Appetite 169 (2022) 105834

Contents lists available at ScienceDirect

Appetite

journal homepage: www.elsevier.com/locate/appet

Changes in temporal discounting, hedonic hunger, and food addiction during recovery from substance misuse

Julia C. Basso [a,b,c], Medha K. Satyal [d], Liqa Athamneh [a], Warren K. Bickel [a,b,*]

[a] Addiction Recovery Research Center, Fralin Biomedical Research Institute at VTC, 2 Riverside Circle, Roanoke, VA, USA
[b] Center for Transformative Research on Health Behaviors, Fralin Biomedical Research Institute at VTC, 1 Riverside Circle, Roanoke, VA, USA
[c] Department of Human Nutrition, Foods and Exercise, Virginia Tech, 1981, Kraft Dr., Blacksburg, VA, USA
[d] Graduate Program in Translational Biology, Medicine, and Health, Virginia Tech, 1981, Kraft Dr., Blacksburg, VA, USA

ARTICLE INFO

Keywords:
Substance use
Recovery
Abstinence
Eating behavior
Food addiction
Temporal discounting

ABSTRACT

Substance use disorders (SUDs) and obesity are both chronic, relapsing, remitting disorders that arise from a heightened preference for immediate-focused rewards (i.e., steep temporal discounting). During recovery from SUDs, overweight and obese outcomes are common as individuals may replace drug rewards for food rewards. However, little has been done to investigate the neuropsychological processes underlying food reward and addiction in individuals recovering from SUDs. Using data collected from the International Quit and Recovery Registry and Amazon Mechanical Turk, we aimed to elucidate the factors that influence the attraction to palatable foods in a population in recovery from substance misuse (n = 114) as well as a population with no history of substance misuse (n = 97). We hypothesized that individuals in recovery from substance misuse would have steeper temporal discounting, an increased drive for palatable foods (i.e., hedonic hunger), and greater food addiction symptoms than non-substance users. Contrary to our hypotheses, we found that individuals in recovery from SUDs show improved outcomes in temporal discounting, hedonic hunger, and food addiction symptoms. Moreover, recovery status and temporal discounting significantly predicted these outcomes. Our findings suggest that the enhanced executive control processes needed for successful SUD recovery may transfer to other reward-related processes such as food reward and consumption. Interventions targeted at improving executive function including episodic future thinking, meditation, or exercise, may be excellent ways to support a successful recovery and improve other reward-related processes, including food consumption, to decrease the risk of overweight or obese outcomes during recovery.

1. Introduction

Body weight gain and quality of food intake are significant concerns amongst individuals in recovery from substance use disorders (SUDs). Research has shown that during recovery from SUDs, individuals may engage in unhealthy eating behaviors, replacing their drug of choice or responding to drug or alcohol craving by consuming highly palatable foods, especially those high in fat and sugar (Hodgkins, Jacobs, & Gold, 2003; Jackson & Grilo, 2002). Additionally, dysfunctional eating patterns and disordered eating, such as bulimia nervosa and binge eating disorder, are often seen early in recovery (Cowan & Devine, 2008; Hodgkins, Cahill, Seraphine, Frost-Pineda, & Gold, 2004; Jackson & Grilo, 2002; Orsini et al., 2014; Williamson et al., 1991). Stress and anxiety, commonly experienced during withdrawal and early recovery, are additional factors that contribute to uncontrolled eating behaviors (Koob & Volkow, 2016). The unhealthy eating behaviors experienced early in recovery can lead to weight gain and overweight or obese outcomes, which can in turn lead to other serious health conditions such as Type 2 diabetes or cardiovascular disease (Cowan & Devine, 2008). Therefore, determining the neuropsychological factors that contribute to increased food consumption is imperative.

SUDs and overeating, which can lead to obesity, involve overlapping behavioral- and endo-phenotypes, with some hypothesizing that obesity is a form of food addiction (Campana, Brasiel, de Aguiar, & Dutra, 2019; Ferrario, 2017; Lerma-Cabrera, Carvajal, & Lopez-Legarrea, 2016). The food addiction hypothesis is based on the idea that obesity and other related disorders such as bulimia and binge eating disorder result from either 1) a heightened sensitivity to foods at both the behavioral and

* Corresponding author. Fralin Biomedical Research Institute at VTC, 2 Riverside Circle, Roanoke, VA, 24016, USA.
E-mail address: wkbickel@vtc.vt.edu (W.K. Bickel).

https://doi.org/10.1016/j.appet.2021.105834
Received 16 August 2021; Received in revised form 26 October 2021; Accepted 30 November 2021
Available online 3 December 2021
0195-6663/Published by Elsevier Ltd.

brain level (especially highly palatable foods high in fat and/or sugar); 2) a reduced response to foods; or 3) a dynamic response whereby the initial response is heightened but over time becomes blunted (i.e., dynamic vulnerability model) (Alonso-Alonso et al., 2015; Ferrario, 2017; Gearhardt, Boswell, & White, 2014, 2011; Stice, Spoor, Bohon, & Small, 2008; Volkow, Wise, & Baler, 2017). However, this hypothesis is not without controversy and recent research suggests that limited behavioral overlap exists between obesity and SUDs (Bickel, Lemos, Tomlinson, & Tegge, n.d; Vainik et al., 2020). Several convincing pieces of evidence exist to support the food addiction hypothesis. First, drugs of abuse and palatable foods are both strong positive reinforcers that activate the brain's reward system, including the nucleus accumbens and prefrontal cortex (Volkow, Wang, Fowler, & Telang, 2008; Volkow & Wise, 2005). Second, research shows that individuals with both SUDs and overeating display increased impulsivity, altered affective and cognitive states, and heightened reward salience and consummatory behavior of their substance of choice (Michaud, Vainik, Garcia-Garcia, & Dagher, 2017). Third, overlapping alterations in neural structure and function exist between these two disorders. For example, microdialysis studies in rodents have shown that both drugs of abuse and highly palatable foods stimulate extracellular dopamine release in the nucleus accumbens (Di Chiara, 2002; Roitman, Stuber, Phillips, Wightman, & Carelli, 2004). Neuroimaging studies in humans have also revealed that both individuals with SUDs and individuals with obesity display a reduction of dopaminergic D2 receptors in the striatum (Trifilieff & Martinez, 2014; Volkow, Fowler, Wang, Baler, & Telang, 2009; Volkow, Wang, Telang, et al., 2008; Wang et al., 2001) as well as reduced neural activity in prefrontal regions including the dorsolateral prefrontal cortex, orbitofrontal cortex, and cingulate gyrus (Volkow et al., 2009; Volkow, Wang, Telang, et al., 2008). Considering this interesting interrelationship, the examination of food salience and food addiction during recovery from SUDs is a relevant area of inquiry.

