# Reward, Reinforcement, and Impulsivity in Obesity

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## Abbreviations

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| BMI | Body Mass Index |
| SR | Sensitivity to reward |
| SP | Sensitivity to punishment |
| GNG | Go/No-Go |
| IGT | Iowa Gambling Task |
| BOLD | Blood-Oxygen-Level-Dependent Signal |
| dlPFC | Dorsolateral prefrontal cortex |
| mPFC | Medial prefrontal cortex |
| ACC | Anterior cingulate cortex |
| Hippocamp. | Hippocampus |
| Operc. | Operculum |
| NS | Nonsignificant |

## Introduction

Societal changes in food production, marketing, and availability have moved eating behavior outside purely homeostatic motives, raising awareness about the relevance of reward, reinforcement, and impulse control systems in regulating food intake [1]. In plentiful environments, where the appeal and size of food products is maximized and exploited, individual differences in reward sensitivity and impulsivity are likely to predict food preferences and food consumption [2]. Moreover, dietary patterns deeply entrenched in current societies (e.g. high-fat and high-calorie diets) have shown to detrimentally impact brain systems involved in reinforcement sensitivity and impulse control [3]. Partly as result of these changes, the prevalence of obesity has sharply increased in recent decades [4]. In response to this challenge, the scientific community has multiplied efforts to understand the contribution of reinforcement and impulse control systems to the risk, progression, and treatment of obesity.

Neuroscientific findings have stressed the relevance of four interrelated brain systems involved in processing food value and regulating food consumption [1]: (1) the hypothalamus, involved in regulating energy intake and maintaining homeostasis; (2) the striatum/limbic system, involved in coding the reward value of available reinforcers and ensuing activation of the impulsive system (approach or avoidance behavior); (3) the somatosensory/interoception system, involved in ongoing mapping of homeostatic signals and subsequent moderation of reward-impulsive and goal-driven systems; and (4) the ventromedial and dorsolateral prefrontal cortical systems, involved in goal-oriented self-regulation of behavior. The hypothalamus has been thoroughly characterized as the main regulator of basic metabolic processes [5] but it also feeds back to brain systems involved in food reward coding, interoception, and self-regulation/decision-making [1, 5]. The striatum/limbic system encompasses brain regions sensitive to

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stimulus-driven behavioral approach or inhibition: the ventral striatum and the amygdala, respectively. These regions represent the hedonic value of food (ventral striatum, amygdala), track the outcome value of food-related reinforcement (hippocampus, extended amygdala), and orchestrate motor responses during approach-avoidance learning (dorsal striatum, cerebellum) [6, 7]. The somatosensory/interoceptive system is essential to link perceived homeostatic signals with motivational states and reward predictions [8, 9]. The insula and the frontal operculum are additionally involved in updating and monitoring cognitive control systems involved in self-regulation [10]. Finally, the prefrontal system computes the relative value of food reinforcers, based both on their basic attributes (e.g. palatability) and more abstract long-term goals (e.g. healthy eating, dieting), and guides behavior accordingly [6]. The balance between ventromedial and dorsolateral prefrontal cortices serves to weigh the rewarding attributes vs. the anticipated outcomes and their consistency with goals [6], whereas ventrolateral prefrontal regions are particularly relevant to performance monitoring and response control [10].

