How can we stop eating? : a global challenge

The worldwide obesity epidemic suggests that while it has become (perhaps too) easy to eat in response to hunger (or to a host of other stimulations), the global problem facing our contemporaries today is to stop eating before over-consumption has occurred. Actually, science has established that biological mechanisms exist to stop eating and/or suppress its occurrence, even in situations of plentiful access to palatable food. One such mechanism is satiety.

From pioneer works to recent contributions

The scientific exploration of satiety developed from physiologically-oriented studies. Enlightening pioneer works have illustrated concepts such as homeostasis and energy regulation that are still at the centre of our understanding of food intake control today. Over time, the contribution of sensory, psychological, and socio-cultural factors has been recognised. Recent scientific developments, notably in the field of brain imaging, now allow the research of satiety mechanisms to scrutinise brain structures and functions directly while increasingly sensitive tools monitor the periphery of the organism, from the mouth to the gut.

A critical contribution to weight control

Once eating has started, various psycho-biological mechanisms come into play to bring the eating episode to an end. Those mechanisms, also very well investigated in scientific works, are those of satiation. After the end of an eating episode, people or animals stop eating for some time before hunger returns. This period without eating, and the psycho-biological mechanisms that support it, are what we call satiety. The duration and intensity of satiety are essential factors in body weight control, as evidenced in laboratory animals with ad libitum access to food: satiety is the crucial mechanism that allows the adequate adjustment of energy intake to energy needs and therefore prevents overeating.

The situation in human consumers is clearly more complicated than in animals confined to the very restricted environment of a laboratory cage. Fortunately! Sensory influences are richer, psychological mechanisms are more complex, social and cultural determinants are powerful. Critically, human consumers are exposed to the highly palatable, energy-dense foods present in their affluent "obesogenic" environment. Under those conditions, it is important to understand satiety mechanisms and, possibly, to optimize them and facilitate body weight control. This is what the present article will address.

Definition and methodology

Scientific progress requires precise definitions of concepts and harmonised use of methods. In the field of satiety research, key definitions exist (*Blundell et al., 2010*). "Satiation" is the multi-dimensional process that leads to the termination of an eating episode after it has started. "Satiety" is the process that leads to the inhibition of further eating, decline in hunger and increase in fullness after the end of an eating episode. A conceptual framework for examining the numerous factors influencing these processes was proposed almost 30 years ago by John Blundell and his group (*Blundell et al., 1987*) and has been regularly updated in order to integrate scientific developments. This framework is known as the "Satiety Cascade" (see Figure 1).

