharine (Sweet'n low)	300
Stevioside	40-300
Sucralose (Splenda)	600

Table 2. Available non-nutritive sweeteners in the market.

Generic name	Commercial name	Caloric value
Based on proteins		
Whey protein	Simplese, Dairy-Lo	1-2 kcal/g
Based on carbohydrates		
Cellulose	Avicel cellulose gel, Methocel, Solka-Floc	0 kcal/g
Dextrins	Amylum, N-Oil	4 kcal/g
Gums	Kelcogel, Keltrol	0 kcal/g
Inulin	Raftiline, Fruitafit, Fibruline	1-1.2 kcal/g
Maltodextrins	Crystal Lean, Lorelite, Lycadex, Maltrin	4 kcal/g
Polydextrose	Litesse, Sta-Lite	1 kcal/g
Polyols	Several brands	1.6-3.0 kcal/g
Fat analogous		
Olestra	Olean	0 kcal/g
Sorbestrin		1.5 kcal/g
Salatrim	Benefat	5 kcal/g
Emulsifiers	Dur-Lo	9 kcal/g

Table 3. Fat replaces available in the market.

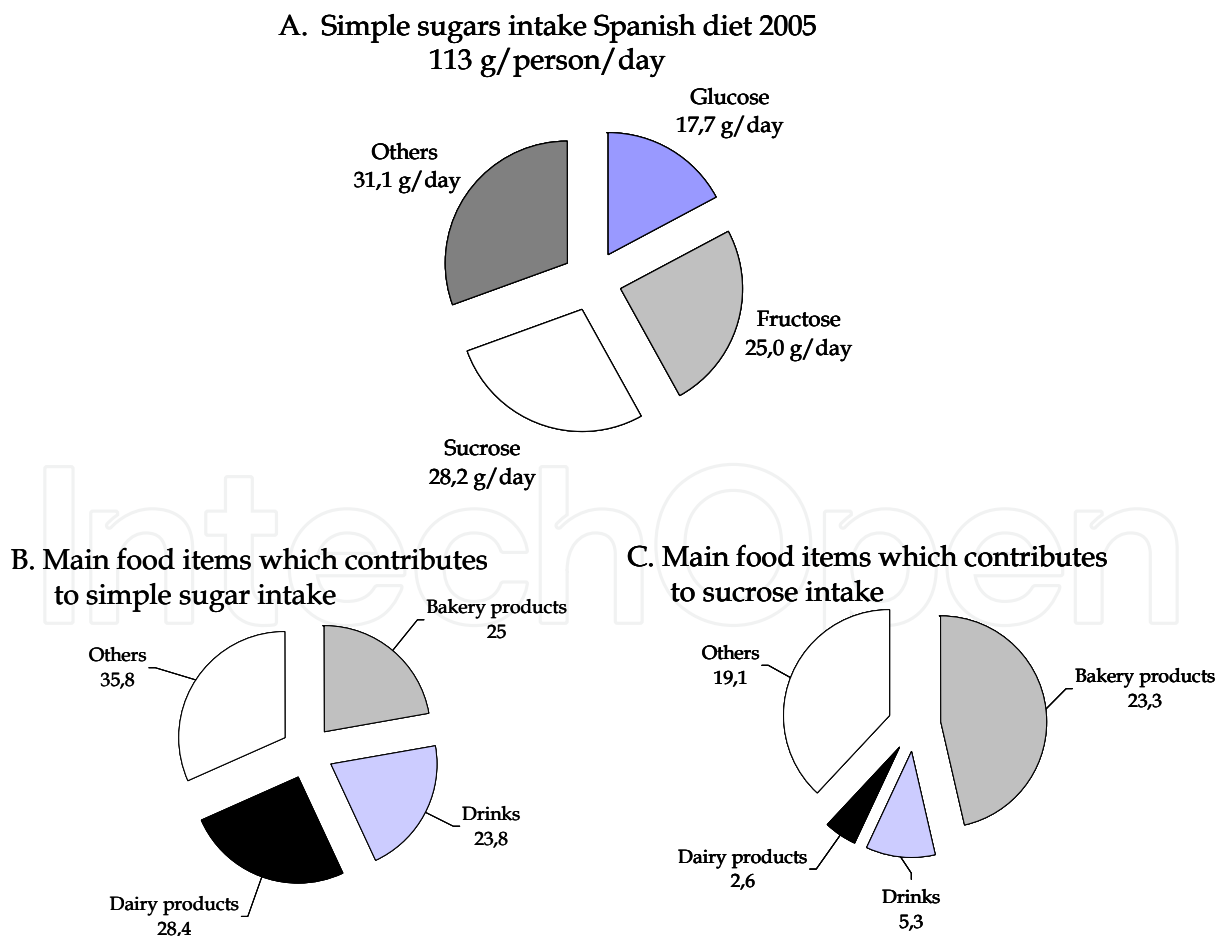


Fig. 5. Simple sugars intake and principal food items that contributes to its intake in the Spanish diet.

With respect to fat substitutes, several fat replacers have been developed to assimilate the sensory properties of fat. It can be divided basically into 3 groups; 1) based on proteins, used mainly in dairy products; 2) based on carbohydrates, used in dressings, meat products, etc. and 3) based on modified fat, that may contain organoleptic characteristics similar to fats but with a lower caloric content. Among them, Olestra approved in 1996 by the United States to replace fats and oils, is a sucrose polyester containing between 6 and 8 fatty acids per molecule, that has organoleptic properties similar to those of typical fat, but without the capacity of being hydrolyzed by lipases; and Salatrim, a mixture of long- (mainly stearic acid) and short-chain fatty acids (acetic, propionic and butyric) esterified with glycerol that greatly reduce the caloric intake per gram of fat. The main drawback of the use of fat-based substitutes is the possible decrease in the absorption of fat soluble vitamins, which must be taken into account in the formulation or modification of food that are sources of fat-soluble vitamins in the diet.

Naturally, foods with lower energy density and satiety properties, such as fruits and vegetables, compared to energy-dense foods, are low in fat and high in water and/or dietary fibre content, since these compounds may add weight and volume to foods without increasing its caloric content. Therefore, increasing the water content through wetting agents and/or increasing the content of dietary fibre could be a possible approach.

