Appetite-Related Eating Behaviours: An Overview of Assessment Methods, Determinants and Effects on Children's Weight

Ana Freitas^a Gabriela Albuquerque^b Cláudia Silva^{a, d} Andreia Oliveira^{a–c}

^aFaculty of Health Sciences, University Fernando Pessoa, Porto, Portugal; ^bEPIUnit-Institute of Public Health, University of Porto, Porto, Portugal; ^cFaculty of Medicine, University of Porto, Porto, Portugal; ^dFP-ENAS (UFP Unit of Research in Energy, Environment and Health), CEBIMED (Center of Studies in Biomedicine), Porto, Portugal

Keywords

Appetite · Epidemiology · Feeding behaviour · Obesity · Paediatrics

Abstract

Identifying the underlying child-eating behaviours that contribute to weight differences across growth has been a constant challenge. This report reviews the various literature approaches for assessing appetite regulation. In doing so, it attempts to understand how appetite control develops and determines the eating habits in early childhood, and its effects on children's weight status. The interaction between homeostatic and hedonic mechanisms largely explains the appetite regulation process. Homeostatic mechanisms are mediated by the biological need to maintain the body's energy reserves, increasing the motivation to eat. On the contrary, the hedonic mechanisms are mediated by food reward, increasing the craving for high-palatable foods and

triggering the release of dopamine and serotonin. There are many biological methods (plasma measurements of hormones, like leptin, ghrelin and insulin) and behavioural evaluation methods of appetite. The Children's Eating Behaviour Questionnaire is most commonly used, due to its adequate psychometric properties tested in several population settings. The development of eating behaviours begins in utero, and several determinants may contribute to a decrease in the ability to self-regulate dietary intake. Examples include genetic predisposition, the first taste experiences and the family environment, a key determinant in this process. Several eating behaviours contribute most to childhood obesity. Among them, are the external eating (eating by external stimuli, such as the mere presence of the food or its smell), food restriction (which may potentiate the uninhibited increased intake of the restricted foods) and emotional eating (intake due to emotional variations, especially negative feelings). These eating behaviours have been linked to childhood obesity.

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Introduction

Understanding the aetiology of children's eating behaviours can be of utmost relevance when designing interventions for tackling childhood obesity. Some studies have shown that eating behaviours seem to tracking from childhood into adulthood [1, 2]. Therefore, early intervention is essential to curb further increases in childhood obesity.

Eating behaviours are closely associated with appetite, and both are modulated by environmental and social factors, and by internal biological mechanisms [3]. Appetite is a complex concept. However, from a biological perspective, it can be defined as "the internal driving force for search, choice and ingestion of food" [3]. Alternatively, in a broader context, appetite represents a set of physiological processes coupled with psychological and environmental factors, which determine the daily eating pattern [4]. Eating behaviours are defined as "the attitudes and psychosocial factors related to the selection and decision of which foods to eat" [5].

The World Health Organisation defines obesity as "abnormal or excessive accumulation of body fat that presents a risk to health" [6]. The disease is associated with a high risk of developing medical, psychological, social and economic consequences, and an increased risk of premature death [7]. Several risk factors for obesity have been described. Besides some hereditary characteristics, the lifestyles, and particularly diet, are the main factors responsible for the accumulation of excess weight [8]. Although there is some research in the areas of eating behaviours and appetite, understanding which children's eating behaviours are associated with weight differences has been a constant challenge [5, 9, 10]. This literature review addresses how the appetite regulation process develops and determines the eating habits in early childhood, the most common methods of assessment of appetite regulation, and the impacts of appetite-related eating behaviour on health, particularly on the weight status.

Appetite Regulation

The process of appetite regulation is mainly explained through a relationship between homeostatic and hedonics mechanisms [11], which have distinct but not independent functions [12]. The homeostatic mechanisms are mediated by the biological need to maintain the body's energy depots [12], increasing the motivation for food intake [11]. Hedonic mechanisms are mediated by food

reward [12] and act in periods of "abundance," surpassing the homeostatic mechanisms, as they increase the desire to consume foods of high palatability [11]. The interaction between these mechanisms aims to achieve a balance between food intake based on need and food intake for pleasure [12].

