Meaning And Assessment Of Satiety In Childhood

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Introduction

Subjective experience of appetite and learnt preferences for foods are important determinants of food choice and intake in children impacting upon what is eaten, how much and when. All of these drive consumption and potentially lead to excessive energy intake. While the operation of appetite can be considered asymmetrical, in that it defends against energy deficit rather than energy excess, it underpins the architecture of meal patterns which characterise human eating behaviour. So while the food environment has the power to overwhelm normal appetite regulation there are endogenous appetite mechanisms that can be boosted or deliberately exploited to restore energy balance. The biological mechanisms underpinning acute appetite regulation are common to both adults and children; however, their operation and effectiveness are likely to change with age and can become comprised by weight gain. Thus understanding the development of appetite in children has the potential to allow us to understand pathways to excessive weight gain through life but also to identify points in childhood where we can potentially intervene to prevent excessive weight gain. However, to understand this it is necessary to define appetite, its core components (both stimulatory and inhibitory) and the mechanisms that underlie each of these. This chapter will focus on appetite expression in children and the way in which it can be measured.

Appetite and the satiety cascade

The selection of specific food and drinks, the motivation to consume, individual preferences and specific cravings and overall calorie intake, in children and adults, are all subsumed by the term appetite. Appetite is regulated by two opposing feelings: hunger, denoting the drive to eat caused by homeostatic need for energy or non-homeostatic environmental influences that trigger and sustain eating behaviour, and satiety, the process that takes place during and after a meal has been consumed leading to the decline of hunger and the inhibition of further intake. Satiety can be divided into two types: intra-meal satiety or satiation which is the process that happens during consumption and leads to meal termination thus controlling meal size, and inter-meal or post-ingestive satiety which denotes the feeling of fullness after a meal has been finished and inhibits further consumption (1).

The strength and duration of post-meal satiety and thus the initial onset of the next meal are controlled by multiple factors like the physicochemical properties of food including energy density, weight, volume, macronutrient composition, bulk, particle size and solidity, and its sensory characteristics like palatability, taste, odour and appearance. Thus after food has been consumed, the way nutrients are absorbed, utilised and stored, can be a powerful determinant of further eating behaviour. The interactions of all these factors are described in the "satiety cascade" which was first introduced by Blundell et al. in 1982 (2) a schematic representation of which is presented in Figure 1 (3).

