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1 **Variability in Children’s Eating Response to Portion Size: A Biobehavioural Perspective**

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14 Running Title: Influences of nature or nurture in children’s portion size response

15 Abbreviations: Energy density, ED; monozygotic, MZ; dizygotic, DZ

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19 **Introduction**

20 An obesogenic environment refers to an environment that facilitates the risk of obesity and
21 includes the built and food environments [1]. Obesogenic food environments, which can be
22 found inside and outside the home, are characterized by ready availability and easy access to
23 large portions of energy-dense, palatable foods and beverages. As children grow older and
24 become more independent, obesogenic environments also begin to affect their food purchasing
25 and thus consumption behavior. A recent analysis by Drewnowski and Rehm [2], which
26 examined energy intakes in children, adolescents, and adults by food purchase location using
27 data from the National Health and Nutrition Examination Survey (NHANES), showed that for
28 each age group stores and restaurants (including full-service and quick service/pizza/take-
29 out/delivery) accounted for at least 85% of total energy intake. For younger children (6-11
30 years), 63% of daily energy intake came from stores, 12% from quick-serve restaurants, and 10%
31 from school cafeterias. For adolescents (12-19 years) 63% of daily energy intake came from
32 stores, 18% from quick-serve restaurants, and 7% from full-service restaurants. Another study by
33 Bourradaile and colleagues [3] showed that for only ~\$1 spent in corner stores (i.e., average
34 amount spent per purchase), children in grades 4 through 6 (ages 9 years to 12 years) from urban
35 elementary schools purchased 357 kcal worth of food and beverage items. Once again, this
36 confirms the ease with which food is cheap, available and purchased when children demonstrate
37 their purchasing power. Children are exposed to the obesogenic food environment and food
38 marketing strategies, including value size pricing, at a young age and may learn to associate the
39 purchase of large food portions with better value when making food purchasing decisions.

40 Current estimates indicate that 31.8% of US children and adolescents, between 2 and 19
41 years of age, are considered overweight or obese (BMI-for-age \geq 85th percentile) [4]. While this

42 causes concern from a public health perspective, it is important to note that the majority of
43 children (68.2%) are able to maintain a healthy weight under the same obesogenic environmental
44 conditions experienced by all. The fact that not all children are equally susceptible to overeating
45 and excess weight gain suggests that differences in genetic predisposition interact with the
46 environment to determine the expressed phenotype. Data from mostly cross-sectional research
47 point to a positive relationship between child BMI and portion sizes consumed. For example,
48 using dietary intake data from the Continuing Survey of Food Intakes by Individuals (CSFII) and
49 a Nationwide Food Consumption Survey, McConahy and colleagues related average quantities
50 (expressed as portion size z-scores) of commonly consumed foods to children's body weight
51 (expressed as percentiles) [5]. Results showed that average portion size z-scores were positively
52 related to children's percentile body weight indicating that children with greater body weights
53 consumed larger food portions. Similarly, when examining associations between eating
54 behaviors and weight status of 3- to 19-year-old children and adolescents using data from the
55 CSFII, Huang and colleagues [6] showed that meal portion size was positively related to BMI-
56 for-age percentiles in boys 6 years and older and in girls 12 years and older. Whilst energy
57 requirements are greater for children who have a high BMI and growth spurts may drive periods
58 of increased hunger, selecting larger portions of foods and beverages can become learned and
59 then expected even when weight is stable and growth is no longer driving intake.

60 Controlled laboratory studies which experimentally modify food and beverage portions
61 and precisely quantify children's food and energy intakes are critical for studying children's
62 response to portion size manipulations. These studies are also able to shed light on the individual
63 differences in susceptibility or resistance to overeating when served large portions. Over the past
64 decade, a series of well controlled laboratory-based studies in children and adults have advanced

65 our understanding of the role of portion size in determining food intake. Interestingly, in contrast
66 to studies with adults, portion size effects in children appear to be more variable across
67 experiments in that some studies demonstrated significant portion size effects for a specific
68 experimental manipulation (e.g., serving method, interaction with energy density) or child
69 characteristic (e.g., age), while others did not (**Table 1**). Explanations to account for these
70 differences might include study design, research methods, or differences among the cohorts
71 studied, but it is also possible that eating behaviors are simply more malleable at a young age as
72 children's eating habits are being formed through genetic predisposition interacting with the
73 environment [7-9]

