Current Nutrition Reports (2020) 9:236–250
https://doi.org/10.1007/s13668-020-00321-5

NUTRITION AND THE BRAIN (J NASSER, SECTION EDITOR)

Neural Mechanisms of Food Decision-Making in Children

Oh-Ryeong Ha[1] · Seung-Lark Lim[1] · Amanda S. Bruce[2,3]

Published online: 27 July 2020

Abstract

Purpose of Review The goal of the current paper is to review the literature on the neural and behavioral factors involved in food decision-making in youth.

Recent Findings Recent neuroimaging studies that employ passive viewing paradigms have found that exposure to food-related cues activate reward, motor planning, and attentional salience signals in children. Greater activations of reward signals and/or lower activations of control signals are associated with overeating and weight gain. Neuroimaging studies with decision-making paradigms have found the reward network in the brain activates during food choices, while control network activates less strongly.

Summary Findings suggest that exposure to food cues activates reward/valuation network, but activation of control network tends to be relatively weaker in children. Hedonic aspects of foods are predominantly considered in children's food choices, and their dietary self-control is not matured yet. The increased activation in reward network and the decreased activation in control network are associated with risk of developing obesity.

Keywords Brain development · Food decisions · Self-control · Value-based decision-making · fMRI · Children

Introduction

Food decisions during childhood can establish lifelong behavioral habits. While caregivers may frequently encourage healthy choices ("eat your vegetables!"), children often prefer calorically dense foods (e.g., candy, soda, chips) with less nutritional value. Compared with adults, self-controlled dietary decisions like choosing healthier but less tasty foods (e.g., vegetables) can be challenging for children whose self-regulation and executive functions are still maturing. Children rarely understand nutrition facts (e.g., calories, carbohydrates) designed for adult consumers, and real-world studies are less supportive of any effect of nutrition labeling on children's food choices [1]. Furthermore, brain development and neural connections in self-control regions such as dorsolateral prefrontal cortex (dlPFC) are not complete until a person reaches his or her mid-20s [2, 3]. Thus, neither serving foods that follow nutritional guidelines nor providing nutritional information is sufficient to prevent childhood obesity. Indeed, one primary limitation of these traditional approaches is that they do not consider how children learn to make independent food choices and exert dietary self-control through their growth and development. To effectively prevent childhood obesity and promote healthy dietary behaviors in children, it is critical to (1) gain a scientific understanding of how children gradually develop to make their own independent food choices at both the behavioral and the neurobiological level and (2) provide appropriate guidance or aids that emphasize children's developmental transition to an independent dietary decision-maker.

Why Do Children's Food Decisions Matter?

Across the world, the prevalence of childhood obesity has dramatically increased during the past several decades [4].

Oh-Ryeong Ha and Seung-Lark Lim contributed equally to this work.

This article is part of the Topical Collection on *Nutrition and the Brain*

✉ Amanda S. Bruce
abruce2@kumc.edu

[1] Department of Psychology, University of Missouri-Kansas City, 5030 Cherry St, Kansas City, MO 64110, USA

[2] Department of Pediatrics, University of Kansas Medical Center, 3901 Rainbow Blvd, Kansas City, KS 66160, USA

[3] Center for Children's Healthy Lifestyles and Nutrition; Children's Mercy Hospital, Kansas City, MO, USA

This is likely due to environmental and lifestyle changes leading to a chronic energy imbalance (intake > expenditure). Childhood obesity is one of the biggest global health challenges of the twenty-first century in both developed and developing countries [5, 6]. Presently, 18.5% of US children and adolescents aged 2 to 19 years are in the obese range [7]. Childhood obesity puts children at higher risk for physical, social, behavioral, and emotional health difficulties, including type 2 diabetes, cardiovascular disease, hypertension, and certain cancers, as well as lower self-esteem, depression, poor academic achievement, social rejection, and discrimination [8–12]. Furthermore, childhood obesity is decisively associated with adulthood obesity [13]. A recent systematic meta-analysis study shows that about 55% of childhood obesity continues through adolescence and about 80% adolescent obesity continues through young adulthood [14]. There are several physiological, psychological, and environmental factors that contribute to childhood obesity, including genetics, metabolism, eating habits, physical activity, economic factors, and social environments. Among these, food decisions ("what to eat") are the primary determinant of children's energy intake. Children's dietary choices frequently do not meet current nutritional recommendations (e.g., highly energy-dense snacks and sugar-sweetened beverages) and therefore increase the child's risk for developing obesity. Given the important role that children's dietary decisions play in preventing childhood obesity, it is important to systematically understand how children learn to make their own food choices and provide the ways in which we can effectively guide children to voluntarily make healthy food choices using knowledge from developmental neuroscience.