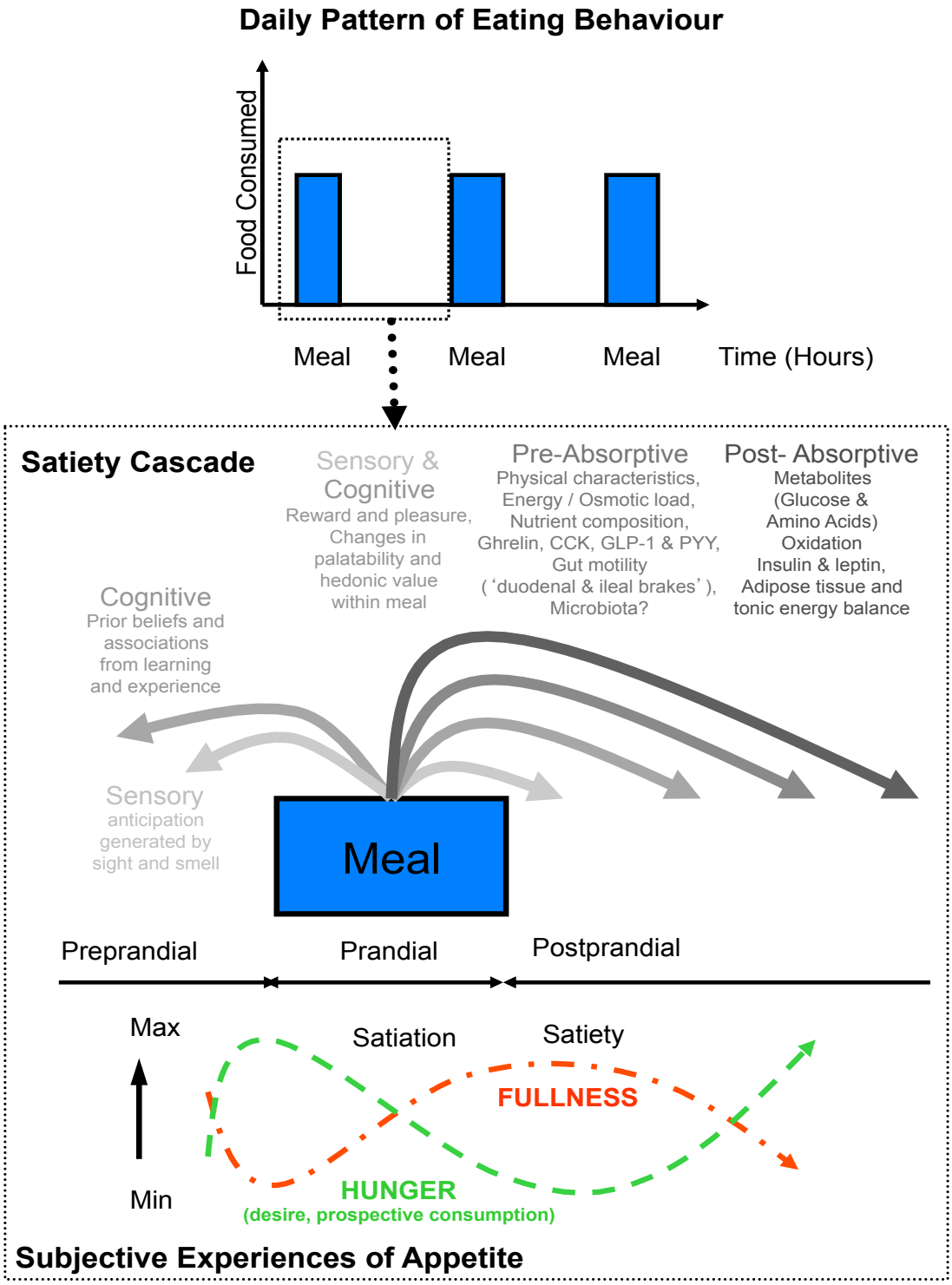

Figure 1. Satiety cascade. *CCK, cholecystokinin; GLP-1, glucagon-like peptide-1; PYY, peptide YY. Source: (3)*

As represented within the satiety cascade, neuronal and hormonal signals released from the gastrointestinal (GI) tract during digestion, lead to a reduction in hunger and this along with other signal triggers generates satiation within a meal. Pre-absorptive factors such as the impact of the volume of food on stomach size (stretching) and the rate of delivery of nutrients into the proximal small intestine are critical to satiation. The release of hormones triggered by the detection of nutrients within the GI tract regulate the passage of food through the tract and this, along with their role as direct signals, has an important impact on subject experience of appetite. Directly or indirectly, they exert their influence on the CNS. These homeostatic mechanisms interact with and influence the operation of reward system within the CNS, and themselves influence sensory and cognitive factors that promotion food consumption. Thus satiation can weaken the drive to consume but conversely the hedonic experience of food can override signals of satiation leading to eating in the in the absence of (homeostatic) hunger. The homeostatic systems underpinning appetite remain effectively the same during lifetime; however, as various parts of the brain are still developing throughout childhood and adolescence, its function and influence of appetite regulation might differ as children develop into adults. This will depend in part on the foods children are exposed to, and whether hedonic factors systematically out influence homeostatic factors in determining intake.