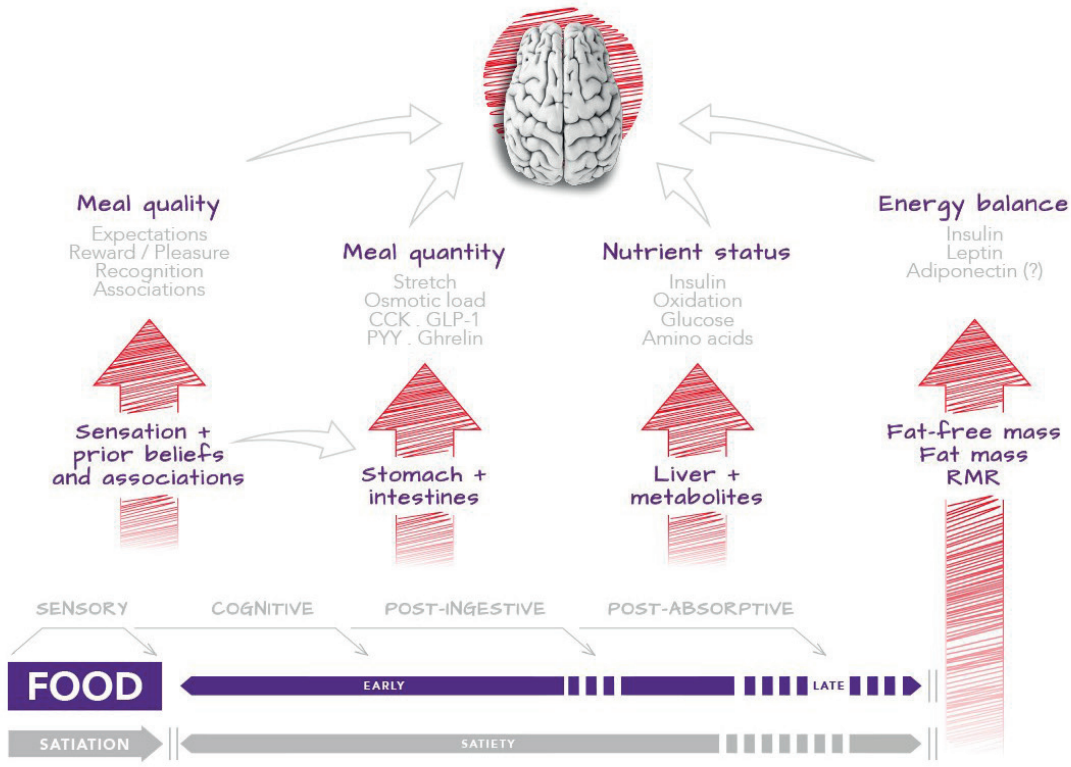


Figure 1: The Satiety Cascade. Multiple influences contribute to satiety from the early to the late phases of the post-ingestive interval. Sensory and cognitive factors are potent immediately after intake. Gastric factors, such as post-intake stretch and fall in ghrelin, as well as the release of various hormones, such as CCK, GLP-1 and PYY contribute to early post-meal satiety. Then elevated circulating levels of nutrients (glucose and amino acids mainly) and the release of hormones such as insulin support satiety until the return of hunger and the onset of the next meal. The body fat mass, via the release of the satiating hormone leptin, and the body fat free mass that determines the Resting Metabolic Rate, significantly modulate satiety.
CCK: Cholecystokinin; GLP-1: Glucagon-Like Peptide-1; PYY: Peptide YY.
Source: Adapted from *Blundell et al., 2010*

How is satiety measured in scientific studies? Three main aspects of the post-ingestive inhibition of intake are classically measured. The first one is the duration of the post-ingestive interval, from the end of one eating event to the spontaneous onset of the next eating episode (*Chapelot, 2013)*. Another approach consists of measuring the amount of food ingested at the next eating event, following the intake of a fixed amount of a particular food and after a time interval of fixed duration. This is the classic "preload – test meal paradigm" which has been used extensively to quantify differences in satiating power between different foods (*Blundell et al., 2010; Chapelot, 2013*). Finally, satiety can be equated with the changes in experienced sensations of hunger or fullness that follow ingestion. Psycho-physical instruments have been validated for the rating of relevant sensations (*Blundell et al., 2010*). The most commonly used is the unipolar unstructured Visual Analogue Scale (VAS), which consists of a straight line with verbal anchors at both ends indicating the extremes of one sensation, for example: "not hungry at all" and "extremely hungry" (see Figure 2). Labelled Magnitude Scales (LMS) are marked at both ends and with descriptors of increasing levels of sensation (see Figure 2). These scales are completed before and after the consumption of a test food, and then at regular time intervals for a few hours or until the start of the next meal. Typically, many aspects of post-ingestive sensations are rated: hunger, fullness, satiety, desire to eat, and prospective consumption. They can be analysed separately or a composite index can be computed.