"You can lead a horse to water, but you can't make it drink." Providing healthy dietary guidelines (e.g., "MyPlate") and environments is important, but it is *not sufficient* in and of itself, to prevent the development of childhood obesity or stop additional weight gain in children with overweight or obesity. Children must learn to consciously *decide* to eat healthy rather than unhealthy foods. In other words, we may more effectively prevent childhood obesity by encouraging children to be successful self-controllers who can make their own healthy dietary choices. Therefore, this selective qualitative review emphasizes the importance of understanding the developmental and neurobiological underpinnings of children's self-regulated dietary choices to prevent childhood obesity.

Determinants of Children's Food Decisions

Children's food choices can be generally understood as an interaction between internal-biological influences (e.g., interoception of hunger, affective states, genetic factors, hormonal influences, neural mechanisms) and external-social influences (e.g., parental feeding behaviors, peer pressures, food marketing). These internal and external factors inextricably overlap [15, 16]. Among those, to understand how children learn to make their own food decisions, parental influence is of particular importance, especially early in life. Children's food decisions follow a developmental trajectory, beginning with early feeding experiences by their parents. Initially, caregivers have control over their children's environments and make most of the food decisions for their children. Parents may limit foods they offer to their children by parents' own preferences. However, as children grow, they gradually learn to make dietary choices independently, increasingly without their parents' presence (i.e., school cafeteria). Furthermore, children can influence what their parents buy at grocery stores, and what they order in restaurants. Not surprisingly, children's dietary behavioral habits continue to be highly influenced by their parents and family environments [17, 18]. Children often establish their dietary behavior patterns by observing and modeling their parents' behaviors [19]. Also, parents' food-related health literacy, ethnicity, and socioeconomic status can significantly shape children's eating behaviors [20]. Thus, the parental influence and the family environment are critical to understand children's dietary decision-making development [16]. After beginning of formal schooling, the educational environment (e.g., lunch menus in school cafeteria) and peers can play a very important role to shape children's autonomous food decisions [21]. Food marketing is another important factor to be considered. Children are exposed to thousands of food and beverage-related commercials per year, and the vast majority of these advertised foods are not healthy (processed food products which are high in fat, sugar, or salt) [22]. External factors like advertising along with internal factors combine to result in the foods that children choose.

Dietary Self-Control in Children

To prevent obesity, children need to make food choices consistent with maintaining healthy energy balance. This, however, is not simple or easy. Healthier food choices require children's *effective dietary self-control* (i.e., resisting tasty but unhealthy foods) [23], which is executed through complex interactions between brain valuation and control networks [24]. Preadolescence and early adolescence are developmental periods that can be characterized by impulsive, suboptimal behaviors (e.g., unhealthy food intake). Neuroscientific evidence explains this critical period as being due, in part, to differing rates of maturation in limbic (bottom-up; valuation) and prefrontal (top-down; control) systems [25]. Emotion-based limbic regions develop more quickly than frontal regions that help children exhibit self-restraint and control [3, 25, 26]. In the preadolescence period, children do not yet have mature development in the prefrontal cortex. Thus, from a

neurobiological perspective, youth tend to be prone to hedonic (tastiness)-based unhealthy food decisions rather than self-regulated healthy food decisions that emphasize longer-term nutritional consequences.

Brain Imaging and Children's Food Decision-Making

Brain imaging techniques like functional magnetic resonance imaging (fMRI)) and positron emission tomography (PET) are helpful in uncovering the neural bases of appetite and energy balance in humans. Typically, neuroimaging studies in obesity have shown irregularities in a distributed network of reward (valuation) and attention/control regions implicated in motivational signals and cognitive control functions [27, 28]. For example, adults with obesity show significantly greater brain activations in the ventromedial prefrontal cortex (vmPFC), ventral striatum, amygdala, insula, and anterior cingulate cortex when shown pictures of high-calorie foods [29, 30]. Successful weight loss is associated with the levels of activation in the vmPFC (reward signals) and the dorsolateral prefrontal cortex (dlPFC; control signals) [31, 32]. However, the neuroimaging research findings with adults may not be directly generalizable to children and adolescents due to the structural and functional changes that occur throughout brain development that continue until one's mid-20s [2, 3]. Thus, it is necessary to separately investigate developing brain mechanisms of eating behaviors and obesity in children and adolescents. Similarly, while advances in decision neuroscience provide useful insights to understand basic computational and neurobiological bases of dietary decision-making in adults [33], our neuroscientific understanding of children's obesogenic *food choice* mechanisms is still in its infancy. Because the neural response to food stimuli is a dynamic process that interacts with the motivational and cognitive status of an individual rather than a stable or trait characteristic [34], it is critical to investigate how people makes their food choices. The neurocomputational understanding of children's "developing" brain mechanisms of dietary decision-making go beyond what children or parents *report*. This will be critical for providing unique translational information to develop age-appropriate obesogenic dietary decision prevention interventions, along with creating practical guidance to children, parents, educators, and policy-makers.

