Review

Neural correlates of pediatric obesity

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Abstract

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Objective. Childhood obesity rates have increased over the last 40 years and have a detrimental impact on public health. While the causes of the obesity epidemic are complex, obesity ultimately arises from chronic imbalances between energy intake and expenditure. An emerging area of research in obesity has focused on the role of the brain in evaluating the rewarding properties of food and making decisions about what and how much to eat.

Method and Results. This article will begin by reviewing some of the challenges associated with neuroimaging in children and the relevant developmental brain changes that occur in childhood and adolescence. The article will then review the current literature on neural mechanisms of food motivation and the ability to delay gratification in children and how these responses differ in obese compared to healthy weight children.

Conclusion. Increasing our understanding about how brain function and behavior may differ in children will inform future research, obesity prevention, and interventions targeting childhood obesity.

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Abbreviations: ACC, anterior cingulate cortex; BMI, body mass index; fMRI, functional magnetic resonance imaging; HW, healthy weight; OFC, orbitofrontal cortex; PFC, prefrontal cortex.

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Introduction

The prevalence of childhood obesity has reached crisis proportions. Mirroring trends in adult obesity, the prevalence of obese and overweight youths in the United States is increasing rapidly. Recent estimates show that approximately one in three youths is overweight (body mass index [BMI] 85–95% for age and sex) or obese (BMI >95% for age and sex) (Ogden et al., 2006). Obesity contributes to a wide range of physical and emotional problems, including cardiovascular disease, diabetes, hypertension, depression, and some forms of cancer (Mokdad et al., 2003; Must et al., 1999). Childhood obesity is a grave concern, as many speculate that current youth will live unhealthier and shorter lives than their parents (Daniels, 2006).

Obesity results from a chronic imbalance between energy intake and energy expenditure. The accumulation of excess weight occurs when the calories consumed chronically surpass the amount of calories expended through homeostasis and physical activity. The brain plays a fundamental role in modulating hunger and regulating motivated behaviors such as eating (Berthoud and Morrison, 2008). It is imperative that we gain a better understanding of the neural mechanisms involved in childhood obesity and weight loss so that prevention and intervention strategies will be more effective in
curbing the childhood obesity epidemic. Two important brain functions related to eating are the motivation toward the rewarding properties of food and decision-making regarding food consumption—these will be the focus of the current review.

Many of the brain regions involved in reward processing and decision-making are developing throughout childhood and adolescence. This article will, therefore, begin by discussing brain development as it relates to health behaviors and reviewing challenges associated with conducting brain research in youth. Brain development, specifically prefrontal cortical development, has important implications for self-control and impulsivity. The behavioral manifestations—impulsivity, self-control, and the ability to delay gratification—will then be discussed in relationship to pediatric obesity. We will discuss functional neuroimaging studies examining neural mechanisms of energy intake (food motivation) in children and obese children. Next, we will review the few studies examining the brain mechanisms of energy expenditure (physical activity) in children. This article will conclude with directions for future research targeting brain mechanisms of pediatric obesity.

The developing brain

Before delving into the neuroimaging studies examining brain responses of food motivation in children, it is necessary to understand some of the challenges associated with pediatric neuroimaging studies. One of the challenges relates to variability in brain development, even among children of similar age. Throughout childhood and into early adulthood, the human brain undergoes significant changes. By the age of 6, a child's total brain size is 90% of the adult brain size yet neural changes, cell migration, changes in synaptic density, and development of myelin continue into adulthood (Bourgeois et al., 1994; Casey et al., 2005; Conel, 1939–1963; Huttenlocher, 1979; Huttenlocher, 1990; Yakovlev and Lecours, 1967). Some of these changes are not apparent using current functional neuroimaging techniques.

Many research studies of human participants have used functional neuroimaging as a useful method of studying the brain regions involved in certain aspects of cognition. Functional magnetic resonance imaging (fMRI) is a neuroimaging method that provides a safe (no ionizing radiation exposure) and noninvasive method to study human cognition. fMRI measures the hemodynamic response, or changes in blood flow and oxygenation, associated with neural activity while a person is engaged in a certain task (i.e. viewing visual stimuli, decision-making, memory tasks). Using functional neuroimaging and specifically fMRI in pediatric populations presents unique challenges (e.g. Bookheimer, 2000; Byars et al., 2002; Casey et al., 2000, 2005). For example, children are smaller than adults and shorter necks can mean their heads are not centered in the radiofrequency receiver coil, resulting in sub-optimal contrast-to-noise ratio, potentially underestimating brain activation (Bookheimer, 2000). Another practical concern is children's difficulty remaining still during the scan. This can often be addressed by having the child do a practice session in a mock scanner that is the same size and produces the same noise as the actual MRI, or through other training and habituation experiences.