Possibly the best option is to reduce the energy density of foods by the incorporation of fibre (with an average energy value of 1-2 kcal/g) to the formulations, which also includes the possibility to increase the water content of food products. Physiologically, fibre may decrease the absorption of macronutrients like fat and carbohydrates reducing its energy value. On the other hand, some fibres obtained from fruits and vegetables may also provide functional antioxidant compounds valuable for obesity treatments.

It should be noted, however, that an increased intake of foods with low energy density is not enough to lose weight, except when they move to higher-energy density. The choice of food can often be influenced also by their quantity, volume or weight, in this sense changes in the appearance of food can be another simple strategy to develop other types of functional foods.

2.1.2 Rheological modifications of foods to induce satiety

The main objective in developing this type of functional foods is to induce satiety by modifying the perception of consumers toward food. That is, sensory (sight, smell and taste mainly) stimulations that able to production of an "apparent" fullness feeling in the early stages of feeding. For example, some studies suggest that the volume of food ingested psychologically affect hunger, satiety and the amount of food that individuals want to eat (Rolls et al., 2000). In this sense, an example could be the development of products with a high volume and low density in both weight and energy (for example by cereal extrusion or air emulsions desserts like "mousse").

Satiety may also be induced through the stimulation of retro-nasal aroma by food (Ruijschop & Burgering, 2007). There are some indications that not all types of foods produce the same quality (flavour) or quantity (intensity) of sensory stimulation. The physical structure largely seems to be responsible for stimulating aroma. For example, solids tend to produce a greater sense of satiety than liquid foods, probably because of the increased contact time of food in the oral cavity and therefore the greater sensory

stimulation. It is also suggested the addition of encapsulated flavours that may extend specialized sensory flavour in the mouth in foods with a low energy density.