Homeostatic Mechanisms of Appetite Regulation

In 1991, Blundell [13] proposed a psychobiological model, explaining the existence of 3 independent but related domains in the regulation of appetite. One domain represents psychophysiological processes (which includes, for example, the hunger, hedonic and satiation sensations), and their behaviours and consequences (e.g., meal and food choices, energy and nutritional intake). A second domain encompasses peripheral physiological and metabolic events (including absorption, use and storage of nutrients), and the third domain comprises neurotransmitters and metabolic interactions in the brain [12–14].

In the domain of psychophysiological processes, is included the "satiety cascade" phenomenon, a conceptual framework that combines the physiological events controlling appetite with the behavioural and psychological experiences associated with the eating process. It describes a series of physiological and behavioural events that occur between the stimulating of hunger sensation (pre-prandial) and the end-of-meal (post-prandial), the later determining the satiation sensation [13, 14]. According to Blundell, hunger is the motivation for the food demand and consumption, associated with the onset of the meal, and satiation is the culmination of a set of processes associated with the end of the meal, which includes the inhibition of behaviours and motivations associated with food intake [13, 14]. The physiological state at the end of a meal, when further eating is inhibited by "fullness," is termed satiety [13, 14].

In the pre-prandial phase, several external and internal signals prepare the body to receive food. For instance, external cues include the sight and smell of food. Conversely, internal signals, such as the rise of ghrelin [15, 16] and hypothalamus peptides' (neuropeptide Y, orexins, agouti-related peptide, melanin-concentrating hormone, endogenous opiates and dopamine) levels [16], also stimulate food intake [17]. In the prandial phase, contact in the mouth with food generates information transmitted to the central nervous system, which in turn signals hunger and promotes the food intake [12] – the domain of neurotransmitters and brain interactions. At this stage, the central nervous system also receives sen-

sory signals from the gut (e.g., from cholecystokinin, glucagon-like peptide-1 and peptide YY). These signals are received through mechanoreceptors that signal gastric distention caused by the presence of food (giving a sense of the amount of food ingested), and chemoreceptors that detect the presence of nutrients (providing information on the nutritional composition of foods ingested). In the peripheral circulation, the detection of nutrients absorbed from the gastrointestinal tract generates prandial and post-prandial signals (rapid decrease of ghrelin and rise of insulin, glucose and amino acid concentrations in the blood and oxidation of nutrients in the liver) – the domain of peripheral physiology and metabolic events [12, 16].

Hedonic Mechanisms of Appetite Regulation

Hedonics mechanisms are triggered by signals of palatability such as smell and taste of food [18]. Lowe and Butryn [19] defined “hedonic hunger” as the motivation created by exposure and repeated consumption of highly palatable energy-dense foods. Their review provided evidence suggesting the pleasure obtained from eating that kind of food may overlap the homeostatic signals, promoting weight gain.

The intake of high-palatable, energy-dense foods triggers the release of neurotransmitters such as dopamine and serotonin [11]. The level of dopamine released is correlated with the level of pleasure obtained by ingestion [20]. Serotonin is associated with the sense of well-being, improved mood and promotion of motivation for food intake [18]. Palatable food, due to its high sugar and fat content, can disrupt appetite regulation because they blunt the response to satiety signals, increasing the duration of meals, and activating the reward system [16]. Nonetheless, animal studies indicate that this effect may be independent of palatability, suggesting post-ingestive effects on dopamine release [21] that should be further studied in humans.

There are individual differences in the responsiveness to food reward, but it seems that foods high in sugar and fat have an enhanced ability to activate the brain reward system [22]. Stewart et al. [23] observed that individuals with lower fatty acids sensitivity tend to ingest significantly higher amounts of fat during meals, associated with a higher body mass index, than individuals with high fatty acids sensitivity. The signalling ability of dopamine appears to be decreased in obese individuals and, consequently, it could compromise the reward system, and the reinforcing properties of food [24, 25].

Formation and Development of Eating Behaviours

Appetite-related eating behaviours arise as the result of a combination of genetic and biological factors. These factors are shaped by influences of the environment in which we find ourselves, including the family environment, school, culture/society, among others.

