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Impulsive choice does not predict binge-like eating in rats

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Abstract

Binge eating disorder (BED) is an addiction-like disorder characterized by recurrent, excessive food consumption within discrete periods of time, and it has been linked to increased trait impulsivity. Within impulsivity components, while impulsive action was shown to predict binge- and addictive-like eating, the role of impulsive choice is instead unknown. The goal of this study was to determine if impulsive choice predicted, or was altered by, binge-like eating of a sugary, highly palatable diet. We utilized a modified adjusting delay task (ADT) procedure in *ad libitum* fed rats to assess impulsive choice behavior, i.e. the tendency to respond for a larger, delayed reward over a lesser, immediate reward. We found that baseline impulsive choice was not a predictor of binge-like eating in 1-hour sessions of palatable diet operant self-administration. Furthermore, binge-like eating of the same palatable diet had no effect on later impulsive choice behavior. Thus, our data suggest that, unlike impulsive action, impulsive choice behavior does not predict binge-like eating in rats.

Keywords

Binge-eating; Impulsivity; Delay Gratification

Introduction

Binge eating disorder (BED) is an addiction-like disorder characterized by recurrent, excessive food consumption within discrete periods of time (American Psychiatric Association, 2013; Schulte et al., 2016; Moore et al., 2017). Impulsivity, which is a trait characteristic of both drug and behavioral addictions including BED (Dawe and Loxton, 2004; Schag et al., 2015), can be broadly defined as a predisposition toward rapid, unplanned reactions to internal or external stimuli, with diminished regard to the negative consequences of these reactions to the impulsive individual or to others (Chamberlain and Sahakian, 2007). Impulsivity is most often broken down into two main components:

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impulsive choice and impulsive action. Impulsive choice reflects an aversion to delay of gratification, while impulsive action reflects the inability to withhold responding when it might be inappropriate or premature.

In this context, a highly relevant question has been whether high trait impulsivity represents a risk factor for drug abuse and eating disorders; such information would, in fact, help refine intervention strategies aimed at preventing and treating these diseases. In clinical studies, understanding whether impulsivity represents a vulnerability factor for addictive behaviors and eating disorders has been a major challenge, given the difficulty of dissecting and tracking the study variables longitudinally. In regards to this issue, preclinical animal studies are indeed particularly valuable, since they allow a greater longitudinal control of experimental variables. Surprisingly, in feeding research, the preclinical investigation of whether impulsivity represents a risk factor for the development of maladaptive feeding behavior has been nearly neglected. Recently, it was shown that impulsive action predicts increased binge and addictive-like eating in rats (Velazquez-Sanchez et al., 2014), mirroring what was found earlier with drugs of abuse such as cocaine and nicotine, but not heroin (Belin et al., 2008; Diergaarde et al., 2008; McNamara et al., 2010). However, to the best of our knowledge, whether impulsive choice predicts binge-like eating is unknown.

The first aim of this study was, therefore, to investigate whether impulsive choice predicts binge-like eating behavior in rats. The second aim of this study was then to evaluate whether exposure to daily 1-hour access to a palatable diet induces any (further) deficits in impulsive choice behavior. To determine impulsive choice, a modified adjusting delay task (ADT) was utilized (Blasio et al., 2012).

Methods

Subjects

Forty-five day old male Wistar rats (N=48) were triple-housed upon arrival (Charles River, Wilmington, MA). Rats were housed in a temperature and humidity-controlled vivarium on a 12:12 hour reverse light cycle. Rats had *ad libitum* access to water and a corn-based chow (5TUM: 65.5% (kcal) carbohydrate, 10.4% fat, 24.1% protein, 3.30 cal/g; TestDiet, Richmond, IN) at all times, except during testing sessions. All experimental procedures adhered to the National Institutes of Health Guide for the Care and Use of Laboratory Animals and were approved by Boston University Medical Campus Institutional Animal Care and Use Committee (IACUC).

Apparatus

Individual operant test chambers enclosed in ventilated, sound-attenuating cubicles were used for all procedures (Med Associates Inc., St Albans, VT). Two retractable levers were located on one wall of the chamber, each with a 28 V stimulus cue-light above them. Between the levers were two stainless steel drinking cups mounted 2 cm above the grid floor of the chamber. Syringe pumps (Med Associates, St. Albans, VT) dispensed solutions into drinking cups for the ADT procedure. The opposite wall had two food magazines on either side of a liquid cup magazine, with 28 V stimulus cue-lights above them. Magazine head

entries were detected by infrared photobeams to deliver food or water reinforcement in food self-administration procedures.

