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Psychological and Neural Contributions to Appetite Self-Regulation

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Abstract

Objective—Review the state-of-the-science on psychological and neural contributions to appetite self-regulation in the context of obesity.

Methods—Three content areas (neural systems and cognitive functions; parenting and early childhood development; and goal setting and goal striving) served as examples of different perspectives on the psychological and neural factors that contribute to appetite dysregulation in the context of obesity. Talks were initially delivered at a workshop consisting of experts in these three content areas and then content areas were further developed through a review of the literature.

Results—Self-regulation of appetite involves a complex interaction between multiple domains, including cognitive, neural, social, and goal-directed behaviors and decision-making. Self-regulation failures can results from any of these factors, and the resulting implications for obesity should be considered in light of each domain. In some cases, self-regulation appears to be amenable to intervention; however, this does not appear to be universally true, which has implications for both prevention and intervention efforts.

Conclusions—Appetite regulation is a complex, multi-factorial construct. When considering its role in the obesity epidemic, it is advisable to consider these various contributions together to best inform prevention and treatment efforts.

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Introduction

The widespread availability of low-cost, highly palatable, energy-dense foods has led to increased opportunities to overeat and we as a society are consistently confronted with provocative cues encouraging us to do so. The modern "obesogenic" environment challenges our ability to self-regulate. Chronic appetitive self-regulation challenge is a leading cause of obesity, which itself has enormous public health consequences. Consequently, better understanding factors that lead to dysregulation of appetite is of critical importance and may help to develop novel approaches to improve self-regulation of food intake.

It is clear that self-regulation in the context of appetite control is influenced by several individual-, group-, and environment-level factors. Individual-level factors influencing appetite regulation in the context of weight management include genes and epigenetics, hormones and other metabolic influences, neurocognition, and behavioral and emotional characteristics; and each of these is influenced by developmental stage and the social and built environment (1–6). Topics contained in this "white paper" include neural and psychological (including cognitive) contributions to appetite self-regulation, and focus on how various mechanisms – neural systems and cognitive functions, parenting and early childhood development, and goal setting and goal striving – contribute to effective and ineffective self-regulation of appetite and ingestive behavior.

The three areas above were addressed in four talks delivered at the July 2015 National Institutes of Health Appetite Self-Regulation expert panel meeting, addressing the theme of cognitive and psychological contributions to appetite self-regulation. Dr. Todd Heatherton spoke about the balance model of self-regulation, involving motivation/reward and executive functions (including inhibitory control and decision-making) and supporting neural circuitry and also addressed the contexts in which these functions and neural systems fail (e.g., in negative or depressed moods). Dr. Cary Savage elaborated on the balance model of self-regulation, summarizing neuroimaging studies, and discussed how self-regulation may change in response to different treatment approaches (e.g., behavioral therapy, weight-loss surgery) and how neuroimaging data can be used to predict treatment outcomes and, one day perhaps, personalize interventions. Dr. Leann Birch then discussed the intergenerational transmission of food intake behaviors and the relationship to obesity. Her talk focused on the importance of early child development in shaping dietary choices and appetitive self-regulatory behavior, with a special emphasis on the role of the parent-child dyad in these process and how these processes influence behaviors and weight trajectories in early life. Dr. Traci Mann closed this series of talks with a presentation on goal setting and goal striving in the context of appetite self-regulation. She emphasized the use of approach (vs. avoidance) and mastery (vs. performance) strategies when setting goals. After goals have been set, emphasis was placed on maintenance: the need for goals to be consistently achieved. Dr. Mann discussed how "willpower" or self-control may have a limited effect in certain

circumstances, and she offered two goal striving strategies that lower the burden on "willpower": planning or prospection, and habitual or less effortful strategies.

It is important to note, however, that there are several additional, related psychological and neural factors that have clear relevance to appetite regulation and obesity that were not covered in the workshop leading to this paper. For example, Epstein and Bickel propose that excessive food intake may be driven by reinforcement pathology where individuals experience high reinforcing value of food which, when accompanied by poor appetitive impulse control, leads to a self-regulatory breakdown and increased risk for excessive caloric intake (7). We recognize that obesity is a complex, multi-faceted medical condition. For example, recent studies indicate that obesity may be associated with increased reward system response in anticipation of food reward but the response to actual delivery is dampened and can show individual variation (8). Thus, the present paper is not intended to be a comprehensive review. Rather, as in the expert panel discussion, we acknowledge the broad and interacting factors that influence self-regulation of appetite but retain a focus on a few illustrative domains explored in more depth. As such, this paper concentrates on three factors thought to influence self-regulation of appetite: 1) brain and cognition, 2) parenting and early childhood development, and 3) goal setting and goal striving.