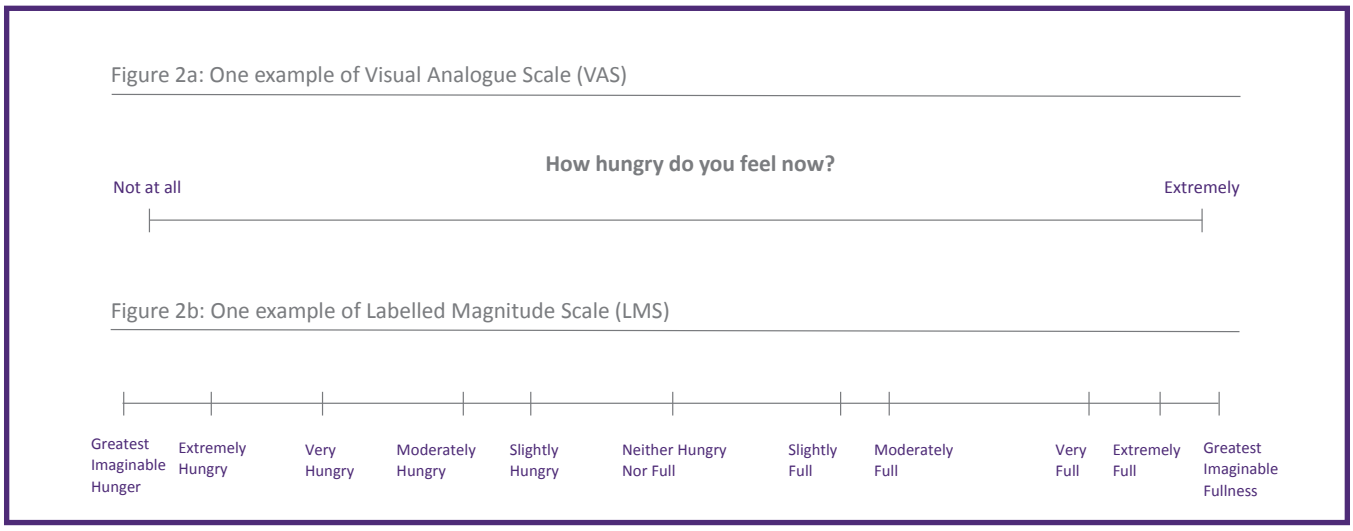


Figure 2: One Visual Analogue Scale and one Labelled Magnitude Satiety Scale
Source: Adapted from *Solah et al., 2015*

Measuring satiety as the spontaneous duration of the post-meal interval or by using the preload paradigm brings complementary information. While satiety effects are thought to be exhausted at the end of the satiety cascade when hunger returns and the next meal is spontaneously initiated, the preload paradigm informs about satiety when both the size of a meal and the following interval are fixed: effects on later eating events are still expected to occur. The latter approach has ethological validity since in human societies eating times are determined by social constraints. Future research in satiety might be directed to other potentially interesting aspects: for example a "satiety threshold" might be identified following one episode of intake, showing what differences in satiety intensity of duration might lower or increase the threshold for initiating the next eating event under particular free-living or laboratory circumstances (*Chapelot, 2013*).

Ratings of present sensations of hunger/satiety can be obtained in any untrained individual with VAS or LMS. In certain cases, however, ratings must discriminate subtle changes, for example, when one wants to compare the satiating power of two relatively similar foods or ingredients. In such cases, consumers can be trained to use rating scales in order to improve their sensitivity and reliability. In a procedure similar to the training of sensory evaluation panellists, participants learn to use the scales descriptors with maximum precision and to pay attention to relevant internal physiological cues. They can then produce highly sensitive satiety evaluations, for example between foods supplemented with various dietary fibres (*Lesdéma et al., 2016; Solah et al., 2015*).
Over time, consumers gradually learn to estimate the satiating efficiency of familiar foods as a result of their repeated experience of sensory, cognitive and/or physiological satiety effects. Because of this, it is recommended that the satiety value of a particular substance be tested several times, particularly after the consumer has had repeated experiences of ingestion (*Yeomans et al., 2014*).

Nutrients and satiety

The consumption of any food or beverage, even pure water, induces some level of satiety. The intensity and/or duration of satiety vary, however, according to the nutritional content of the food or beverage. The total energy content is a critical factor directly affecting satiety (for example *Yeomans and Chambers, 2011*).

Then, for a given energy content, satiety varies according to macronutrient composition, the presence of non-nutrients such as fibre, and some other bioactive food constituents (*Tremblay and Bellisle, 2015*).

Macronutrients differ in their satiety power. For an identical energy load, a nutrient-specific hierarchy of satiating power exists, with protein showing the highest potency, and carbohydrates (CHO) being more satiating than dietary fats (*Veldhorst et al., 2008*). A number of mechanisms could contribute to the high satiating value of protein, including increased Diet Induced Thermogenesis, the effects of circulating amino acids, and the release of satiety-related intestinal signals such as GLP-1 and PYY. The rise in glycemia that follows the intake of CHO is considered to contribute to satiety. CHO foods that induce a sharp high peak in post-ingestive glycemia, however, might be less satiating than foods with a lower Glycemic Index (GI) that produce a more moderate but sustained elevation in glycemia in the hours following consumption (*Poppitt, 2013*). In addition to available CHO, the presence of fibre (soluble and insoluble) in foods and drinks promotes satiety. Dietary fibres can act in the stomach or the gut, where they affect fullness or transit responses.