There are also underlying anatomical differences between adults and children that can pose challenges to fMRI data analysis. The ratio of gray to white matter decreases with age. Children experience myelination at different rates, potentially resulting in variability in the magnitude of activation (Bookheimer, 2000), even in children of similar age. Conceptually, if subject groups under comparison are not matched for performance levels, observed activation differences could be due to the difficulty and/or the perceived difficulty of the task (Kotsoni et al., 2006).

Perhaps most relevant to studies of eating behavior and obesity, neuroimaging studies consistently indicate that the prefrontal cortex (PFC) is among the last brain regions to mature (Casey et al., 2000). The PFC is an area of the brain that, among other functions, helps to control behavior, inhibits impulsive responses, and evaluates and makes decisions about stimuli in the environment. A recent theoretical model specific to youth posits that the increase in risk-taking behavior seen in youth is due to an uneven neurobiological development in brain regions (See Fig. 1; Somerville and Casey, 2010). Choices, and particularly those choices that may have negative health consequences, are biased in favor of perceived immediate rewards (Somerville and Casey, 2010; Somerville et al., 2010; Casey et al., 2008a,b; Fareri et al., 2008). Research using both animal and human samples supports this framework. Health-related decision making in children is also affected by PFC and corresponding control processes that are not yet fully developed. Therefore, without the necessary inhibitory processes to aid in decision-making, youth are particularly susceptible to making poor health behavior choices and these differences may be particularly pronounced when evaluating appetitive cues (Somerville et al., 2010).

Many functional neuroimaging studies of children are data-driven rather than theory-driven. Working within the framework of a theory, such as described above, provides distinct advantages and offers the opportunity for a more complete understanding of the brain's role in behavior. The first fMRI study using children was published in 1995 (Casey et al., 1995), and since that time, over 600 studies have been published. Researchers have and continue to successfully manage the challenges associated with using functional neuroimaging techniques in children (e.g. Crone et al., 2010).

Components of healthy decision making

As described above, one cause of obesity is a chronic imbalance between energy intake and energy expenditure. In order to avoid an imbalance of energy, individuals must evaluate the rewarding properties of food (taste, texture) in comparison with the nutritional

**Fig. 1.** Illustration of striatal and prefrontal interactions across development. Deeper color indicates greater regional signaling. Line represents functional connectivity, with solid line indicating mature connection and dotted line indicating immaturity. Reprinted with permission from publisher.
value of the food (nutrients and caloric content). Next, individuals must make decisions about which behaviors to engage in. That is, they make choices, such as whether to eat (energy intake) or to go play or exercise (energy expenditure). Recent research has focused on the differential contributions of the PFC and limbic system to child and adolescent behavior (Somerville and Casey, 2010). The development of “drive” regions (limbic system and ventral striatum) and “control” regions (PFC) is particularly relevant to decision-making about health behaviors. Choosing a carrot instead of a cookie, or choosing to exercise instead of watching a favorite movie, requires self-control and the ability to make decisions based on long-term rewards. McClure and colleagues (2004) describe a model, emphasizing the role of the limbic system role in processing immediate rewards, and the role of PFC in choosing larger, delayed rewards. The neural networks of “drive” originate in the ventral tegmental area and project throughout limbic system, basal ganglia, and neocortex. The projections to the ventral striatum (including nucleus accumbens) are crucial to reward processing (Cardinal et al., 2002; Duffy, 1997; Rolls, 1997). The reward regions of the brain are typically considered ventromedial PFC, amygdala, and basal ganglia (ventral and dorsal striatum; Van Leijenhorst et al., 2010a). Relative to the majority of prefrontal control regions of the brain, these drive regions develop at an earlier age (See Fig. 1; Somerville and Casey, 2010). In particular, youth are susceptible to appetitive cues, meaning that if one choice has a particularly alluring outcome, their ability to inhibit such a response is much weaker. A recent study showed that compared to pre-adolescents and young adults, there is a peak in ventral striatum activation in mid-adolescence in response to reward (Van Leijenhorst et al., 2010a). These findings are consistent with a growing number of fMRI studies demonstrating the relatively hyperactive reward regions of the brain in adolescence: (Galvan et al., 2006; Geier et al., 2010; Van Leijenhorst et al., 2010b).