This paper summarizes the evidence, opinions and perspectives presented by experts at the workshop. By focusing on the leading candidate psychological and neural factors discussed at the meeting, we hope to improve understanding of the relationship of self-regulation of appetite to food intake behavior and obesity by: (1) identifying potential applications of the scientific evidence presented in the talks and discussed across all experts in attendance (i.e. inform the potential for novel intervention strategies) and (2) reviewing the gaps in knowledge identified by these experts, as wells as areas where the state-of-the-science could lead to different interpretations and approaches to application in order to advance research in the area of appetite regulation and obesity. This information can further serve to educate the many stakeholders with an interest in obesity and inform public policy, thereby suggesting strategies to target psychological and neural influences on appetite regulation, and help change the current course of the obesity epidemic.

State of the Science

"Motivational, Reward-based, and Cognitive Contributions to Appetite Self-Regulation"

Self-regulation of appetite shares many similarities with other self-control challenges, such as the ability to moderate or avoid substance use. Tempting situations of various kinds require the capacity to delay gratification and make decisions based on anticipated long-term consequences. Self-regulatory capacity is not static; rather, it can be compromised by external factors such as cue exposure and negative affect (7–9). For example, exposure to appetitive cues, such as food images used in advertising (see Advertising and Policy paper in this issue), increases both conscious and nonconscious craving, particularly among individuals who are hyper-sensitive to specific cues, such as food cues among the obese or smoking cues in smokers (10). In one study, food cue reactivity demonstrated in the nucleus accumbens predicted weight gain during the first year of college (11). Thus, one common

cause of self-regulation failure is the desire produced by being exposed to things that people are trying to control, such as food cues for eaters.

Another common cause of self-regulation failure across all domains is negative affect. There are well reported associations between depression and obesity and laboratory induction of negative mood is associated with increased eating among dieters and reduced ability to choose delayed rewards (12). Negative mood also enhances reward cue reactivity in the brain (7). A recent meta-analysis (13) found that depression was the most common comorbid psychiatric condition among bariatric surgery candidates.

Investigators also found moderate evidence for reduced rates of depression post-surgery. Self-regulation requires making choices based on longer-term goals rather than more immediately rewarding impulsive options. Individuals vary in self-regulatory capacity and in their sensitivity to external forces that strain the ability to make healthy decisions. Thus individual variation in cue responsivity and factors compromising the ability to choose healthy behaviors likely contribute to today's most pressing public health problems, including obesity.

The ability to self-regulate relies on brain networks involved in processing reward and regulating behavior. In particular, effective and enduring self-control may be dependent on a balance between top-down regulation from areas such as the prefrontal cortex (PFC) and bottom-up signals from subcortical reward processing centers in the limbic system (7, 9). The PFC is not exclusively composed of cognitive control networks. Large territories in PFC, particularly along midline and ventral surfaces, are connected to limbic networks and the dopamine reward system, and play a role in evaluating expectations and probabilities of reward. Thus, self-regulation likely requires a complex balance between subcortical reward signals, ventromedial PFC circuit evaluation of these inputs, and self-regulatory control mediated by lateral PFC circuits. The PFC-subcortical balance model of self-regulation (7) posits that poor self-regulation arises from an imbalance in these brain networks, either as a result of hyper-responsive reward networks, ineffective control systems, or a breakdown in connectivity between frontal control systems and limbic systems. Indeed, there is recent evidence from a neuroimaging study (14) in which participants were scanned during food cue and response inhibition paradigms and then surveyed with experience sampling methods for one week. Baseline food cue reactivity in the nucleus accumbens positively predicted future ratings of food desire, initiation of eating behavior, and amount of food consumed. In comparison, baseline activity in the lateral PFC was associated with subsequent resistance to temptation. Thus, PFC networks, particular lateral regions, have been implicated in inhibitory and regulatory processes needed to make non-impulsive choices under experimental conditions and in daily life.