Dietary fats bring more energy per gram than protein or CHO (9 kcal versus 4) but produce paradoxically less satiety. The relative "satiety deficit" of dietary fats could be accounted for by their high energy density so that a high energy load can be ingested in a small volume of food, which could favour overconsumption (*Rolls, 2000*). Hormonal factors also seem to contribute: for example the appetite-stimulating hormone ghrelin falls to lower levels following intake of a CHO-rich meal than after consumption of a similar fat-rich meal (*Gibbons et al., 2013*). Figure 3 shows changes in hunger ratings in parallel with ghrelin status in the hours that follow the intake of high fat versus high CHO test meals.

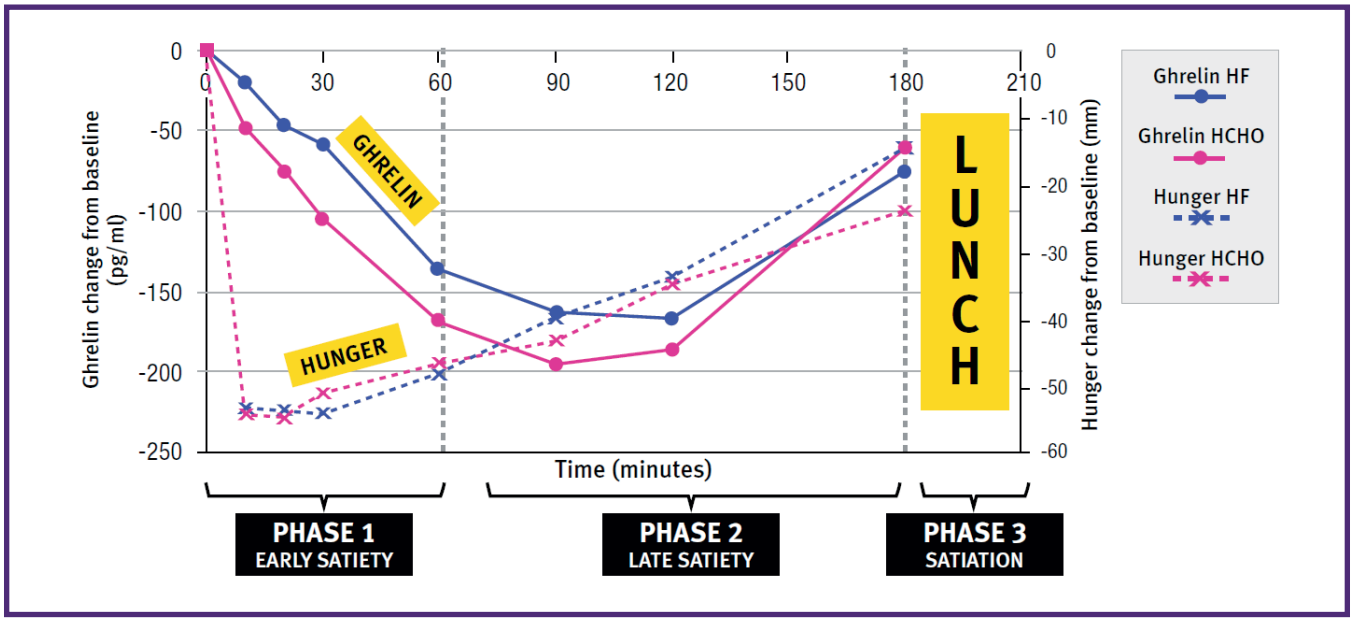


Figure 3: Ghrelin and hunger fluctuations following high fat (HF) or high CHO (HCHO) test meals
Source: Adapted from *Gibbons et al., 2013*

Micro-nutrients such as calcium and vitamins could play a modulatory role in satiety. Calcium supplementation in obese consumers significantly increases their plasma concentration of the satiety-related hormone PYY (*Jones et al., 2013*). Multi-vitamin supplements reduce hunger sensations in obese women on a weight loss program (*Major et al., 2008*). Non-nutritive constituents of foods and beverages, such as caffeine and capsaicin, also enhance satiety via activation of the systematic Nervous System (*Tremblay et al., 1988; Westerterp-Plantenga et al., 2005*).