Modified Adjusting Delay Task (ADT)

Non-deprived rats (i.e. with *ad libitum* food and water) were trained on the modified ADT procedure to assess impulsive choice between isocaloric reinforcers. In this task, modified from Perry et al. (2005) and described previously (Blasio et al., 2012), subjects were first trained in two overnight self-administration sessions under a Fixed Ratio 1 (FR1) schedule of reinforcement. A lever press on either lever resulted in delivery of 0.1 ml of 1.5% w/v glucose solution.

For the next training phase, sessions consisted of a 30-min pre-session followed by 15-blocks. The session ended following the completion of the 15-blocks or after 2 hours, whichever came first. The 30-min pre-session phase, where both levers were in the retracted position and all stimulus lights were off, served as an acclimation period and signal of the imminent availability of the reinforcers. During training, each block consisted of 4 forced-choice trials, signaled by the extension of one of the two levers and illumination of the stimulus light above it. A lever-press response in the forced-choice trials resulted in the immediate retraction of the lever. One lever resulted in delivery of 0.1 ml of the glucose solution delivered immediately, while the other resulted in 0.1 ml of a “supersaccharin” solution (1.5% w/v glucose + 0.4% saccharin, >80% preferred to 1.5% glucose (Blasio et al., 2012)) delivered after a constant delay of 6 sec. After learning to complete at least 10/15 trials, rats began the modified ADT.

In this task, the first two trials of each block were forced-choice trials (as during training), while the third and fourth trial were free-choice trials. In the free-choice trials, signaled by the illumination of both stimulus lights, both levers were extended, and rats were free to choose between the immediate delivery of glucose or a delayed delivery of supersaccharin. The delay was initially set to 6 sec, and was then adjusted after each free-choice trial, depending on which lever was chosen (immediately reinforced lever = 1 sec decrease in delay; delayed reinforced lever = 1 sec increase in delay). An inter-trial-interval (ITI) followed each lever press, adjusted so that all trials would be a maximum of 36 sec in duration (i.e., delay + ITI = 36 sec). During the delay and ITI, stimulus lights were extinguished and responses on either lever had no programmed consequences. At the end of each session, a mean adjusted delay (MAD) was calculated by averaging all adjusting delays on free-choice trials. MADs ranged from 0.5 sec (in the event that the immediately reinforced lever was chosen every time) to 21.5 sec (in the event that the delayed reinforced lever was chosen every time). ADT sessions were run until MAD stabilized (<3 sec variation across 5 days with no increasing or decreasing trends). Five rats were removed for unstable responding during the ADT procedure. MAD values were used as a quantitative measure of impulsive choice behavior, where lower MADs indicate higher levels of impulsive choice. MAD values was transformed into an impulsivity score (IS) using the following equation:

$$IS = \frac{MAD_{\max} - MAD}{MAD_{\max} - MAD_{\min}}$$

The IS serves as a more intuitive measure of impulsivity; whereas high IS indicates high impulsivity and vice versa. The IS is a dimensionless variable with an inverse relationship to the MAD, ranging from 0 to 1. Following a median split of baseline (i.e. average last 5 days) MAD values, rats that fell above the median MAD were assigned to the Low-impulsive group, and rats that had a MAD lower than the group median were assigned to the High-impulsive group.

Operant binge-like eating procedure

Rats previously trained in the ADT task and assigned to Low- or High-impulsive groups were then trained in an operant binge-like eating procedure (Blasio et al., 2014; Velazquez-Sanchez et al., 2014). Briefly, in daily 1-hour sessions, rats were trained to self-administer 45-mg precision pellets, identical in composition to the standard chow diet provided in the home cage (5TUM; TestDiet, Richmond, IN) and water (100 μ l) under an FR1 schedule of reinforcement. Each head entry into the food magazine resulted in the delivery of 1 pellet, followed by a 1.0 sec timeout period, during which no additional pellets were delivered.