Central control of appetite

The satiety cascade demonstrates the variety of signals generated by the consumption, digestion and absorption of food and the subsequent metabolism of the nutrients this yields. Such factors indicate the fulfilment of biological need. However, to inhibit eating behaviour these signals must be integrated to collectively exert influence on the motivational systems within the brain dampening the drive to consume. The hypothalamus, others areas in the limbic system, the brainstem, the amygdala and the cerebral cortex, all appear critical in the regulation of appetite. Peripheral satiety signals interact with neuropeptides and neurotransmitters. Moreover, prominent anabolic effector pathways such as pro-opio melanocortin / cocaine- and amphetamine regulated transcript (POMC/CART) are stimulated by leptin and insulin. This in turn down regulates energy intake. Prominent catabolic effectors such as Neuropeptide Y / Agouti-related protein (NPY/AGRP) are suppressed by leptin and insulin and upregulate food intake. Other neurotransmitters within the CNS can act to stimulate or inhibit eating behaviour including classic monoamine neurotransmitters such as noradrenaline, dopamine and serotonin (4, 5). In addition, certain substances, most notably glucose and certain amino-acids, have the ability to cross the blood-brain barrier to be directly utilised by the brain and effectively alter key parts of the appetite regulating regions.

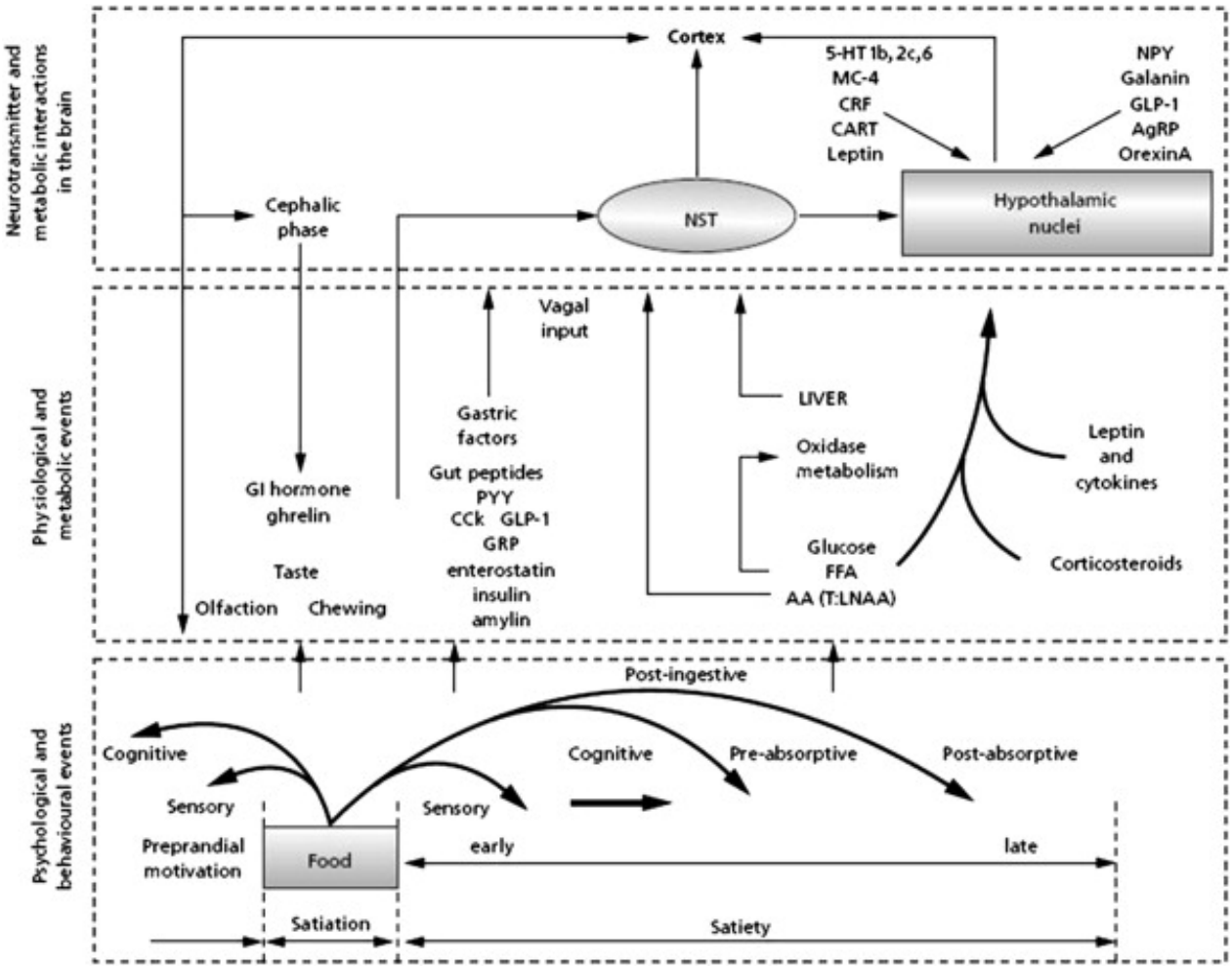

Figure 2. Action of peripheral signals on the CNS in parallel with the satiety cascade. *5-HT, serotonin; AA, amino acids; AgRP, agouti-related peptide; CART, cocaine and amphetamine-regulated transcript; CCK, cholecystokinin; CRF,corticotrophin releasing factor; FFA, free fatty acids; GI, gastrointestinal; GLP-1, glucagon-like peptide-1; GRP, gastric releasing peptide; MC, melanocortin; NPY, neuropeptide Y; NST, nucleus tractus solitarius; PYY, peptide YY; T:LNAA, tryptophan large neutral amino acid ratio. From: (5)*

Cephalic phase response (CPR) and sensory specific satiety (SSS)

Cephalic Phase Response encapsulates a number of innate responses to eating including changes in temperature, heart rate, gastric activity and saliva. CPR appears critical to both meal initiation and termination and thus it influences meal size (6). Salivary flow decreases when the same food is presented during a meal which is part of a process called habituation (7). In children the habituation process can be disrupted by other foods that differ in sensory characteristics or by activities other than eating that