Rheological modifications for satiety peptide release are mainly based in the induction of a higher gastric distension and a reduction in gastric emptying which may promote the release of CCK and the decrease of ghrelin levels. Viscous fibres are the main nutrient associated with the ability to slow gastric emptying as discussed above.

2.1.3 Digestive enzyme inhibition and gut energy absorption inhibition

Another possible strategy is to limit the absorption of nutrients in the intestinal tract, by limiting the action of digestive enzymes and/or interacting with them to physically interrupt its absorption in the intestinal tract. The most common example is the use of fibre, which modulates the intestinal transit time resulting in increased satiety, reduced physical accessibility of nutrients for being absorbed and a reduced the calorie intake of the formulation.

An interesting example of functional fibre is chitosan (product obtained from chitin, located in the shells of shellfish), which is a positively charged polymer that could bind to negatively charged fat molecules in the intestinal lumen (mainly free fatty acids) therefore inhibiting its absorption. On the other hand there are other bioactive compounds with the ability to inhibit the activity of digestive enzymes, the most common digestive enzyme inhibitor found in foods are condensed tannins, which have the ability to precipitate proteins (including enzymes), reducing its action.

In connection with the absorption of carbohydrates, some researchers have shown that certain polyphenols (for example from tea) have the ability to inhibit *in vitro* the translocation of glucose transporter, GLUT2, in the intestinal epithelial cells, thus inhibiting the absorption of glucose (Kwon et al., 2007). This same effect has been demonstrated *in vivo* by oral sucrose tolerance test in the presence of epigallocatechin gallate, observing a decrease in blood glucose values (Serrano et al., 2009).

2.2 Thermogenesis and possible modifications in energy expenditure

Small differences in energy expenditure might have long-term effects on body weight. The human energy budget is usually divided into four major components (Figure 6), which, together, constitute the total energy expenditure: 1) basal metabolic rate; 2) diet-induced thermogenesis; 3) physical activity and 4) adaptative thermogenesis.

One of the suggested metabolic factors involved in the development of obesity is adaptive thermogenesis. It is defined as the regulated production of heat in response to environmental temperature or diet. It can be seen as a mechanism for dissipation, in a regulated manner, of the food energy as heat instead of its accumulation in fat. Skeletal muscle is potentially one of the largest contributors to adaptive thermogenesis in humans. For example, an adrenaline infusion may stimulate muscles to consume over a 90% more oxygen, thus increasing energy expenditure.

The possible mechanism of action of the increase in heat production may be due to the higher stimulation/synthesis of uncoupling proteins at the mitochondria. The uncoupling protein can dissipate a proportion of metabolic energy through uncoupled metabolism that creates heat rather than the generation of ATP. Consequently, these have been the topic of extensive research as possible targets to fight against obesity. The favoured route has been to develop specific β 3-adrenergic compounds that might stimulate UCP1 without the undesirable β 1 and β 2 effects on heart rate and blood pressure.

