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Explaining Excessive Weight Gain during Early Recovery from Addiction

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Abstract

Background: Many people receiving treatment for addiction gain an excessive amount of weight during early recovery. We outline two hypothesized mechanisms that might explain weight gain: The *Addiction Transfer Hypothesis*, which suggests that some individuals respond to cravings with non-nutritive eating behavior, and the *Propensity for Behavioral Addiction Hypothesis*, which suggests that some people are at higher risk for addiction, and that excess weight gain results from a rebound of appetitive processes that were temporarily suppressed during active addiction.

Method: We evaluate the extent of support for these alternative hypotheses using repeated measures of cravings and eating behavior collected in real time using a combination of ecological momentary assessment methodology and interviewer-based 24-hour dietary recall. Participants included N=111 individuals receiving treatment for substance use disorder who were currently abstaining from use, but who had used their primary treatment substance within the past 12 months.

Results: Using linear mixed models to test the temporal effects of cravings on subsequent eating behaviors hypothesized by the *Addiction Transfer Hypothesis* and generalized linear models to evaluate the effect of a common propensity for behavioral addiction factor on eating behaviors (a test of the *Propensity for Behavioral Addiction Hypothesis*), we find no evidence to support the *Addiction Transfer Hypothesis*, but we find modest support for the *Propensity for Behavioral Addiction Hypothesis*. Findings do not account for appetitive effects of psychotropic medications.

Conclusions: General nutrition education and encouragement of health eating behaviors may be useful for reducing excessive weight gain among people recovering from substance dependence.

Keywords

Behavioral addiction; Addiction transfer; Addiction recovery; Excessive weight gain; Ecological momentary assessment; 24-hour recall

People who are in the early stages of recovery from substance use disorders—including recovery from alcohol, cocaine, opioids, tobacco, and cannabis—tend to gain extra weight,

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beyond the amount necessary to recover their pre-addiction body weight. Whereas modest weight gain during recovery may be a compensatory mechanism to restore weight lost during active addiction, recent research has demonstrated that weight gain is excessive among many individuals in recovery (Billing & Ersche, 2015; Cocores & Gold, 2009; Cowan & Devine, 2008; Hodgkins, Cahill, Seraphine, Frost-Pineda, & Gold, 2004; Nolan & Scagnelli, 2007; VanBuskirk & Potenza, 2010). Addressing excessive weight gain among individuals in recovery is a major public health priority, as excess weight gained during recovery is a risk factor for obesity-related disorders and may cause significant body image distress and increase the risk of relapse (Billing & Ersche, 2015; Gold, Frost-Pineda, & Jacobs, 2003).

The central aim of this manuscript is to outline and test two alternative theories explaining excess weight gain during recovery from addiction. The *Addiction Transfer Hypothesis* posits that highly palatable foods are used as a substitute for drugs or alcohol when cravings arise, leading to over-consumption of high-fat and high-sugar foods for non-nutritive purposes. The *Propensity for Behavioral Addiction* posits that there are common, trait-level underpinnings to binge eating and substance dependence, and that these different behavioral addictions may emerge under different situations.

The Addiction Transfer Hypothesis

Addictive behavior is characterized by a *spiral of dysregulation*, whereby the habitual behavior (e.g., drinking 15 ounces of liquor between 8 and 10 pm each night or eating a bar of chocolate daily at 3 pm) that was once a positive reinforcer resets the homeostatic set-point such that the individual with the habitual behavior must now compulsively engage in the behavior, in higher and higher amounts, simply to attain homeostasis (Koob & Le Moal, 2001; Volkow, Koob, & McLellan, 2016). In other words, the addictive behavior becomes negatively reinforcing. It follows from this spiraling dysregulation model that individuals in early recovery may be at high risk for addiction transfer, specifically to foods and beverages that share similar neurobiological mechanisms of action to the substance(s) for which they are receiving treatment. For instance, high-fat, high-sugar, and high-caffeine foods interact with endogenous opioid and dopamine systems that are involved in liking and craving, respectively (Blumenthal & Gold, 2010; Gearhardt et al., 2011; Levine, Kotz, & Gosnell, 2003; Schulte, Avena, & Gearhardt, 2015; Solinas et al., 2002; Thornley, McRobbie, Eyles, Walker, & Simmons, 2008a; Volkow, Wang, Fowler, Tomasi, & Baler, 2011; Zhang, Gosnell, & Kelley, 1998), and so individuals recovering from substance use may transfer addictions to these food types.