Genetic Predispositions

Genetic inheritance seems to play a major role in the development of the appetite. Recent literature highlights the valuable contribution of twin studies, to increase knowledge on the diversity of genetic factors associated with the development of eating behaviours. In a study of 8–11-year-old twins [26], the authors determined 63 and 75% heritability for “satiety responsiveness” and “food responsiveness” sub-dimension scores in the Children’s Eating Behaviour Questionnaire (CEBQ) respectively. The Gemini study [27] of infants with exclusive milk feeding in the first 3 months of life showed a large and moderate genetic impact of the Baby Eating Behaviour Questionnaire (BEBQ) constructs “slowness in eating” and “satiety responsiveness” (84 and 72% respectively) and “food responsiveness” and “enjoyment of food” (59 and 53% respectively).

In attempts to explain these genetic predispositions, some authors have supported a hypothesis related to the *FTO* (fat mass and obesity-related) gene, which is predominantly expressed in the hypothalamus [28]. This gene has been associated with the “satiety responsiveness” and “food responsiveness” in children [29, 30]. Some studies have also shown an apparent influence of the *FTO* gene in the development of obesity [31].

Food preferences also play a key role in the process of developing the eating behaviour. Food preferences for basic tastes (sweet, salty, bitter, sour and umami) are genetically determined [1]. By observing facial expressions of newborns, it was understood that they had a preference for sweet taste and an aversion to bitter and sour tastes [32]. The umami taste is associated with good acceptance [33] and the salt preference is increased at 4 months of age [34].

It is believed that this genetic predisposition of food preferences may result from the evolution of species, at a time when few foods were available [35]. The preference for sweet tastes would lead to the consumption of foods associated with a high energy density, thereby ensuring extended survival [35]. However, these preferences can be changed by behavioural experiences [32].

First Taste Experiences

The development of eating behaviours, during the first years of life, has its origins in utero [33, 36]. During foetal life, the foetus often swallows amniotic fluid that allows the first contact with some tastes and smell from the maternal diet (e.g., garlic [37], cumin [38] and curry [38]). These prenatal exposures influence the acceptance of foods in the first years of life [33, 39, 40].

As in amniotic fluid, there are characteristic tastes from the mother's diet in breast milk, such as garlic [36], alcohol [41] and vanilla [42]. Compared to the consumption of infant milk formulas, which always have the same taste over time [33], breast milk seems to expose the child to a greater variety of tastes [43], thereby also enabling the acceptance of new tastes in the early years of life [33, 43]. In addition, the consumption of breast milk is indicated to allow better self-regulation of the energy intake by infants [33]. With the bottle, the child has more "facilitated" access to milk than through the breast, exerting less effort and having less control of the amount ingested, enabling excess feeding [33]. Furthermore, bottle-feeding may potentially override the child's ability to self-regulate [33]. Thus, the child who is breastfed has a more active role in deciding the timing and the amount of intake. Some authors have shown that this learning about self-regulation of food intake will later translate into an enhanced response to internal signals of satiety [44, 45].

Other effects of maternal diet during pregnancy on children's food intake and preferences have been reported. A British study [46] determined that maternal macronutrient intake (especially protein and fat) during pregnancy has a stronger influence on child macronutrient intake at 10 years old than post-natal maternal intake or paternal macronutrient intake. Wardle et al. [47] observed that twin children from families with obese/overweight parents had modestly higher preferences for the taste of fatty foods, less liking for low-energy-dense foods, such as vegetables, and displayed stronger positive appetitive reactions to food and drink than children of lean parents. Other studies have reported that individuals with severe intrauterine growth restriction have altered nutritional intake later in life [48, 49]. Lussana et al. [50] demonstrated that middle-aged adult participants exposed to famine in early gestation were twice as likely to consume a high-fat diet (>39% energy from fat) than time controls, supporting long-term effects of intrauterine exposures. Overall, these results could reflect in utero programming of offspring appetite by maternal diet during pregnancy.

Family Environment and Its Influence

The family environment is critical in the development of children's eating behaviour. Children tend to imitate models and, in this sense, eating behaviour is also acquired by observing their models [33, 51], often parents, siblings, other relatives and even children of the same age.

Parents may create environments that foster the development of healthy eating behaviours and maintenance of appropriate weight or may promote overweight and eating disorders. There are main factors that contribute to this phenomenon, such as food availability [52], parental eating behaviours [1] and parental practices/styles used in child's feeding [1].