Previous neuroimaging studies that have examined food intake in children can be categorized into two different types: (1) response to food cue exposure (please see Table 1) and (2) decisions based on food choices (please see Table 2). Here, we summarize children's neuroimaging studies that have been conducted in the past five years (2016–2020).

Exposure to Food Cues

Exposure to food cues can elicit multiple brain responses related to physiological, motivational, and cognitive processes of eating behavior [35, 36]. Reward and valuation circuitry of individuals with overweight/obesity demonstrate hypersensitively to highly palatable (high-caloric) food (particularly when fasting), increasing consumption of foods [37, 38]. Most food cue reactivity studies employed simple passive-viewing paradigms, in which experimental food images (alternating with control images) were presented without a required task for participants. Findings have suggested that children show similar patterns of neural activations to adults while engaging in passive viewing paradigms. That is, studies show increased activations of reward signals on exposure to palatable unhealthy foods in children. They also show decreased activations of control signals in children with obesity risks. For example, exposure to appetizing foods activates regions implicated in reward (e.g., orbitofrontal cortex [OFC], striatum), gustatory processing (e.g., insula, postcentral gyrus), emotion (e.g., amygdala), memory (e.g., hippocampus), and attentional and visual processing (e.g., occipital and parietal cortex) relative to nonfood cues [39]. Exposure to unhealthy food cues activates regions implicated in attention and memory (e.g., inferior frontal gyrus, hippocampus), motor planning (e.g., precentral gyrus), and inhibitory control (e.g., dlPFC) compared with healthy food cues [40]. The greater level of activation in regions implicated in inhibitory control (e.g., medial prefrontal cortex [mPFC], lateral orbitofrontal cortex [lOFC], posterior parahippocampal gyrus [PPHG], dorsomedial prefrontal cortex [dmPFC], dlPFC) on exposure to palatable, high-calorie, unhealthy foods is associated with lower body mass index (BMI) or lower percent body fat [39–41]. When levels of portion size and energy density are varied, exposure to images of high energy-dense foods has activated regions implicated in reward and sensory signals (e.g., insula, fusiform gyrus, thalamus) compared with low energy-dense foods [42, 43]. Exposure to large portion sizes has reduced activations in regions implicated in control compared with small portion sizes (e.g., inferior frontal gyrus), which suggests that exposure to large food portions may suppress inhibitory control that could result in overconsumption [42•]. One study used words (text) for food and nonfood items, and similar to studies using food images, exposure to food words activates regions related to taste and reward signals (e.g., insula, basal ganglia nuclei) compared with nonfood words [44]. Compared with adults, children have shown greater activation in the region implicated in motor planning of ingesting foods (i.e., precentral gyrus) in response to unhealthy relative to healthy food images, which suggests that children may be more susceptible to tempting unhealthy foods than adults [40].

Some studies have adopted a paradigm that delivers liquid foods to participants (e.g., milkshake) after simulating anticipations to foods by viewing food images (e.g., a glass of milkshake or water) as cues for anticipated foods during

Table 1 An abridged summary of recent neuroimaging studies investigating brain responses on exposure to food cues in children

Study	Participants	Topics	Stimuli	Study design	Behavioral findings	Brain regions	Brain activations	Conclusions
Adise et al., 2018	7 to 11 years	Anticipation to food rewards; food intake	Food, money, neutral images (types of anticipated rewards)	Anticipated rewards in winning vs. no winning condition while playing a card-guessing paradigm (fMRI); food intake (ad libitum baseline, overindulgence, satiation conditions); healthy weight vs. overweight group comparison	A greater food intake was found in baseline and overindulgence conditions in children with overweight/obese compared with healthy weight children	mPFC (reward); dlPFC (inhibitory control)	Greater activation in the mPFC for anticipation to food relative to money was positively correlated with food intake in the baseline and overindulgence conditions. Greater activation in the dlPFC for winning food relative to money was positively correlated with food intake in the overindulgence and satiation conditions. No weight status difference was found	Heightened reward value of foods and hypersensitivity to anticipatory food cues are associated with the vulnerability of overeating. The inhibitory system becomes activated in the context of overindulgence and satiation. Being exposed to various temping foods even in the absence of hunger increases overeating, despite an attempt to suppress eating behaviors by exerting inhibitory control
Bohon, 2017	6 to 8 years	Food taste and anticipation to food cue	Images of glasses of beverages (milkshake and water)	Passive viewing of images of glasses of beverages as cues for anticipated tastes of beverages and beverage tasting (fMRI); healthy weight vs. overweight group comparison		Insula (taste), operculum (gustatory processing), precentral gyrus (motor planning), angular gyrus (taste reward), precuneus (attentional salience) and cingulate (reward)	Greater activations occurred in the insula, operculum, precentral gyrus, angular gyrus, precuneus, and cingulate in response to milkshake relative to water in overweight children compared with healthy weight children. Activations to anticipated food cues were not different between groups	Tasting sweet liquid foods activates taste oriented, reward signals in young children with overweight
Carnell et al., 2017	14 to 19 years	Familial obesity risk, energy density (ED)	Words (high ED, low ED foods and nonfood items); food intake	Passive viewing of words representing high ED, low ED, and nonfood items; food intake (ad libitum); weight status and familial obesity risk group comparisons (overweight, healthy weight with obese/overweight mothers, and healthy	The amount of food intake was greatest for the overweight, high-risk healthy weight group, and then low-risk healthy weight group	Insula (taste), ACC (attention allocation, reward anticipation, cognitive control); dlPFC (inhibitory control), and basal ganglia nuclei (reward processing)	Greater activations occurred in the insula and ACC in response to food relative to nonfood words in all three groups. As the risk of obesity increased across three groups, the intensity of activations in the dlPFC, ACC, and basal	Words representing foods activate a reward system. Words representing foods suppress a control system in adolescents as the risk of obesity increases