In a series of qualitative interviews with 25 men at different stages of recovery from drug and alcohol addiction, Cowan and Devine found that men in early recovery described dysfunctional eating practices, including emotional and binge eating, substituting food for drug use, and using food to satisfy cravings (Cowan & Devine, 2008). We have heard similar anecdotes in our own (unpublished) qualitative interviews with women in recovery, and such accounts have been reported widely in popular media (Castaneda, 2017; Ellin, 2014). Furthermore, this behavior is recommended explicitly in the Alcoholics Anonymous tome, *The Big Book*: "...all alcoholics should constantly have chocolate available for its quick

energy value in times of fatigue....,” and night cravings “would be satisfied by candy” (1975; pp.133–134).

In addition to qualitative accounts, there is some empirical support for the concept of addiction transfer. Known as “consummatory cross-sensitization” in experimental animal research, several studies have found that abstinence from addictive substances leads to binge eating behaviors in animals and vice versa (Avena, Rada, & Hoebel, 2008). Although human studies have found significant relationships between being in substance use recovery and having an increased liking for sweet foods, less common has been research evaluating the potential for addiction transfer to caffeine, fat, or general binge eating behavior, in spite of plausible mechanisms for addiction transfer to these other high-reward eating behaviors. Thus, we provide a more rigorous test of the *Addiction Transfer Hypothesis* by evaluating the degree to which an individual’s various behavioral eating patterns are preceded by self-reported cravings in real-time.

The Propensity for Behavioral Addiction Hypothesis

An alternative explanation for excess weight gain during recovery is that certain individuals might be more prone to compulsive, reward-seeking behaviors including both substance abuse and impulsive food intake (e.g., binge eating, high sugar consumption) (Kampov-Polevoy et al., 2014; Munn-Chernoff & Baker, 2016). This explanation relies on the concept of behavioral addiction described by Volkow and colleagues (2016). The concept of behavioral addiction, particularly as it relates to eating behaviors, is hotly contested in the literature (Davis et al., 2011; Hebebrand et al., 2014; Pursey, Stanwell, Stroman, Collins, & Burrows, 2017; Ziauddeen & Fletcher, 2013). Regardless of whether compulsive, non-nutritive eating patterns are labeled as a form of “behavioral addiction,” or are given a less controversial label (e.g., “disorder of self-regulation”(Volkow et al., 2016)), abundant evidence suggests that some individuals are prone to engage in compulsive behaviors *as if* in an addicted state, and that non-nutritive eating is one relatively prevalent form of such behavior.

Thus, under the *Propensity for Behavioral Addiction Hypothesis*, an individual’s general tendency to engage in binge eating practices are (intentionally or unintentionally) overshadowed by the metabolic or appetite-suppressing effects of alcohol or drugs, or by the preference to spend money on these substances rather than food (Jeynes & Gibson, 2017). During recovery, this temporary suppression of excessive eating behavior is removed, leading individuals to re-establish overweight BMI trajectories.