The parental child-feeding practices more often described, include pressure to eat, restriction of certain foods, food reward and monitoring of food intake [53–55]. Several studies have emphasised the negative interference of these practices on the development of healthy eating habits and, consequently, on the child's weight [53, 56, 57]. In 1985, Costanzo and Woody [58] proposed that parental control of food is triggered by concerns about the child's risk of becoming obese. However, such authority could have adverse effects on the child's eating behaviour and weight, as it could hamper the child from developing self-regulation mechanisms [58].

Children have the innate ability to regulate their energy intake [59–62]. Nevertheless, these imposed feeding practices can render ineffective the ability to respond to internal stimuli, as children will focus on external stimuli, to the detriment of the internal signals of hunger and satiety (e.g., when the child does not want to eat more yet the parents insist that the child eat the food remaining on the plate [63]). This reduced ability to respond to internal signals of hunger and satiety has been associated with increased weight in childhood [64–66]. More recent data indicates these associations between parental child-feeding practices and child's weight may be bidirectional, meaning that the feeding practices could influence child's weight, but they also could be a reaction to the child's weight (e.g., parents pressure imposed on a child to eat more often in reaction to a child's low weight) [56, 67].

Appetite and Related Eating Behaviours Assessment

There are several methods of assessing appetite and related eating behaviours. These measures are based on the biological mechanisms of appetite regulation. The most commonly used procedures are the biological (short- and long-term appetite regulation biomarkers [3]

and magnetic resonance techniques, able to detect variations in blood flow in response to neuronal activity [68]) and behavioural methods (laboratory observation and psychometrics).

Biological Methods

Biomarkers of appetite may be used as an objective tool for understanding the regulation of food intake and energy balance. The most commonly used biomarkers of appetite can measure satiation, satiety, or both, depending on whether their action is peripheral or at the central nervous system level [3]. At the peripheral level, the physical and chemical measures of distention of the stomach and the concentrations of cholecystokinin and glucagon-like peptide-1 are useful for assessing short-term satiation [3]. Concentrations of leptin, ghrelin and insulin are helpful in regulating long-term mechanisms of eating behaviours [3].

Neuronal techniques, such as functional magnetic resonance imaging and positron emission tomography, can also be used to understand how appetite modulates brain activity. These techniques are based on the detection of changes in blood flow associated with neuronal activation [68]. It has been suggested that obese individuals experienced greater reward from food consumption and anticipated food intake than lean individuals using functional magnetic resonance imaging [69, 70]. Still, these neuronal techniques may have limited use [3], given their poor accessibility for large-scale sample analysis at the population level.

Genetic markers for the early detection of obesity susceptibility may be beneficial in the perception of eating behaviour deregulation in the future. Polymorphisms within the *FTO* gene have been identified [71]. *FTO* is highly expressed in the arcuate nucleus of the hypothalamus, which is a key appetite control centre [28]. Thus, *FTO* may influence weight through effects on appetite, and other weight-related genes may operate similarly, representing a promising approach.

Behavioural Methods

Behavioural methods are indirect measures of appetite assessment, which allow the evaluation of different eating behaviour constructs, either in the laboratory or the natural environment of the individual, or even through psychometric tests (scales or questionnaires). According to the literature, the behavioural methods can be organised into 2 broad categories/domains: “food approach” and “food avoidance” [68]. The food approach can be defined as the impact that external signs, such as the sight or smell

of food, can have on food consumption, especially when it is in excess. In the food avoidance, the main aspect to be measured is the sensitivity to the fullness or satiety response capacity (when individuals can respond to internal signals, ceasing the consumption) [68].

Various psychometric instruments to evaluate appetite-related eating behaviours have been developed. Among the most used are the Dutch Eating Behaviour Questionnaire (DEBQ) [72, 73], the BEBQ [74], and the CEBQ [10]. The DEBQ was designed so that it can be reported by parents or caregivers [72] or by children from 7 to 12 years old [73]. The BEBQ is intended to be completed by mothers of babies between 0 and 3 months of age during exclusive breastfeeding or retrospectively [74]. The CEBQ has been described in several studies [5, 10, 68, 75] as the questionnaire most currently used to assess eating behaviours in children.