Table 1 (continued)

Study	Participants	Topics	Stimuli	Study design	Behavioral findings	Brain regions	Brain activations	Conclusions
				weight with healthy weight mothers)			ganglia nuclei decreased in response to high ED relative to low ED foods	
English et al., 2016	7 to 10 years	Portion size; energy density (ED)	Food and nonfood images	Passive viewing of food images varying portion sizes and EDs		Inferior frontal gyrus (inhibition); fusiform gyrus (visual cue processing), insula (taste), superior temporal gyrus (gustatory processing)	Lower activation occurred in the inferior frontal gyrus in response to high PS relative to low PS. Greater activations occurred in the fusiform gyrus and insula in response to high ED relative to low ED. An interaction between PS and ED was found in the superior temporal gyrus	Exposure to large portions of food suppresses inhibitory control. Exposure to high energy-dense foods activates reward and taste processing signals
Fearnbach et al., 2016	7 to 10 years	Portion size; body composition (fat free mass, FFM) and energy density (ED)	Food and nonfood images	Food intake varying PSs and EDs for visit 1–4; passive viewing of high and low ED food images at visit 5 (fMRI)		Thalamus (sensory processing); substantia nigra (reward, motivational drives)	Greater activation occurred in the thalamus in response to high ED relative to low ED. Greater activation elicited in the substantia nigra in response to high relative to low ED was positively correlated with FFM	High energy-dense foods stimulate the gustatory network. Low body fat status is related to higher reward values of high energy-dense foods, which may be linked to the required greater energy consumptions
Gearhardt et al., 2020	13 to 16 years	Impact of food advertising; food intake	Unhealthy and healthier fast-food commercials, nonfood commercials	Passive viewing of commercials (fMRI); commercial preference ratings; food intake in a simulated fast-food restaurant	Unhealthy fast-food commercials were preferred to healthier fast-food commercials. Nonfood commercials (i.e. phone) were preferred to both food commercials. The amounts of unhealthy and healthier fast-food intake were negatively correlated. The hunger level was correlated with total and unhealthy food intake,	Caudate nucleus (reward), hippocampus (memory), anterior cerebellum (sensory motor), precuneus (attentional salience), nucleus accumbens (reward)	Greater activations in the caudate nucleus while viewing unhealthy food commercials compared with nonfood commercials were related to greater total food intake. Greater activations in the nucleus accumbens, hippocampus, and anterior cerebellum in response to healthier relative to nonfood	The exposure to unhealthy food commercials increases overeating through reward systems. Reducing exposure to unhealthy food commercials and enhancing the ability to suppress attentional bias to food cues in commercials will be important for obesity prevention and intervention

Table 1 (continued)