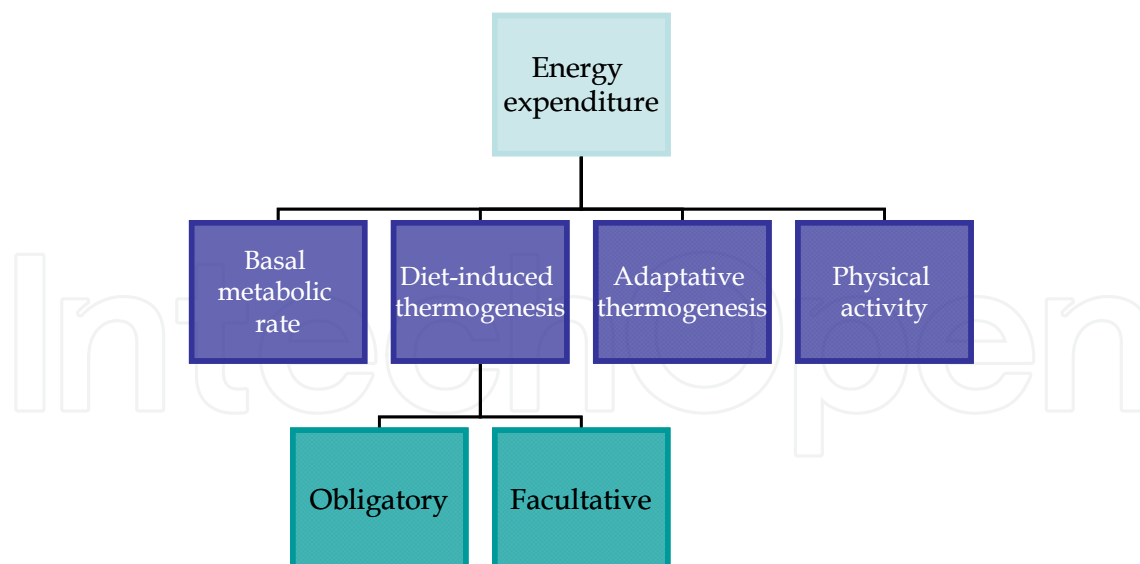


Fig. 6. Principal components of energy expenditure in humans.

In relation to nutrition, it has been shown that carbohydrates induced an increased adrenaline concentration, resulting in increased muscle thermogenesis (Astrup et al., 1986). Other authors have reported a variety of compounds that can alter energy expenditure. For example, in rodents, acute treatment with retinoic acid increases the thermogenic capacity in brown adipose tissue, which further in time may induce a significant decrease in body weight and adiposity (Bonet et al., 2000). Green tea extracts have been also described to stimulate thermogenesis in brown adipose tissue, mainly due to the interaction between its high content of catechins and caffeine that may stimulate the noradrenaline released by the sympathetic nervous system. Overall, the action of caffeine and catechins may prolong the stimulatory effects of noradrenaline on energy metabolism and lipid.

In another context, it is interesting to observe that after overfeeding, the same amount of excess energy intake and nutrients does not always invoke the same body weight gain in all people because of differences in diet-induced thermogenesis (energy expenditure in response to food intake).

Diet-induced thermogenesis can be divided in two categories: obligatory and facultative thermogenesis. The obligatory part consists of all process related to the digestion, absorption and processing of food. Stimulation of adenosine triphosphate (ATP) hydrolysis during intestinal absorption, initial metabolic steps and nutrient storage, are responsible for this food thermic effect. For example, measured thermic effects of nutrient digestion are 0-3% for fat, 5-10% for carbohydrates and 20-30% for proteins. While, the facultative component enables wasting of energy after a high caloric meal and prevents the storage of energy.

In relation to facultative thermogenesis, it has been observed that the thermic effect of food is reduced in obese and insulin-resistant patients, possibly because of the effect of the autonomic nervous system activation by certain nutrients and the insulin secretion stimulation. Insulin resistances may induce a decrease turnover of ATP by muscle tissue, therefore reducing its metabolic rate. It has also been suggested that insulin, via unidentified receptors, most probably located in the central nervous system, may stimulate muscle sympathetic nerve activity and facultative thermogenesis, and therefore its probably reduce in obese insulin resistance patients. Functional foods against insulin resistance may also help to reduce obesity by increasing insulin sensibility thus optimizing the use of energy. This aspect is described deeply in the next section.

And finally, protein turnover defined as degradation of proteins into amino acids and resynthesis of new proteins could be responsible for large part of the energy expenditure, around 15-20% of basal metabolic rate. Most tissues exhibit protein turnover, specifically the skeletal muscle tissue, liver, skin and small intestine. In humans, it has been shown that after carbohydrate overfeeding, protein turnover is increased by 12%, may be because of the reduction in protein intake.

2.3 Insulin metabolism and the regulation of adiposity

The causal links between obesity and insulin resistance are complex and controversial. Weight gain from overfeeding induces insulin resistance, whereas weight loss by caloric restriction reverse insulin resistance. The main concern should be to reduce the insulin secretion, associated to an increase in fat deposition in the adipocyte; as well as to increase its sensibility which is associated to an increase in metabolic rate.