Therefore, given its scope, a more detailed description of the CEBQ is provided here. The CEBQ was developed in the United Kingdom by Wardle et al. [10] to be filled by parents of school-age children, to distinguish differences in eating behaviour that contribute to high or low weight. The questionnaire consists of 35 items, answered on a 5-point Likert scale (where 1 = “never” and 5 = “always”). It is conceptually organised in 8 sub-dimensions: satiety responsiveness, slowness in eating, food fussiness, emotional undereating, food responsiveness, enjoyment of food, emotional overeating and desire for drinks [10] (defined in Table 1). The first 4 sub-dimensions refer to the “food avoidance” domain and the last 4 sub-dimensions highlight the “food approach” [9]. The CEBQ has been validated in different populations (Table 2), showing good psychometric properties.

Method Considerations (Advantages and Disadvantages)

There is no ideal method of assessing appetite and related eating behaviour. Nonetheless, it is important to consider their advantages and limitations.

Biological methods have the advantage of capturing various elements of the individual’s response to diversified stimuli more objectively and completely, unlike behavioural and laboratory methods that use only one measure. Behavioural methods provide only part of the individual’s food response [68], devaluing several extrinsic factors that may interfere with the results [9], being more vulnerable to social desirability bias, a disadvantage applicable to all behavioural methods. Biological methods are costly, which makes their frequent use unfeasible [3, 76], as opposed to behavioural psychometric measures that are cheaper [68].

Table 1. Definition of the sub-dimensions used in the CEBQ

Sub-dimension of the CEBQ	Definition
SR	It represents the degree of self-regulation of the amount of food consumed by a child, based on the sensation of satiety by itself [9, 77].
SE	Measures the speed of food intake during a meal. A higher score in this sub-dimension reflects a gradually reduced interest in the meal [75, 77].
FF	Reflects a lack of interest in food and for trying novel foods (neophobia), resulting in a poorer diet [77].
EUE	Represents a decreased food intake in response to negative emotions such as anger and anxiety [77].
FR	Measures food intake in response to external stimuli from food. It represents an interest in eating and the desire to spend time eating [123].
EF	Represents a general liking for eating. Captures the extent to which the child enjoys eating and wants to eat [123].
EOE	Represents an increased food intake in response to negative emotions such as anger and anxiety [75, 77].
DD	Aims to identify the desire to drink, particularly sugary drinks [10].

CEBQ, Children's Eating Behaviour Questionnaire; SR, satiety responsiveness; SE, slowness in eating; FF, food fussiness; EUE, emotional undereating; FR, food responsiveness; EF, enjoyment of food; EOE, emotional overeating; DD, desire for drinks.

Psychometric methods may have problems when self-applied in children who cannot understand or who do not have the self-knowledge necessary to answer questions about their eating behaviour [9]. For this reason, the instruments usually used are questionnaires to be answered by parents/caregivers, who have a privileged position to observe the child's eating behaviours [9]. However, parents/caregivers may only develop socially desirable responses or rely solely on some observed occasions [9], which may limit the validity of their responses. Nonetheless, particularly regarding the CEBQ, several studies have demonstrated good internal consistency, reproducibility and construct validity of this instrument [5, 9, 10, 75, 77–87]. For all these reasons, the CEBQ has been considered a useful tool to comprehensively evaluate children's eating behaviours.

Effects of Eating Behaviours on Children's Weight Status

Childhood obesity rates are massive throughout the world [88]. Obese children are at increased risk of developing both short-term [88–90] and long-term [91, 92], adverse health consequences. It is known that in comparison to normal weight children, obese children are significantly more likely to be obese adults [93]. Hence, there

is a strong need to invest in studies and more effective interventions to reduce the prevalence of childhood obesity.

Genetic factors, eating habits and other lifestyles are mainly responsible for children's weight status [8]. Parental weight is also a risk factor for children's weight; children of obese parents are at a higher risk of becoming obese in adulthood than children of normal weight parents [94–96].

Some aspects of eating behaviour have also been associated with the development of childhood obesity. According to Braet and Van Strien [72], the main eating behaviours leading to childhood obesity are the pleasure in eating, the preference for foods rich in sugar and fat, the high responsiveness to external stimuli associated with food intake, the difficulty in self-regulation of the hunger and satiety signals and emotional factors.