These areas of “drive” and “control” have also been implicated in fMRI studies of food motivation (Bruce et al., 2010). On the inhibitory side, the PFC is vital to successful decision-making. The PFC receives input from a variety of sources to determine the value of a potential reward and mobilize the behavioral action necessary or control response inhibition (Bechara et al., 2000; Tanji and Hoshi, 2008; Wallis, 2007). Control areas of the brain help to inhibit impulsive decisions in favor of deliberate, rational, calculated decisions. Areas of prefrontal cortex are commonly considered as areas of higher order reasoning (Balleine et al., 2007; Rubia et al., 2006; Delgado et al., 2004). As described above, these are the last areas to develop, not reaching full maturity until the mid 20’s. Youth, therefore, do not have the benefit of fully matured regulatory regions of the brain. Structurally and functionally, these brain regions do not mature until early adulthood (Luna et al., 2001; Giedd et al., 1999). Once PFC regions are more developed, the prevalence of risk-taking behaviors such as drug and alcohol experimentation, deviant behavior, and unsafe sex practices, decreases (Spear 2000; Eaton et al., 2008).

Only a few neuroimaging studies have simultaneously examined the reward processing and control brain regions in youth, observing relative activity and interactions between regions. One way to do this is to examine functional connectivity. Somerville and colleagues (2010) determined that the connectivity between “drive” and “control” areas is weaker in younger people (the sample ranged from age 6 to 29). In addition, findings suggest that with proper incentives, adolescents are able to improve cognitive control (Geier et al., 2010; Hardin et al., 2009). These findings are encouraging, suggesting that cognitive control and, therefore, self-control can be improved such that adolescents could reap the potential health benefits of more controlled decision-making.

Results from neuroimaging studies have important implications for regulating behaviors, particularly health behaviors. If one’s ability to inhibit impulses is weaker, it makes it more difficult to resist eating that second or fifth cookie. In relation to obesity, children and adolescents may have both increased sensitivity to cues and reduced cognitive control needed for effective behavioral regulation. This means that parents, schools, and policy makers may need to take a more active role in providing healthy food options and physical activity opportunities for youth.

Food motivation and “drive” brain regions

Food motivation and eating behaviors are inextricably linked to behavioral self-control and the ability to delay gratification. The brain is instrumental in controlling hunger and associated eating behaviors. Both homeostatic and motivational processes contribute to ingestive behaviors. The hypothalamus and caudal brain stem help control homeostasis, detecting internal cues and adjusting appetite/hunger accordingly (Berthoud and Morrison, 2008). Homeostatic eating refers more to how much a person needs to eat to maintain an adequate energy balance. As for how much a person wants to eat, limbic and paralimbic brain regions are implicated in the hedonic aspects of food motivation and play an important role in reward, motivation, decision-making, inhibition, and cognitive control (Zheng and Berthoud, 2007). Using visual, olfactory, and gustatory stimuli paradigms, functional neuroimaging studies have examined neural mechanisms of food motivation in adults (e.g. Dell’Angi et al., 2002, 2005; LaBar et al., 2001; Simmons et al., 2005; Tataranni et al., 1999).

Using functional neuroimaging to examine food motivation is a relatively new field. Research initially focused on food motivation and adults, but applying the same techniques to children is innovative. The brain regions most commonly associated with food motivation in healthy weight adults are those associated with taste (insula), motivation/reward (orbitofrontal cortex [OFC], medial PFC, anterior cingulate cortex [ACC], amygdala, striatum) and behavioral control (lateral PFC) (Gordon et al., 2000; Killgore et al., 2003; LaBar et al., 2001; Morris and Dolan 2001; O’Doherty et al., 2002; Rothemund et al., 2007; Simmons et al., 2005; Tataranni et al., 1999).

Only a few studies have examined the neural mechanisms of food motivation in children. In one study, healthy weight children aged 10–17 showed significant amygdala, medial PFC, ACC, and insula activations to visual food stimuli during a pre-meal (hungry) condition that decreased after eating (Holsen et al., 2005). These findings closely replicate previous results in adults and indicate normal patterns of neural activity of food motivation likely begin in childhood. Another study examined brain activations to high and low calorie food images in female youth ages 9–15 (Killgore and Yurgelun-Todd, 2005). Images of high calorie food produced activation in hippocampus and subgenual cingulate. Age was positively correlated with activation in the OFC but negatively correlated with brain activation in the ACC. The brain regions identified in these studies that are involved in processing appetizing stimuli were the same as those in drive regions that have been implicated in reward processing.