For food formulation it is important to know that increased plasma glucose levels increase the secretion of insulin from pancreatic β -cells, although other substrates such as free fatty acids, ketone bodies, and certain amino acids can also directly stimulate insulin's release or augment glucose's ability to trigger insulin release.

It has been suggested that a low-glycemic diet can help control obesity because of the ability to reduce the rate of glucose uptake in the small intestine and therefore the insulin secretion, as well as to increase satiety value of food and appetite regulation (Brand-Miller et al., 2002; Roberts, 2003). While, high glycemic index foods, by its higher stimulation in insulin secretion, may promote a higher postprandial oxidation of glucose at the expense of fat oxidation, which can lead to increased body weight gain.

The glycemic index of a food is directly proportional to the degree of intestinal absorption of carbohydrates, in this sense the incorporation of factors that replace simple carbohydrates or decrease the absorption of carbohydrates such as fibre, fat or by inhibiting the action of digestive enzymes can reduce the glycemic index of food.

However, not all strategies to reduce the glycemic index of foods are widely accepted by the scientific community. For example, replacing simple sugars like sucrose with fructose, as a sweetener with low glycemic index, has been associated with increased body adiposity (Bray et al., 2004). After the ingestion of fructose, insulin is not increased, leptin is reduced, and ghrelin is not inhibited. Because these hormones play important roles in regulating food intake, the combined effects of excessive fructose intake could result in a lower induction of satiety and increase in total intake. Moreover, these foods should be low in saturated fatty acids, especially *trans* fatty acids, which are associated with a higher induction in adiposity. In this context, the addition of conjugated linoleic acid appears to have an effect based on the inhibition of the activity of lipoprotein lipase, which may reduce the uptake of lipids by the adipocyte.

3. Functional diets based on traditional foods

The use of appropriate combinations of nutrients that affect different processes of energy intake and expenditure could be the best strategy to tackle obesity control, usually a multi-causal problem. A higher effectiveness can be obtained from the combination of functional ingredients that inhibit the appetite sensations, the bioavailability of macronutrients and induce a thermogenic response to an individual. An interesting approach could be the formulation of "Ready Meal" type product.

A ready meal is a type of convenience food that consists of a pre-packaged meal that needs little preparation. In 2009 the global market for ready meals was worth \$71.6bn. In the Western Europe and the US the ready meals markets are fairly mature and well-established with moderate growth rates. The only pre-requisite for such diets is that they should be nutritionally appropriate and balanced with a low caloric content that allows consumers to replace any meal time with some of these diets on the market.

4. Conclusions

To create and consume a satisfying diets and foods with high intrameal and intermeal satiety feeling that may help to fight against obesity, ideally should consist of low-energy dense foods with high palatability; however, such foods do not commonly exist. Moreover, individual genetic variation may influence the effectiveness of such preparations against obesity.

Notwithstanding, the diversity of strategies to combat the obesity problem makes possible to develop a variety of food products that may satisfy individual requirements. However, before its market launch, the metabolic effects should be widely contrasted as well as it should be defined the concrete profile of the people who can benefit from the consumption of the formulated functional food.

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This book presents the wisdom, knowledge and expertise of the food industry that ensures the supply of food to maintain the health, comfort, and wellbeing of humankind. The global food industry has the largest market: the world population of seven billion people. The book pioneers life-saving innovations and assists in the fight against world hunger and food shortages that threaten human essentials such as water and energy supply. Floods, droughts, fires, storms, climate change, global warming and greenhouse gas emissions can be devastating, altering the environment and, ultimately, the production of foods. Experts from industry and academia, as well as food producers, designers of food processing equipment, and corrosion practitioners have written special chapters for this rich compendium based on their encyclopedic knowledge and practical experience. This is a multi-authored book. The writers, who come from diverse areas of food science and technology, enrich this volume by presenting different approaches and orientations.

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