The *Propensity for Behavioral Addiction Hypothesis* is supported by research on the comorbidity of eating disorders (ED) and substance use disorders (SUD) that suggests having one disorder greatly increases the likelihood of experiencing others. For instance, a person with a history of drug dependence has 8.0 times greater odds of developing Bulimia Nervosa than a person without a history of drug dependence (Hudson, Hiripi, Pope, & Kessler, 2007). Even more strikingly, heritability of comorbid ED and SUD has been estimated to be on the order of .23 and .61, depending on the specific disorders (Munn-Chernoff & Baker, 2016). This means that genetics alone explain a substantial portion of an

individual's predisposition for developing comorbid ED and SUD. Along the molecular pathway, it is suggested that individuals with the highest genetic risk for ED or SUD are more likely to have dopamine deficiency, also known as a reward deficiency syndrome, so individuals with a heightened propensity to engage in addictive behaviors may exhibit other characteristics, such as impulsivity and sensation-seeking (Comings & Blum, 2000; Dawe & Loxton, 2004; Volkow et al., 2011).

If it is true that weight gain during recovery is, at least in part, a return to pre-addiction tendencies to engage in compulsive eating behaviors that are consistent with behavioral addiction, then there should be no systematic relationship between momentary cravings and food choices, but instead a general tendency for people with more severe addictions to consume rewarding, or "highly palatable" foods. In terms of specific nutritional content, food that is high in sugar, or high in both fat and sugar, have been flagged as having this addictive potential (Avena et al., 2008; Schulte et al., 2015; Thornley et al., 2008a).

Study Overview

Our primary study aim was to compare the degree of evidence favoring the *Addiction Transfer Hypothesis* versus the *Propensity for Behavioral Addiction Hypothesis* in explaining food choices and weight gain amongst individuals early in recovery from addiction. The *Addiction Transfer* model was tested by evaluating whether momentary cravings were linked with consumption of added sugar, fat, caffeine, or calories within two hours of the self-reported craving. The *Propensity for Behavioral Addiction* model was tested by constructing a latent construct that reflected shared covariance amongst the following indicators: low self-control (Baler & Volkow, 2006), high impulsivity (Crews & Boettiger, 2009; Garavan, 2011), greater food addiction symptoms (Volkow et al., 2011; Volkow & Wise, 2005), a family history of addiction (Agrawal et al., 2012), and a greater number of treatment substances (Smith et al., 1992; Vanyukov et al., 2003). Nutritional outcomes were then regressed on this *Propensity for Behavioral Addiction* construct.

To date, a few studies have used self-report, cross-sectional survey methods to assess individual perceptions of their own eating behavior during recovery from addiction to begin to understand eating habits of individuals recovering from addiction to specific substances (Nolan & Scagnelli, 2007; Peles, Schreiber, Sason, & Adelson, 2016; Shrestha et al., 2018). However, self-reports of underlying behavior patterns are notoriously unreliable; respondents may rely on incorrect beliefs or heuristics when asked to self-report on patterns of eating behavior during recovery. Researchers have called for improved measurement of food intake in future studies that assess the relationship between substance use and nutrition (Nolan & Scagnelli, 2007).

The current study relies on a gold-standard, 24-hour dietary recall methods in which interviewers were trained to reliably assess daily nutritional intake over many days. To establish whether and how eating patterns relate to drug and alcohol cravings during recovery, dietary data were mapped on to ecological momentary assessments (EMA) that assessed drug and alcohol cravings at four random times per day. Given that eating behavior and cravings were frequently assessed, we were able to establish temporal precedence of

cravings and subsequent eating behavior without asking people to retrospectively report on these patterns.

Method

Participants

Participants were recruited via in-person visits to addiction clinics, flyers, brochures given to new patients at intake at their addiction clinic, directed phone calls, and paper mailings.

Prospective participants were screened to ensure that they met the following study criteria: (1) at least 18 years of age; (2) not actively using their primary substance of abuse; (3) use of primary substance within the past 12 months; (4) access to a personal mobile phone; and (5) not currently pregnant. Pregnant women were excluded from the study until after giving birth due to their different nutritional requirements.