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

80

81 **Genetic Influences Underlying Food Intake and Meal Size (Nature)**

82 Typically, genetic susceptibility to obesity is identified through twin studies and via
83 linkage and association studies connecting the functional role of specific genes to the expression
84 of differences in body mass, appetite regulation or eating traits. There are multiple, complex
85 routes to obesity, but certain behavioural traits are linked to overeating and obesity risk. These
86 might include traits, which reflect high approach tendencies towards food (such as opportunistic

87 eating, heightened sensitivity to food as a reward) and low avoidance tendencies (such as
88 impaired satiety, weak short term energy compensation) or an interaction between the two (such
89 as excessive snacking of high energy-dense foods, consuming large portions of highly palatable
90 items). For example, a recent cross-sectional observational study by Llewellyn et al. [10] of a
91 population-based cohort of 2258 twins (Twins Early Development Study) tested if satiety
92 responsiveness may serve as an intermediate behavioral phenotype associated with a genetic
93 predisposition to obesity in children. The results of the study showed that associations between
94 the polygenic risk score, which was comprised of 28 common obesity-related single nucleotide
95 polymorphisms (SNPs), and child adiposity were significantly mediated by satiety
96 responsiveness. Thus, the genetic influence on overconsumption might operate through different
97 routes – increasing salience of food, reduced responsiveness to satiation and satiety or a
98 combination of these. The heritable component of BMI could then be expressed through specific
99 eating traits conferred by parents to their children.

100 Given that BMI is highly heritable with heritability estimates ranging between 70 – 80%
101 for children and adolescents [11], what is the basis of the resemblance? Family and twin studies
102 investigating eating phenotypes among nuclear family members have provided evidence that
103 many dietary and eating behaviors are shared and heritable. For example, an analysis of dietary
104 data collected from adult twins over a 7-day period provided heritability estimates of 42% for
105 daily energy intake, 28% for meal size, and 34% for meal frequency, respectively [12]. Genetic
106 influences have also been observed for meal energy intake in children. In a study by Faith and
107 colleagues [13], 36 monozygotic (MZ) and 18 dizygotic (DZ) twins were invited to the
108 laboratory to consume lunch *ad libitum* from a multi-item buffet. Children could freely select
109 both the types and amounts of foods and beverages, which showed a range in energy density

110 (ED; kcal/g). The results of the study indicated that MZ twin pairs were more similar in their
111 meal energy intake ($r = 0.80$) than DZ twin pairs ($r = 0.68$) with genetic variations accounting for
112 24 – 33% of the variance in age- and sex-adjusted total energy intake at the meal.

113 The control of human appetite is expressed as a complex interaction between
114 psychological, physiologic and metabolic factors involving nutrients in the blood and a host of
115 peripheral hormones, and metabolic and neurotransmitter interactions in the brain. The
116 overlapping sensory, cognitive, hormonal, and metabolic signals that are triggered by the
117 ingestion of food and beverages have been conceptualized within the ‘satiety cascade’ [14, 15].
118 This cascade identifies the concepts of satiation, defined as processes that bring an eating
119 episode to an end (intra-meal satiety), and satiety, defined as processes that inhibit further eating
120 in the postprandial period until the next meal (inter-meal satiety). Both satiation and satiety are
121 influenced by physiological signals, which arise from a complex network of hormones and
122 neuropeptides controlling the size of an eating episode (amount consumed) and the interval until
123 the next meal (post-prandial suppression of appetite). Genes which encode these complex
124 appetite and satiety signals are involved therefore in the susceptibility to overeat and in the
125 extent to which external factors such as portion size influence amount eaten.

126 Specific single gene variants associated with obesity have been identified using genome-
127 wide association study (GWAS) techniques. However, single gene mutations linked to obesity
128 are rare and account for less than 5% of severe obesity [16]. Nevertheless when these are
129 observed they are generally associated with disruption in appetitive pathways and extreme
130 hyperphagia [17]. Disruption to the leptin-melanocortin pathway produces dramatic effects on
131 food intake and body weight. Specifically, congenital deficiency in the leptin receptor is
132 characterized by early onset, severe obesity, and hyperphagia [18].