Delayed gratification and “control” brain regions

The observed neurobiological and cognitive development in childhood and adolescence is mirrored in behavioral differences. In general, adolescents engage in more risk-taking behaviors, ignoring the potential negative consequences of actions in pursuit of the immediately rewarding, pleasurable aspects of behavior. Eating unhealthy foods and forgoing exercise are two types of health behaviors associated with long-term negative consequences. Intuitively, the ability to forego an immediate pleasurable reward for a postponed benefit should be related to health outcomes such as obesity.

In fact, the relationship between obesity and delayed gratification in adults is well documented (Brogan et al., in press; Davis et al., 2004; Pignatti et al., 2006). Recent research more closely investigates the relationship between delayed gratification and obesity in children. In general, studies show overweight and obese children are more impulsive and less able to delay gratification than healthy weight
children. Cross-sectional studies have determined that obese children are more impulsive than HW children (Braet et al., 2007; Nederkoorn et al., 2006). However, evidence is mixed regarding whether obese children’s difficulties delaying gratification apply specifically to edible rewards (Bonato and Boland, 1983) or apply more generally to all types of rewards including nonedible rewards (Sigal and Adler, 1976). Although one longitudinal study showed that impulsivity can predict weight loss, more impulsive children lost less weight in a behavioral intervention (Nederkoorn et al., 2006); another study found the opposite (Pauli-Pott et al., 2010). These two studies used different measures of impulsivity, and Nederkoorn and colleagues (2006) used a sample of exclusively preadolescents, while Pauli-Pott and colleagues (2010) included youth up to age 15.

The majority of these studies use either self-report, caregiver-report, or experimental cognitive tasks to assess impulsivity and self-control. A recent study used an ecologically valid measure of delayed gratification to test the hypothesis that preadolescents with higher BMI are less likely to delay gratification (Bruce et al., in press). During a family-based obesity intervention, participants in this study earned a point each week if they completed their goals worksheet. They could spend that point immediately on a small toy prize (e.g., stickers) or save points to use on a larger prize (e.g., basketball). A higher BMI percentile was associated with reduced point savings. These results support the theory that obese children are less likely to delay gratification than overweight and healthy weight children. Even for nonfood rewards, preadolescent children with higher BMIs preferred the immediate reward over the delayed, larger reward (Bruce et al., in press).

Only a few longitudinal studies have been conducted on the relationship between self-control and pediatric obesity. Children with more self control, as measured by self-report and parental-report measures, tend to remain leaner in the transition to adolescence (Duckworth et al., 2010; Francis and Susman, 2009). One study used the classic “marshmallow” Mischel test (Mischel and Ebbesen, 1970) at age 4 and determined that children who were less able to delay gratification were at a increased risk of being overweight at age 11 (Seevaye et al., 2009). A recent study used an even younger sample to demonstrate that self-regulation skills as early as 2 years of age can predict the risk for becoming overweight/obese at age 5 (Graziano et al., 2010). More research is needed to clarify the relationship between the ability to delay gratification and obesity. This has important implications for developing specific strategies within obesity prevention programs or obesity treatments aimed at improving delayed gratification.

**Studies comparing obese and healthy weight groups**

As mentioned above, functional neuroimaging investigation of food motivation and obesity is a new area of research. Very few studies have examined obese children’s brain responses in comparison to healthy weight children’s brain responses. However, research has shown that obese individuals find food more reinforcing and, therefore, exhibit higher levels of food motivation, than healthy weight individuals (Epstein et al., 2007; Saelens and Epstein, 1996). Functional neuroimaging studies of obese adults have observed differential activation between obese and healthy weight groups in brain regions associated with food motivation. DelParigi and colleagues (2002) scanned obese and HW participants in two sessions: when hungry and while consuming a liquid meal. Upon tasting liquids while hungry, the obese group showed greater increases in regional cerebral blood flow to liquid tasting in insula and cingulate cortex. Post-prandial differences were observed in hippocampal formation, cingulate cortex, and amygdala. Rothemund and colleagues (2007) used fMRI to compare obese and HW women in response to visual food stimuli differing in caloric density. Obese women showed increased dorsal striatum activations in response to high calorie food images when compared to HW controls. The dorsal striatum is involved in reward anticipation and the authors hypothesized this increased activation contributes to overeating and obesity.