demand attention and this disruption can sometimes lead to overconsumption and increased energy intake (8).

Sensory-specific satiety (SSS) characterises declining sensory feelings generate by a specific food eaten during a meal while hunger for other types of food remains unchanged or may even be enhanced (9). SSS begins in the early pre-absorptive stages, does not rely on gastric signalling (10) and can influence intake (11). Presenting children with a variety of foods that differ in their sensory and nutritional characteristics can lead to a disruption in habituation and positive orosensory feedback which enhances pleasure and thus can increase energy intake (12). For children, SSS appears to be bound to specific foods, in contrast to adults who can transfer their sensory-specific satiety to other foods that share similar sensory characteristics (13).

Homeostatic mechanisms for appetite control

Episodic signals: The gastrointestinal tract has mechanical and functional properties that affect appetite. Mechanical factors such as gastric distension that creates a feeling of fullness and gastric emptying (the rate at which food leaves the stomach entering the duodenum) contribute to meal termination and postprandial satiation modulating satiety in the short-term (14). Changes in these mechanical factors as well the presence of nutrients and pH changes lead to the release of gut peptides (15) some of which have been found to have an effect on appetite. However, there can be various factors that determine children's gut hormone responses (16). Ghrelin is the only one of the gut peptides which has been found to have an orexigenic effect. It can reach the hypothalamus either directly or indirectly and it can also be synthesized there in small amounts, thus affecting the NPY/AgRP neurons. It is synthesized in the stomach by endocrine cells but it is also found in smaller quantities in the large intestine. Ghrelin in children has been found to be associated with BMI irrespective of feeding status (17) and obese children have demonstrated lower ghrelin levels than lean children while their ghrelin levels improved after weight loss (18).