A total of N=111 participants receiving addiction treatment contributed data to this study (see Figure 1 for a chart depicting criteria for inclusion in this analysis). All participants included in this study had used their primary substance(s) within the past 12 months. The mean participant age was 38.52 (SD=9.94). Seventy-four percent of participants were female, 72% were White, 25% were African American, and 3% were Hispanic. Mean participant BMI was 29.17 (SD=8.83). Thirty-five percent of participants reported earning a high school diploma or less, 47% attended some college or earned an Associate's Degree, and 18% earned a Bachelor's Degree or higher. Eight percent of participants reported having received an eating disorder diagnosis in their lifetime.

Alcohol was the most common substance for which participants reported receiving treatment (53%), followed by cocaine (38%), heroin (27%), prescription opioids (27%), marijuana (21%), methamphetamines (7%), and other substances (4%). As is normative for individuals receiving addiction treatment, treatment categories were not mutually exclusive: whereas 45% of individuals reported receiving treatment for only one substance, 36% reported receiving treatment for two substances, and 19% reported receiving treatment for three or more substances. Of participants reporting receiving treatment for only one substance, 40% used only alcohol, 20% used only prescription opioids, 20% used only heroin, 16% used only cocaine, 2% used only methamphetamine, and 2% used only marijuana. Of individuals reporting receiving treatment for more than one substance, paired substances were: alcohol and cocaine (44%), alcohol and marijuana (33%), heroin and prescription opioids (22%), cocaine and heroin (19%), cocaine and marijuana (17%), prescription opioids and cocaine (14%), heroin and alcohol (13%), prescription opioids and marijuana (11%), and heroin and marijuana (11%).

At baseline, 25% of participants reported that they had used their primary treatment substance(s) within the past month, 26% reported using within the past 1–3 months, 31% reported using within the past 3–6 months, and 18% reported using within the past 6–12 months.

Procedures

This study was approved by the UNC Institutional Review Board and all participants provided written informed consent. Participants completed in-person intake interviews, which included a computer-based survey, and they were trained in measuring portion sizes and were instructed to note all ingredients and the cooking method used to cook their food. They were given a small notebook and pen to carry with them so that they could record meals in real time, along with a wallet-sized handout for estimating portion size. Participants selected a time for the interviewer to call them each day for a nutritional assessment. During the intake session, participants chose whether to receive EMA surveys via a text message that linked to a survey or via a phone call that permitted them to use an interactive voice messaging system (IVRS). Twenty-three percent chose to receive text-based EMA surveys and 77% chose IVRS. Participants provided their typical wake and sleep times and EMA surveys were scheduled to not interfere with participant sleep schedules.

This intake session initiated a series of three, week-long, measurement bursts during which they recorded all their food and beverage intake (except for water) and conducted daily 24-hour dietary recalls over the telephone with an interviewer trained to ask questions to extract high-quality nutritional data through using the Nutrition Data System for Research (NDSR) (Schakel, 2001). During these interviews, participants were prompted to report the timing, ingredients, and cooking method for every eating episode in the previous 24 hours. Participants were reimbursed \$3 per nutritional assessment completed.

In addition to these dietary recall calls, participants received four brief EMA surveys each day during the week-long period, which collected information regarding substance cravings. These surveys were scheduled to occur within four random uniform intervals that were defined uniquely by each participant's sleep and wake times, with a constraint that no two surveys could be sent within one hour of each other. Participants were reimbursed \$1 for every EMA survey that they completed. Participants were instructed to skip an EMA survey if they received their next survey before they had an opportunity to fill out the previous survey. These week-long "measurement bursts" were repeated two additional times with six weeks between each burst.

To ensure that each of our data points were valid and unique, we manually removed from the dataset any EMA survey that was taken within two hours of the previous survey. A two hour increment was chosen because this was the time frame during which nutritional outcomes were measured for each EMA survey measure. We also removed EMA surveys from the analysis if they were provided on days on which no nutritional data were collected. Finally, because occasional lapses were inevitable among this population, we excluded burst-level data when participants indicated use during that burst; however, we did not exclude these individuals from the full analysis sample.

