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Research report

Variability in children's eating response to portion size. A biobehavioral perspective

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ABSTRACT

The portion size of foods and beverages offered at meals has been shown to significantly affect human food intake. While portion size effects appear to be fairly robust across studies in adults, findings from studies in children are generally more variable and do not reliably predict a significant portion size effect. Eating behaviors are still forming at a young age and individual differences in children's response to portion size will depend upon genetic predisposition interacting with the child's environment. The aim of this review is to present and discuss evidence that innate controls of appetite and eating, which involve genes that encode key hormones and neuropeptides implicated in processes of satiety and satiation, may differentially affect meal size. We also present evidence that children's response to portion size is learned and this is in turn shaped by upbringing, the early family and home environment. The review will conclude with a conceptual model that illustrates how biological and environmental factors may interact to shape child eating traits including a behavioral susceptibility to overeating when large portion sizes are available.

Introduction

An obesogenic environment refers to an environment that facilitates the risk of obesity and includes the built and food environments (Lake & Townshend, 2006). Obesogenic food environments, which can be found inside and outside the home, are characterized by ready availability and easy access to large portions of energy-dense, palatable foods and beverages. As children grow older and become more independent, obesogenic environments also begin to affect their food purchasing and thus consumption behavior. A recent analysis by Drewnowski and Rehm (2013), which examined energy intakes in children, adolescents, and adults by food purchase location using data from the National Health and Nutrition Examination Survey (NHANES), showed that for each age group stores and restaurants (including full-service and quick service/pizza/take-out/delivery) accounted for at least 85% of total energy intake. For younger children (6–11 years), 63% of daily energy intake came from stores, 12% from quick-serve restaurants, and 10% from school cafeterias. For adolescents (12–19 years) 63% of daily energy intake came from stores, 18% from quick-serve restaurants, and 7% from full-service restaurants. Another study by Borradaile and colleagues (Borradaile et al., 2009) showed that for only ~$1 spent in corner stores (i.e., average amount spent per purchase), children in grades 4 through 6 (ages 9 to 12 years) from urban elementary schools purchased 357 kcal worth of food and beverage items. Once again, this confirms the ease with which food is cheap, available and purchased when children demonstrate their purchasing power. Children are exposed to the obesogenic food environment and food marketing strategies, including value size pricing, at a young age and may learn to associate the purchase of large food portions with better value when making food purchasing decisions.

Current estimates indicate that 31.8% of US children and adolescents, between 2 and 19 years of age, are considered overweight or obese (BMI-for-age ≥85th percentile) (Ogden et al., 2014). While this causes concern from a public health perspective, it is important to note that the majority of children (68.2%) are able to maintain a healthy weight under the same obesogenic environmental conditions experienced by all. The fact that not all children are equally susceptible to overeating and excess weight gain suggests that differences in genetic predisposition interact with the environment to determine the expressed phenotype. Data from mostly cross-sectional research point to a positive relationship between child BMI and portion sizes consumed. For example, using dietary intake data from the Continuing Survey of Food Intakes by Individuals (CSFII) and a Nationwide Food Consumption Survey, McConahy and colleagues related average quantities (expressed as portion size z-scores) of commonly consumed foods to children's body weight (expressed as percentiles) (McConahy et al., 2002). Results showed that

Abbreviations: ED, energy density; MZ, monozygotic; DZ, dizygotic.

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average portion size z-scores were positively related to children's percentile body weight indicating that children with greater body weights consumed larger food portions. Similarly, when examining associations between eating behaviors and weight status of 3- to 19-year-old children and adolescents using data from the CSFII, Huang and colleagues (Huang et al., 2004) showed that meal portion size was positively related to BMI-for-age percentiles in boys 6 years and older and in girls 12 years and older. While energy requirements are greater for children who have a high BMI and growth spurts may drive periods of increased hunger, selecting larger portions of foods and beverages can become learned and then expected even when weight is stable and growth is no longer driving intake.

Controlled laboratory studies which experimentally modify food and beverage portions and precisely quantify children's food and energy intakes are critical for studying children's response to portion size manipulations. These studies are also able to shed light on the individual differences in susceptibility or resistance to overeating when served large portions. Over the past decade, a series of well controlled laboratory-based studies in children and adults have advanced our understanding of the role of portion size in determining food intake. Interestingly, in contrast to studies with adults, portion size effects in children appear to be more variable across experiments in that some studies demonstrated significant portion size effects for a specific experimental manipulation (e.g., serving method, interaction with energy density) or child characteristic (e.g., age), while others did not (Table 1). Explanations to account for these differences might include study design, research methods, or differences among the cohorts studied, but it is also possible that eating behaviors are simply more malleable at a young age as children's eating habits are being formed through genetic predisposition interacting with the environment (Fisher & Kral, 2008; Small et al., 2013; Zlatevska, Dubelaar, & Holden, 2014).