Over the last decade, neuroscientific research has revealed several mechanisms by which dispositional differences or adaptations in the function of these systems can contribute to the risk and progression of obesity. Specifically, there is evidence to support: (1) that individual differences in reward responsivity and impulsivity are associated with the risk for weight gain and obesity [11, 12]; (2) that high-fat diets and excessive adiposity impair efficient communication between peripheral homeostatic regulators (e.g. insulin, leptin) and central reinforcement nuclei (e.g. striatum, insula) [13, 14]; and (3) that dysfunctional reinforcement functioning can ultimately "hijack" top–down inhibitory control and decision-making systems relevant to the self-regulation of eating in the context of lifestyle goals or treatment commitment [15]. There is also ongoing discussion on (4) parallels between obesity and addiction, mainly based on these overlapping deficits in reward and inhibitory control systems [16, 17]. However, this overlap seems to fit better with specific patterns of overeating (e.g. binge eating) [18, 19] than with the typical obesity phenotype [20, 21]. Therefore, in this chapter, I will discuss evidence hinting to the first three notions, taking a multimodal approach that will address findings from personality, neuropsychology, and brain imaging studies. The theoretical advantage of personality studies is the reliable measurement of stable dispositions purportedly associated with vulnerability to obesity [12, 22]. Complementarily, neuropsychological tools provide more accurate estimations of the current function (and malleability) of specific cognitive processes, including reward prediction and reinforcement value, impulsive action, and decision-making (i.e. impulsive choice) [23, 24]. Finally, neuroimaging studies offer insight about the brain underpinnings of trait and cognitive measures, as well as about the dynamic interplay between different brain systems, or between hormonal signaling and brain systems relevant to obesity [25]. The evidence provided by these multimodal assessments will be discussed in the framework of maturational stages, differentiating findings from pediatric/adolescent populations vs. adult populations. This distinction is based on evidences showing that brain maturational processes impact trait and cognitive aspects of reward sensitivity and impulsivity [26], and that obesity is better characterized as a dynamic process in which predisposing traits (e.g. child hypersensitivity to reward) may turn to opposite states as the condition evolves (e.g., hyporesponsivity to reward in adult chronic populations) [5]. To avoid further confounders, I will only review evidence directly related to obesity, leaving aside related conditions such as binge eating disorder, or relevant comorbidities like diabetes or hypertension. Moreover, I will focus on studies lacking pre- and post-prandial manipulations, in order to attain a more uniform account of long-lasting (vs. transient) alterations associated with the condition.

## Assessment Tools: Multidimensionality and Convergence of Personality, Cognition, and Neuroimaging

During this chapter I will review evidence from self-reported personality questionnaires, neuropsychological tests, and neuroimaging tools that

have been applied to the study of sensitivity to reward and impulsivity in the context of obesity. These varied tools provide different but complementary insights into reward processing and impulsivity constructs, which I discuss in this section prior to description of the specific findings from these studies.

Self-reported personality questionnaires assess general dispositional traits of the individual: how the individual would typically behave in a given situation, or to what extent the subject agrees or disagrees with particular statements. Conversely, neuropsychological tests are designed to provide objective "current state" indices of behavioral performance linked to the function of specific cognitive systems and processes. Both personality and neuropsychological measures are often used in combination with different neuroimaging techniques. Structural neuroimaging tools (e.g. Magnetic Resonance Imaging, MRI) serve to quantify gray and white matter, total or regional brain volumes through different statistical approaches [27]. Resting-state functional connectivity is useful to characterize synchronized activation of large-scale inter-connected brain systems [28]. In the obesity literature, both of these measures have been successfully correlated with personality traits or performance on neuropsychological tests [25, 29]. However, the more widely used approach is that of functional neuroimaging (with PET or functional MRI) which measures regional brain activity (or neurotransmitter activity) during actual performance on specific cognitive tasks. This latter approach offers the opportunity of characterizing the neural underpinnings of normal (or abnormal) responsivity and function of reward sensitivity and impulse control systems and processes [30, 31] and I will mainly rely on this evidence to describe the brain underpinnings of these processes.

Several questionnaire measures have been developed based on major personality theories. For the purpose of this chapter, I will focus on three main personality models and measures: the Gray's behavioral inhibition and behavioral approach systems model [32], which is typically measured with the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) [33]; the Costa and McRae Five-Factor model, which is measured with the NEO-PI inventory [34]; and the UPPS pathways model, which is measured with the UPPS-P scale [35]. Gray's model defines two dimensions of personality that represent the sensitivity of two neuropsychological systems involved on motivated response towards environmental stimuli. The behavioral inhibition system is sensitive to environmental stimuli signaling potential punishment or non-reward, therefore inhibiting behaviors associated with perceived threat. Conversely, the behavioral activation system is sensitive to environmental stimuli signaling potential reward or non-punishment, resulting in behavioral activation linked to the prospect of reinforcement. The SPSRQ assesses the differential sensitivity of these systems, with high sensitivity to reward scores representing increased sensitivity of the behavioral activation system, and high sensitivity to punishment scores representing increased sensitivity of the behavioral inhibition system.