Problematic eating behaviours in childhood and pre-adolescence can manifest in more severe eating complications in adolescence [97], as is the case of overweight [98–101]. Several eating behaviours related to appetite signalling are harmful to the individual's health, but 3 types seem to contribute more to overweight: external eating, restriction and emotional eating [102]. The idea that these behaviours are not independent and interact with each other has recently been consolidated [73, 103, 104].

Table 2. Summary of different validation studies using the Children's Eating Behaviour Questionnaire

Auhor, years	Country	Study sample	Structure
Wardle et al. [10], 2001	United Kingdom	<i>n</i> = 131; <i>n</i> = 187; <i>n</i> = 218 3–8 years old	35-item, 8-factor structure: DD, EF, EOE, EUE, FF, FR, SE, SR
Carnell and Wardle [9], 2007	United Kingdom	<i>n</i> = 111 4–5 years old	Validity of 3 CEBQ sub-scales (EF, FR, SR) as assessed by a variety of behavioural tests as indicators of the traits of responsiveness to internal satiety cues and to external food cues
Viana and Sinde [5], 2008	Portugal	<i>n</i> = 249 3–13 years old	35-item, 6-factor structure: DD, EF/FR, EOE, EUE, FF, SE/SR
Sleddens et al. [75], 2008	Netherlands	<i>n</i> = 135 6–7 years old	35-item, 7-factor structure: DD, EF, EUE, FF, SE, SR, FR/EOE
Svensson et al. [77], 2011	Sweden	<i>n</i> = 174 1–6 years old	34-item, 7-factor structure: DD, EF, EUE, FF, SE, SR, FR/EOE
Spence et al. [82], 2011	Canada	<i>n</i> = 1,730 4–5 years old	35-item, 8-factor structure: DD, EF, EOE, EUE, FF, FR, SE, SR
Santos et al. [85], 2011	Chile	<i>n</i> = 294 6–12 years old	35-item, 8-factor structure: DD, EF, EOE, EUE, FF, FR, SE, SR
Cao et al. [79], 2012	China	<i>n</i> = 219 12–18 months	35-item, 7-factor structure: SE, EUE, FF, FR1, DD, EOE, FR2
Sparks and Radnitz [80], 2012	EUA	<i>n</i> = 229 2–5 years old	15-item, 3-factor structure: disinhibition, food interest, undereating
Loh et al. [83], 2013	Malaysia	<i>n</i> = 362 13 years old	30-item, 8-factor structure: FR, EF, EOE, DD, SE, EUE, FF1, FF2
Mallan et al. [84], 2013	Australia	<i>n</i> = 244; 24 months Australian <i>n</i> = 203; 1–5 years old Indian <i>n</i> = 216; 1–4 years old Chinese	35-item, 8-factor structure: DD, EF, EOE, EUE, FF, FR, SE, SR
Domoff et al. [81], 2015	EUA	<i>n</i> = 1,002 Preschool aged children	35-item, 8-factor structure: DD, EF, EOE, EUE, FF, FR, SE, SR
Albuquerque et al. [78], 2017	Portugal	7 years old	35-item, 2-factor structure: appetite restraint, appetite disinhibition
Behar et al. [86], 2017	USA	<i>n</i> = 295 5–11 years old Latino children	CEBQ Shortened version 14-item, 3-factor structure: FR, SR, SE
Quah et al. [87], 2017	Singapore	<i>n</i> = 636 3 years old	35-item, 7-factor structure: EF, FF, EOE, DD, EUE, SR, SE

CEBQ, Children's Eating Behaviour Questionnaire; DD, desire for drinks; EF, enjoyment of food; EOE, emotional overeating; EUE, emotional Undereating; FF, food fussiness; FR, food responsiveness; SE, slowness in eating; SR, satiety responsiveness.

Eating by external stimuli corresponds to the eating behaviour triggered by external environmental stimuli, particularly, the presence of food, smell and/or taste or even the time of day [102]. Food restriction results from a combination of eating inhibition and overeating [105], can lead to overweight or even long-term obesity and

may be the precursor of the development of eating disorders, such as binge eating [106–108]. Some literature indicates that it is the excess weight that precedes the binge eating [109, 110] and that these compulsive behaviours are not the drivers of obesity, when it arises [111]. Binge eating may be associated with several fac-

tors, such as excessive concern with food [112], a negative body image, and shame or concern with public appearance [113].