The PFC is known to support a number of high-level control processes collectively termed "executive functions" which include the ability to regulate emotions and behaviors and cognitive control processes such as inhibition, mental flexibility, and working memory (15). These various components of executive functioning are coordinated to support effective self-regulation, including the capacity to consistently make health decisions based on anticipated consequences (16). Hall and Marteau (16) recently reviewed the connections between

executive functioning and health behavior and concluded that executive functioning likely has a causal influence on health behaviors, including healthy food decisions and adherence to physical activity guidelines. They also note that causal links are complicated by potential reciprocal relationships between executive functioning and health behaviors. Specifically, better executive functioning supports consistent healthy choices, which may in turn strengthen brain function and lead to better executive functioning over time, so-called "positive feedback loops." By comparison, "negative feedback loops" are also likely, in which poor executive functioning leads to consistently unhealthy choices, which in turn compromise brain function and weakens the capacity for strong executive function. Of note, weaker executive functioning has been linked to frequency of snacking, particularly in the presence of facilitating cues (17). This highlights potential differences in susceptibility to the effects of food advertising. For example, children with obesity show reduced lateral prefrontal cortical activity in comparison to children of healthy weight when presented with food advertising logos (18). Moreover, children with obesity also showed greater activity in somatosensory regions associated with mouth actions when viewing commercials (19).

Obesity is a common and growing problem arising, at least in part, from chronic failures in self-regulation of food intake and energy expenditure (physical activity). Neuroimaging studies are providing important information regarding brain function in obese and healthy weight groups. Though results can be variable, studies point to hyper-responsiveness in reward processing networks alone, or in combination with, decreased activation in control processing regions of PFC in persons who are obese. Chronic self-regulation failure leading to obesity might, therefore, arise from greater reward system activation, control system activation failure, or a combination of both (see (20) and (21) for reviews). In relation to reward processing, Burger and Stice (22) proposed a dynamic vulnerability model of obesity, whereby striatal reward hyper-responsivity is a risk factor for obesity that changes over time. According to this model, at risk individuals initially show increased striatal responsiveness to hedonically appealing foods. However, the human brain is highly adaptive and, over time, chronic overconsumption of palatable foods leads to down-regulation of dopamine receptors and, eventually, blunted reward response to food consumption. Similar to those with addiction, affected individuals continue to over-consume, seeking the previously experienced reward, creating a vicious cycle of overeating and further blunted reward response. This theory reconciles apparently discrepant observations from neuroimaging studies of obesity showing increased reward system activation to food cues and anticipation of food reward alongside blunted activation to the actual delivery of food reward. In support of this theory, Burger and Stice (23) found that increased ventral pallidum activation to food cues and decreased caudate activation to repeated milkshake delivery predicted increases in body mass index (BMI) over a two-year follow-up in a group of initially healthy weight and adolescent and young adult females who were overweight. To further complicate the picture, however, there is growing evidence that reward system activation is influenced by individual genotype, such as carriers of the A1 allele of the Taq1A polymorphism ((25), see paper in this issue: Biologic Complications). Thus, evidence is emerging supporting alterations in brain response to food reward in obesity and highlight dynamic malleability and modulation associated with genotype, in processes leading to self-regulatory failure and the development of obesity.

In addition to the domain of motivation/reward, obesity has also been linked with problems in cognitive self-regulation, particularly in the domain of executive functioning (1). For example, in a recent review of studies in midlife adults with obesity, Prickett et al. (24) concluded that there is consistent evidence of impairment in several cognitive domains, including executive functioning. They also noted, however, that it is difficult to separate the effects of obesity from those of comorbid conditions such as diabetes (also see (25)). While most studies try to evaluate and exclude comorbid conditions, it is impossible to do so completely and conditions such as diabetes can affect cognition long before the diagnosis. Fitzpatrick et al. (26) also found that obesity was consistently associated with comparatively weaker executive functioning, but noted that directionality was difficult to establish in the relationships between obesity and decreased executive functioning. To further complicate understanding the relationships of cognitive and physiologic mechanisms, it appears that the so-called “Western” diet may negatively impact brain function before the onset of obesity (27, 28). Obesity might be the outcome of chronic problems in decision making and resultant unhealthy food choices, but it might also be causative of brain dysfunction and poor executive functioning, either directly or through metabolic and other co-morbid dysfunction (25). Though cause and effect have not been established, the links between obesity and comparatively weaker executive functioning are supported, and this is likely both a contributor to, and outcome of, obesity.