The Five-factor model defines five broad personality dimensions (factors) that represent different constellations of traits: Openness, Conscientiousness, Extraversion, Agreeableness, and Neuroticism. In this chapter I will address three of these factors, since they encompass specific facets of impulsive personality [34]. Conscientiousness represents the tendency to orientate behavior according to long-term goals, such that low conscientiousness comprises poor deliberation and low self-regulation. Extraversion represents proneness to seek novel stimulation and experiences, including exciting things linked to potential harm. Similarly, neuroticism represents increased sensitivity to negative emotions, including proneness to trigger impulsive responses when under these feelings. Obesity has been mainly associated with low conscientiousness and high neuroticism, whereas there is mixed evidence hinting to both high and low extraversion.

Building on the Five-factor model and on factor analysis of an array of well-validated measures tapping on impulsive traits, Whiteside and Lynam [35] originally developed the UPPS model and scale. This model defines impulsivity as a behavioral outcome that can be potentially triggered through different personality pathways.

They originally proposed four different pathways: negative urgency, representing the tendency to succumb to strong impulses under the influence of negative emotions; lack of perseverance, representing difficulty to stay through tasks until completion, especially when they are long, boring, or difficult; lack of premeditation, representing the tendency to act without sufficient regard of potential consequences; and sensation seeking, representing proneness to engage in novel, exciting activities that can be risky or not [35]. A fifth dimension of positive urgency (the tendency to succumb to strong impulses under the influence of positive emotions) was later added to the scale and model to represent a positive emotionality pathway to impulsive behavior [36]. The dimensions of lack of perseverance and premeditation stems from the Five-factor dimension of conscientiousness, whereas negative urgency stems from neuroticism, and sensation seeking from extraversion [35].

All of these scales have demonstrated sound psychometric properties and validity. However, it is worth noting that there are a number of well-known caveats inherent to the self-report methodology. Some of these caveats are particularly relevant for obese populations. For example, individuals with obesity are at higher risk of social marginalization and may therefore be more susceptible to social desirability biases. Furthermore, similar to other impulsive populations, individuals with obesity are purportedly more prone to exhibit a careless approach to questionnaires themselves.

In contrast to self-report (which primarily addresses stable and broad domains) neurocognitive measures are designed to obtain precise estimations of the current function of cognitive processes, and to relate the function of these processes to that of relevant brain systems. In the framework of sensitivity to reward and impulsivity, four major families of measures have been put forward. Sensitivity to reward can be measured with (1) cue-related attention/motivation tests, and (2) relative reinforcement value tests. Impulsivity can be measured with (3) response inhibition tests, and (4) impulsive choice tests including delay discounting and decision-making tasks [22, 37].

Cue-related attention/motivation can be evoked through different probes. These probes include attentional bias tests (e.g. Dot Probe), measuring the strength of the attentional engagement toward (or the difficulty to disengage from) spatial locations formerly paired with incentive cues (i.e. food stimuli); and cue-delayed incentive tasks (e.g. Monetary Incentive Delay), measuring degree of responsivity to cues associated with prospective rewards [38]. The dependent measures of these tests include behavioral reaction times, eye-tracking based time estimations of attention allocation, and cue-related physiological and brain activations. Conversely, relative reinforcement value procedures measure the amount of time or effort invested on particular reinforcers (e.g. food procurement) relative to other competitive options (e.g. reading magazines). Both types of tasks engage the dorsomedial prefrontal–ventral striatal brain circuit typically involved in stimulus-driven motivation [39].

With regard to impulsivity, response inhibition tests are based on the ability or difficulty to suppress an automated (prepotent) response. Some of these tests stress the ability to suppress perception or attention-based primed responses (e.g. similar to target "non-target stimuli" in Continuous Performance tests, or the reading response in the Stroop test), whereas other measures stress the ability to suppress a previously reinforced or a previously initiated motor response, such as in Go/No-Go and Stop-Signal tasks respectively [40]. Both types of tests seem to engage overlapping brain circuits encompassing ventrolateral and dorsolateral prefrontal cortices, and dorsal striatal regions [41, 42]. Within the impulsive choice family, measures of delay discounting define impulsivity in terms of choice preference for a small reward available immediately (or after a short delay) over a larger reward available at some point in the future [43, 44]. Decision-making measures involve choices between a safe option and a more risky option that offers a "superficially appealing" gain. These measures