The competing neurobehavioral decision systems (CNDS) theory posits that the reward-driven impulsive system and the future-oriented executive system work in conjunction to govern behavior (Bickel, Mellis, et al., 2018). The impulsive system consists of limbic and paralimbic brain regions (e.g., nucleus accumbens, amygdala), and the executive system consists of prefrontal and temporal regions (e.g., prefrontal cortex, hippocampus). Dysregulation of these systems, namely a hyperactive reward system and hypoactive executive system, leads to maladaptive health behaviors including both substance use and overeating (Bickel et al., 2021; Kekic et al., 2019; Levitt, Sanchez-Roige, Palmer, & MacKillop, 2020). Behaviorally, the balance between the two systems can be measured using a temporal discounting task termed delay discounting, which assesses an individual's preference for smaller, immediate rewards compared to larger, delayed rewards (McClure, Laibson, Loewenstein, & Cohen, 2004). Here, we use the framework of the CNDS theory to examine the hypothesis that heightened temporal discounting may underlie the heightened response to highly palatable foods during recovery and increased body weight.

Using data collected from the International Quit and Recovery Registry (IQRR; www.quitandrecovery.org) and Amazon Mechanical Turk (mTurk), we aimed to elucidate the neuropsychological factors that influence the attraction to palatable foods in a population in recovery from substance misuse as well as a population with no history of substance misuse. Specifically, we examined hedonic hunger and food addiction as metrics of food reward. Hedonic hunger refers to the preoccupation with and desire to consume foods for pleasure in the absence of hunger (Espel-Huynh, Muratore, & Lowe, 2018), whereas food addiction (as measured by the Yale Food Addiction Scale) examines the DSM-IV criteria of substance dependence in relation to high-fat, high-sugar foods. We hypothesized that individuals in recovery from substance misuse would have an increased drive for palatable foods (i.e., hedonic hunger), greater symptoms of food addiction, and greater temporal discounting compared to non-substance users. We expected that temporal discounting and recovery status would predict food addiction symptoms, hedonic hunger, and body mass index (BMI).

2. Methods

Data were collected through the IQRR, an online community and registry for individuals in self-reported recovery from substance misuse or behavioral addictions. The initial assessment, completed upon registration, includes contact information, demographic information, history of substance use and behavioral addictions, and recovery history (Table S1, Table S2). After registering, members may complete assessments that aim to advance the understanding of phenotypes of recovery. Participants earn a predefined number of points for completion of each assessment, which are redeemable for $1.00 per 100 points. The current investigation includes data from the initial assessment, which was completed upon registration, and 3 monthly assessments, all of which were programmed and administered through LimeSurvey.

Data for the non-SUD control group were collected through mTurk, an online crowdsourcing platform that allows members to complete Human Intelligence Tasks (HITs) for compensation. The task was available to mTurk workers with a high (>90%) HIT approval rate, indicating that they provided high-quality data on at least 90% of previously completely HITs. Participants were screened to ensure they met inclusion criteria, and were required to pass a CAPTCHA test to screen out machine input. To be included in the study, participants needed to be non-smokers and have no self-reported history of substance abuse or over-consumption. History of substance abuse and overconsumption were assessed using three questions: 1) "Are you currently, or have you in the past been abusing alcohol or drugs?"; 2) "Do you now think, or have you in the past thought you may be over-consuming drugs or alcohol?"; and 3) "Are you currently in recovery from substance use or addiction?". Participants who responded "No" to all three questions qualified for the study. Participants were compensated for completion of the questionnaire and awarded bonus compensation if their data passed attention checks. All questions were programmed and administered through Qualtrics.

This study was approved by the Institutional Review Board at Virginia Polytechnic Institute and State University and was performed in accordance with relevant guidelines and regulations.

2.1. Measures

Demographics: Demographics were collected in the IQRR initial assessment and on mTurk, as demographic variables have been previously associated with temporal discounting (Bickel, Moody, Quisenberry, Ramey, & Sheffer, 2014; Stanger et al., 2012) and BMI (Berry et al., 2010; Claassen, Klein, Bratanova, Claes, & Corneille, 2019). Demographic information collected includes age, gender, race, ethnicity, education, and income. Age in the IQRR sample was calculated by subtracting the participant's year of birth from the year the assessment was completed.

Days in abstinence: Self-reported quit date was collected in the IQRR initial assessment. In the current assessment, participants were asked whether they engaged in their primary substance since registering in the IQRR. Those who answered yes were subsequently asked if their use was ongoing. Those who answered "No" were asked to report an updated quit date. The most recent quit date was subtracted from the assessment completion date to calculate the number of days in abstinence. Participants who reported a quit date that was the same day that they completed the assessment were considered ongoing users.

Body mass index (BMI): BMI was calculated from self-reported height in inches and weight in pounds as: BMI = weight/[height]^2 x 703.

Food addiction: The Yale Food Addiction Scale is a valid and reliable (Kuder-Richardson alpha = 0.86) self-report questionnaire to assess symptoms and diagnosis of food addiction (Gearhardt, Corbin, & Brownell, 2009). The questionnaire consists of 25 items that probe the DSM-IV criteria of substance dependence in relation to the consumption of high-fat, high-sugar foods. Items are scored for a symptom count ranging from 0 to 7. A diagnosis of food addiction occurs when clinical significance is indicated and the symptom count is at least 3.

Hedonic hunger: The Power of Food Scale is a valid and reliable (Cronbach's alpha = 0.91; test-retest reliability r = 0.77) self-report questionnaire to assess hedonic hunger in food-rich environments (Cappelleri et al., 2009; Lowe et al., 2009). The questionnaire consists of 15 items scored from 1 (*don't agree at all*) to 5 (*strongly agree*). Items are summed for a total score, ranging from 15 to 75, and three subscale scores including food available (range 6–30), food present (range 4–20), and food tasted (range 5–25). The full text of the questionnaire is available in (Cappelleri et al., 2009).

Temporal Discounting: The five-trial adjusting delay discounting task was used to assess future valuation and impulsivity (Koffarnus & Bickel, 2014). In this task, participants were asked if they would rather receive $500 now or $1000 in three weeks. The time delay in the subsequent trial is increased or decreased based on the participant's response. The delays continue to adjust in this manner for a total of five trials. The delay at which the reward loses 50% of its value compared to the immediate reward (ED_{50}) is provided by the indifference point. The inverse of the ED_{50} ($1/ED_{50}$) was calculated to provide an estimate of the discount rate (k). The natural log transformed discount rate [ln(k)] was used for analysis.

2.2. Data cleaning and statistics

Participants who did not complete all questionnaires, provided an invalid BMI, reported a primary behavioral addiction in the IQRR, were ongoing substance users, or who did not pass attention checks were excluded from all analyses. In the case of multiple submissions, only one record from each participant was included in the analyses.

Chi-square tests of independence were performed to compare the distributions of gender, race, ethnicity, household income, and education between individuals in recovery and the non-SUD control group. Independent samples t-tests were conducted to compare continuous variables including age, BMI, food addiction symptoms, hedonic hunger, and temporal discounting between groups. If a variable had unequal variance between groups, as determined by Levene's test for homogeneity of variance, Welch's *t*-test was used. ANCOVA was used to compare food addiction symptoms, hedonic hunger, and temporal discounting between groups after controlling for significant demographic variables. Multiple linear regression analyses were conducted to assess the association of recovery status and temporal discounting to food addiction symptoms, hedonic hunger, and BMI. Additionally, demographics (i.e., age, gender, race, household income, education) were included in multiple linear regressions to assess the association of recovery status and temporal discounting to food addiction symptoms, hedonic hunger, and BMI after adjusting for these demographic variables. Relationships among all neuropsychological variables, BMI, and time in abstinence were probed using Pearson correlations. All analyses were conducted in SPSS Statistics 26.0 with statistical significance determined at an alpha level of 0.05.