Study	Participants	Topics	Stimuli	Study design	Behavioral findings	Brain regions	Brain activations	Conclusions
					but not with healthy food intake		commercials were related to greater total and unhealthy food intake. Lower activation in the precuneus in response to unhealthy relative to heartier commercials was related to healthier food intake	
Luo et al., 2019	7 to 11 years	Healthy and unhealthy food images	Appetizing food and nonfood images	Passive viewing of food images (fMRI); healthy weight vs. overweight group comparison		OFC (reward), striatum (motivation), insula (taste), postcentral gyrus (taste), hypothalamus (metabolic signaling), amygdala (emotion), hippocampus (memory), occipital and parietal cortex (attention and visual processing); mPFC (cognitive control), lOFC (cognitive control)	Greater activations occurred in the OFC, striatum, insula, postcentral gyrus, hypothalamus, amygdala, hippocampus, occipital cortex, and parietal cortex in response to food relative to nonfood cues. Greater activations in the mPFC and lateral OFC in response to food relative to nonfood cues were related to lower percent body fat when age and sex were controlled	Food cues activate reward and motivation, emotion, memory, taste, and attention and visual processing in children. Activation of control signals to food cues is associated with low fat mass
Masterson et al., 2019	7 to 10 years	Impact of food brands; food intake	Food brands, nonfood brands, control images	Passive viewing of food brands, nonfood brands, and control images (fMRI); food consumption in food packaging with familiar brands, novel brands, and nobrand conditions	There was no energy take difference between food packaging conditions	Lingual gyrus (visual attention), fusiform gyrus (visual cue processing)	Greater activation occurred in the lingual gyrus in response to food relative to nonfood brands. Greater activation occurred in the fusiform gyrus in response to food and nonfood brands relative to control. Lower activation in the fusiform gyrus to food and nonfood brand images was negatively	Food logos activate a stronger attentional bias than nonfood logos. Attenuated activation to brand logos in the fusiform gyrus might indicate susceptibility to food advertising

Table 1 (continued)

Study	Participants	Topics	Stimuli	Study design	Behavioral findings	Brain regions	Brain activations	Conclusions
							correlated with food intake	
Samara et al., 2018	8 to 10 years	High caloric food cues	High-calorie food and nonfood images	Passive viewing of food images (fMRI); healthy weight vs. overweight group comparison		Posterior parahippocampal gyrus (PPHG-memory encoding and retrieval), dmPFC (cognitive control, complex decision making)	Greater activations in the PPHG and dmPFC in response to food images occurred only in healthy weight children	High-calorie food cues activate cognitive control and memory process in healthy weight children, but not in overweight children. Healthy weight children may retrieve and process the value of healthiness formed in past experience
Shearrer et al., 2018	14 to 16 years	Parental obesity risk, the impact of fat and sugar on food intake	Images of glasses of liquid foods (milkshake and water), liquid foods varying in fat and sugar content; food images (appetizing, unappetizing, and glass of water)	Passive image viewing of glasses of liquid foods as cues for anticipated tastes and liquid food tasting (fMRI); passive viewing of food images (fMRI); 3-year follow-up for measuring weight gain	High-obesity-risk adolescents had a significantly higher BMI compared with low-obesity-risk adolescents at the 3 year follow-up, although there was no difference in their baseline BMI between two groups	Anterior insula, lOFC, postcentral gyrus, and ventral precentral gyrus (gustatory and reward processing, motor planning); caudate, central operculum, superior temporal gyrus, juxtapositional lobule, thalamus (gustatory processing, salience, motivation, reward learning)	Greater activations occurred in the anterior insula, lOFC, postcentral gyrus, and ventral precentral gyrus in response to milkshakes (in all fat and sugar combinations) relative to water in high-obesity-risk compared with low-obesity-risk participants. Greater activations occurred in the caudate, central operculum, superior temporal gyrus, juxtapositional lobule, and thalamus in response to high-sugar/low fat milkshake relative to water in high-obesity risk compared with low-obesity-risk participants	Elevated gustatory, somatosensory, striatal, hedonic-based reward processing in response to food cues with high sugar contribute to the increased risk of developing obesity
Stice & Yokym, 2016	14 to 16 years	Food taste and anticipa-tion to	Images of glasses of liquid foods (milkshake and water), liquid	Food reward paradigm: passive image viewing of glasses of liquid foods as cues for	Adolescents who gained body fat over 3-year follow-up showed a higher body fat	Putamen (reward), insula (taste), Rolandic operculum (gustatory	Greater activations occurred in the putamen, insula, and Rolandic operculum in	Elevated gustatory and reward processing in activation to food cues

Table 1 (continued)