include the Iowa Gambling Task [45] and the Risky Gains Task [46]. Impulsivity can be indexed by selection of the highly rewarding option despite the clear potential for negative outcomes. A separate aspect of the decision-making process is reflection impulsivity, which represents the tendency to gather and evaluate sufficient information before making complex decisions [47]. This aspect can be measured with specific probes (e.g. the Information Sampling Task) or through indirect indices of planning time and increased errors in Maze tasks (Austin or Porteus Maze tests) and Tower tasks (Tower of Hanoi or Tower of London tests) [37]. The latter are based on the assumption that poor reflection at the planning/pre-decisional stage will reduce the accuracy of the eventual decision [48]. Delay discounting and decision-making tests engage overlapping brain systems that encompass the midbrain and striatum, the insula, and the medial orbitofrontal cortex [49].

There is a classic controversy in the literature concerning notable mismatch between results from self-report and neurocognitive measures of impulsivity. The different targets of these complementary approaches (trait vs. state, general dispositions vs. specific operations) partly explain this mismatch. Recent studies have traced tentative links between trait measures of premeditation and perseverance and cognitive measures of response inhibition, and there is also evidence of correspondence between emotional dispositions to impulsivity (positive and negative urgency) and medial orbitofrontal brain regions involved in decision-making. However, readers must be aware of the existence of frequent disparities in reconciling findings from these approaches.

## Reward and Punishment Sensitivity

In this section, I review available evidence on sensitivity to reward and punishment in overweight and obesity populations, across personality, neuropsychological and neuroimaging methodologies relevant to these constructs. I initially review evidence obtained in pediatric populations (children and adolescents), to then move to adult populations. In those instances in which systematic or meta-analytic reviews are available, I primarily draw my conclusions from the findings from these studies. In addition, I consider case-control studies providing detailed biometric descriptions of the clinical populations and utilizing well-validated measures and statistical control.

### Adolescents

#### Personality Measures

Trait sensitivity to reward is regarded as a vulnerability marker for obesity, due to positive associations with overeating and increased BMI in children of 6- to 13-years-old [50]. During mid-adolescence (12- to 17-years-old) there is also a significant association between sensitivity to reward and poor premeditation and sensation seeking traits, which may promote external eating patterns [51]. However, the association between sensitivity to reward and BMI in child and adolescent populations (10- to 15-years-old) seems to be nonlinear: there is a positive correlation between sensitivity to reward and BMI in normal weight and overweight children/adolescents, but there is a negative correlation between sensitivity to reward and BMI in obese children/adolescents [52]. Considering that sensitivity to reward emerges early in life and promotes BMI gain, and that this positive association turns negative along the BMI continuum, the data seems to support a dynamic vulnerability model [11], by which hypersensitivity to reward originally fosters weight gain to then being detrimentally impacted by fat accumulation. Considerably less is known about sensitivity to punishment, but available evidence indicates that conjointly increased sensitivity to punishment and reward is particularly associated with excess weight problems in adolescence [53].

#### Cognitive Measures

With regard to attentional bias towards food cues, there is no consistent behavioral evidence of this

phenomenon in children or adolescents, although neuroimaging studies have shown suggestive findings that I discuss in the following section. In relative reinforcement value tasks, children and adolescents with greater BMI levels exhibit faster rates of motivated responses associated with actual food rewards relative to competitive reinforcers [54]. Furthermore, the relative reinforcing value of food prospectively predicts BMI gain across one year [55].

### Neuroimaging

Structural neuroimaging studies have demonstrated that in adolescents who are overweight or obese, there is a positive correlation between gray matter in somatosensory cortices (SII, parietal operculum) and sensitivity to reward, whereas the directionality of this association is negative in normal weight peers [29]. Functional neuroimaging studies have further shown that adolescents who are overweight or obese exhibit increased functional activation of anterior cingulate, frontal/rolandic operculum, and somatosensory regions (insula and parietal operculum) during anticipation of highly palatable food, coupled with increased activation of the frontal/rolandic operculum regions during the intake of highly palatable food [56]. During attentional bias towards food visual cues, adolescents with excess weight also exhibit increased activation of the insula and the frontal operculum [57]. Moreover, during attentional orientation towards and passive observation of visual food cues, adolescents with higher BMI show increased activation of dorsolateral and ventrolateral prefrontal regions, which purportedly reflects food-evoked attentional "hijacking" [57, 58]. Increased ventrolateral activation toward cues prospectively predicts BMI gain [57]. Therefore, current evidence indicates that both trait sensitivity to reward and state sensitivity to actual food reward are associated with abnormal function of brain somatosensory regions, while observation of food stimuli hyper-engages attention/executive control regions.