3. Results

3.1. Demographics

The final analysis included 211 participants (97 non-SUD controls, 114 in recovery). Participant characteristics are displayed in Table 1. Participants in the recovery group were older than those in the non-SUD control group. Additionally, gender, race, household income, and education were significantly different between groups.

3.2. Between groups comparisons

Independent samples t-tests showed that individuals in recovery have significantly fewer food addiction symptoms ($t(189.5) = 3.396$, $p < 0.001$), and lower hedonic hunger ($t(209) = 5.489$, $p < 0.001$), including when food is available ($t(209) = 3.608$, $p < 0.001$), present ($t(209) = 4.597$, $p < 0.001$), and tasted ($t(209) = 7.640$, $p < 0.001$) compared to the control group (Fig. 1, Table S3). Additionally individuals in recovery have significantly lower rates of temporal discounting ($t(209) = 3.038$, $p = 0.003$) (Fig. 1, Table S3).

After controlling for significant demographic characteristics, between groups comparisons revealed that individuals in recovery have significantly lower hedonic hunger ($F(1, 204) = 12.160$, $p < 0.001$) including when food is present ($F(1, 204) = 10.531$, $p = 0.001$) and food is tasted ($F(1, 204) = 31.723$, $p < 0.001$) (Table S4). No significant differences were observed between groups in food addiction symptoms ($F(1, 204) = 1.077$, $p = 0.301$) or temporal discounting ($F(1, 204) = 0.309$, $p = 0.579$) (Table S4).

Table 1
Participant characteristics.

	Non-SUD	Recovery	t/X2	p
N	97	114		
Age[a]	32.6 (0.95)	44.5 (1.48)	−6.770	<0.001
BMI[a]	26.6 (0.58)	27.3 (0.57)	−0.816	0.416
Days Abstinent[a b]	–	3071.9 (369.77)	–	–
Gender			17.780	<0.001
% Female	31 (32.0%)	64 (56.1%)		
% Male	66 (68.0%)	46 (40.4%)		
% Other/Prefer not to answer	0 (0.0%)	4 (3.5%)		
Race			16.446	<0.001
% Asian	32 (33.0%)	13 (11.4%)		
% Black/African American	8 (8.2%)	7 (6.1%)		
% White/Caucasian	51 (52.6%)	80 (70.2%)		
% Other[c]	6 (6.2%)	14 (12.3%)		
Ethnicity			1.904	0.168
% Hispanic	10 (10.3%)	6 (5.3%)		
% Non-hispanic	87 (89.7%)	108 (94.7%)		
Household income			22.872	<0.001
% <$30,000	24 (24.7%)	45 (39.5%)		
% $30,000-$49,999	25 (25.8%)	16 (14.0%)		
% $50,000-$69,999	12 (12.4%)	8 (7.0%)		
% $70,000-$89,999	16 (16.5%)	11 (9.6%)		
% $90,000+	20 (20.6%)	20 (17.5%)		
% Prefer not to answer	0 (0.0%)	14 (12.3%)		
Education			43.821	<0.001
% High school/GED or lower	7 (7.2%)	32 (28.1%)		
% Some college	14 (14.4%)	34 (29.8%)		
% Bachelor's degree	64 (66.0%)	25 (21.9%)		
% Advanced degree	12 (12.4%)	23 (20.2%)		

[a] All variables are reported as frequencies and percentages with the exception of age, BMI, and days since last use which are reported as the mean and standard error.
[b] No statistical tests reported for days since last use as it is only collected in the recovery group.
[c] Other race includes those who identified as American Indian/Alaskan Native, Native Hawaiian/Pacific Islander, more than one race, and Other.

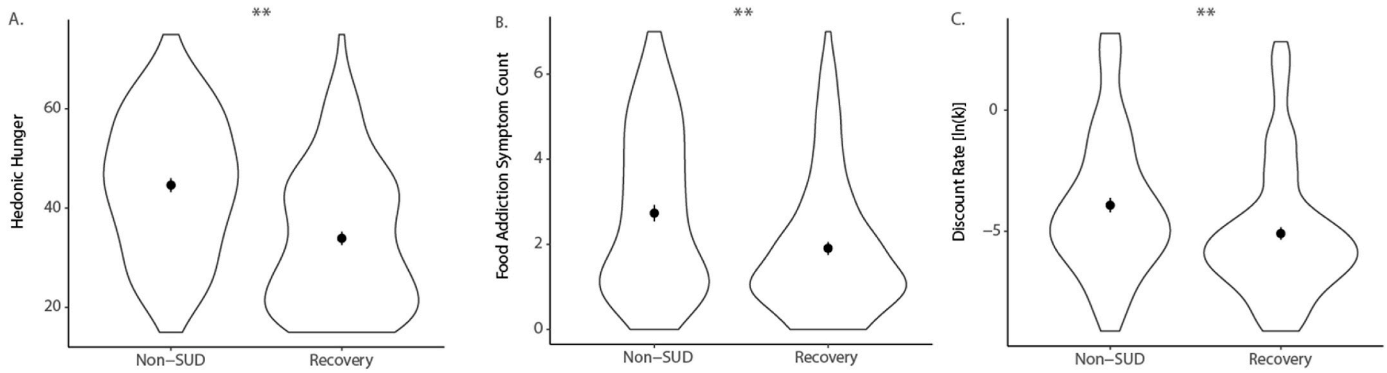

Fig. 1. Between groups comparisons of A) hedonic hunger, B) food addiction symptoms, and C) temporal discounting. Plotted as distributions, means (black dots), and SEM. **p < 0.01.

3.3. Correlations

Pearson correlation analysis revealed that food addiction symptoms were significantly correlated with increased hedonic hunger and greater temporal discounting, and hedonic hunger was significantly correlated with greater temporal discounting. Additionally, days in abstinence were correlated with higher BMI and decreased temporal discounting (Fig. 2).