Study	Participants	Topics	Stimuli	Study design	Behavioral findings	Brain regions	Brain activations	Conclusions
		food cue; weight gain	foods (milkshake and water)	anticipated tastes and liquid food tasting (fMRI); money reward paradigm: passive image viewing of three coins followed by winning or losing outcomes; food and monetary reinforcement paradigms (behavioral); pleasanting, wanting, and familiarity ratings of beverages; 3-year follow-up for measuring weight gain	percentage, BMI, and milkshake wanting ratings compared with those who lost body fat. Over time, adolescents who gained body fat showed a significant increase in milkshake wanting and a decrease in working time to earn food and monetary rewards	processing); precuneus (attentional salience)	response to anticipation to milkshake relative to water in adolescents with body fat gain compared with those with stable body fat. Greater activation occurred in the precuneus in response to anticipation to milkshake relative to water in adolescents with body fat gain compared with those with body fat loss or stable body fat	are related to body fat gain
Stice & Yokym, 2018	14 to 16 years	Food taste and anticipation to food cue; Weight gain	Images of glasses of liquid foods (milkshake and water), liquid foods varying in fat and sugar content; food images (appetizing, unappetizing, and glass of water)	Passive image viewing of glasses of liquid foods as cues for anticipated tastes and liquid food tasting (fMRI); passive viewing of food images (fMRI); 3-year follow-up for measuring weight gain	11.3% of participants developed overweight or obesity over 3-year follow-up	Pre-SMA (motor inhibitory control); precentral gyrus (motor planning), Rolandic operculum (gustatory processing); vmPFC (reward)	Lower activation in the pre-SMA in response to high-fat/low-sugar milkshake relative to water predicted BMI gain and lower level of dietary restraint. Greater activations in the precentral gyrus and Rolandic operculum in response to appetizing food images relative to control predicted BMI gain. Lower activation in the vmPFC in response to appetizing relative to unappetizing food images predicted BMI gain	Lower motor inhibitory control in activation to high-calorie food taste contributes to weight gain. Increased activations of motor planning and oral somatosensory processing in activation to palatable food cues are risk factors for overeating and weight gain. Elevated reward region signal was not evidenced for future weight gain
van Meer et al., 2016	10 to 12 years; adults (parents)	Unhealthy and healthy food cues; healthiness and liking attributes	Healthy and unhealthy food images	Passive viewing of food images (fMRI); healthiness and liking ratings: children and adults (parent) comparison		Inferior frontal gyrus (attentional control), precentral gyrus (motor planning), hippocampus (memory), dlPFC (inhibitory control)	Greater activations occurred in the inferior frontal gyrus, precentral gyrus, hippocampus, and dlPFC in response to unhealthy relative to healthy food images in	Unhealthy food cues activate stronger attention, memory processing, and motor planning (ingesting of food) compare with healthy food cues in children. Activation of

Table 1 (continued)

Study	Participants	Topics	Stimuli	Study design	Behavioral findings	Brain regions	Brain activations	Conclusions
							children. Greater activation in the dlPFC in response to unhealthy food images was related to lower BMI in children. Greater activation occurred in the precentral gyrus in response to unhealthy relative to healthy food images in children compared with adults	inhibitory control in response to unhealthy foods is associated with lower BMI

mPFC, medial prefrontal cortex; dlPFC, dorsolateral prefrontal cortex; ACC, anterior cingulate cortex; OFC, orbitofrontal cortex; lOFC, lateral orbitofrontal cortex; PPHG, posterior parahippocampal gyri; pre-SMA, pre-supplementary motor area; vmPFC, ventromedial prefrontal cortex;

Table 2 An abridged summary of recent neuroimaging studies investigating neural mechanisms of food decision-making in children

Study	Participants	Topics	Stimuli	Study design	Findings	Brain regions	Brain activations	Conclusions
Bruce et al., 2016	8 to 14 years	Food choices; taste and health attributes; commercials	Food and nonfood commercials; healthy and unhealthy food images	Food choices after watching commercials (fMRI); taste, healthiness, preference ratings	The importance of taste and decision times in food choices were increased after watching food commercials. Health ratings did not but taste ratings significantly predicted food choices	vmPFC (reward)	Greater activation in the vmPFC occurred at the time of food choices after watching food commercials compared with nonfood commercials. Greater activation in the vmPFC in response to food relative to nonfood commercial was related to the higher hunger level	Taste attribute (immediate reward) predominantly determines food choices. Unlike adults, health attribute does not determine decisions. Exposure to food commercials provokes youth to make eating decisions more impulsively while incorporating more hedonic aspects of foods (i.e., taste) through reward value computation systems. Taste-oriented, impulsive eating decisions become profound as there is an increase in hunger
Lim et al., 2016	8 to 14 years	Food choices (own and projected maternal decisions); taste and health attributes	Healthy and unhealthy food images	Food choices: (1) own choice (children's own food decisions) and (2) projected mom's choice (projected maternal food decisions for them) (fMRI); taste, healthiness, preference ratings	Taste attribute predicted own food choices; both health and taste attributes predicted projected maternal food choices for children. Both own food preferences and projected maternal food choices predicted children's own food choices	vmPFC (reward), dlPFC (inhibitory control)	Own food choices: activation in the vmPFC was positively correlated with own food preferences. Activation in the dlPFC was positively correlated with projected maternal food choices for them. The peak of the dlPFC signal was delayed relative to that of the vmPFC signal in the course of decision times. The dlPFC showed negative (inhibitory) functional connectivity with the vmPFC. The greater inhibitory functional connectivity between vmPFC and dlPFC was related to higher BMI and lower self-control scale scores. Projected maternal food choices: the activation in the dlPFC was positively correlated with the projected maternal food choices for them, not with	Reward value (preferences) and inhibitory control (projected maternal guidance) determine eating decisions. The initially activated, primary reward signal is suppressed by the inhibitory signal that is stimulated by projected maternal guidance for food choices. The regulatory role of perceived maternal guidance is especially important when the inhibitory control is not fully internalized, and health attribute, a determinant for healthy eating decisions in adults, is not meaningfully incorporated in the food decision-making process yet