### Interim Conclusion and Future Directions

Concerning reward/punishment sensitivity, obesity in adolescent populations is associated with BMI-related reductions in trait sensitivity to reward, higher explicit motivation towards food reinforcement, atypically increased positive association between brain somatosensory regions volume and activation and reward excitability, and increased involvement of brain attentional control regions during presentation of food stimuli.

## Adults

### Personality Measures

Similar to the case in children and adolescents, the association between sensitivity to reward and BMI in adults is non-linear: there is a positive correlation between sensitivity to reward and BMI in normal weight and overweight adults, but there is a negative correlation between sensitivity to reward and BMI in moderate to morbidly obese adults (BMIs~>35) [59]. Interestingly, both overweight and obesity have been associated with increased sensitivity to punishment [60]. Therefore, the reward/punishment profile of moderate to morbidly obese adults is better characterized by reduced sensitivity to reward and increased sensitivity to punishment. It is worth mentioning that these temperamental traits are regarded to be genetically mediated, such that there is particular interest on dopamine and opioid receptor genes implicated in both reward processing, and in the pathogenesis of obesity [22]. There is evidence showing that individuals with excess weight and binge eating disorders carrying gene variants associated with decreased dopamine D2 receptor availability (*DRD2/ANKK1 Taq1A* A1 carriers) are characterized by increased sensitivity to reward relative to individuals with excess weight and binge eating disorder not carrying the "high risk" A1 allele [61]. Moreover, it has been shown that the rare *G118 variant* of the *mu* opioid receptor gene (*OPRM1*), associated

with gain of hedonic function, is overexpressed in excess weight individuals with binge eating disorder compared to excess weight individuals who do not binge [62]. However, the association between dopamine or opioid gene polymorphisms and BMI has not been supported by larger epidemiological studies [63]. Therefore, while it is plausible that dopamine and opioid gene variants mediate individual differences on sensitivity to reward and eating habits, its relevance to obesity has not yet been solidly established.

### Cognitive Measures

A systematic review of available evidence has shown that obese populations consistently and robustly demonstrate increased sensitivity to food in relative reinforcement value tasks [22]. Moreover, it has been shown that the relative reinforcement value of food is a significant predictor of BMI, and this association is moderated by disinhibited dieting [64]. There is no evidence to support an increased motivational/attentional bias towards food rewards on implicit association tests [65]. However, this motivational/attentional bias has been observed in individuals with excess weight and binge eating disorder when utilizing fine-grained eye-tracking tools measuring orientation of attention towards food images, and difficulty to disengage attention from the spatial locations associated with those images [66, 67].

### Neuroimaging

Cue-reactivity functional neuroimaging studies have demonstrated that obese individuals exhibit significantly increased activation in medial prefrontal/anterior cingulate cortex, striatum, insula, and hippocampal regions during visual processing and anticipation of high vs. low rewarding food [68, 69]. Interestingly, a similar pattern of hyperactivations has been observed in obese individuals relative to normal weight controls during anticipation of monetary rewards [20]. Moreover, individuals with obesity show increased effective connectivity between the caudate nucleus and amygdala and insular regions during visual processing of high- vs. low-calorie food [70]. Importantly, the strength of functional activation in the anterior cingulate, insula, and caudate regions is prospectively associated with lower long-term weight loss following a lifestyle weight management intervention [71].

### Interim Conclusion and Future Directions

Concerning reward/punishment sensitivity, obesity in adult populations is associated with increased trait sensitivity to punishment, decreased trait sensitivity to reward, increased explicit (but not implicit) motivation towards food-related reinforcement, and hyperactivation and hyperconnectivity of brain reward systems during anticipation of high-calorie food and money.

## Impulsivity

In this section, I review available evidence on impulsivity in overweight and obese populations, across personality, neuropsychological and neuroimaging methodologies. I will first review evidence obtained in pediatric populations (children and adolescents), and then move to adult populations. In those instances where systematic or meta-analytic reviews are available, I will ground my discussion on the findings from these analyses. In addition, I primarily review case-control studies providing detailed biometric descriptions of the clinical populations and utilizing well-validated measures and statistical control.