One important question regarding the relationships between brain, cognitive dysfunction and obesity is the extent to which they are modifiable. There are encouraging findings that intentional weight loss is associated with modest improvements in executive function ((29); see (30) for a meta-analysis). Bariatric surgery has particularly been shown to result in rapid improvement in cognitive functioning, including executive control, that can endure over at least several years ((31) for a review). Consistent with these observations, successful weight-loss with both behavioral based diets and bariatric surgery have been associated with changes in brain function as measured with fMRI. (see Biologic Complications paper in this issue.) Results from two recent studies indicate that successful bariatric surgery and behavioral weight-loss interventions result in unique patterns of change in brain function (32, 33). In these studies, bariatric surgery resulted directly in decreased activity in reward evaluation regions of PFC, while successful behavioral dieters showed increased functional connectivity with executive control networks to adaptively support self-regulation. Critically, both interventions can lead to successful weight-loss, and impact function in prefrontal brain networks. Finally, other interventions have been shown to improve executive functioning, including aerobic exercise ((34) for a meta-analysis) and food response inhibition and facilitation training ((35) for a review). Thus, there is reason to be optimistic regarding the reversibility of brain and cognitive dysfunction associated with obesity, and potential strategies for intervention.

“Intergenerational Transmission: Parenting and Early Self-Regulation”

The first 1000 days of life are marked by major developmental milestones, as children learn how to sit, crawl, stand, walk, and talk (36). They also learn to eat, making a dramatic dietary transition as they are introduced to many of the foods of the adult diet of their culture. By the end of the first 1000 days, children’s diets closely resemble those of their

parents. As children's motor skills develop, they transition from the exclusive milk diet of infancy to consuming many "table foods" (non-pureed fruit and vegetables, grains, meats, and eggs). They also become increasingly autonomous, making their own food choices from among those offered, using a spoon, feeding themselves, and regulating how much to eat.

However, despite increasing autonomy, children are dependent on parents or other caregivers for sustenance, and parents' decisions about what foods are offered how and when infants are fed shape this early learning and the development of self-regulation of intake (36). Because children's diets resemble those of their parents, they tend to be too high in sugar, fat, salt, and calories and too low in fruits, vegetables, and complex carbohydrates (37, 38); such diets are implicated in obesity among children and adults.

Because early rapid growth in infancy increases risk of obesity and comorbidities later in life, a clear understanding of how caregivers' feeding practices affect early learning and the development of self-regulation in feeding has implications for promoting healthier diets and reducing obesity risk (36).

Familiarization

To obtain a healthy diet, children must learn to accept and consume a variety of foods. As they are introduced to the diet of their culture, all foods are new. However, children are neophobic. "Neophobia", literally fear of the new, describes the child's normal and adaptive response to reject new foods. Fortunately, this neophobia can be reduced through experience with the flavors and textures of new foods. Extensive research confirms that what becomes familiar tends to become preferred and what is unfamiliar is often disliked and avoided (36, 39, 40). This exposure process begins in the earliest days of life. Because a variety of flavors from the mothers' diet are introduced to the infant through breast milk, breastfed infants become familiar with flavors of the adult diet (41). This "flavor bridge" (also described in an earlier paper in this issue as a biologic mechanism) that eases the transition to table foods (36, 42), ultimately promotes acceptance of new foods. Compared to formula-fed infants who lack this early flavor experience, breastfed infants more readily accept pureed vegetables than formula-fed infants (43). Parents may facilitate or impede the process of familiarization as they select food to offer to the child, as well as the frequency of exposure (36, 44). Studies have shown that parent-led, exposure-based interventions involving daily tasting of vegetables increased children's liking of those vegetables (45–47), and this experience can play a role in establishing subsequent eating behaviors and related-health outcomes (48).

Associative Learning

Because parents control the child's feeding environment, their expectations of what, when, and how much a child should eat can yield positive or negative results: children's food like and dislikes are also affected by associative learning, particularly affected by emotional valence and context (see Biologic Complications paper in this issue.) Evidence indicates that a better approach is to offer healthy choices and let the child decide what and how much to eat. For example, offering children foods as a reward or pairing foods with positive attention increased children's liking of those foods (45). By contrast, parents who encourage or coerce

children to consume foods can foster food dislikes and avoidance. Evidence from experimental studies indicates that children who were pressured to eat “healthy” foods were more likely reject those foods than children who were not pressured to eat (49, 50). Moreover, children whose access to “unhealthy” foods was restricted were more likely to choose and to eat more of the restricted foods relative to similar foods that were not restricted (51–53). There is some evidence that restrictive feeding practices may increase intake of the “forbidden” or restricted foods, leading to overconsumption and excessive intake of those foods. Additionally, parents may inadvertently teach children to ignore their own hunger and fullness when “forbidden” foods become readily available, which can result in eating in the absence of hunger (36, 52, 54) and result in disinhibited, dysregulated eating, potentially compromising self-regulation of intake and predisposing children to excessive intake and obesity.