Recent findings from our group (Martin et al., 2010) were consistent with these studies. We used fMRI to examine changes in the hemodynamic response in obese and healthy weight adults while they viewed food and non-food images in pre-meal and post-meal states. During the pre-meal condition, obese participants showed increased activation, compared to healthy weight participants, in ACC and medial PFC. During the post-meal condition, obese participants also showed greater activation than healthy weight participants in the medial PFC. These results indicate that obese groups are hyperresponsive to food cues in reward processing areas, and may have implications for understanding brain mechanisms contributing to overeating and obesity, and variability in response to diet interventions. Overall, these studies demonstrated that obese individuals exhibit increased food motivation and subsequent brain activations in rewarding “drive” areas.

Researchers have also examined associations between brain responses and behavioral measures. Subjective ratings of appetite correlate with insular activation (Porubská et al., 2006) and a behavioral measure of reward sensitivity correlates with ventral striatum, amygdala, and orbitofrontal cortex activation (Beaver et al., 2006). In our recent study, we found that for the obese group of participants, self-report measures of disinhibition negatively correlated with ACC activations in the premeal condition (Martin et al., 2010). In addition, self-report measures of hunger positively correlated with medial PFC activations in the premeal condition.

Neuroimaging of food motivation in children is beginning to appear in the literature (e.g. Bruce et al., 2010; Holsen et al., 2005). A recent study reported that obese children show higher activation of dorsolateral PFC than HW children in response to food images, which they hypothesize to be associated with increased inhibitory activation in the obese group (Davids et al., 2010). It is possible that the increased activation to food cues in control areas could be due to less mature, less focused brain activity than that of healthy weight peers. Notably, research indicates that, during normal development, maturity is associated with more focused patterns of brain activation (Luna et al., 2001). Our results (Bruce et al., 2010) were consistent with the above studies as obese children showed PFC and OFC hyperactivation compared to HW children in response to visual pictures of appetizing food when hungry. After eating, the obese children’s brains remained hyperactivated compared to healthy weight children’s brains in PFC, OFC and visual processing areas. When comparing premeal to postmeal brain activations, the healthy weight children showed decreased activation in medial PFC, PFC, cingulate, insula, nucleus accumbens, ventral putamen, and amygdala. In the obese group, however, only PFC and insula decreased after eating; significant changes were not observed in reward processing regions. The healthy weight children were modulating brain responses, but the obese children did not show that same modulation.

There are some discrepancies between several published studies, with some results identifying areas of increased activity and others observing decreased activity in reward regions. For example, Stice and colleagues (2008) observed decreased activity in obese youth in striatum compared to healthy weight individuals. They proposed that obese individuals may have reduced dopamine receptor density and impaired dopamine signaling in reward regions of the brain. It should be noted that this study scanned young adults during actual feeding, while the majority of the other studies examined youth while viewing food images. Future studies are clearly needed to elucidate the brain mechanisms of “drive” related to eating behaviors.

No studies have yet been conducted comparing pediatric brain activation to food cues in “control” regions with behavioral measures of impulsivity or self-control. Little is known about how activity in these regions may correspond with general aspects of impulsivity or the ability to delay gratification related to health behaviors.
Takentogether, results from the pediatric obesity neuroimaging studies suggest that neural networks of food motivation are differentially responsive in obese groups, even in childhood, though longitudinal studies are needed to better characterize this process. It is not yet known if this develops over time, or if it is an individual difference present early in life due to hereditary factors.

**Physical activity and the pediatric brain**

Another approach to understanding the relationship between obesity and the brain is to examine associations between energy expenditure (e.g., physical activity) and brain function. Examining the relationship between physical activity and brain function in children is also a very new field. Only a few studies have looked at brain structure and function, measures of intellectual ability, and physical fitness. Hillman and colleagues (2005, 2009a,b) have examined the relationship between physical activity and cognitive functioning in children. Studies show that cardiovascular fitness generally, and acute bouts of exercise specifically, positively influence neuropsychological functioning. Children who are more physically fit perform better on tests of self-regulatory processes including cognitive control (Buck et al., 2008; Hillman et al., 2009b). Another study demonstrated improved executive function with exercise in overweight children (Davis et al., 2007). These findings are consistent with neuroimaging studies of food motivation and behavioral studies of self-control and the ability to delay gratification. It is not yet definitively known whether improving physical fitness can lead to improvements in cognition and self-control in children, though studies in adults and one study in children suggest that this may be the case. A recent study (Davis et al., in press) randomized overweight children to a three-month high intensity, low intensity, or no exercise groups. Results demonstrated a dose response benefit to exercise on executive functioning (control regions of the brain) and mathematics achievement. In addition, fMRI results using an antisaccade task showed increased activity in bilateral PFC. These findings warrant future longitudinal studies in children of the relationship between physical fitness, obesity, and brain functioning.