## Adolescents

### Personality Measures

Meta-analytic evidence has shown small clinically nonsignificant effect sizes for questionnaire measures of impulsivity in pediatric populations [24]. Nonetheless, it has been shown that increases in BMI are significantly associated with elevations of negative urgency [51]. These findings indicate that impulsive personality is not

a major characteristic of adolescents with excess weight; however, higher levels of adiposity are linked to elevations in negative urgency. These elevations in negative urgency are malleable and relevant to treatment outcome, in the sense that the adolescents who reduce urgency levels to a greater extent also achieve greater reductions in BMI during multicomponent interventions (including dieting, physical activity, and psychological interventions) [23].

### Cognitive Measures

Meta-analytic evidence has shown moderate to large effect sizes for motor and decision-making measures of impulsivity in pediatric populations [24]. Specifically, adolescents with obesity are characterized by lower vigilance and poorer response inhibition on motor impulsivity measures (e.g. Go/No-Go or Stop Signal), preference for high immediate rewards at the expense of larger delayed punishments on gambling tasks, and preference for immediate rewards over larger delayed rewards on delay discounting measures [24, 72, 73]. Noteworthy, steeper rates of delay discounting are associated with steeper gains in BMI over time [74]. Conversely, obesity is not associated with significant alterations on attentional impulsivity or reflection impulsivity in adolescents.

### Neuroimaging

Structural neuroimaging studies have linked higher BMI with decreased orbitofrontal gray matter, mid-cingulate cortex gray and white matter, and body of corpus callosum white matter [75–77], both relevant to increased trait impulsivity and response disinhibition [78, 79]. Conversely, BMI levels have been positively associated with white matter volumes in rolandic operculum, mid-temporal bundles, dorsal striatum and splenium [77, 80] which are relevant systems for sensory/emotional memories and habits. Functional imaging of motor impulsivity during a Go/No-Go task has shown that in obese individuals BMI negatively correlates with activation of dorsolateral and ventrolateral prefrontal cortices and frontal operculum/anterior insular regions during inhibition trials [81]. Risky decision-making in an impulsive choice task is associated with decreased anterior insular and increased midbrain activation in overweight and obese adolescents [82].

### Interim Conclusion and Future Directions

Concerning impulsivity, obesity in adolescent populations is associated with BMI-related elevations in trait negative urgency, significant cognitive deficits on response inhibition and decision-making, structural alterations in orbitofrontal, mid-temporal and dorsal striatal regions, and abnormal activations of the anterior insula during response control and risky decisions.

## Adults

### Personality Measures

Evidence summarized in a systematic review has mapped personality findings to the Five-Factor Model, concluding that obesity is characterized by increased neuroticism and decreased conscientiousness [22]. This notion is in fitting with findings obtained with the multidimensional UPPS-P scale, demonstrating that obesity is specifically linked to elevations in negative urgency and lack of perseverance [59]. Higher neuroticism and negative urgency reflect negative emotion-driven impulsive behavior, as manifested by obesity-related elevations in specific measures of emotional eating [5]. Similarly, *lack of* conscientiousness, or perseverance, as manifested in ADHD-like symptoms has been associated with both emotional eating and external eating [12], both triggered by affective or environmental cues in the absence of hunger.

### Cognitive Measures

Evidence from systematic reviews has shown that obesity is associated with significantly poorer performance on measures of motor impulsivity (e.g. Stop Signal, Hayling test), delay discounting

(particularly when food is involved) and risk-taking [22, 83]. There is also evidence showing that obese individuals have deficits in solving maze planning tasks involving components of reflection impulsivity [22, 83]. There is however no consistent evidence about obesity-related deficits on attentional impulsivity or decision-making tests involving uncertainty about punishment [22, 83, 84].

### Neuroimaging

Structural neuroimaging studies have demonstrated that obese individuals have reduced gray matter in the dorsolateral prefrontal cortex, frontal operculum and dorsal striatum [27], key brain regions for response inhibition. It has been shown that in female obese individuals BMI negatively correlates with activation of response control specialized regions, including the supplementary motor area, the insula, and the inferior parietal lobe during the "stop" trials of the Stop-signal task [85]. Comparatively less is known about functional brain alterations associated with impulsive decision-making. However, there is longitudinal evidence showing that decreased activation in dorsolateral and ventrolateral prefrontal cortices and inferior parietal lobe regions during delay discounting predict weight gain across 1–3 years in obese female individuals [86].