A number of hormones secreted from the small intestine have a powerful effect on appetite. CCK is released in response to nutrients, mainly fat and protein, and it facilitates digestion but also has a satiating effect by limiting meal size and duration by acting on the CNS through the vagus nerves. CCK regulates appetite in the short-term but ongoing research is taking place about its long-term effects making CCK a promising target for more effective appetite control (19) but there's not enough research about it in children. Glucagon Like Peptide (GLP)-1 is co-secreted by the intestine together with PYY and it can also be produced in the brain. It can cause insulin secretion the same way glucose does. GLP-1 levels fall in the fasted state and rise with meal anticipation, during the meal and post-prandially. Peptide YY (PYY) is found almost exclusively in the large intestine and has an anorexigenic effect. PYY levels are low pre-prandially and high post-prandially and are influenced by the caloric content and macronutrient profile of the food, with fat possibly having a greater effect (20).

Tonic signals: In addition to the gut the adipose tissue and pancreas release hormones/cytokines associated with appetite regulation including insulin, leptin and adiponectin (adipokines) (19). Leptin is secreted by the adipose tissue and reaches the hypothalamus through the bloodstream or through the vagal nerve (21). In humans with congenital leptin deficiency, recombinant leptin administration does indeed reduce hyperphagia and fat mass. However, the vast majority of the obese appear to be able to produce leptin although they appear relatively insensitive to its effects. Leptin resistant is characterised by blunted

anorexigenic effects (20). Endogenous leptin levels are elevated in obese children with levels dropping after weight loss with both features being especially pronounced in girls (22, 23). Adiponectin, in contrast to leptin , has been found to have an orexigenic effect and it is theorised that the adiponectin coding gene has been acting as a "starvation gene" in the course of evolution by promoting fat storage (24). Adiponectin has been found to be inversely correlated with children's adiposity (25) and adiponectin levels in obese children have been shown to rise after weight loss interventions (26).

Hedonic Factors: The hedonic characteristics of food promote consumption and have the potential to override homeostasis. The reward system (27) in the brain includes key pathways such as endocannabinoids, opioids and dopamine pathways that interact with key homeostatic system (28). The developing reward system in childhood and adolescence is relatively hyper-active which leads towards greater reward-seeking and that could influence children's and adolescent's food choices (29). Palatable foods seem to transmit information to the reward system that upregulate reward mediators which connect with neurones that control appetite in the hypothalamus. These, in turn, increase the expression of orexigenic peptides like NPY and orexins and blunt anorexigenic peptides like insulin, leptin and CCK (30).

Ingredients and Appetite Control

The effect of energy on satiety is not proportional and it depends on the macronutrient profile and the reward capacity of the food. Protein is considered the most satiating of the macronutrients. High-protein diets in adult studies have been shown to significantly increase subjective appetite ratings and it is possible that they can reduce energy intake (31-33) however its effects in children are unclear (34, 35). Dietary fat, while being the most energy dense, is the macronutrient with the least satiating capacity (36) and high-fat foods can lead to overconsumption in children as they are also usually highly palatable (37, 38). Carbohydrates, seem to be somewhere between fat and protein on their effects in satiety. However, the effects of sugar are often compared to dietary fat due to its effect of increasing energy intake possibly because of their sweetness (39). Sugar-sweetened beverages with their high sugar content have been tightly linked to higher weight status in children of all ages and are widely considered to promote childhood obesity (40-42) suggesting that they provide excess energy with low satiating value. Non-caloric substitutes have been suggested as a healthier choice but there is not enough evidence about their effect on satiety in the short or long term in children. **Dietary fibre is a form of complex carbohydrate found in fruits, vegetables, legumes and whole grains.** Besides its other health benefits, dietary fibre is thought to reduce energy intake, body weight and subjective appetite ratings and it is thought as the most potent type of ingredient for enhancing satiety, however more research is needed to establish its effects, especially in children (43, 44).