133 More common than single gene mutations are SNPs in candidate genes. To date more
134 than 127 SNPs in candidate genes have been identified which can lead to impaired functionality
135 in the central and peripheral regulation of energy balance and have been associated with the
136 human obesity phenotype [19, 20]. **Table 2** depicts examples of some SNPs that have been
137 shown to affect food intake. These include, but are not limited to, polymorphisms in the agouti-
138 related protein (AGRP), fat mass and obesity associated gene (FTO), cholecystokinin (CCK),
139 leptin, monamine oxidase A (MAOA), catechol-o-methyltransferase (COMT),
140 hydroxytryptamine receptor 2A (HTR2A), and peroxisome proliferator-activated receptor
141 gamma (PPARG), which have been shown to be implicated in behavioral traits such as
142 hyperphagia, satiety responsiveness, meal size, snacking behavior, food reinforcement, and
143 macronutrient intake [21-29].

144 Following a meal, the key peptide signaling satiety is CCK, secreted by the intestine. The
145 CCK1 receptor plays a role in regulating food intake, and CCK generally acts to suppress further
146 food intake. However, in animals who are naturally CCK-1 receptor deficient adult onset
147 diabetes and obesity are observed [30]. Functionality then is compromised when the receptor is
148 absent or impaired. In humans, variations in the H3 haplotype of CCK are linked to extreme
149 portion size consumption [17]. De Krom and colleagues [22] employed an “extreme discordant
150 phenotype” approach by identifying obese adults from the large scale population based European
151 Prospective Study into Cancer and Nutrition (EPIC) cohort who were ranked at the top 5th
152 percentile for self-reported extreme snacking behavior and portion sizes. The results of the study
153 showed significant associations between four of the five CCK SNPs and increased meal size but
154 not snacking frequency, thus carriers of these specific polymorphisms are at risk of consuming
155 large portion sizes, inferring a link to impaired satiety signaling. Interestingly, two of the four

156 leptin SNPs and one of the eight leptin receptor SNPs were associated with frequent snacking but
157 not with meal size. Therefore, demonstrating two different pathways to extreme eating traits,
158 only one of which relates to the tendency to eat large portions.

159 Other eating traits have been identified which have been shown to be in part under
160 genetic control and which link to the tendency to overeat. For example, heritability has been
161 established for eating in the absence of hunger (EAH; susceptibility to eating when satiated in
162 response to the presence of palatable snacks; $h = 51\%$) [31], eating rate ($h = 62-84\%$) [32, 33],
163 satiety responsiveness (degree to which an individual ceases eating or chooses not to start eating
164 based on their perceived fullness; $h = 65\%$) [34], and food cue responsiveness (tendency to eat in
165 response to food cues; $h = 75\%$) [34]. While no study to date has established heritability
166 estimates for children's response to portion size, data from the above mentioned studies can be
167 used as a proxy for genetic influences underlying susceptibility to overeat in childhood.

168 A recent study conducted in weight-discordant siblings provided evidence for significant
169 family correlations for caloric compensation and EAH in children [35]. Caloric compensation,
170 expressed as percentage compensation index (%COMPX), refers to adjustments in intake in
171 response to changes in the ED of a compulsory preload. In this study, 47 same-sex sibling pairs
172 (55% full siblings), ages 5 – 12 years, were invited to consume dinner in the laboratory once a
173 week for three weeks. Twenty minutes before an *ad libitum* dinner meal, children were asked to
174 consume in full or not to consume one of two pudding preloads which varied in ED (0.57 kcal/g
175 or 0.97 kcal/g). On the day when no preload was served, children were given access to a variety
176 of snacks after they completed the dinner meal. %COMPX was computed as the difference in
177 energy intake at dinner in the two preload conditions divided by the difference in energy intake
178 from the compulsory preloads multiplied by 100. EAH referred to the energy consumed from the

179 snacks while satiated. The results of the study showed that overweight and obese siblings
180 showed poor caloric compensation and significantly more EAH when compared to their normal-
181 weight siblings. Further, the data showed familial associations for %COMPX and EAH that were
182 significant for full siblings (%COMPX: ICC = 0.36; EAH: ICC = 0.37, $P < 0.05$) but not for half
183 siblings (%COMPX: ICC = 0.02; EAH: ICC = 0.16, $P > 0.05$), which suggests that genetic
184 influences underlie both of these eating traits.