### Interim Conclusion and Future Directions

Concerning impulsivity, obesity in adult populations is associated with negative urgency and lack of conscientiousness/perseverance traits, response inhibition deficits, and decreased frontostriatal gray matter coupled with decreased activation in dorsolateral and ventrolateral prefrontal cortices and inferior parietal regions during impulsivity tasks, which prospectively predicts weight gain. Most imaging evidence stems from studies on obese female populations, but preliminary evidence suggests significant sex-related differences in structural measures and brain-behavior associations [87]. Therefore, a closer look to sex differences is warranted to allow generalization of findings.

## Concluding Remarks

The research on reward processing and impulsivity in obesity has been increasing exponentially during the last decade, and is expected to continue to grow over the coming years. Current findings have used a range of methods, and the field is in need of further multimodal and longitudinal studies addressing questions of causality and progression of illness. Notwithstanding these limitations, some points have been well-established across different methodologies and age cohorts. This evidence is summarized in Table 2.1, which provides a snapshot of findings across personality, neuropsychological and neuroimaging measures across pediatric and adult populations. Based on the evidence that I have reviewed in this chapter, it can be concluded that obesity (customarily defined as BMI levels above 30 $kg/m^2$) is characterized by dispositionally lower sensitivity to reward (reward deficiency), coupled with *higher* responsivity to food rewards, response disinhibition, and steeper discounting of delayed rewards, all of which are longitudinally associated with weight gain. These deficits are also manifested as dysfunctions in neural systems involved on somatosensory processing (insula/frontal operculum), reward seeking (striatum, extended amygdala, cerebellum), stimulus-oriented attention (dorsolateral prefrontal cortex), and decision-making (orbitofrontal cortex). More research is needed on the negative affective pathway to reward seeking and impulsivity, since both sensitivity to punishment and negative urgency (the tendency to make impulsive acts under negative affect) are elevated in populations with obesity. Critically, more research is warranted to address the dynamic association between reward processing, impulsivity and obesity, since both variables are purportedly involved in vulnerability to weight gain, but there is growing evidence on mechanistic pathways by which unhealthy diets and adiposity have a detrimental impact on these characteristics.

**Table 2.1** Summary of findings from self-report personality questionnaires, neuropsychological tests and neuroimaging tools that have been applied to the study of sensitivity to reward and impulsivity in the context of obesity

| | Personality | | | Neuropsychological tests | | | | Brain systems | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | SR/SP | Neuroticism | Conscient. | Cue-induced attention | Reinforce. value | Response inhibition | Impulsive choice | Interoception | Reward | Cognitive control |
| Children/ Adolescents | ↓ SR | ↑ Urgency with ↑ BMI | NS | NS | ↑ Food reinforcing value | ↓ Motor Inhibition: (Stop Signal & GNG) | ↑ Delay discounting<br>↑ Preference Reward/Risk choices (IGT) | ↑ Correlation Gray matter SII and SR<br>↑ BOLD SII, Insula, Frontal Oper. & ACC during Food Anticipation<br>↓ BOLD Insula during Impulsive Choice | ↑ Gray matter Hippocamp.<br>↑ BOLD Midbrain during Impulsive Choice | ↑ BOLD activation of dlPFC during Food Visual Cues |
| Adults | ↓ SR<br>↑ SP | ↑ Urgency | ↑ Lack of perseverance | NS Reaction Times<br>↑ Eye-tracked Attention to Food<br>↑ BOLD signal during Money Anticipation | ↑ Food reinforcing value | ↓ Motor Inhibition (Stop Signal)<br>↓ Primed Words Inhibition (Hayling Test) | ↑ Delay discounting<br>NS Preference Reward/Risk choices (IGT)<br>↓ Reflection (Maze Tests) | ↓ Gray matter Frontal Operculum<br>↓ Connectivity Insula | ↓ Gray matter Dorsal Striatum & Cerebellum<br>↑ BOLD mPFC, Striatum & Hippocampus during Food (& Money) Anticipation | ↓ Gray matter Dorsolateral Prefrontal Cortex<br>↓ BOLD Anterior Insula on Stop Signal |

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