3.4. Regression models

The unadjusted regression model of temporal discounting and

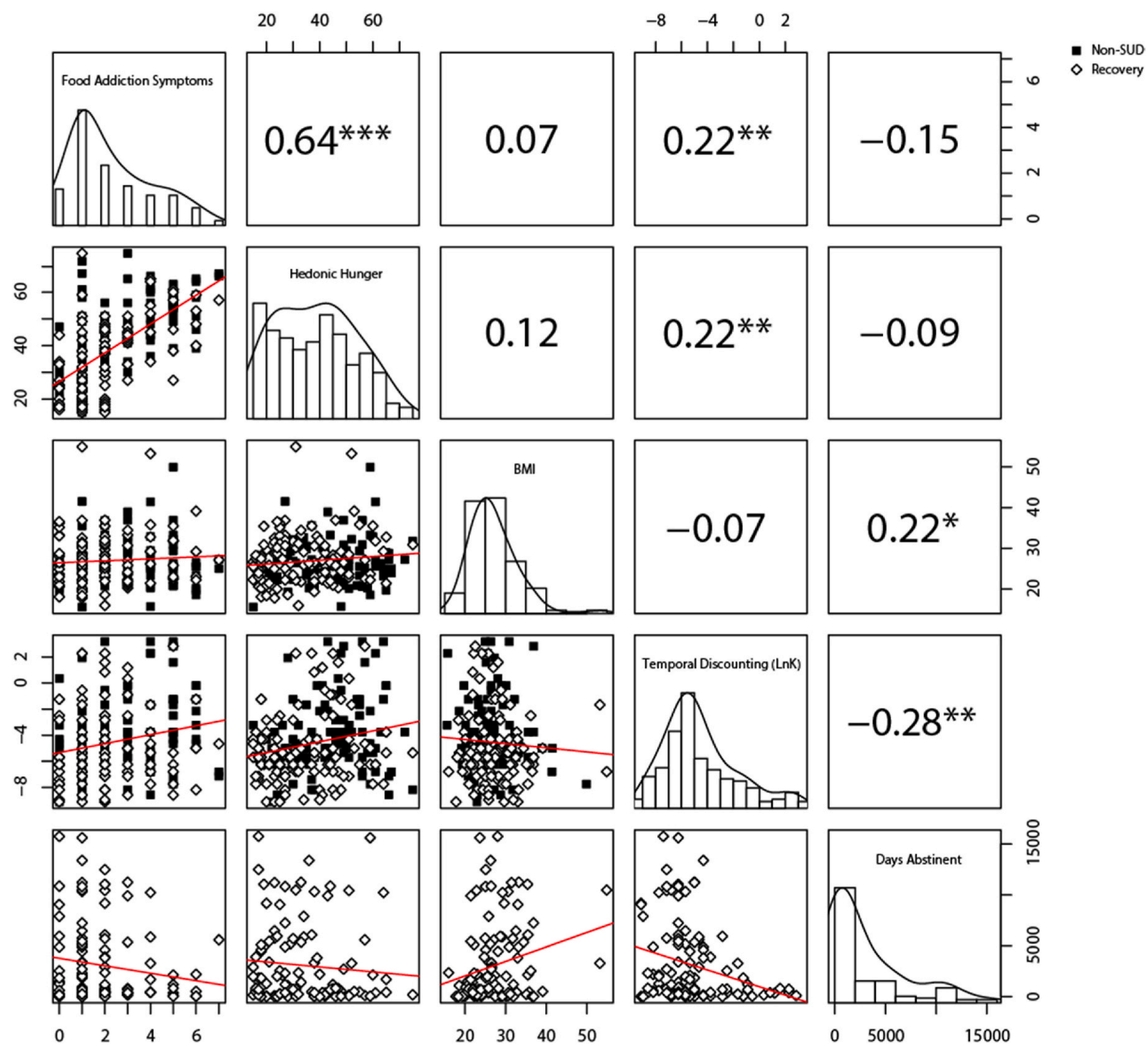

Fig. 2. Pearson correlation coefficients (top), histograms (diagonal), and correlation scatterplots (bottom). Data for days abstinent were collected only in the recovery group (n = 114). *p < 0.05, **p < 0.01, ***p < 0.001.

recovery status predicting food addiction symptoms was significant and explained 7.5% of the variance in the outcome. The model indicated that greater temporal discounting and non-SUD recovery status significantly predicted increased food addiction symptoms (Table 2). After adjusting for demographic variables, the overall model was significant and explained 11.4% of the variance in the outcome. Temporal discounting and recovery status did not significantly predict food addiction symptoms in the adjusted model (Table 2).

The unadjusted regression model of temporal discounting and recovery status predicting hedonic hunger was significant, and explained 14.1% of the variance in the outcome. The model indicated that greater temporal discounting and non-SUD recovery status significantly predicted increased hedonic hunger (Table 3). After adjusting for demographic variables, the overall model was significant and explained 18.5% of the variance in the outcome. Non-SUD recovery status, but not temporal discounting, was significantly associated with increased hedonic hunger in the adjusted model (Table 3).

The unadjusted and adjusted regression models of temporal discounting and recovery status predicting BMI were not statistically significant (Table 4).

4. Discussion

Recent work has identified that drugs of abuse and food, especially high calorically dense foods, activate similar reward circuitry of the brain, with some even conceptualizing obesity as a food addiction. During recovery from SUDs, overweight and obese outcomes are common as individuals may replace drug reward for food reward. However, little has been done to investigate hedonic hunger and food addiction symptoms in individuals recovering from SUDs. Considering that our work and others have shown that steep temporal discounting is a *trans*-disease process that underlies both SUD and obesity (Bickel et al., 2019, 2021), we were interested in investigating the relationship between temporal discounting, hedonic hunger, and food addiction symptoms in a group of individuals abstinent from substance misuse and non-substance users. Therefore, in this cross-sectional investigation, we utilized regression analyses to examine the predictive validity of recovery status and temporal discounting on hedonic hunger and food addiction symptomatology. We found that individuals in recovery from SUDs show improved outcomes in temporal discounting, hedonic hunger, and food addiction symptoms and that both recovery status and temporal discounting significantly predicted these outcome measures, which we hypothesize are due to the enhanced executive abilities needed to sustain abstinence. We also show that these effects are significantly affected by demographic variables. We discuss how our findings relate to the current literature as well as the clinical implication of these findings.

4.1. *Recovery status predicts decreased hedonic hunger and food addiction symptoms*

Our results revealed that recovery status predicts both hedonic hunger and food addiction symptoms. Specifically, compared to non-SUD controls, individuals in SUD recovery display lower levels of hedonic hunger as well as lower levels of food addiction, though the latter effect dissipated after controlling for demographic variables. This is the first time that hedonic hunger and food addiction have been examined in a group of individuals in recovery from SUDs. These findings suggest that somewhere in the process of active SUD or SUD recovery, changes in the reward-related response to food may occur, though future longitudinal, controlled studies will be needed to investigate this relationship.

Some have conceptualized that SUDs and obesity share many overlapping behavioral phenotypes and that obesity may be a food addiction (Blanco-Gandía, Miñarro, & Rodríguez-Arias, 2020; Ifland et al., 2009; Takgbajouah & Buscemi, 2021). Specifically, both drugs of abuse and food activate similar brain circuitry (i.e., the mesocortical pathway). The motivational drive that supports drug/food seeking and consumption is regulated by these dopaminergic pathways. Repeated consumption of drugs or food (especially high-calorically dense foods) alter the dopaminergic circuitry and response, causing habitual and inflexible responses that lead to SUD or obesity, respectively (Alonso-Alonso et al., 2015; Nora D. Volkow, Wang, & Baler, 2011, 2017). To this point, animal studies have shown that cross-sensitization occurs between drugs of abuse and food (Le Merrer & Stephens, 2006), additionally suggesting that the brain changes induced by substances may cause behavioral alterations to food reward and responsivity. In addition, SUDs are associated with a variety of physical changes that affect food consumption and absorption. For example, SUDs are associated with altered levels of hunger, impaired taste, malnutrition, constipation, damaged stomach lining, and altered metabolism and hormonal regulation. These physical changes in turn affect food-related brain processes, contributing to the interaction between drugs and food. During recovery from SUDs, these physical aspects may begin to improve as the maladaptive behavior of drug use is replaced by healthier behaviors such as improved eating, which again in turn cause changes to the brain (Mahboub, Rizk, Karavetian, & de Vries, 2020; Neale, Nettleton, Pickering, & Fischer, 2012).