Temperament

Because influence in parent-child interactions is bidirectional, parents’ controlling or indulgent feeding practices may be at least in part a response to the child’s characteristics, particularly to differences in child temperament, eating behavior, or weight status. In this context, temperament is defined in terms of a balance in reactivity (ease of arousal) and self-regulation. Children with high reactivity and low self-regulation are more likely to find palatable foods highly reinforcing and may be susceptible to excessive intake and obesity (55–57). One possible explanation for this finding is that children high in negative reactivity are prone to frequent expressions of distress; parents may use feeding as a first response to this infant distress, which increases opportunities for intake (57, 58). However, in a recent study, only infants high in negative reactivity and who had mothers likely to use feeding to soothe their distress gained excessive weight; infants high in negative reactivity whose mothers used alternative soothing strategies did not show excessive weight gain, suggesting that parents’ feeding practices can affect the development of self-regulatory skills. Differences in older children’s temperaments have also been shown to be associated with more susceptibility to the adverse effects highly restrictive practices (53, 57): girls with low self-regulation (inhibitory control) and high parental restriction were particularly prone to excessive weight gain and risk of obesity (59). Finally, the increased risk of obesity in early childhood due to underdeveloped self-regulatory skills occurs during a period of rapid neurodevelopment in the subcortical and prefrontal brain systems that mediate the motivation/reward and executive functions critical for appetite regulation as discussed in detail above (see (60); (4) for recent reviews). Thus, there appear to be developmental/social pathways leading to brain based changes in motivation/reward and executive function in children, which could be impacted through parental interventions.

“Goal Setting and Goal Striving in the Context of Appetite Self-Regulation”

In the first two sections, self-regulation was defined in the context of inhibitory control. However, self-regulation may also be conceptualized as the processes people use to set, pursue, and attain goals; these processes can be divided into two main categories: goal setting and goal striving (101). Goal setting involves deciding which goals to pursue and what will count as successful achievement of those goals. Research shows that people are more likely to set eating goals if these goals are consistent with other goals they value (102,

61). However, it may be necessary to challenge an individual's defensiveness regarding the negative aspects of behavior before they are willing to set a goal for healthy eating. Indeed, interventions in which people affirm their positive values have been shown to reduce defensiveness and lead to healthier behavioral intentions (62); (103).

People are more likely to set and pursue a goal if they are motivated by the pleasure inherent in pursuing the goal, rather than by the outcome of the goal (e.g., improved health markers such as lower blood pressure, reaching a certain body weight, etc.); or, worse, because others pressure them to pursue it (104). If the process of attaining a goal is not considered pleasurable, which is the case for most dieting goals, then people are more likely to set a diet goal if they focus on the outcome of that goal (63). There are two kinds of goals that have been shown to be relatively effective at leading to successful behavior change. The first is called an approach goal, which is a goal to achieve a certain outcome (e.g., to eat more vegetables), in contrast to an avoidance goal, which aims to *prevent* a particular outcome (e.g., to *not* eat candy, or to *not* get fat) (64). The second is a mastery goal, which focuses on developing a skill (such as learning to cook) rather than a performance goal, which aims to attain a certain standard (such as reaching a certain weight) (65). Mastery goals are particularly helpful when dealing with setbacks, because the setback can be seen as useful information on how to proceed, whereas with performance goals, a setback becomes an evaluation, highlighting that the individual has failed.

Once individuals have set goals, they strive to achieve them, and these goals must be shielded from disruptions and distractions from competing goals or desires (66). To do so, one often has to use willpower, or self-control, to effortfully resist temptations (e.g., resisting delicious foods that are nearby). As discussed previously, willpower is prone to failure. Because many common daily experiences cause willpower to fail, goal striving strategies that don't require willpower show promise in leading to successful goal achievement. One category of strategies that does not involve willpower is termed planning or prospective strategies, and there is evidence that they can lead to healthy eating, at least in brief studies (105, 106). These strategies involve changing daily routines or altering surroundings so that tempting goal-inconsistent foods are not encountered. Another form of prospective strategy would be to pre-commit to a healthy course of action when temptation isn't present (e.g., packing a healthy lunch to bring to work), so that individuals are locked in to the healthy behavior later (67). Prospection or planning is akin to aspects of self-regulation discussed above, where self-regulation is maximized when choices are made based on longer-term goals rather than more immediately rewarding options. Episodic future thinking, mentioned earlier as a behavioral channel for altering appetitive behavior, which involves thinking about future goals and outcomes, has been demonstrated to reduce temporal discounting or increase an individual's relative preference for future vs. immediate rewards, and also reduces ad libitum food intake in people with obesity. Episodic future thinking has been proposed as a potential strategy in the obesity therapeutics arsenal (68, 69).