After applying these restrictions, a total of N=104 currently abstinent individuals in the sample provided both nutrition data and EMA survey data for at least one day during Burst 1, N=91 currently abstinent individuals provided both types of data for at least one day during Burst 2, and N=73 currently abstinent individuals provided both types of data for at least one day during Burst 3. As shown in Figure 1, N=111 individuals contributed data to at

least one burst and were thus included in the analytic sample. Among respondents eligible for the present analyses, the average number of EMA observations per person was 16.41(SD=7.95) during Burst 1, 15.40 (SD=7.09) during Burst 2, and 16.65 (SD=7.41) during Burst 3. In total, there were an average of 38.20 unique EMA surveys with same-day food data per person (SD=22.71) across all individuals and all bursts.

Measures

Nutritional Outcomes.—We used estimates tabulated from the NDSR software to quantify number of (kilo)calories, grams of fat, grams of added sugar, and milligrams of caffeine consumed within each two-hour period following an EMA survey. When no eating episodes were reported during a specific two-hour period but nutrition data had been collected on that day, these nutritional values were coded as ‘0.’ When multiple eating episodes were reported within a two-hour span following the EMA, nutritional information was summed across the relevant episodes.

Cravings.—In line with other EMA surveys with people addicted to substances, we used a single item to assess momentary cravings (Carter et al., 2008; Epstein et al., 2009). Participants were asked: “How much do you crave drugs or alcohol right now?” Response options were: “Not at all,” “Just a little,” “A moderate amount,” “Quite a lot,” and “It is all I can think about.”

Propensity for Behavioral Addiction Hypothesis.—We generated factor score estimates that measured common variation amongst items and scales that have been theoretically linked to an underlying propensity to engage in addictive behaviors. These measures, all of which were collected during the intake assessment, included: the total number of different substances for which the individual reported receiving treatment, whether the individual reported a family history of substance problems, trait impulsivity (as measured by the short UPPS impulsive behavior scale)(Cyders, Littlefield, Coffey, & Karyadi, 2014), trait self-control(Hoyle & Davisson, 2016), and number of food addiction symptoms as measured by the modified Yale Food Addiction Scale (e.g., “I find myself consuming certain foods even though I am no longer hungry;” “My behavior with respect to food and eating causes me significant distress”(Flint et al., 2014).

Before generating factor score estimates, we compared the fit of one-, two-, and three-factor models using exploratory factor analysis. The two- and three-factor models did not generate a superior fit to the one-factor model and all items loaded significantly on the single factor, indicating that all of the indicators were reflective of a single, underlying construct (i.e., propensity for behavioral addiction). We used robust maximum likelihood estimation in Mplus version 7.4 to generate factor score estimates that accounted for the non-normal response distributions of some of the items. To evaluate the sensitivity of our results to the inclusion of food addiction symptoms, we also estimated factor score estimates excluding this indicator.

Weight Gain and Appetite Increase.—On the intake survey, individuals were asked to report whether they had experienced weight gain or loss, or whether they had noticed an

increase or decrease in the appetite during recovery. We scored each variable as “1” if the participant endorsed the item, and “0” otherwise.

Control Variables.—We controlled for measurement burst, age at intake (mean centered), gender, time since last use of treatment substance (measured at intake), and treatment substance. We included dummy variables for use of opiates, cocaine, and alcohol, thereby allowing for poly-substance use.

Data Analysis

Testing the Addiction Transfer Hypothesis.—We used linear mixed models with restricted maximum likelihood estimation to predict nutritional outcomes from cravings, accounting for nesting of observations within individuals. We included random effects of the intercept and cravings using an unstructured covariance matrix. We disaggregated cravings into their within-person and between-person components (Curran & Bauer, 2011) to evaluate whether eating patterns differ *between* individuals who experience consistently more cravings, or whether eating patterns are influenced by *within-person* fluctuations in cravings. The latter is theoretically more consistent with the *Addiction Transfer Hypothesis* because a significant temporal within-person association suggests that individuals respond to cravings by consuming certain types of food. We compared fit of models with burst treated as a nominal variable versus a continuous variable and chose to treat burst as continuous (centered at burst=1). After testing the main effects of cravings on subsequent nutritional outcomes, we explored the moderating effects of gender and primary substance on the effect of craving.