The aim of this review is to discuss how genetic susceptibility may interact with factors in children's early environment to predispose some children to overeat when served large food portions. We present evidence for the proposal that children's response to portion size may in part be determined by innate (genetic) appetite and eating traits, which can affect meal size. We further discuss evidence for children's response to portion size as a learned behavior influenced by upbringing (parenting style and feeding practices) and early environment.

Genetic influences underlying food intake and meal size (nature)

Typically, genetic susceptibility to obesity is identified through twin studies and via linkage and association studies connecting the functional role of specific genes to the expression of differences in body mass, appetite regulation or eating traits. There are multiple, complex routes to obesity, but certain behavioral traits are linked to overeating and obesity risk. These might include traits, which reflect high approach tendencies toward food (such as opportunistic eating, heightened sensitivity to food as a reward) and low avoidance tendencies (such as impaired satiety, weak short term energy compensation) or an interaction between the two (such as excessive snacking of high energy-dense foods, consuming large portions of highly palatable items). For example, a recent cross-sectional observational study by Llewellyn et al. (2014) of a population-based cohort of 2258 twins (Twins Early Development Study) tested if satiety responsiveness may serve as an intermediate behavioral phenotype associated with a genetic predisposition to obesity in children. The results of the study showed that associations between the polygenic risk score, which was comprised of 28 common obesity-related single nucleotide polymorphisms (SNPs), and child adiposity were significantly mediated by satiety responsiveness. Thus, the genetic influence on overconsumption might operate through different routes – increasing salience of food, reduced responsiveness to satiation and satiety or a combination of these. The heritable component of BMI could then be expressed through specific eating traits conferred by parents to their children.

Given that BMI is highly heritable with heritability estimates ranging between 70 and 80% for children and adolescents (Maes, Neale, & Eaves, 1997), what is the basis of the resemblance? Family and twin studies investigating eating phenotypes among nuclear family members have provided evidence that many dietary and eating behaviors are shared and heritable. For example, an analysis of dietary data collected from adult twins over a 7-day period provided heritability estimates of 42% for daily energy intake, 28% for meal size, and 34% for meal frequency, respectively (de Castro, 2004). Genetic influences have also been observed for meal energy intake in children. In a study by Faith and colleagues (Faith et al., 1999), 36 monozygotic (MZ) and 18 dizygotic (DZ) twins were invited to the laboratory to consume lunch *ad libitum* from a multi-item buffet. Children could freely select both the types and amounts of foods and beverages, which showed a range in energy density (ED; kcal/g). The results of the study indicated that MZ twin pairs were more similar in their meal energy intake ($r = 0.80$) than DZ twin pairs ($r = 0.68$) with genetic variations accounting for 24–33% of the variance in age- and sex-adjusted total energy intake at the meal.

The control of human appetite is expressed as a complex interaction between psychological, physiologic and metabolic factors involving nutrients in the blood and a host of peripheral hormones, and metabolic and neurotransmitter interactions in the brain. The overlapping sensory, cognitive, hormonal, and metabolic signals that are triggered by the ingestion of food and beverages have been conceptualized within the 'satiety cascade' (Blundell, 2010; Cotton & Blundell, 1994). This cascade identifies the concepts of satiation, defined as processes that bring an eating episode to an end (intra-meal satiety), and satiety, defined as processes that inhibit further eating in the postprandial period until the next meal (inter-meal satiety). Both satiation and satiety are influenced by physiological signals, which arise from a complex network of hormones and neuropeptides controlling the size of an eating episode (amount consumed) and the interval until the next meal (postprandial suppression of appetite). Genes which encode these complex appetite and satiety signals are involved therefore in the susceptibility to overeat and in the extent to which external factors such as portion size influence the amount eaten.

Specific single gene variants associated with obesity have been identified using genome-wide association study (GWAS)

Table 1
Examples of pediatric studies showing inconsistencies in portion size effects.

Child characteristics/type of portion size modification	Observed portion size effects?	References
Age (toddler vs. older)	Yes and No	Fisher, 2007; Rolls, Engell, & Birch, 2000
Weight status (normal-weight vs. overweight/obese)	Yes, No, Maybe	Fisher et al., 2012; Kral et al., 2010, 2014; Savage et al., 2012; Sharafi, Fisher, & Birch, 2009
Serving method (self-serve vs. pre-portioned)	Yes and No	Fisher, 2007; Fisher et al., 2013; Savage et al., 2012
Health foods (fruits and vegetables)	Yes and No	Kral et al., 2010; Mathias et al., 2012
Interaction with energy density	Yes and No	Fisher et al., 2007a; Leahy et al., 2008; Looney & Raynor, 2011