In the present study, we investigated two aspects of food reward: hedonic hunger and food addiction. Food addiction is associated with increased BMI, obesity, and binge eating disorder (Burrows, Skinner, McKenna, & Rollo, 2017; Davis et al., 2011; Gearhardt et al., 2014; Ivezaj, White, & Grilo, 2016). Similarly, hedonic hunger is affected by food consumption and is known to be heightened in individuals with obesity (Rabiei, Sedaghat, & Rastmanesh, 2019; Ribeiro et al., 2018). Additionally, weight loss during weight loss interventions results in decreased hedonic hunger (O'Neil, Theim, Boeka, Johnson, & Miller-Kovach, 2012; Theim, Brown, Juarascio, Malcolm, & O'Neil, 2013), indicating that hedonic hunger may be a flexible process that is

Table 2
Multiple linear regression models predicting food addiction symptoms.

	R	R^2	Adjusted R^2	F	β	t	p
Dependent variable: Food Addiction Symptoms							
Unadjusted**	0.289	0.083	0.075	9.459			<0.001
Temporal Discounting*					0.176	2.599	0.010
Recovery Status**					−0.195	−2.879	0.004
Adjusted**	0.379	0.114	0.114	4.873			<0.001
Temporal Discounting					0.094	1.293	0.197
Recovery Status					−0.076	−0.989	0.324
Age**					−0.231	−2.783	0.006
Gender					−0.065	−0.964	0.336
Education*					0.188	2.545	0.012
Income					−0.079	−1.105	0.270
Race					0.041	0.562	0.575

*$p < 0.05$, **$p < 0.01$.

Table 3
Multiple linear regression models predicting hedonic hunger.

	R	R^2	Adjusted R^2	F	β	t	p
Dependent variable: Hedonic Hunger							
Unadjusted**	0.386	0.149	0.141	18.214			<0.001
Temporal Discounting*					0.155	2.373	0.019
Recovery Status**					−0.323	−4.943	<0.001
Adjusted**	0.460	0.212	0.185	7.805			<0.001
Temporal Discounting					0.060	0.871	0.385
Recovery Status**					−0.253	−3.449	<0.001
Age					−0.152	−1.904	0.058
Gender					−0.097	−1.506	0.134
Education					0.100	1.406	0.161
Income					−0.082	−1.190	0.236
Race*					0.152	2.147	0.033

*$p < 0.05$, **$p < 0.01$.

Table 4
Multiple linear regression models predicting BMI.

	R	R^2	Adjusted R^2	F	β	t	p
Dependent variable: BMI							
Unadjusted	0.079	0.006	−0.003	0.660			0.518
Temporal Discounting					−0.057	−0.810	0.419
Recovery Status					0.045	0.631	0.529
Adjusted	0.222	0.049	0.017	1.510			0.166
Temporal Discounting					0.016	0.208	0.835
Recovery Status					−0.062	−0.763	0.446
Age**					0.236	2.701	0.008
Gender					−0.015	−0.219	0.827
Education					−0.075	−0.955	0.341
Income					−0.067	−0.891	0.374
Race					−0.028	−0.360	0.719

*$p < 0.05$, **$p < 0.01$.

associated with improved outcomes during recovery from obesity. Here, we newly show that in the process of recovery from SUDs, hedonic hunger and food addiction may show similar decreases as during recovery from obesity.

Others have investigated food preference, choice, and consumption in individuals in SUD recovery. For example, several studies have shown that individuals in SUD recovery show preference for and heightened consumption of sweet and high-calorically dense foods, especially in the early stages of recovery (Gambera & Clarke, 1976; Janowsky, Pucilowski, & Buyinza, 2003; Kampov-Polevoy, Tsoi, Zvartau, Neznanov, & Khalitov, 2001; Nolan & Scagnelli, 2007). Additionally, a recent study found that individuals in treatment for SUD, compared to the general population, showed significantly higher food cravings and positive emotional eating as well as theoretical energy consumption in an online food choice task (i.e., all-you-can-eat buffet task) (Nolan, 2019). Though our findings seem contrary to this work, our population of individuals in recovery include individuals who have from 1 day to 43.2 years of abstinence with an average of 8.4 (±1.01) years. This other work examined individuals who were early in recovery, when brain function is impaired by recent drug use and decision-making processes are geared towards immediate gratification. Therefore, our work suggests that distinct changes in food reward and addiction may occur over the course of recovery.

4.2. *Steep temporal discounting predicts heightened hedonic hunger and food addiction symptoms*

As hypothesized, temporal discounting predicted hedonic hunger and food addiction symptomatology, although these effects dissipated after adjusting for demographics. Specifically, steep temporal discounting was associated with heightened levels of hedonic hunger and food addiction. Our group and others have previously found that temporal discounting, a behavioral indicator of the balance between the executive and reward systems, is associated with heightened hedonic hunger and food addiction (Satyal, Basso, Tegge, Metpally, & Bickel, 2021; VanderBroek-Stice, Stojek, Beach, vanDellen, & MacKillop, 2017), though this is the first time this relationship has been investigated in individuals recovering from substance misuse.

We additionally found that abstinent individuals with a history of substance misuse discount the future less than non-substance users. Further, we found that as time in recovery progresses, the discounting rate decreases. This is in line with previous findings indicating that individuals who discount less may be more likely to succeed in abstaining from substances (Sheffer et al., 2014; Stanger et al., 2012; Washio et al., 2011). This finding is akin to one of our recent cross-sectional studies comparing individuals who maintained substantial weight loss to weight-matched controls, showing that the weight loss maintenance group discounted the future less (Bickel, Moody, Koffarnus, Thomas, & Wing, 2018). These findings suggest that individuals in recovery (either from drugs or overeating) show heightened executive control processes. As individuals with SUDs are hypersensitive to drug rewards (Kalivas & Volkow, 2005; Lawn et al., 2015), this enhanced level of executive control is needed to successfully resist the urge to consume drug rewards and maintain abstinence. If steep temporal discounting is a driver of hedonic hunger and food addiction, and temporal discounting is heightened during the early stages of recovery, then interventions that target temporal discounting, such as episodic future thinking, may help to improve food salience and eating behaviors in early recovery.

4.3. *No association between BMI and temporal discounting or recovery status*

Contrary to our initial hypothesis, there was no association between temporal discounting or recovery status and BMI. A recent meta-analysis found that about half of existing studies found a positive association between temporal discounting and body weight while the other half found no association (Tang, Chrzanowski-Smith, Hutchinson, Kee, & Hunter, 2019). The current study adds a sample of individuals in SUD recovery to this body of literature. Although some have reported weight gain during recovery (Nolan, 2013), no difference in BMI has been reported in other samples of individuals in SUD recovery compared to the general population (Nolan, 2019). In the current study, both the recovery and control groups have an average BMI that is considered overweight and is slightly lower than the average BMI of the general U.S. population (Fryar, Kruszan-Moran, Gu, & Ogden, 2018).