Table 2 (continued)

Study	Participants	Topics	Stimuli	Study design	Findings	Brain regions	Brain activations	Conclusions
							health attribute. The vmPFC activation in the projected maternal food choices was not significant. The peak of the dlPFC signal was faster in projected maternal food choices relative to own food choices	
van Meer et al., 2017	10 to 12 years; adults (parents)	Food choices; taste and health attributes	Healthy and unhealthy food images	Food choices: making food choices based on preference in three attention conditions, (1) health (2) taste (3) natural (whole aspects of foods) (fMRI); taste and healthiness ratings	In the natural condition, taste rating was a positive predictor of food choices (yes choice) in both children and adults. Health rating was a positive predictor of food choices in adults, but a negative predictor of food choices in children. In the health condition, both children and adults made healthier choices	vmPFC (reward), mPFC (reward), dlPFC (inhibitory control)	Across all attention conditions, greater activation occurred in the vmPFC in response to the yes relative to the no choice in both children and adults. Greater activation occurred in the dlPFC in response to the yes relative to no choice in adults. The dlPFC activation was weaker in children in food choices compared with adults. Greater activations in the vmPFC were related to higher taste rating in children and adults. While attending to food healthiness, greater activation in the mPFC was related to higher health rating in adults, but to lower health rating in children. Considering food healthiness increased connectivity between dlPFC and mPFC in adults, but not in children	Reward signals mainly involve in children's food choices. Tasty foods activate reward signals in both children and adults. When considering health aspects of foods, adults find healthy foods rewarding, but children do not. Children are more likely to make food choices predominantly by taste attributes, while control signals are weak in children's food choices

vmPFC, ventromedial prefrontal cortex; dlPFC, dorsolateral prefrontal cortex; mPFC, medial prefrontal cortex

scanning [45–48]. In addition, scientists have investigated the relationship of neural activations and excessive weight gain. Findings suggest that anticipations to food tastes and the actual tasting of sweet and high-fat foods activate hedonic reward signals and suppress inhibitory signals, which all contribute to an increased risk of developing obesity. For example, tasting foods varying in sweet and fat levels have activated regions implicated in hedonic gustatory processing (e.g., insula, operculum, caudate, thalamus, angular gyrus, superior temporal gyrus, cingulate) and motor planning (e.g., precentral gyrus) compared with control beverage (water) [45–48]. Attenuated activation in the region implicated in motor inhibitory control (e.g., pre-supplementary motor area [pre-SMA]) in response to high-fat food taste is associated with weight gain and lower level of dietary restraint [48]. Anticipations to foods have activated regions implicated in gustatory processing (e.g., insula, Rolandic operculum), reward/motivation (e.g., putamen), attentional salience (e.g., precuneus), and motor planning (e.g., precentral gyrus) compared with anticipation to control [45–48]. Greater activations of these regions occur in children with overweight [45], youth with parental obesity risk [46••], and youth with body fat and BMI gain over 3-year follow-up [47, 48]. In one study, instead of using passive viewing paradigms, anticipations to food rewards have simulated using a card-guessing paradigm during fMRI scanning, and actual amounts of food consumption have measured in various meal conditions (i.e., ad libitum, overindulgence, and eating in the absence of hunger) [49]. Still, anticipations to food rewards have activated the regions implicated in the reward signals (e.g., mPFC) compared with monetary rewards, and greater activation in the reward system is associated with overeating.

Food commercial exposure is another important variable in a comprehensive understanding of children's eating behavior, because food marketing often targets children and the vast majority of advertised food and beverage products do not meet nutrition recommendations [50]. Recent findings have suggested that food commercials activate hedonic reward systems and children are susceptible to attentional bias for food brand logos, which increase vulnerability to overeating and weight gain [51, 52]. For example, exposure to fast-food commercials in passive viewing paradigms has activated the regions implicated in rewards signals (e.g., caudate, nucleus accumbens), memory (hippocampus), and sensory-motor processing (e.g., anterior cerebellum) compared with nonfood commercials [51]. Exposures to food brands have activated regions implicated in attention salience (e.g., lingual gyrus) compared with nonfood brands [52]. Lower activations in regions implicated in visual cue processing (e.g., fusiform gyrus) in response to both food and nonfood brands are related to greater food intake. More research is necessary to investigate the breadth of the influence that food marketing has on children's brain activations, subsequent food choices, and prospective excess weight gain (development of obesity).