Individuals are not equal in their responses to nutrient-related satiety. The Satiety Cascade acknowledges the importance of the body fat-free mass which directly determines the person's metabolic rate. The metabolic rate in turns generates a biologically based drive to eat which modulates all aspects of appetite, including hunger, satiation, and satiety (*Blundell et al., 2012; Blundell et al., 2015*).

Sensory factors and satiety

The sensory experience of ingestion is an important contributor to satiety effects. Preloads studies have shown that the same load of energy or nutrient is more satiating when actually ingested by an individual than when directly infused into the stomach or the intestine (*Cecil et al., 1998*). The critical aspects of the sensory experience of consumption that modulate satiety have been extensively studied.

While palatability directly affects intake at one eating occasion, and therefore critically determines satiation, its influence on satiety is unclear: hunger/fullness sensations and the size of the next eating event does not seem to be predictably affected by the palatability of the previously consumed foods (*De Graaf et al., 1999; Sørensen et al., 2003*).
Sensory Specific Satiety (SSS) refers to the decline in pleasantness that follows consumption of a substance (*Hetherington, 2013*). Many sensory properties of the ingested food undergo SSS: its taste and smell, but also its texture, its colour, and even its shape (*Sørensen et al., 2003*). SSS is related to the sensory stimulation rather than to the energy content and post-absorptive effects. The pleasantness of foods sharing sensory characteristics with the ingested food also declines while that of substances with different sensory characteristics remains unchanged or even increases. This is why dessert is still appealing at the end of a savoury meal. SSS is apparent immediately after intake and then tends to decrease with time but it can be detected over several months.

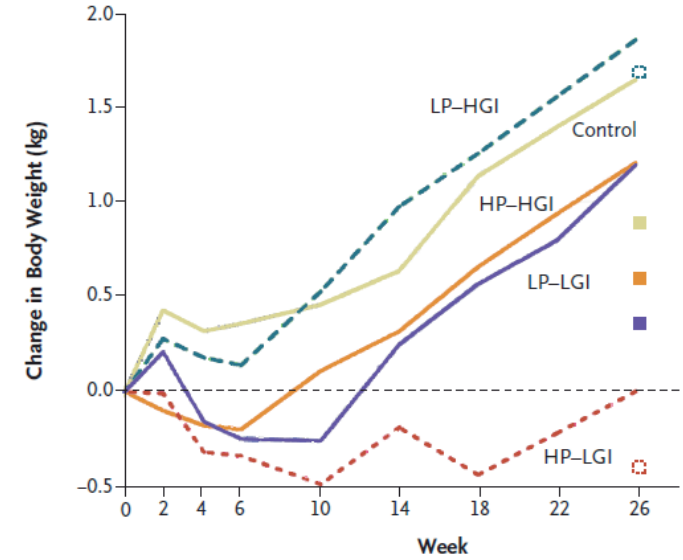
One important aspect of the sensory experience associated with consumption is the liquid or solid phase of the ingested substance. It is often reported that beverages induce less satiety than solid foods (*Mattes, 2006; Mattes et al., 2011*; with a few exceptions for example *Almiron-Roig and Drewnowski, 2003; Gadah et al., 2016)*. Lower energy compensation for a beverage than for a solid food is generally observed at the next eating occasion, although many factors modulate the extent of energy compensation (*Almiron-Roig et al., 2013*). The difference in satiating value could be explained, at least partly, by the shorter time of oral processing (sensory influence) and the quicker gastric emptying (gastric influence) of liquid versus solid substances.

Beliefs and expectations are key factors affecting the experience of satiety. The anticipated satiating effect of a food is linked to previous experience. Sensory aspects such as viscosity, creaminess, and thickness, which are usually linked with high calorie content in foods, are learned satiety cues that enhance expectations of satiety (*McCrikerd et al., 2015*). In practice, making a high-energy or high-protein beverage taste thicker and creamier enhances its inhibitory effects on intake at the following test meal (*Bertenshaw et al., 2013; Yeomans and Chambers, 2011*).
Recent scientific literature distinguishes between two aspects of the reward value of foods: "liking" and "wanting" (*Finlayson et al., 2007*). Liking is the hedonic evaluation (pleasantness) of tasting a particular substance whereas wanting refers to the desire to actually ingest a particular substance. Liking affects wanting but wanting is also directly influenced by physiological/psychological state and by environmental factors.