After 1-hour chow self-administration performance was stable, rats were divided into 'Chow' control groups ('Low-impulsive/Chow,' N = 9; and High-impulsive/Chow,' N = 12), which continued to self-administer the 45-mg chow pellets offered during training, and 'Palatable' groups ('Low-impulsive/Palatable,' N = 12; and High-impulsive/Palatable,' N = 10), which were allowed to self-administer a nutritionally complete, chocolate-flavored, high sucrose (50% kcal) AIN-76A-based diet (45-mg precision pellets, chocolate-flavored, 5TUL: 66.7% (kcal) carbohydrate, 12.7% fat, 20.6% protein, metabolizable energy 3.44 cal/g; TestDiet). The 5TUL is >90% preferred over the standard chow diet (Cottone et al., 2008; Cottone et al., 2009).

Following >15 days of operant food self-administration sessions, which encompassed both escalation of intake and stabilization in rats administering the palatable diet (<10% group variability in responding compared to previous 3 sessions), rats underwent additional ADT sessions every other day to assess changes in impulsive choice behavior. Average MAD, IS, and latency (log) of five ADT sessions that took place after the binge-like eating procedure were used to compare to pre-binge impulsive choice behavior.

Statistical analysis

Baseline (last 5 days) MAD, IS, and latency (log(sec)) values were analyzed by a two-way, random effect intraclass correlations (ICC) of absolute agreement (Shrout and Fleiss, 1979; Cottone et al., 2008; Cottone et al., 2009) to evaluate stability of performance. ICC coefficients range between 0 and 1, with values closer to 1 denoting smaller within-subject variation across sessions. MAD, IS, and latency were analyzed using two-way ANOVA, with impulsivity and diet as between-subjects factors. To analyze impulsivity and diet effects on operant binge-like food self-administration, a three-way ANOVA was used, with impulsivity and diet as between-subjects factors and day as a within-subjects factor. Differences in impulsive choice variables before and after the binge-eating procedure were assessed using a three-way ANOVA, with impulsivity and diet condition as between-subjects factors and MAD score as a within-subject factor. The software/graphic packages used were SigmaPlot

12.0 (Systat Software Inc., Chicago, IL), SPSS Statistics 24 (IBM Corp., Armonk, NY), and Statistica 7.0 (StatSoft, Tulsa, OK).

Results

Impulsive choice: performance on modified ADT

Intraclass correlation analysis showed good internal consistency in MAD and IS, and latency (log(sec)) [intraclass correlation, **MAD** and **IS**: ICC (2,5) = 0.68, $F(42,168) = 3.10$, $p < 0.0001$; **latency(log(sec))**: ICC (2,5) = 0.78, $F(42,168) = 4.63$, $p < 0.0001$) across the last 5 days (Mean $\pm$ SEM: **MAD**: Low-impulsive: 15.41 ± 0.38 ; High-impulsive: 10.98 ± 0.37 ; **IS**: Low-impulsive: 0.29 ± 0.02 ; High-impulsive: 0.50 ± 0.02 ; $t(41) = 8.41$, $p < 0.0001$). The high intraclass correlation coefficients and the wide range of MADs or ISs ($2.90 < \text{MAD} < 21.50$ sec, or $0.00 < \text{IS} < 0.89$) revealed strong and stable individual differences.

Effect of impulsive choice on binge-like eating of palatable food

Prior to diet group assignment, there were no differences in chow food self-administration between groups (Mean $\pm$ SEM: Low-impulsive/Chow: 12.2 ± 1.2 kcal; High-impulsive/Chow: 11.5 ± 1.0 kcal; Low-impulsive/Palatable: 11.7 ± 1.0 kcal; High-impulsive/Palatable: 12.7 ± 1.1 kcal; $F(3,39) = 0.23$, not significant (NS)). Once rats were allowed to self-administer their respective diets, rats with access to palatable food escalated their intake over time compared to the Chow control group (Diet $\times$ Day: $F(14,490) = 6.38$, $p < 0.0001$), however there was no influence of prior impulsive choice on food intake in either diet condition (Diet $\times$ Impulsive choice: $F(1,35) = 0.05$, NS; Fig 1A; Diet $\times$ Impulsive Choice $\times$ Day: $F(14, 490) = 0.45$, NS). A Pearson correlation analysis showed no relationship between IS and amount of food eaten in the operant binge-like self-administration paradigm (average intake (kcal) on days 11–15 of food self-administration (Palatable $r^2 = 0.00$, NS; Chow $r^2 = 0.07$, NS; Fig 1B)), further demonstrating that there is no relationship between impulsive choice behavior and subsequent binge-like eating.