Testing the Propensity for Behavioral Addiction Hypothesis.—Because addiction propensity was measured at the individual level with a factor score estimates, we used linear regression models to predict the average number of calories, grams of fat and added sugar, and milligrams of caffeine, that were consumed within each two-hour episode from the factor score estimate. We also used logistic regression to predict the odds of self-reported weight gain or increased appetite during recovery from addiction propensity. All models controlled for control variables mentioned above.

Results

Addiction Transfer Hypothesis.

Results of the *Addiction Transfer Hypothesis* tests are presented in Table 1. People reported consuming fewer calories ($B=-22.71$, $SE=9.98$), and marginally significantly less fat ($B=-.91$, $SE=.50$, $p=.07$), per two-hour interval with increasing bursts.

At the between-person level, after controlling for burst, age, gender, time since last use of primary substance, and primary substance, people who tended to experience more cravings tended to consume *fewer* calories per two-hour interval compared to people who experienced fewer cravings ($B=-43.81$, $SE=21.10$). At the within-person level, people tended to eat marginally significantly *less* added sugar in the two-hour interval after experiencing cravings that were greater than their average cravings ($B=-.79$, $SE=.46$). The

random intercept was significant in all models, but the only random effect of craving that was significant was for the effect on added sugar. This suggests that there are individual differences with respect to whether individuals consume less sugar after experiencing cravings.

Propensity for Behavioral Addiction Hypothesis.

Results of the *Propensity for Behavioral Addiction Hypothesis* tests are presented in Table 2. Over and above age, gender, time since last use, and primary substance, addiction propensity factor scores were marginally significantly associated with increased calorie consumption ($B=113.14$, $SE=67.20$) and consumption of more added sugar ($B=6.78$, $SE=3.89$). Additionally, addiction propensity predicted significantly higher odds of reporting weight gain or increased appetite during recovery ($B=4.15$, $SE=1.44$; $OR = 63.43$). The pattern of results was not sensitive to the inclusion of the food addiction scale as an indicator of the addiction propensity score.

Discussion

Excessive weight gain is a commonly reported problem during recovery from addiction to a variety of substances. On its own, excessive weight gain promotes risk for a variety of health outcomes. Additionally, body dissatisfaction may trigger relapse in a subset of individuals. Given the risks associated with weight gain, it is important to identify the mechanisms that predict excessive weight gain among people in recovery.

We evaluated the extent of evidence for two hypotheses about how excessive weight gain might arise during recovery. The *Addiction Transfer Hypothesis*, which is widely cited in qualitative accounts, and is also plausible given shared neurobiological pathways between highly palatable foods and drugs of abuse, suggests that individuals might use food in place of drugs and alcohol in response to cravings. Added sugar in particular has been identified as having strong addictive potential (Avena et al., 2008; Erica M. Schulte et al., 2015; Thornley, McRobbie, Eyles, Walker, & Simmons, 2008b). The *Propensity for Behavioral Addiction Hypothesis*, which is supported by strong comorbidities and shared heritability between binge eating and substance use disorders, suggests that some individuals may be highly prone to engage in addictive behaviors, and that weight gain during recovery represents a return to a natural state after having a restricted diet during active addiction.

We found no evidence to support the *Addiction Transfer Hypothesis*. Rather, we found that some people respond to drug or alcohol cravings by consuming *less* added sugar than they otherwise would have, and people who tended to report regular cravings consumed fewer calories and less fat overall. On the other hand, there was moderate support for the *Propensity for Behavioral Addiction Hypothesis*: people who had a family history of addiction, high impulsivity, low self-control, were addicted to a greater number of substances, and reported more food addiction symptoms tended to eat more calories and consume more added sugar overall. The propensity for behavioral addiction was also strongly associated with self-reported weight gain or appetite increase during recovery.