A second type of goal striving strategy that minimizes self-regulatory demands is making healthy eating operate automatically, with little to no conscious awareness. If a behavior is automatic, it is less subject to interference or disruption from moods, energy level, distractions, or competing temptations (70). Habits are behaviors that have been paired with

a particular context so often that the behavior will occur automatically, without conscious effort, whenever the person is in that context (107). Habits take many repetitions to form, and making healthy eating a habit is particularly difficult because it requires the consolidation of many separate habits to account for the many contexts in which individuals eat. A quicker alternative is to form an implementation intention, which is a specific automatic association that can be formed with just a single pairing (108). The pairing must be in the form of an if-then plan that specifies a particular behavior to be performed in a particular context (e.g., "If I am offered a donut at work, then I will have coffee instead"). Implementation intentions are highly effective in increasing the amount of healthy food individuals eat, although somewhat less effective at decreasing the consumption of unhealthy food (71).

A final type of goal striving strategy is to change the way one thinks about temptations. To resist eating something tempting, it is more effective to think of that food in an abstract, distanced, or rational way, instead of in a concrete, immediate, or emotional way (109). Thinking abstractly is helpful because it includes goal-relevant thoughts (e.g., "this food will lead to weight gain") instead of thoughts about the tempting details and immediate rewards of the food. For example, when children were instructed to think about a marshmallow as a fluffy cloud instead of focusing on how delicious it looked, they were able to resist it longer (72, 73). In addition, individuals were more likely to choose an apple over a cookie if they were put into an overall abstract mindset instead of a concrete one (72).

Potential Applications and Gaps in Knowledge

In this section, the important gaps in knowledge regarding the factors discussed in this paper are highlighted. The potential near-term and far-term applications of the reviewed knowledge are discussed. The goal is to translate basic science into clinical research and trials (when appropriate), generating ideas for future research studies that can lead to improved prevention and treatment of disorders of appetitive behavior leading to obesity.

The "vicious cycle" of obesity and cognitive dysfunction

Although obese and healthy weight groups consistently differ in brain imaging studies, little is known regarding cause and effect. It is likely that both "positive" and "negative" feedback loops are operating (16), in which brain function is strengthened by healthy behaviors –– protecting from obesity – or weakened by poor diet and sedentary lifestyle, making people more susceptible to obesity. This has also been described as a "vicious cycle" of obesity and cognitive decline (74). Without intervention, this cycle contributes to continued weight gain and metabolic dysfunction and then further dysregulation of brain and cognitive functioning. As discussed previously, there is evidence from animal studies that, to at least some degree, poor diet impacts cognitive function and this precedes the development of obesity (27, 28). This suggests that at least some of the cognitive and brain function differences in obesity arise from eating behavior itself. Overall, the preponderance of evidence suggests that causal effects likely flow in both directions: cognitive and brain function impacts self-regulation of eating and the physiological changes associated with poor eating further degrade brain function and cognition (see (25) for further discussion)

Cognitive training and neuromodulation as potential therapies for obesity

Though there is evidence that many of the cognitive and brain function differences in obesity are modifiable, the extent to which this is possible and the conditions in which they can occur are not well delineated. As noted, there is consistent evidence of changes in cognitive and brain function after intentional weight-loss in adults, whether by calorie restriction and behavioral diet (29, 30) or bariatric surgery (31). This raises the interesting possibility that cognitive and brain processes underlying self-regulation might become targets for intervention in order to develop new treatments that are biologically informed. These interventions might be indirect, such as cognitive training approaches, or direct, using new neuromodulation techniques such as transcranial direct current stimulation (tDCS). Though previous efforts to train cognitive abilities have produced mixed results with little evidence for training transfer to other skills (75), there is some evidence that cognitive retraining efforts aimed at altering eating behavior by improving impulse control can result in improved weight loss (76)(35), decreased food intake (77) and reduce perceived value of snack foods (78). Brain function may be more directly impacted using methods such as tDCS, in which low amplitude currents are applied to the brain via scalp electrodes, altering membrane potentials of some targeted neurons and increasing or decreasing their spontaneous firing rate. Previous studies have shown effects on food craving and food intake with positive (anodal tDCS) to left or right PFC. Gluck and colleagues (79) recently used anodal tDCS to increases neural excitability in a left dorsolateral prefrontal cortical target in nine obese individuals during a 9-day inpatient stay in which they ate *ad libitum* from a computerized vending machine. Individuals consumed fewer calories showed greater weight loss with anodal stimulation of left DLPFC. Thus, there is early evidence that brain function might be directly and indirectly targeted to facilitate self-regulation (for a review, see (80)). Of note is that evidence is derived from studies in adults, and does not address the issue of neural plasticity early in development.