Assessment of satiety

There are a number of means of assessing satiety, primarily through measuring the effect of a food on subjective feelings of appetite, and by qualifying its effects upon the amount consumed whilst consuming the test food itself, or on subsequent eating occasions. In addition to these other indices of the motivation

to consume can be measured through cognitive tests, eye tracking and or neurological techniques such as EEG and functional imaging (fMRI). Moreover, the distinct peripheral mechanisms underlying satiation and satiety provide key biomarkers of appetitive effects. Finally, in studies of long term consumption psychometric tools offer the possibility of examining the impact of satiety on perceptions of control, cravings and mood.

Although a well-developed methodological platform exists compromise is require in study design. Researchers must decide if they wish to examine appetite in free-living or laboratory conditions. Studies in free-living environments have high external validity but low internal validity because they are prone to errors and bias due to under-reporting, over-reporting or mis-reporting. Laboratory studies have high internal validity when they are carried out carefully, however it cannot be said that they clearly mirror real-life situations. A solution is often to carry out overlapping protocols in a variety of situations in order to examine the hypotheses at hand from all possible angles (45). Whilst laboratory studies possess the greatest opportunity for experiment control, and therefore the isolation of distinct mechanism under pinning appetite, children are a particular difficult group to assess in these paradigms. Participants usually need to be followed for several hours during the course of a day, with measurements taken at precise and regular interventions. In free-living conditions participants are required to self-report most of the information which can be a problem when children are not able to understand how to report them.

Preload study design

The preload study design is a standardised approach to study short-term appetite regulation. The preload provides the nutritional or sensory manipulation to challenge appetite control. It is best conducted with a within subjects design; however this is not always possible when eating is involved as manipulations in food are not always easy to disguise and repeated measure can prove demanding for participants, particularly for children. The preload is generally, a meal, a snack or even part of a meal, which has been designed to match a control meal in all its aspects, including appearance and taste, except for the one thing the aim of the study wants to examine. After the preload has been consumed in its entirety the participants' either self-report their own intake or, for better accuracy, receive a weighed test meal, after a certain amount of time, so energy intake can be calculated. Allowing the participants to decide when they want their next meal allows them to better express their appetite outside the constraints of limited food options. Providing a range of food items allows the experiment to examine food choice and macronutrient intake, but too much variety may encourage overconsumption (see next section).

Subjective ratings are usually completed by the participants before and after the meals. For children, it can be a challenge to finish a meal they don't like or are unfamiliar with, thus the ideal preload meal consists of as few items as possible, to reduce the risk of rejection of items, and where possible the inclusion of items children are familiar with and have accepted to eat in the past. The main challenge of preload designs is the lack of standardization of the aspects of a preload meal, resulting in results that cannot be compared because various studies have used different kinds of preloads.

Ad libitum meal

The ad libitum test meal can either be a buffet-style meal or a single meal and is served in ad-libitum quantity. These are generally presented in a quantity much beyond what is normally expected to be consumed in order to make sure satiety is reached before meal termination. The test meal is weighed before and after consumption to measure the amount eaten by the child participants as accurately as possible. In the buffet-style meal a variety of items, which should differ in energy density and taste characteristics, is offered, which allows examination of the effect of the preload on food choice. The results gleaned from the use of buffet meals might not be that representative if they differ dramatically from the participants' standard lunch or evening meal. The single course meal on the other hand aims at the assessment of energy intake and short-term energy regulation rather than food choice; however, the effects of sensory specific satiety might lead to energy under-consumption.

Subjective appetite ratings

Subjective appetite ratings are a range of measures usually in the form of a questionnaire which are completed by the participants themselves and examine the state of hunger or satiety, prospective consumption, motivation to eat and other questions about the meal or the state of well-being of the participants, and are completed before and after the meal. The participants note their answers on a scale which should be age-appropriate and their answers should in theory reflect the results gained by measuring the test meal. The most widely accepted scale for adults are Visual Analogue Scales (VAS) where the participant marks a straight line of specific length to indicate their answer. They are regarded as a reliable and valid means of assessment (46, 47) which predict eating behaviour in the normal environment and in the controlled conditions of the laboratory. Adolescents, may also be able to complete VAS scales. However, for children it is more appropriate to use 5-point likert scales, typically of pictorial format (Figure 3). Whilst these are widely used in psychometric analyses for children, few validated measures currently exist that allow children to accurately and reliably rate their appetite.