4.4. Demographic variables influenced our outcomes of interest

Our findings indicate that our outcomes were significantly affected by demographic variables. Specifically, our regression models revealed that age was significantly associated with food addiction symptoms and BMI, education was significantly associated with food addiction symptoms, and race was significantly associated with hedonic hunger in this dataset. Despite these associations between demographic variables and outcomes, some demographic variables may not be determinants of the independent and/or dependent variables and therefore may not truly be sources of confounding bias (Bartram, 2021). For instance, socioeconomic measures such as education have been previously associated with temporal discounting (Bickel et al., 2014). However, it is unclear whether education determines temporal discounting or the other dependent variables in our models (e.g., food addiction, hedonic hunger, BMI). Therefore, we have provided results from all analyses both with and without controlling for demographic variables.

4.5. Limitations and future directions

While this study provides new insights into the relationships among recovery status, temporal discounting, and psychological responses to food, several limitations are worth noting. Participants recruited from the IQRR may not accurately represent the general SUD recovery population. As many IQRR members join the registry as a source of accountability and/or inspiration, the results of this study may reflect individuals in recovery who are prone to seek support. Similarly, participants recruited from mTurk may not accurately represent the general population. Although mTurk produces reliable data, some characteristics of mTurk participants may differ from traditional samples (Behrend, Sharek, Meade, & Wiebe, 2011; Goodman, Cryder, & Cheema, 2013). Additionally, characteristics of both traditional and mTurk sample characteristics may differ from characteristics of the general population. For example, the control group in this study, which was collected on mTurk, is 32% female and 10.3% Hispanic whereas the general U.S. population is 50.8% female and 18.5% Hispanic (United States Census Bureau, n.d.).

The cross-sectional design of our study limits the ability to examine these relationships over time and to establish causality or temporal relationships among outcomes. Future longitudinal investigations are warranted to examine food addiction symptomatology, hedonic hunger, temporal discounting, and BMI in a repeated-measures design to clarify the trajectory of these outcomes throughout SUD recovery and abstinence. Considering that our sample of participants in recovery utilized a variety of treatment and maintenance strategies (e.g., twelve-step programs, therapy and counseling, detoxification with medication), future research may examine the particular benefits of different treatment modalities on these outcomes. Future research may also investigate these outcomes specifically in users of different substances (e.g., alcohol, stimulants).

5. Conclusions

In this cross-section study, we found that both recovery status and temporal discounting predicted hedonic hunger and food addiction symptoms, though these predictive relationships were significantly affected by demographic characteristics. Our results support the idea that drugs of abuse and food engage similar brain circuitry and that individuals in recovery from SUDs may experience changes in not only drug-related reward processes but food-related reward processes as well. Our findings suggest that individuals in recovery from SUD actually show improved outcomes in temporal discounting as well as hedonic hunger and food addiction. This may be due to the fact that our dataset included individuals who were abstinent with a range of times in recovery up to 43.2 years (mean 8.4 years), rather than including individuals only early in recovery. Our findings suggest that the enhanced executive control processes needed for successful SUD recovery may transfer to other reward-related processes such as food reward and consumption. Indeed, previous research has shown that executive function improves over the course of recovery from SUDs, and our data support the idea that this executive recovery may support other reward-related processes as well. Additionally, considering that steep temporal discounting was associated with food addiction symptoms, our results support the idea that interventions targeted at improving executive function including episodic future thinking, meditation, or exercise may be excellent ways to support a successful recovery and improve in other reward-related processes including food consumption in support of decreasing the risk of overweight or obese outcomes during recovery.

Author contributions

JCB: conceptualization, methodology, formal analysis, writing original draft. MKS: methodology, formal analysis, visualization, writing original draft. LA: writing, review & editing. WKB: conceptualization, supervision, writing, review & editing. All authors approved the final article.

Funding

This work was supported by the Fralin Biomedical Research Institute at Virginia Tech Carilion, NIH/NIAAA [grant number R01AA027381], and National Center for Advancing Translational Science of the NIH Award [UL1TR003015/KL2TR003016].

Data availability

The dataset obtained in the current study is available from the authors upon request.

Ethical statement

This study was approved by the Institutional Review Board at Virginia Polytechnic Institute and State University [protocols 11–716 and 17–460] as reported in section 2 of the article. Electronic informed consent was obtained for all participants involved in the study.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.appet.2021.105834.

References

Alonso-Alonso, M., Woods, S. C., Pelchat, M., Grigson, P. S., Stice, E., Farooqi, S., et al. (2015). Food reward system: Current perspectives and future research needs. *Nutrition Reviews, 73*(5), 296–307.

Bartram, D. (2021). Age and life satisfaction: Getting control variables under control. *Sociology, 55*(2), 421–437.

Behrend, T. S., Sharek, D. J., Meade, A. W., & Wiebe, E. N. (2011). The viability of crowdsourcing for survey research. *Behavior Research Methods, 43*(3), 800–813.

Berry, T. R., Spence, J. C., Blanchard, C., Cutumisu, N., Edwards, J., & Nykiforuk, C. (2010). Changes in BMI over 6 years: The role of demographic and neighborhood characteristics. *International Journal of Obesity, 34*(8), 1275–1283.

Bickel, W. K., Athamneh, L. N., Basso, J. C., Mellis, A. M., DeHart, W. B., Craft, W. H., et al. (2019). Excessive discounting of delayed reinforcers as a trans-disease process: Update on the state of the science. *Current Opinion in Psychology, 30*, 59–64.

Bickel, W. K., Freitas-Lemos, R., Tomlinson, D. C., Craft, W. H., Keith, D. R., Athamneh, L. N., et al. (2021). Temporal discounting as a candidate behavioral marker of obesity. *Neuroscience & Biobehavioral Reviews*. https://doi.org/10.1016/j.neubiorev.2021.07.035

Bickel, W. K., Lemos, R., Tomlinson, D. C., & Tegge, A. N. (n.d.). Behavioral economic cigarette demand among individuals who smoke with and without obesity (in press). Health psychology: Official journal of the division of health psychology, American Psychological Association.

Bickel, W. K., Mellis, A. M., Snider, S. E., Athamneh, L. N., Stein, J. S., & Pope, D. A. (2018). 21st century neurobehavioral theories of decision making in addiction: Review and evaluation. *Pharmacology, Biochemistry, and Behavior, 164*, 4–21.

Bickel, W. K., Moody, L. N., Koffarnus, M., Thomas, J. G., & Wing, R. (2018). Self-control as measured by delay discounting is greater among successful weight losers than controls. *Journal of Behavioral Medicine, 41*(6), 891–896.

Bickel, W. K., Moody, L., Quisenberry, A. J., Ramey, C. T., & Sheffer, C. E. (2014). A Competing Neurobehavioral Decision Systems model of SES-related health and behavioral disparities. *Preventive Medicine, 68*, 37–43.