Biomarkers as predictors of treatment outcome

Finally, it is possible that neuroimaging, genetic, and hormonal biomarkers might be used to predict response to particular interventions aimed at increasing self-regulation of eating and other health behaviors. These studies may be able to identify biomarkers that predict treatment response and, ultimately, classify and assign individuals to particular interventions that are most likely to be successful (see Schlogl et al., 2016 for a review). For example, Murdaugh et al. (81) scanned 25 obese participants with fMRI, while participants viewed high calorie food pictures, before and after a 12-week behavioral weight loss program. Higher activity in reward networks, including nucleus accumbens, anterior cingulate cortex, and insula predicted less weight loss after 12 weeks, indicating that participants with higher reward system activation were less likely to lose weight. More recently, Szabo-Reed and colleagues (Szabo-Reed et al., under review) scanned 67 obese adults with fMRI while they viewed appetizing food pictures before initiating a 12-week diet program. In this study, bilateral PFC activation positively predicted future weight-loss and the effects of bilateral PFC activation were linked to diet adherence behaviors (number of sessions attended, healthy foods consumed, physical activity) in path analyses. These initial studies are correlational and focus at the group level. Recent advances in data analytic techniques, such as machine learning, raise the possibility of individual classification by combining data from

multiple modalities (e.g., neuroimaging, genetics, hormones; see (82) for an example in addiction) in cross-validated classification analyses. While these approaches are new and not fully tested, they at least raise the possibility of personalized medicine approaches, which assign individuals to particular interventions that are most likely to be effective and can be monitored over time as response to treatment, physiology, and behavior change (see (83) for an example of single person longitudinal phenotyping). While cost is often cited as a limitation, the cost of brain imaging, genetics and other biomarker collection is less than the personal, financial, and societal cost of multiple failed interventions.

Responsive Parenting

As noted earlier, parents' use of traditional feeding practices, including coercion to consume healthy foods to promote a healthful diet can be counterproductive; parenting and feeding strategies that are not responsive to the child can also have unintended negative consequences for children's food selection, preferences, and the development of behavioral control of food intake, especially given our obesogenic environment (39, 52, 54). A promising alternative may be the use of responsive parenting, which fosters parent-child interactions that are prompt, contingent, and developmentally appropriate. Responsive parenting, known to promote cognitive, social, and emotional development (84, 85), may be particularly important in infancy to support the emergence of self-regulatory skills that can shape subsequent eating behavior, effect growth rates, and weight status in childhood. Responsive parenting interventions show promise for obesity prevention in early life and might guide future studies for parents and their children (86, 87). Two recent randomized trials (86)(88) have demonstrated the effectiveness of responsive parenting approaches and demonstrated positive effects on infant self-regulation, eating behavior, sleep duration, emotion regulation and rate of growth. Taken together, these findings indicate that the use of responsive parenting shows efficacy in influencing infant outcomes, including early indicators of self-regulation and rapid growth (86).

The evidence is more mixed regarding effects of responsive parenting interventions on preventing rapid weight gain and obesity, and additional research is needed (86, 87, 89, 90). Establishing efficacy and testing theoretical frameworks is compromised by the paucity of validated, reliable measures of key processes (e.g. intergenerational transmission of food exposures; parenting styles in the context of scarcity of healthy foods) and proximal outcomes included in study designs. Measures appropriate for use with groups differing in race and ethnicity, particularly for underserved high-risk populations, are also needed, as are accurate measures of child growth and body composition that can be used in community settings. Longitudinal studies are essential to determining the efficacy of responsive parenting interventions; it is also unclear how early responsive parenting practices affect preferences, inhibitory control, and weight status in the context of environmental influences. (see Marketing and Policy paper in this issue.)