185 Data by Cecil and colleagues [21] provide further evidence for genetic factors influencing
186 children's compensation ability. In a study with 84 children, ages 4 – 10 years, from 47 schools
187 in Scotland, children were asked to consume in full either a no-energy, low-energy, or high-
188 energy preload, consisting of an orange drink (or water) and a muffin (or no muffin),
189 midmorning on three occasions followed by an *ad libitum* lunch 90 minutes later. They
190 examined if variants in the nuclear fatty acid receptor peroxisome proliferator-activated receptor
191 gamma (PPAR γ) gene (Pro12Ala, C1431T, C-681G) and the beta-adrenergic receptor (ADRB3)
192 gene (Trp64Arg) were associated with %COMPX. The results of the study showed indeed that
193 children's genotype was a significant factor in children's ability to compensate. Children with
194 polymorphisms in the PPAR γ gene (T1431 allele) showed poor compensation whereas children
195 with polymorphisms in the ADRB3 gene (Trp64Arg allele) showed good compensation. When
196 the same cohort of children were enriched for the A allele of the FTO gene, it was found that
197 carriers of this allele were heavier, had a higher fat mass, consumed more energy (even adjusting
198 for their larger body size) and selected more energy as fat in a self-selection test meal, but did
199 not differ in %COMPX. Thus, in this group the FTO risk allele was associated with increased
200 intake, which could be related to opportunistic eating or to a preference for energy-dense,
201 palatable foods.

202 A study by Wardle and colleagues [23] aimed to test the hypothesis that higher risk FTO
203 alleles would be associated with greater EAH. In this study, 131 4-year-old children from the
204 Twin Early Development Study (TEDS) were given access to three different varieties of biscuits
205 in their homes one hour after children finished eating a meal. Further, children's FTO single
206 nucleotide polymorphism (rs9939609) was determined. The results of the study showed that
207 biscuit intake differed significantly across the three genotype groups (TT, AT, AA). Children
208 with higher risk FTO alleles (AA) showed 25% greater snack intake compared to children with
209 the more protective genotype (TT); an effect which was independent of children's BMI z-score.

210 In a highly innovative series of studies combining mechanistic analyses of the function of
211 FTO in mice with fMRI scans of human carriers of the AA risk allele, Karra and colleagues [36]
212 found that FTO has a specific regulatory effect on the orexigenic hormone ghrelin. Normal-
213 weight participants with the AA genotype showed a blunted postprandial hunger and ghrelin
214 response to a standard meal and they responded differently to the presentation of food images in
215 the scanner, in both homeostatic (hypothalamus) and reward-relevant brain regions whether
216 satiated or fasted. They also responded differently to the administration of ghrelin, suggesting a
217 perturbation in ghrelin signaling, which is a putative mechanism for observed differences in
218 eating behaviour. The authors suggest that the FTO rs9939609 AA genotype is characterized by
219 an eating phenotype, which could link to obesity risk since these observations were made in
220 normal-weight participants. Clearly this is relevant to identifying characteristics of the pre-obese
221 phenotype in children since enhanced food responsiveness, preferences for high-fat foods,
222 increased appetite and food cue-potentiated eating, part of the FTO phenotype [36], could be
223 identified in children.

224 Together, these examples illustrate both the eating traits that might be associated with the
225 tendency to respond to portion size manipulations as well as possible mechanisms by which
226 genetic influences shape the underlying phenotype. It is proposed that genes encoding gut
227 hormones and neuropeptides act in concert to control appetite and eating determining meal size
228 and in particular children's response to portion size. While a genetic predisposition may increase
229 some children's susceptibility to overeating when served large portions of foods and beverages,
230 evidence suggests that children's responsiveness to portion size can also be learned behavior.
231 Therefore, biology is not destiny with respect to how much children choose to eat.