Food Choices

Only a few studies have investigated the neural mechanisms of food *decision-making* in children. Food decision-making paradigms employ food choice tasks that require participants to make food choices while viewing food images during scanning. Health and taste attributes are important determinants of food choices in adults [24]; thus, food healthiness and taste ratings on food images presented during food choice tasks are used to predict eating decisions at the individual level in children as well. Recent findings on children's food decision-making have supported that limbic regions (valuation network) develop earlier than prefrontal systems (control network) [25], which hinders exertion of dietary self-control in demanding healthy food choices. In children, similar to adults, food decision-making activates the reward/valuation network (e.g., vmPFC, mPFC) for computing reward values of foods at the time of choices [53–55]. Yet, in the course of food choices, activation of control network (i.e., dlPFC) is delayed compared with that of the valuation network, which challenges healthy food choices due to delayed incorporation of health attributes [53••]. Considering the health aspects of foods during food choices increases the reward value of healthy foods in adults; however, attending to health attributes does not change (increase) the reward value of healthy foods in children [54]. Moreover, food choices are predominantly determined by perceived food taste and preferences at where the activation of cognitive network is much weaker than that of adults [53–55]. Thus, children are highly susceptible to appetitive cues including food commercials given that mostly hedonic aspects of food (taste) are incorporated through activation of reward network, while the activation of inhibitory network is either lacking or weak [55•]. Interestingly, in our recent neuroimaging study [53••], food decision-making based on perceived maternal choices for children (i.e., would *my mom* like me to eat this?) activates the control network (i.e., dlPFC). In addition, the activation timing of the control network is much earlier in the time course of food choices compared with the timing in own food choices. In other words, children's brains encode and utilize their caregiver's preferences when children make food choices. This *neural internalization of parental regulation* encoded in the dorsolateral prefrontal cortex of children's brains may serve as a critical self-regulator of children's decision-making, leading to more optimal dietary choices in the preadolescent period.

Promoting Children's Dietary Self-Control

Despite the increase in prevalence of childhood obesity over the past several decades and the major public health problems

associated with it [4], obesity prevention and intervention programs for children are still limited or have only modest effects on BMI changes. Even if there are effects on BMI, they are not sustained [56, 57]. Related to children's food intake, restrictive parental feeding practices have little effect on childhood obesity prevention. In fact, it often counterproductively increases children's uninhibited consumption of forbidden foods (e.g., candy) or obstinate rejection of pressured foods (e.g., vegetables) [58]. Thus, it is important to encourage and help children to make healthy choices *voluntarily* by enhancing children's dietary self-control. Scientific evidence suggests that self-control is modifiable [59], and researchers are actively exploring how to improve children's self-control skills using a variety of interventions [60–62]. Also, positive child-parent relationships support children's successful development of self-regulation [63, 64]. Parents or other family members can encourage children's self-regulation through behavioral modeling, interactive responses to children's behavior, assistance, and motivating children's behavior through rewards and punishments [65, 66]. Thus, especially, for young school-age children who typically begin to make their own food choices, dietary self-control training to take to take their parent's perspective [67] (i.e., would *my mom* like me to eat this?) in positive child-parent relationships at both behavioral and neural levels may hold strong promise to promoting resilience to childhood obesity [68].

Conclusions

This selective review focuses on some of the known behavioral and neural mechanisms of food decision-making in youth. Despite recent advances in our understanding of food choices, and how these change over time for youth, much is still unknown. In addition, just understanding the determinants of food intake and eating behavior in and of itself is not sufficient to translate our scientific discoveries to clinical practice and interventions designed to produce significant behavior change. In order to effectively prevent and treat childhood obesity, a more comprehensive and interdisciplinary approach including decision-making about other health behaviors (physical activity, sedentary time) will be necessary. Finally, it will be crucial to examine some of the interpersonal (trans-generational) and cultural factors related to energy intake and expenditure. This will allow us to then develop tailored, individualized interventions—precision medicine—approaches to improving health behavior choices in youth.

Compliance with Ethical Standards

Conflict of Interest All authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent All reported studies/experiments with human or animal subjects performed by the authors have been previously published and complied with all applicable ethical standards (including the Helsinki declaration and its amendments, institutional/national research committee standards, and international/national/institutional guidelines).

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- • Of importance
- •• Of major importance

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systems in response to sweet liquid foods were greater in adolescents with parental obesity risk compare to those without parental obesity risk. Increased level of reward network activation is related to high-obesity-risk.

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