In addition, some studies have examined the impact of physical activity on brain structure as opposed to brain function. A recent study showed that the structure of the brain, specifically the volume of the hippocampus, is greater in physically fit children (as measured by VO2 max) when compared to age matched non-physically fit children (Chaddock et al., 2010a). The hippocampus plays a critical role in learning and memory. There were no observed volume differences in the nucleus accumbers (part of ventral striatum). Another study reported several additional brain regions that are structurally different based on the child's level of physical fitness. The ventral striatum (part of the basal ganglia) did not differ in size based on the child's level of physical fitness (Chaddock et al., 2010b). However, the dorsal striatum, believed to play a role in cognitive control and inhibition, was larger in those children who were more physically fit. In general, obese children are less physically fit than healthy weight children (Shulz et al., 2009). It is possible that the observed structural differences between physically fit and unfit children may partly underlie the foundation for the functional brain differences seen in food motivation studies of obese versus healthy weight children. Future neuroimaging studies need to include measures of both physical fitness and obesity to increase understanding of the association between cardiovascular fitness and excess body weight on brain function and structure.

**Conclusion and future directions**

Obesity and its related health conditions are serious problems for youth that can follow them throughout their lives, causing personal, medical, financial, and societal burden. As eating habits are established early in life, it is important to learn more about the underlying brain activity involved in responses to food stimuli and decisions to eat. Our understanding of the brain mechanisms of pediatric obesity is still in its infancy and neuroimaging studies of brain function in pediatric obesity are only beginning.

Continued research is needed based on strong theoretical frameworks. Operating from a theory allows interdisciplinary collaboration and mutual benefit to all fields. For example, additional work is needed to better define “drive” and “control” regions of the brain in relation to normal development of food motivation and cognitive control in healthy weight groups. A better understanding of typical, healthy “drive” regions and “control” regions will serve as a necessary basis for then comparing how neural mechanisms differ between healthy weight and overweight or obese children. Studies of both healthy weight and obese children should also begin to apply network level analyses in order to understand interactions between brain regions. This will be more informative than observing the separate contributions of each region. Testing the interaction between these regions using functional connectivity and other developing techniques will be crucial to gaining a better understanding of the influence of brain development on appetitive and eating behaviors.

To expand our knowledge, longitudinal neuroimaging studies of children will also be necessary. Following children who are at risk for developing obesity over time could be one way of learning more about the brain’s contribution to the development of obesity. Another area of study would be to scan children before and after an obesity intervention. One study has been published, but more are needed. The goal would be to identify predictors of successful response to treatment and to examine how brain function changes in successful and unsuccessful dieters. Identifying brain activation patterns that predict success in a behavioral obesity intervention could provide important clues for developing more effective interventions.

Ultimately, research must examine whether cognition and brain function can be modified to improve health-related decision-making. One example could be developing ways to improve the cognitive mechanisms of restraint, inhibition, and self-control. Initial work has shown that it is possible to train children to delay gratification and early results also indicate that, with proper incentives, adolescents are able to improve cognitive control. These findings are encouraging and suggest that there may be ways to improve obesity prevention and treatment programs. Prevention and childhood intervention may help provide more long-standing patterns of healthy behavior.

Behavioral planning and decision-making are influenced by both reward-seeking (“drive”) and inhibitory processes (“control”)—making healthy decisions regarding food and beverage choices and physical activity and sedentary behavior involves a balance between these two processes. For healthy behaviors, decisions must be based upon long-term consequences (weight control, good health, etc.) rather than the immediate reward (taste of food) requiring the “control” brain areas to override input from the “drive” areas. Research on the neural correlates of health behaviors in children has the potential to help us better understand, prevent, and treat obesity, in turn providing a healthier, happier future for our youth.

**Conflict of interest statement**

The authors declare that there are no conflicts of interest.

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