232

233 **Environmental Influences Shaping Eating Traits (Nurture)**

234 The early home food environment plays an important role in shaping children's food
235 preferences and eating behaviors [37-39]. Parents and caregivers influence their children's food
236 choices and eating in a variety of ways. For example, parents serve as important role models for
237 eating [40, 41]. A number of studies have shown significant mother-child relationships in dietary
238 intake including significant positive correlations between maternal and child consumption of
239 sweets and daily energy intake [42] as well as fruits and vegetables [42, 43]. The observed
240 mother-child associations in dietary intake may in part be explained by mothers providing a
241 model of food choices and dietary intake, as well as a marker of shared environmental factors,
242 such as access to the same foods in the same home. Further, the specific feeding strategies and
243 practices parents use have been shown to significantly impact their children's food intake and
244 weight regulation [44]. Parents also decide on the types and quantities of foods and beverages
245 that are being brought into the home and the manner in which meals are being consumed (e.g.,

246 family meals, self-serve). Additionally, parents influence the physical home environment by
247 selecting dishware (e.g., plates, utensils, cups) sizes and styles and by setting the social norm for
248 appropriate serving sizes. Besides shaping the home food environment, parents make decisions
249 about where to shop for groceries (e.g., grocery stores, wholesale clubs), what promotional tools
250 to use (e.g., grocery coupons), and which restaurant to frequent (e.g., quick serve restaurants that
251 offer value pricing). All of these combine to form the important early home environment.

252 Data from a recent observational study by Johnson and colleagues [45] in parents and
253 their preschoolers who were recruited from Head Start centers showed that a major driver of how
254 much food children consumed at a meal at home was how much they were served by their
255 parents. In this study, research staff measured amounts served and consumed at a meal by
256 children and parents using digital photography during three home visits. The results of this study
257 showed that amounts served to children by their parents accounted for 73% of the variance in
258 children's intake and children who were served more food showed significantly greater intakes (r
259 = 0.88). Interestingly, amounts served to children was significantly correlated with the amounts
260 parents served themselves ($r = 0.51$). These data not only highlight the important role that
261 parents play in establishing portion norms early in children's lives, but they also suggest that
262 parents who consistently serve large portions at home meals may be imparting an expectation
263 that their children will learn to consume them. In a setting where children are expected to "clean
264 the plate" and to avoid waste, especially where families are low income and disadvantaged,
265 setting high social norms for how much is eaten may set in train a pattern of overeating relative
266 to energy requirements.

267 Besides portions served at home meals, parents influence children's decisions
268 surrounding portion size selection by the feeding styles and practices they use on a day-to-day

269 basis. One of the aims of a controlled laboratory experiment by Fisher and colleagues [46] was to
270 identify child and family predictors of individual differences in children's self-served portions.
271 In this crossover study, 4- to 6-year-old children were asked to serve themselves macaroni and
272 cheese from a serving dish that contained different portions (275 vs. 550g) of the pasta meal.
273 Parents were asked to complete the Caregiver Feeding Style Questionnaire [47] which assessed
274 the extent to which they used the following four feeding styles: 1) authoritative feeding style,
275 which is characterized by parental involvement, nurturance, reasoning, and structure; 2)
276 authoritarian feeding style, which is characterized by restrictive, punitive, rejecting, and power-
277 assertive parental behaviors; 3) indulgent feeding style, which is characterized by warmth and
278 acceptance in conjunction with a lack of parental monitoring of child's behavior; and 4)
279 uninvolved feeding style, which is characterized by parents showing little control or involvement
280 with the child. The results of this study showed that children of parents who used indulgent and
281 authoritarian feeding styles served themselves about twice as much of the pasta meal and also
282 consumed significantly more calories during the meal than children of parents who used
283 authoritative and uninvolved feeding styles. These data provide evidence for a link between
284 specific parenting styles, feeding practices and consumption of larger food portions even when
285 the child is not under direct supervision of parents.

286 Parents and caregivers influence child eating via structuring of family meals, modeling
287 eating behaviors, and use of certain feeding practices. They also are in charge of creating the
288 physical home environment. Aspects of the physical home environment that relate to family
289 meals and eating, such as dishware size, can also significantly impact children's selection of food
290 portions. For example, the aim of a recent study by DiSantis and colleagues [48] was to test the
291 effects of dishware size (including plates and bowls) on self-selected portion sizes and intake in a

292 group of 42 elementary school-aged children who were observed on repeated occasions during
293 school lunch. Children were instructed to serve themselves from three serving bowls at a buffet
294 table containing a main dish, a vegetable side dish, and fruit using either child- or adult-size
295 dishware. The adult-size dishware represented a 100% increase in surface area/volume compared
296 to the child-size dishware. This study showed that children served themselves 90 calories more at
297 lunch when using the adult-size dishware. Further, for every additional calorie that children
298 served themselves, they added 0.43 more calories to their total meal energy intake. Interestingly,
299 the results of this study also showed that food insecurity was a significant predictor of children's
300 response to dishware size in that children from food insecure households self-served significantly
301 more compared to children from food secure households. By way of explanation the authors
302 suggested that larger dishware may have inflated children's norms for consumption and/or may
303 have also altered their visual perception of portion sizes.