Blanco-Gandía, M. C., Miñarro, J., & Rodríguez-Arias, M. (2020). Common neural mechanisms of palatable food intake and drug abuse: Knowledge obtained with animal models. *Current pharmaceutical design*. https://doi.org/10.2174/1381612826666200213123608

Burrows, T., Skinner, J., McKenna, R., & Rollo, M. (2017). Food addiction, binge eating disorder, and obesity: Is there a relationship? *Behavioral Sciences, 7*(3). https://doi.org/10.3390/bs7030054

Campana, B., Brasiel, P. G., de Aguiar, A. S., & Dutra, S. C. P. L. (2019). Obesity and food addiction: Similarities to drug addiction. *Obesity Medicine, 16*, 100136.

Cappelleri, J. C., Bushmakin, A. G., Gerber, R. A., Leidy, N. K., Sexton, C. C., Karlsson, J., et al. (2009). Evaluating the power of food Scale in obese subjects and a general sample of individuals: Development and measurement properties. *International Journal of Obesity, 33*(8), 913–922.

Claassen, M. A., Klein, O., Bratanova, B., Claes, N., & Corneille, O. (2019). A systematic review of psychosocial explanations for the relationship between socioeconomic status and body mass index. *Appetite, 132*, 208–221.

Cowan, J., & Devine, C. (2008). Food, eating, and weight concerns of men in recovery from substance addiction. *Appetite, 50*(1), 33–42.

Davis, C., Curtis, C., Levitan, R. D., Carter, J. C., Kaplan, A. S., & Kennedy, J. L. (2011). Evidence that "food addiction" is a valid phenotype of obesity. *Appetite, 57*(3), 711–717.

Di Chiara, G. (2002). Nucleus accumbens shell and core dopamine: Differential role in behavior and addiction. *Behavioural Brain Research, 137*(1–2), 75–114.

Espel-Huynh, H. M., Muratore, A. F., & Lowe, M. R. (2018). A narrative review of the construct of hedonic hunger and its measurement by the Power of Food Scale. *Obesity Science & Practice, 4*(3), 238–249.

Ferrario, C. R. (2017). Food addiction and obesity. Neuropsychopharmacology. *Official Publication of the American College of Neuropsychopharmacology, 42*(1), 361.

Fryar, C. D., Kruszan-Moran, D., Gu, Q., & Ogden, C. L. (2018). *Mean body weight, weight, waist circumference, and body mass index among adults : United States, 1999–2000 through 2015–2016*. https://stacks.cdc.gov/view/cdc/61430.

Gambera, S. E., & Clarke, J. A. (1976). Comments on dietary intake of drug-dependent persons. *Journal of the American Dietetic Association, 68*(2), 155–157.

Gearhardt, A. N., Boswell, R. G., & White, M. A. (2014). The association of "food addiction" with disordered eating and body mass index. *Eating Behaviors, 15*(3), 427–433.

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale food addiction Scale. *Appetite, 52*(2), 430–436.

Gearhardt, A. N., Yokum, S., Orr, P. T., Stice, E., Corbin, W. R., & Brownell, K. D. (2011). Neural correlates of food addiction. *Archives of General Psychiatry, 68*(8), 808–816.

Goodman, J. K., Cryder, C. E., & Cheema, A. (2013). Data collection in a flat world: The strengths and weaknesses of mechanical Turk samples: Data collection in a flat world. *Journal of Behavioral Decision Making, 26*(3), 213–224.

Hodgkins, C. C., Cahill, K. S., Seraphine, A. E., Frost-Pineda, K., & Gold, M. S. (2004). Adolescent drug addiction treatment and weight gain. *Journal of Addictive Diseases, 23*(3), 55–65.

Hodgkins, C. C., Jacobs, W. S., & Gold, M. S. (2003). Weight gain after adolescent drug addiction treatment and supervised abstinence. *Psychiatric Annals, 33*(2), 112–116.

Ifland, J. R., Preuss, H. G., Marcus, M. T., Rourke, K. M., Taylor, W. C., Burau, K., et al. (2009). Refined food addiction: A classic substance use disorder. *Medical Hypotheses, 72*(5), 518–526.

Ivezaj, V., White, M. A., & Grilo, C. M. (2016). Examining binge-eating disorder and food addiction in adults with overweight and obesity. *Obesity, 24*(10), 2064–2069.

Jackson, T. D., & Grilo, C. M. (2002). Weight and eating concerns in outpatient men and women being treated for substance abuse. *Eating and Weight Disorders: EWD, 7*(4), 276–283.

Janowsky, D. S., Pucilowski, O., & Buyinza, M. (2003). Preference for higher sucrose concentrations in cocaine abusing-dependent patients. *Journal of Psychiatric Research, 37*(1), 35–41.

Kalivas, P. W., & Volkow, N. D. (2005). The neural basis of addiction: A pathology of motivation and choice. *American Journal of Psychiatry, 162*(8), 1403–1413.

Kampov-Polevoy, A. B., Tsoi, M. V., Zvartau, E. E., Neznanov, N. G., & Khalitov, E. (2001). Sweet liking and family history of alcoholism in hospitalized alcoholic and non-alcoholic patients. *Alcohol and Alcoholism, 36*(2), 165–170.

Kekic, M., McClelland, J., Bartholdy, S., Chamali, R., Campbell, I. C., & Schmidt, U. (2019). Bad things come to those who do not wait: Temporal discounting is associated with compulsive overeating, eating disorder psychopathology and food addiction. *Frontiers in Psychiatry/Frontiers Research Foundation, 10*, 978.

Koffarnus, M. N., & Bickel, W. K. (2014). A 5-trial adjusting delay discounting task: Accurate discount rates in less than one minute. *Experimental and Clinical Psychopharmacology, 22*(3), 222–228.

Koob, G. F., & Volkow, N. D. (2016). Neurobiology of addiction: A neurocircuitry analysis. The lancet. *Psychiatry, 3*(8), 760–773.

Lawn, W., Freeman, T. P., Hindocha, C., Mokrysz, C., Das, R. K., Morgan, C. J. A., et al. (2015). The effects of nicotine dependence and acute abstinence on the processing of drug and non-drug rewards. *Psychopharmacology, 232*(14), 2503–2517.

Le Merrer, J., & Stephens, D. N. (2006). Food-induced behavioral sensitization, its cross-sensitization to cocaine and morphine, pharmacological blockade, and effect on food intake. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 26*(27), 7163–7171.

Lerma-Cabrera, J. M., Carvajal, F., & Lopez-Legarrea, P. (2016). Food addiction as a new piece of the obesity framework. *Nutrition Journal, 15*, 5.

Levitt, E., Sanchez-Roige, S., Palmer, A. A., & MacKillop, J. (2020). Steep discounting of future rewards as an impulsivity phenotype: A concise review. *Current topics in behavioral neurosciences*. https://doi.org/10.1007/7854_2020_128

Lowe, M. R., Butryn, M. L., Didie, E. R., Annunziato, R. A., Thomas, J. G., Crerand, C. E., et al. (2009). The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite, 53*(1), 114–118.