Of note, measurement burst was significantly associated with lower calorie consumption, and marginally significantly associated with consumption of less added sugar per two-hour interval. This finding suggests that unhealthy food consumption may naturally decrease over time during recovery, at least on average. This supports previous work that found the liking for sweetness among individuals in treatment for alcohol dependency decreases over time. (Krahn et al., 2006)

These results suggest that the commonly held belief that foods help individuals in recovery to combat cravings may be incorrect. If anything, our empirical results suggest the opposite: People with a stronger general propensity for addiction tend to consume more unhealthy food, and there is a general trend for individuals to consume fewer calories and less added sugar over the course of recovery. Given the potential negative consequences associated with excessive weight gain during recovery, these results suggest that patients might benefit from learning general, practical approaches to weight gain prevention. Recently, some clinicians have called for treatment programs to promote healthy eating patterns during recovery as a part of self-care education for relapse prevention (Ellin, 2014; Jeynes & Gibson, 2017). In practice, clinical attitudes toward eating vary widely across treatment programs. Our results suggest that these programs are a step in the right direction, and that provision of candy or other unhealthy foods at group treatment meetings (a common practice at many clinics) may be unhelpful.

Limitations and Future Directions

Our study relied on high-quality, interviewer-based, 24-hour nutrition recall taken over many days, together with EMA-based assessment of cravings. By combining these two sources of information, we were able to assess patterns of eating behavior in real time without relying on retrospective reports or participant introspection and use these data to rigorously evaluate support for two hypotheses regarding mechanisms for weight gain during addiction recovery. In addition to these strengths, our study also had some limitations.

As is typical among people with addiction, many participants in our sample reported receiving treatment for more than one substance. Although this feature of our sample enhances external validity of our results, it limits our ability to isolate the unique effects of individual substances. Although many substances of abuse exert effects on similar neurological pathways, there are also differences, particularly for alcohol since this is both a food and a substance of abuse. Related to this point, individual differences in metabolism and nutritional deficiencies may be influenced by addiction history (Jeynes & Gibson, 2017). Animal studies have been used to provide controlled experiments regarding the unique effects of specific substances on eating behaviors (Avena & Hoebel, 2003; Avena et al., 2008; Zhang & Kelley, 2002).

People receiving treatment for addiction tend to have multiple comorbidities, and many are prescribed many medications to address these comorbidities, in addition to medications to assist with relapse prevention. These medications have a variety of effects on appetite: whereas antidepressants may increase appetite, ADHD medications may decrease appetite. Because medications change frequently during early recovery as patients work with clinicians to manage comorbidities and possibly to wean from medications to manage

addiction, it is possible that medication management may influence weight gain during recovery. Clinicians and patients must balance the risks and benefits of different medications, perhaps choosing an alternative medication if it causes excessive weight gain.

Finally, we used multiple indicators to measure individual differences in addiction propensity. These indicators were drawn from the literature. In isolation, the indicators are not necessarily indicative of a predisposition to general addiction, but the shared variance amongst the indicators was assumed to indicate a propensity for addiction. Future research should replicate these results using different measures, including biological measures such as polygenic risk scores.

Conclusions

People who are more highly predisposed to engage in addictive behaviors generally may also be at higher risk for excessive weight gain during early recovery. Our findings suggest that attempts to suppress cravings with unhealthy food may be misguided, and that support for healthy eating behaviors during recovery may greatly benefit patients.

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Figure 1.
Flow chart illustrating derivation of analytic sample.

Table 1.