Health implications

A diet including many highly satiating foods may have the capacity to limit intake and facilitate body weight management. However, the potential impact of modulating satiety on body weight management on the long term is a complex issue. Satiety is one of many factors influencing energy intake, which in turn is one of the numerous factors influencing body weight changes.

Although there are numerous demonstrations that enhancing satiety can reduce food intake in the short term, the hopes that satiety enhancement per se could produce significant weight loss have not up to now been supported by convincing scientific evidence. Evidence exists, however, that a diet rich in high-satiety foods can facilitate the maintenance of weight loss. The DIOGENES project followed 773 obese Europeans from eight countries for 26 weeks after a diet-induced weight loss. Five maintenance diet conditions were compared. The weight loss was best maintained on a diet combining increased protein content and low GI (*Larsen et al., 2010*) (see Figure 4).



No.								
LP–LGI	150	116	121	118	112	104	101	97	106
LP–HGI	155	118	114	118	108	104	95	91	97
HP–LGI	159	132	136	131	125	116	118	114	124
HP–HGI	155	130	124	121	118	114	100	104	107
Control	154	126	131	125	131	125	118	110	114

Figure 4: Weight loss maintenance over 26 weeks is facilitated by a diet enriched in high-satiety foods. At the end of follow-up, a diet with higher protein content and lower glycemic index significantly improved weight loss maintenance
LP: low protein; HP: high protein; LGI: low GI; HGI: high GI
Source: *Larsen et al., 2010*

Beside direct effects on body weight control, enhanced satiety can also exert indirect benefits. Psychological and behavioural benefits include improved hunger management, reduced opportunistic eating in dieters, reduced hunger dysphoria (a state of feeling unwell or unhappy), and improved compliance with a weight management program (*Hetherington, 2013*).

While several food-associated factors affect satiety, responsiveness to satiety cues also varies largely between individual consumers. A "low-satiety phenotype" has been identified in a proportion of obese but also normal-weight individuals, characterised by weak changes of appetite sensations following standardised meals (*Drapeau et al., 2013*). Persons with a low-satiety phenotype exhibit a higher Resting

Metabolic Rate, higher levels of Disinhibition as assessed by the Three Factor Eating Questionnaire, a lower control over food cravings, and a higher "wanting" for high-fat foods than individuals with a "high satiety phenotype" who show a high sensitivity to post-ingestive satiety cues (*Dalton et al., 2015*). The low satiety phenotype can be seen as a marker of increased risk of overeating and weight gain over the long term. The causes of individual differences in satiety responsiveness are not known, although anxiety/stress could act as modulating factors.

Do consumers understand satiety and can they use high satiety foods for their own benefits? Consumer research has shown that people identify satiety effects from the perception of physical cues such as gastric fullness and/or from psychological states such as hunger satisfaction or lack of desire to eat (*Murray and Vickers, 2009*). Can consumers make use of the science presently available to optimise their diet in terms of satiety value and intake control? This could be useful particularly in a post weight loss period when adequate food selection could support weight maintenance. In order for this to happen, knowledge and understanding of scientific results should be improved among consumers (*Stubbs, 2013*) with the help of informed health professionals.

Perspectives

Many novel perspectives on satiety and its mechanisms have opened in recent years. Both the brain and the periphery of the organism are now seen in a new light.