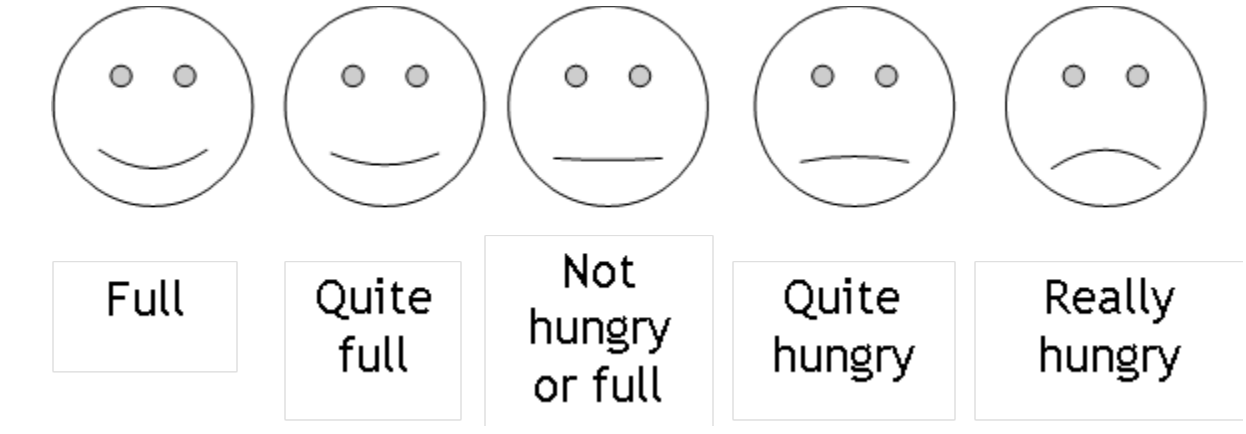

Figure 3. *Children's 5-point likert scales for subjective appetite ratings.*

Biomarkers

Plasma concentrations of glucose, leptin and ghrelin are often used as biomarkers of satiety and satiation in adult studies (48). However, their use in children's studies is limited due to the ethical and practical issues that arise from drawing multiple blood samples from children.

Measurement of Satiety in Children

Whilst the development of eating behaviour in children is a matter of considerable scientific investigation, the underlying mechanisms influencing meal by meal episodic appetite regulation and their influence on appetite expression are poorly understood. The assumption remains, as these mechanisms are largely physiological, that they operate in generally a similar manner to those in adults. This assumption would underpin any satiety-based approach to weight management targeted at children. Limited data suggest that nutritional manipulations which increase the bulk and energy content of foods increase their satiating potential. Conversely, low volume energy density foods appear to possess less satiating potential. This mirrors findings in adults. Yet, measures of satiety in children are not sufficiently developed and as a consequence appetite-based weight status differences are infrequently reported. This primarily reflects temporal profiles, with obesity not being manifest until the behavioural changes that underlie weight gain occur. Additionally, the level of obesity per se is much lower in children reflecting the shorter time scale for weight gain to develop.

One potential resolution to this challenge is to examine satiety in children from families with two obese parents, where the children are likely to be predisposed to weight gain. However, it remains logistically difficult to accurately measure intake and appetitive behaviour in children. Classically such measures are rarely achieved in a laboratory settings but the need to provide food is an integral component. It is technically easier to measure responses to food cues in child participants. Whilst such approaches focus investigation on hedonic rather than homeostatic regulatory mechanisms the potential remains for manipulations that enhance satiety to modulate the hedonic systems and reconnect homeostatic control. Interestingly, weight status differences may emerge in studies examining food cue responses both pre-and post-consumption. In a fasted state all participants would be expected to demonstrate a response to cues. However, in the sated state obese individuals, or those predisposed to obesity, may demonstrated an inappropriate response reflecting disconnect between hedonic and homeostatic control mechanisms. Studies incorporating both feeding states will therefore provide a more complete overview of regulatory control.