Results from Addiction Transfer Hypothesis Test

	Average Number of Calories			Average Grams of Fat			Average Grams of Added Sugar			Average Milligrams of Caffeine		
	Est	SE	P	Est	SE	P	Est	SE	P	Est	SE	P
Intercept	337.16	56.39	<.001	14.71	2.72	<.001	11.71	3.07	<.001	13.63	6.56	.041
Burst	-22.71	9.98	.023	-.91	.50	.071	-.71	.46	.124	.25	1.11	.82
Age	-2.86	1.35	.034	-.14	.06	.032	-.09	.07	.218	.00	.16	.978
Female	-.94.52	32.38	.004	-5.05	1.56	.001	-3.03	1.78	.089	-8.00	3.77	.034
Last Use	-.52	6.83	.940	.11	.33	.736	.02	.38	.966	.47	.80	.555
Alcohol	-29.52	31.46	.348	-1.43	1.52	.347	-.68	1.74	.700	4.65	3.68	.207
Cocaine	21.27	27.05	.432	1.09	1.30	.402	-1.01	1.50	.502	-1.31	3.18	.680
Opiates	19.73	36.47	.589	-.99	1.76	.573	2.92	2.03	.151	10.63	4.28	.013
Person Mean Craving ^a	-43.81	21.10	.038	-1.42	1.02	.163	.04	1.17	.970	.36	2.48	.883
Person Mean Centered Craving ^a	-3.36	10.23	.744	-.05	.50	.914	-.79	.46	.092	-.99	1.15	.394

Abbreviations: Est = Point Estimate; SE= Standard Error; $p = p\text{-value}$ ^aThe effect of person mean craving represents the between-person association between tendency toward cravings and general eating patterns; the effect of person mean centered craving represents the within-person association between momentary fluctuations in cravings and subsequent fluctuations in eating behaviors.^bIndividual diurnal time trends have been removedNote. Bolded estimates are statistically significant at $p < .05$.

Table 2.

Results from Propensity for Behavioral Addiction Hypothesis Test

	Average Number of Calories		Average Grams of Fat		Average Grams of Added Sugar		Average Milligrams of Caffeine		Weight Gain		Appetite Increase	
	Est (SE)	p	Est (SE)	p	Est (SE)	p	Est (SE)	p	Est (SE)	p	Est (SE)	p
Intercept	326.37 (58.21)	<.001	15.00 (2.92)	<.001	12.96 (3.37)	<.001	19.83 (6.75)	<.01	1.09 (1.01)	-	- .05 (1.04)	.96
Age	-1.52 (1.59)	.34	-.09 (.08)	.29	-.04 (.09)	.63	.20 (.18)	.29	.00 (.03)	1.00	-.01 (.03)	.99
Female	-133.11 (33.88)	<.001	-6.65 (1.70)	<.001	-4.59 (1.96)	.02	-11.02 (3.93)	.01	-.35 (.60)	.71	-.76 (.62)	.47
Last Use	11.63 (7.44)	.12	.50 (.37)	.18	.53 (.43)	.23	.38 (.86)	.66	-.05 (.13)	.95	.29 (.15)	1.34
Alcohol	-75.43 (36.03)	.04	-4.12 (1.81)	.03	-2.81 (2.08)	.18	2.12 (4.18)	.61	-1.24 (.61)	.61	-1.24 (.68)	.29
Cocaine	-5.73 (32.43)	.86	.29 (1.63)	.86	-3.01 (1.88)	.11	-6.05 (3.76)	.11	-.20 (.55)	.82	.26 (.57)	1.30
Opiates	-26.47 (40.78)	.52	-2.22 (2.05)	.28	.07 (2.36)	.98	6.49 (4.73)	.18	-.20 (.67)	.82	-.90 (.73)	.41
Addiction Propensity Score	113.14 (67.20)	.09	4.18 (3.37)	.22	6.78 (3.89)	.09	12.68 (7.79)	.11	2.82 (1.25)	16.82	4.84 (1.53)	126.97

Abbreviations: Est = Point Estimate; SE= Standard Error; p = p-value; OR = Odds Ratio

Note. Bolded estimates are statistically significant at $p < .05$.