Striving and Goal Setting

The most serious gaps in knowledge in the areas of setting and striving toward healthy eating goals involve the capacity to scale up from mechanisms and interventions that help with a single isolated act of self-control to multiple acts of self-control, and then

maintenance over extended periods of time. Controlling appetite and limiting food intake require many acts of self-control each day, every day. While many interventions have been found to effectively help people control their eating of one food in a single laboratory session, or minimize their total consumption at any one meal, or even succeed at a diet for six months, few interventions have been found successful over extended time periods, especially in free-living conditions. Effective long-term interventions will need to continue to function effectively after initial weight loss, or during extended periods of caloric deprivation, as cognitive, metabolic, and hormonal changes make controlling appetite even harder. Interventions that function in these circumstances are missing from the literature (as are, presumably, interventions that alter or prevent these circumstances in the first place).

Discussion

Self-regulation invokes a complex balance of cognitive, neural, social, and goal-directed activities. The capacity for self-regulation is prone to failure and is negatively affected by factors such as cue exposure and negative affect (7). With regard to cognitive and brain function, self-regulation requires a balance between subcortical reward signaling, ventromedial PFC evaluation of these inputs, and top-down executive control regulation from lateral PFC. There is evidence that executive function is comparatively weaker and reward processing is altered or enhanced in obesity (16, 22). This combination likely contributes to the development and maintenance of obesity in children and adults. In addition to directly contributing to obesity, an unhealthy diet also negatively impacts brain health. It is therefore likely that both positive and negative feedback loops operate in obesity related health behaviors, such that brain function is strengthened by healthy diet and physical activity - protecting from chronic self-regulation failure and obesity - but also weakened by chronically poor health choices, increasing vulnerability to obesity (16). While this can create a vicious cycle of obesity and cognitive decline, it also raises the interesting possibility that differences in reward processing and cognitive control may become targets of new interventions utilizing cognitive retraining and/or direct neural modulation approaches. There is encouraging evidence that cognitive deficits improve with weight loss, and brain dysfunction in obesity may be reversible. The identification and reliable measurement of biomarkers may also be used to predict treatment response and, ultimately, personalize interventions.

The social world also has a tremendous impact of self-regulation; this is especially true for the impact of parents on young children. Early childhood is a period of developmental plasticity in which children learn food preferences and eating behaviors that have long-term implications for the development of self-regulatory skills, diet, and health. Parents and caregivers control the eating environment and facilitate children’s food and flavor learning (1). However, there is a mismatch between traditional parenting and feeding practices and the current food environment; while parents’ use of “feeding to soothe” and pressure to “clean up your plate” evolved to protect children in the context of food scarcity, these practices are no longer appropriate given the widespread availability of palatable, inexpensive, energy-dense foods (1, 15, 16). Moreover, these overt strategies have been shown to produce unintended adverse consequences on food preferences, self-regulation of intake, and weight status (18, 20). It should also be noted that while promoting dysregulation

of food intake, children are not actively making food choice decisions (see comments on "flavor bridges"). An alternative is responsive parenting, which fosters the parent-child feeding dynamic and has been shown to improve early indicators of children's risk of obesity. While responsive parenting interventions hold promise, findings are inconsistent. More research is warranted to better understand the short- and long-term effects on children's eating behaviors and overall health (28, 30), especially in non-laboratory settings.

Finally, transitioning from other direct influences on development of self-regulatory skills, to volitional behavioral control, self-regulation may also be conceptualized as the setting, pursual, and attainment of goals. For all but young children, goal setting is the process whereby individuals decide which goals to pursue and how successful attainment will be defined. These processes are affected by individual values and the innate enjoyment of goals, and these characteristics can be used to impact capacity for goal-setting. Striving, another aspect of goal achievement, involves maintaining focus and perseverance toward goals. Striving relies heavily on the capacity for self-regulation. As noted throughout this paper, self-regulation is prone to failure. While self-regulatory capacity may become a target for intervention, it is also possible to identify goal striving strategies that bypass or minimize the need for reducing self-regulatory failure or conversely, increase capacity. Examples of this approach include prospection or planning, automaticity or habit formation, and altering the conceptualization of "temptations." Thus, studies reviewed in this paper indicate that self-regulation may be a target for strengthening or bypassed altogether. One implication is early prevention: development of self-regulatory appetitive behavior during early childhood such that behavior is habitual in the face of regulatory challenges.

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