304 In summary, these data illustrate the importance of early influences in children's
305 upbringing and home food environment, which together can help shape children's response to
306 portion size. Neither genetic nor environmental factors work in isolation, however, and it
307 therefore is important to study the interactions between these influences.

308

309 **Gene-Environment Interactions Underlying Behavioural Susceptibility to Portion Size** 310 **Response**

311 As discussed in this review, any behavioral tendency to overeat when large portions are
312 available is likely to occur by way of gene-environment interactions. Thus, a genetic
313 predisposition interacts with behavioural and physical aspects of the child's early environment to

314 facilitate expression of the underlying genotype. In families carrying risk alleles predicting
315 overweight and obesity, genetic effects may be moderated by healthy lifestyle, authoritative
316 parenting, moderate portion sizes, and physical activity. For example, the effects of the FTO risk
317 allele can be attenuated in children by offering healthy diets characterized by lower dietary
318 energy density [49] and in adults by physical activity [50].

319 **Figure 1** provides a conceptual model that illustrates the genetic and environmental
320 influences that can help shape individual eating traits in children. These include genetic factors
321 such as polymorphisms in a multitude of candidate genes that regulate hunger and fullness
322 during and after a meal as well as evidence for select eating behaviors to be heritable. Together,
323 these factors can confer a genetic susceptibility for impaired satiation and/or hyperphagic eating
324 traits in children. Factors in children's early home environment, which include, but are not
325 limited to, parenting styles, feeding practices, family meals, and grocery shopping experiences
326 can also help shape children's eating traits.

327 The relationship between individual eating traits and the early home environment is likely
328 to be bidirectional in that individual eating traits in children can also influence the type of
329 feeding practices parents use or what stores they frequent to shop for groceries, for example. The
330 greater structural and built environment, which includes the physical home and school
331 environment, children's exposure to advertising, neighborhood characteristics, and the type of
332 restaurants they frequent with their families, can also help shape child eating traits or perceptions
333 of portion size. At the same time, children with a genetic susceptibility to heightened food
334 responsiveness may be actively seeking out environments that offer large portions of palatable
335 foods. Children's response to portion size and accompanied energy intake at meals in turn will in

336 part be determined by eating traits (e.g., experience of satiety / satiation, responsiveness to visual
337 cues), which have been shaped by children's biological endowment and early home environment.

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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	[51, 52]
Weight status (normal-weight vs. overweight/obese)	Yes, No, Maybe	[53-57]
Serving method (self-serve vs. pre-portioned)	Yes and No	[46, 52, 56]
Health foods (fruits and vegetables)	Yes and No	[53, 58]
Interaction with energy density	Yes and No	[59-61]

Table 2: Examples of common gene polymorphisms affecting food intake

Gene	Intake Trait	Reference
AGRP ¹	Macronutrient intake, hyperphagia	[24]
FTO ²	Satiety, energy intake, energy density, eating in the absence of hunger	[23, 25, 26]
CCK ³	Meal size	[22]
Leptin	Extreme snacking behavior, hyperphagia	[22, 27]
MAOA ⁴ , COMPT ⁵	High-sugar, high-fat intake, food reinforcement	[28, 29]
HTR2A ⁶ (rs6314)	Food reinforcement	[29]
PPARG ⁷	Caloric compensation	[21]

¹AGRP = agouti-related protein; ²FTO = fat mass and obesity associated gene; ³CCK = cholecystokinin; ⁴MAOA = monamine oxidase A; ⁵COMPT = catechol-o-methyltransferase; ⁶HTR2A = hydroxytryptamine receptor 2A; ⁷PPARG = peroxisome proliferator-activated receptor gamma

Figure Legend

Figure 1: Conceptual Model for Child Behavioural Susceptibility to Portion Size Response

(Adapted from [62])