Mahboub, N., Rizk, R., Karavetian, M., & de Vries, N. (2020). Nutritional status and eating habits of people who use drugs and/or are undergoing treatment for recovery: A narrative review. *Nutrition Reviews, 79*(6), 627–635.

McClure, S. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2004). Separate neural systems value immediate and delayed monetary rewards. *Science, 306*(5695), 503–507.

Michaud, A., Vainik, U., Garcia-Garcia, I., & Dagher, A. (2017). Overlapping neural endophenotypes in addiction and obesity. *Frontiers in Endocrinology, 8*, 127.

Neale, J., Nettleton, S., Pickering, L., & Fischer, J. (2012). Eating patterns among heroin users: A qualitative study with implications for nutritional interventions. *Addiction, 107*(3), 635–641.

Nolan, L. J. (2013). Shared urges? The links between drugs of abuse, eating, and body weight. *Current Obesity Reports, 2*(2), 150–156.

Nolan, L. J. (2019). Food selection, food craving, and body mass index in persons in treatment for substance use disorder. *Appetite, 138*, 80–86.

Nolan, L. J., & Scagnelli, L. M. (2007). Preference for sweet foods and higher body mass index in patients being treated in long-term methadone maintenance. *Substance Use & Misuse, 42*(10), 1555–1566.

O'Neil, P. M., Theim, K. R., Boeka, A., Johnson, G., & Miller-Kovach, K. (2012). Changes in weight control behaviors and hedonic hunger during a 12-week commercial weight loss program. *Eating Behaviors, 13*(4), 354–360.

Orsini, C. A., Ginton, G., Shimp, K. G., Avena, N. M., Gold, M. S., & Setlow, B. (2014). Food consumption and weight gain after cessation of chronic amphetamine administration. *Appetite, 78*, 76–80.

Rabiei, S., Sedaghat, F., & Rastmanesh, R. (2019). Is the hedonic hunger score associated with obesity in women? A brief communication. *BMC Research Notes, 12*(1), 330.

Ribeiro, G., Camacho, M., Santos, O., Pontes, C., Torres, S., & Oliveira-Maia, A. J. (2018). Association between hedonic hunger and body-mass index versus obesity status. *Scientific Reports, 8*(1), 5857.

Roitman, M. F., Stuber, G. D., Phillips, P. E. M., Wightman, R. M., & Carelli, R. M. (2004). Dopamine operates as a subsecond modulator of food seeking. *Journal of Neuroscience: The Official Journal of the Society for Neuroscience, 24*(6), 1265–1271.

Satyal, M. K., Basso, J. C., Tegge, A. N., Metpally, A. R., & Bickel, W. K. (2021). A novel model of obesity prediction: Neurobehaviors as targets for treatment. *Behavioral Neuroscience, 135*(3), 426–442.

Sheffer, C. E., Christensen, D. R., Landes, R., Carter, L. P., Jackson, L., & Bickel, W. K. (2014). Delay discounting rates: A strong prognostic indicator of smoking relapse. *Addictive Behaviors, 39*(11), 1682–1689.

Stanger, C., Ryan, S. R., Fu, H., Landes, R. D., Jones, B. A., Bickel, W. K., et al. (2012). Delay discounting predicts adolescent substance abuse treatment outcome. *Experimental and Clinical Psychopharmacology, 20*(3), 205–212.

Stice, E., Spoor, S., Bohon, C., & Small, D. M. (2008). Relation between obesity and blunted striatal response to food is moderated by TaqIA A1 allele. *Science, 322*(5900), 449–452.

Takgbajouah, M., & Buscemi, J. (2021). Applying the developmental model of use disorders to hedonic hunger: A narrative review. *Journal of Addictive Diseases, 1*, 9.

Tang, J., Chrzanowski-Smith, O. J., Hutchinson, G., Kee, F., & Hunter, R. F. (2019). Relationship between monetary delay discounting and obesity: A systematic review and meta-regression. *International Journal of Obesity, 43*(6), 1135–1146.

Theim, K. R., Brown, J. D., Juarascio, A. S., Malcolm, R. R., & O'Neil, P. M. (2013). Relations of hedonic hunger and behavioral change to weight loss among adults in a behavioral weight loss program utilizing meal-replacement products. *Behavior Modification, 37*(6), 790–805.

Trifilieff, P., & Martinez, D. (2014). Imaging addiction: D2 receptors and dopamine signaling in the striatum as biomarkers for impulsivity. *Neuropharmacology, 76 Pt B*, 498–509.

United States Census Bureau, & QuickFacts, n.d.). United States [Data set]. Retrieved July 12, 2021, from https://www.census.gov/quickfacts/fact/table/US/LFE046219.

Vainik, U., Misic, B., Zeighami, Y., Michaud, A., Mõttus, R., & Dagher, A. (2020). Obesity has limited behavioural overlap with addiction and psychiatric phenotypes. *Nature Human Behaviour, 4*(1), 27–35.

VanderBroek-Stice, L., Stojek, M. K., Beach, S. R. H., vanDellen, M. R., & MacKillop, J. (2017). Multidimensional assessment of impulsivity in relation to obesity and food addiction. *Appetite, 112*, 59–68.

Volkow, N. D., Fowler, J. S., Wang, G. J., Baler, R., & Telang, F. (2009). Imaging dopamine's role in drug abuse and addiction. *Neuropharmacology, 56*(Suppl 1), 3–8.
Volkow, N. D., Wang, G.-J., & Baler, R. D. (2011). Reward, dopamine and the control of food intake: Implications for obesity. *Trends in Cognitive Sciences, 15*(1), 37–46.
Volkow, N. D., Wang, G.-J., Fowler, J. S., & Telang, F. (2008). Overlapping neuronal circuits in addiction and obesity: Evidence of systems pathology. *Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences, 363*(1507), 3191–3200.
Volkow, N. D., Wang, G.-J., Telang, F., Fowler, J. S., Thanos, P. K., Logan, J., et al. (2008). Low dopamine striatal D2 receptors are associated with prefrontal metabolism in obese subjects: Possible contributing factors. *NeuroImage, 42*(4), 1537–1543.
Volkow, N. D., & Wise, R. A. (2005). How can drug addiction help us understand obesity? *Nature Neuroscience, 8*(5), 555–560.
Volkow, N. D., Wise, R. A., & Baler, R. (2017). The dopamine motive system: Implications for drug and food addiction. *Nature Reviews Neuroscience, 18*(12), 741–752.
Wang, G. J., Volkow, N. D., Logan, J., Pappas, N. R., Wong, C. T., Zhu, W., et al. (2001). Brain dopamine and obesity. *The Lancet, 357*(9253), 354–357.
Washio, Y., Higgins, S. T., Heil, S. H., McKerchar, T. L., Badger, G. J., Skelly, J. M., et al. (2011). Delay discounting is associated with treatment response among cocaine-dependent outpatients. *Experimental and Clinical Psychopharmacology, 19*(3), 243–248.
Williamson, D. F., Madans, J., Anda, R. F., Kleinman, J. C., Giovino, G. A., & Byers, T. (1991). Smoking cessation and severity of weight gain in a national cohort. *New England Journal of Medicine, 324*(11), 739–745.