Emotional eating is the eating behaviour resulting from responses to emotional variations, especially negative feelings [102]. People tend to change their eating behaviour according to negative emotional experiences [114–116], which usually happens to people with eating disorders [117]. Van Strien et al. [118] consider that emotional eating has increased noticeably in recent years, which may partially explain the continuing rise of obesity at the population level.

Contrary to that observed in adults, in a study with Dutch children [73], a low prevalence of emotional eating was seen, and childhood overweight was more prevalent when there was food restriction. In an Italian study using the DEBQ, food restriction was found to be higher in overweight or obese pre-adolescents than thinner individuals [119]. Similar results were reported in another study, in which the DEBQ was used in a sample of Belgian children [103].

Autism spectrum disorders are a particular case that illustrates the impact that eating behaviours can have on weight status. The quality of diet of these children can be affected, as these individuals are more picky eaters and tend to refuse food because of an aversive reaction to certain textures, smells, colours or temperatures [120–122]; this tendency may compromise their growth and natural development, due to nutritional deficiencies.

Conclusions

Several factors contribute to the complexity of human eating behaviour, such as genetics, physiological and environmental factors. Currently, it is perceived that nutritional and pharmacological interventions are becoming more ineffective for the prevention and treatment of obesity, and there is an urgent need to develop more advanced mechanisms to intervene in this public health problem. The study of the eating behaviour is a crucial aspect in this health area. It is necessary to determine what overlaps the hedonic control of intake over energy homeostasis and the interaction between both in the regulation of appetite. Such information will lead to developing ways to balance the food pleasure for high-palatable foods with the energy depots that the body needs.

Early identification of children at high risk of developing overweight or obesity is necessary for more efficient

and effective interventions. The CEBQ is recognised as a valid method that allows the identification of children with problematic eating behaviours, regardless of its aetiology. However, in the future, the use of neuroimaging techniques and genetic markers for early detection of obesity susceptibility may help in the perception of eating behaviours deregulation.

If parental child-feeding practices influence the child's weight, then this knowledge could be used to intervene in obesity prevention. However, if these practices are used in reaction to the child's weight, interventions should also be aware and deal with parental concerns about the child's weight. Education of parents/caregivers on different aspects that compromise children's health should be privileged, including the influence that early taste experiences may have, namely, through the maternal diet during the intrauterine duration, as well as by the type of milk used in the first months of life. The importance of parents in shaping children's eating behaviours should also be emphasised. The parents are responsible for food availability, and their eating behaviours may impact on the child's behaviours. Also, certain parental child-feeding practices may negatively interfere with children's eating behaviours. Thus, parents should be key targets in educational actions aiming to improve child's health. Future studies on the determinants of children's eating behaviours may encourage the development of useful tools for prevention, support and treatment of obesity.

Key Messages

- Eating behaviours (external eating, restriction and emotional eating) are associated with overweight. They are not independent and could interact with each other.
- The use of the CEBQ allows the identification of children with problematic eating behaviours, regardless of its aetiology. It is a valid method, largely applied in different population settings.
- The reduced ability of children to respond to internal signals of hunger and satiety can be imposed by parents. Parental child-feeding practices could influence a child's weight, but they could also be a reaction to the child's weight – this bidirectional effect should be considered in future studies.
- Given the close association between appetite-related eating behaviours and weight status, future studies

on the determinants of eating behaviours may favour the development of useful tools for prevention, support and treatment of childhood obesity.

Acknowledgements

A.O. received funds from the FCT Investigator Programme (IF/01350/2015), with FEDER funds through the Operational Programme Competitiveness and Internationalisation, in addition to national funding from the Foundation for Science and Technology (FCT; Portuguese Ministry of Science, Technology and Higher Education), co-funded by the FCT and the POPH/FSE Program.

tion to national funding from the Foundation for Science and Technology (FCT; Portuguese Ministry of Science, Technology and Higher Education), co-funded by the FCT and the POPH/FSE Program.

Disclosure Statement

The authors declared that there are no conflicts of interest to disclose.

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