The composition of the gut microbiota differs between obese and lean individuals (*Delzenne et al., 2013*). Recent research has explored how nutrients can affect the gut microbiota and how in turns it affects satiety responses and body weight. In animal studies, high-fat diets induce alterations in the composition of the gut microbiota that favour the development of obesity (*Neyrinck et al., 2011*). Conversely prebiotics, a class of nondigestible/fermentable CHO that increase gut bifidobacteria, decrease food intake in animal models of obesity. In human consumers, evening meals rich in dietary fibres with prebiotic properties reduce appetite on the following day in parallel with an increase in the satiogenic peptide GLP-1 (*Johansson et al., 2013*). Administration of prebiotics supplements to men and women increases satiety sensations and reduces energy intake (*Cani et al., 2006*), stimulates the release of GLP-1 and PYY (*Parnell and Reimer, 2009; Verhoef et al., 2011*) and, in obese individuals, decreases body weight, waist circumference, and fat mass (*Parnell and Reimer, 2009; Genta et al., 2009*). The microbiota-gut-brain axis appears a very promising area of research in the perspective of understanding and possibly enhancing satiety.

Recent years have seen the development of neuroimaging techniques, such as functional Magnetic Resonance Imaging (fMRI) and Positron Emission Tomography. These powerful and non-invasive tools are used to study the activity of the brain, including the brain processes associated with appetite and satiety. A first observation is that the basic processes of food intake control such as hunger, appetite, cravings, fullness, and satiety are organised in a broad neural network involving most parts of the brain (*Berthoud, 2002; Berthoud, 2012*). During the satiety period, nutrient sensing mechanisms, neural or hormonal, send information to the brain about the presence of nutrients in the gastro-intestinal tract. A brain "homeostatic regulator" integrates these signals with others that reflect energy needs in order to maintain the body energy balance. The brain processes involved in Sensory Specific Satiety have been explored using fMRI: eating a test food to satiety induces significant response decrements in the left dorsomedial amygdala and significant differential activity in multiple areas of the orbitofrontal cortex (*Gottfried et al., 2003*). One interesting development is the identification of changes that can occur in the "hungry brain" of obese patients following attempts to lose weight. Neural activity was examined

in obese individuals before and after the loss of 10% of their initial body weight. Figure 5 (*Rosenbaum et al., 2008*) shows exaggerated activity induced in many brain areas by pictures of foods after weight loss. Most of these exaggerated responses were normalised by the administration of the hormone leptin which is considered a long-term satiety factor.

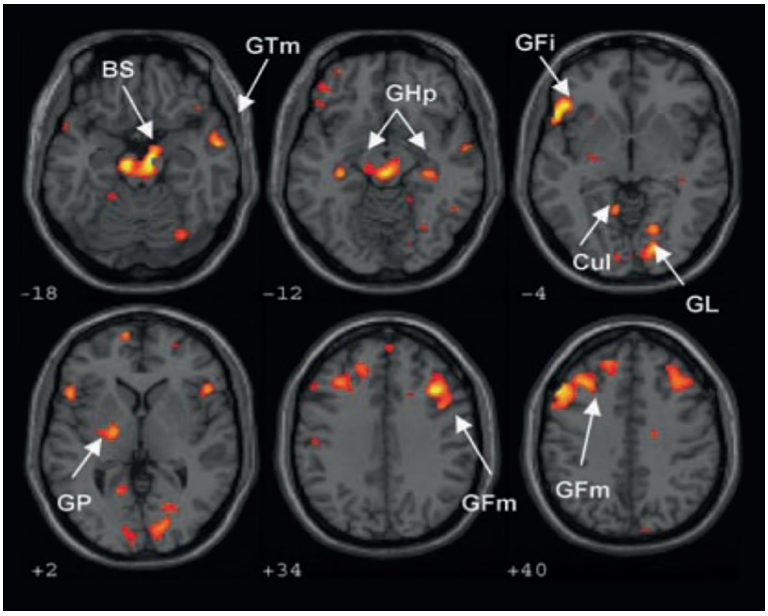


Figure 5: Exaggerated activity of neural brain structures following a 10% weight loss in obese subjects
BS: brainstem; Cul: culmen; GF: fusiform gyrus; GP: globus pallidus; GTm: middle temporal gyrus; GHp: parahippocampal gyrus.
Source: *Rosenbaum et al., 2008*

Abbreviations

CCK: Cholecystokinin

CHO: Carbohydrate

fMRI: functional Magnetic Resonance Imaging

GI: Glycemic Index

GLP-1: Glucagon-Like Peptide-1

LMS: Labelled Magnitude Scale

PYY: Peptide YY

SSS: Sensory Specific Satiety

VAS: Visual Analogue Scale

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