An alternative approach is to undertake observational measurement of eating behaviour. Children, typically being less self-conscious than adults, are less likely to modify their behaviour in response to observation. The Child Eating Behaviour Questionnaire (CEBQ) is a parent-completed tool which assesses eight dimensions of eating behaviour including responsiveness to food, satiety responsiveness

and enjoyment of food and has been validated against measures of food intake (49, 50). Strong associations between CEBQ scores and development of obesity have been demonstrated in a number of European studies. The CEBQ may therefore prove a useful means of examining precursors of obesity in children for example satiety responsiveness, or the extent to which an individual responds to fullness by stopping eating or failing to initiate consumption, which is hypothesised to be low in obese individuals or those predisposed to obesity, leading to a failure in regulation of intake and consequently overconsumption.

Conclusion

Whilst **biological appetite control mechanisms are common to both adults and children, their operation and effectiveness are likely to change with age.** To gain an understanding of these control processes a strong scientific methodology has been developed that proves reliable and valid across different testing environments and research teams. However application of these methodological approaches and tools across a varying age and developmental profile **can prove a challenge.**

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~ About the Authors ~

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Vassiliki Sinopoulou is a PhD student in the Appetite and Obesity research group in the Department of Psychological Sciences, University of Liverpool. She is studying the effects of dietary fibre on satiety in children. She carried out her undergraduate studies in Nutrition and Dietetics at Harokopio University of Athens and gained an MSc specializing in Molecular Nutrition from Wageningen University in the Netherlands

Joanne Harrold

Dr Joanne Harrold is a Senior Lecturer in Appetite and Obesity in the Department of Psychological Sciences, University of Liverpool. She is a behavioural neuroendocrinologist with an interest in the processes involved in food intake and energy balance. Her research covers a broad spectrum of the control of the appetite system from preclinical models through to man. Her early research characterised the homeostatic and hedonic control systems within the brain before progressing to examine pharmacological, dietary and psychological manipulations of human eating behaviour. She has carried out a number of weight management interventions. Recently, her research has focused on early eating experiences and the development of dietary behaviour. She is Director of the Human Ingestive Behaviour Laboratory where she established an Infant feeding laboratory. Here studies examine the development of food preferences in children and in particular the impact of prenatal nutrition, maternal feeding style and parental feeding practices.

Jason Halford

Professor Jason Halford is Head of the Department of Psychological Sciences at the University of Liverpool, former Chair of the UK Association for the Study of Obesity – ASO (www.aso.org.uk), and Treasurer of the European Association of Obesity (EASO). He is a Chartered Health Psychologist. His early research focused on anti-obesity drugs and appetite and this has progressed to the behavioural assessment of potential anti-obesity drugs in early clinical development. Over the past 10 years his research has focused on drug-induced

weight gain, the effects of nutrients and fibre on appetite and hormone release, the effects of stress on eating behaviour, the effect of marketing of children, and on lean-obese differences in the expression of appetite. In 1999 he co-founded the Human Ingestive Behaviour Laboratory at Liverpool and in 2004 he also cofounded the Liverpool Obesity Research Network (LORN). Professor Halford is the co-ordinator of the 8 million Euro EU Framework Seven Satiety Innovation SATIN project (www.satin-satiety.eu) to develop novel foods for appetite control using novel processing technologies to alter food structure. He is also a leading scientist on the WRAP trial investigating the role of commercial weight management providers in primary care and the lead investigator on a new trial to examine the impact of artificial sweeteners on appetite in the context of active weight management.

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