Exposure to 1-hour daily intermittent palatable diet on impulsive choice

When tested in the ADT procedure following operant food self-administration, group IS had remained stable (Pre-Post: $F(1,39) = 0.06$, NS; Fig 2A–B). Additionally, there were no differential effects of diet type (Pre-Post $\times$ Diet: $F(1,39) = 2.64$, NS) or prior impulsive choice (Pre-Post $\times$ Impulsivity: $F(1,39) = 0.63$, NS). Analysis of the log-transformed latency to respond in the ADT showed that latency decreased across all diet and impulsivity groups when comparing pre- and post- food self-administration (Pre-Post: $F(1,39) = 46.30$, $p < 0.0001$; Fig 2C). Latency was not differentially altered by diet (Pre-Post $\times$ Diet: $F(1,39) = 1.66$, NS) or prior impulsive choice group (Pre-Post $\times$ Impulsivity: $F(1,39) = 0.98$, NS).

Discussion

This study shows that impulsive *choice* does not predict increased binge-like eating of palatable food. This finding is especially interesting, as impulsive *action* was instead found to be a strong predictor of both binge-like and addictive-like eating in a study performed previously by our group (Velazquez-Sanchez et al., 2014).

Importantly, the modified ADT was performed in *ad libitum* fed and watered rats. The majority of experiments that test impulsivity in rats have utilized a food restriction protocol to increase motivation to complete the tasks. Unfortunately, food and water restriction is known to cause a myriad of behavioral and neurobiological changes (Carr, 2016), which confounds the investigation into risk factors for development of binge-eating and other disordered eating behavior. Therefore, we can confidently exclude any energy-balance influence in the results of this study. In addition, compared to other models which use food as reinforcer, the caloric content of reinforcers here is negligible (0.006 kcal), resulting in only ~0.3% of rat daily caloric intake after completion of a session (Blasio et al., 2012); this eliminates possible influences of feeding on impulsivity. Notably, in our modified ADT procedure the two reinforcers are isocaloric and, therefore, lever choice cannot result from energy-homeostatic factors.

In control animals, no effects of impulsive choice on the later training and self-administration of a chow diet were observed, and multiple weeks of operant food self-administration did not affect performance on the modified ADT. In animals allowed to self-administer a palatable diet, although intake dramatically escalated over time, the degree of binge-like eating of palatable food was not predicted by impulsive-choice behavior. Furthermore, binge-like eating of palatable food did not modify impulsive-choice behavior.

Similar to the results presented here, studies investigating impulsivity in animal models of drug addiction have also found that baseline impulsive choice does not seem to predict self-administration of cocaine (Mitchell et al., 2014) or nicotine (Diergaarde et al., 2008) under an FR1 schedule of reinforcement. However, on the other hand, impulsive choice appears to be associated with reinstatement of nicotine (Diergaarde et al., 2008) and cocaine (Broos et al., 2012a) seeking behavior; thus, this impulsivity component may be involved in other distinct phases addiction (e.g. maintenance, relapse), rather than the initiation and development of these disorders. The different facets of drug addiction and pathological eating have distinct neurobiological substrates (Moore et al., 2018), as do the components of impulsivity (Wang et al., 2016); it would, therefore, be of interest in future studies to investigate whether other aspects of pathological eating are associated with impulsive choice.

Previous work from our laboratory found that impulsive action, a component distinct from impulsive-choice, predicted binge-like and compulsive eating behavior (Velazquez-Sanchez et al., 2014). Similarly, impulsive action was predictive of cocaine and nicotine self-administration in rats (Belin et al., 2008; Diergaarde et al., 2008), while impulsive choice was not (Diergaarde et al., 2008; Mitchell et al., 2014), suggesting key differences between the two impulsivity components in predicting drug use and binge eating. Indeed, distinct neurobiological systems underlie impulsive action and impulsive choice (Pattij and Vanderschuren, 2008), thus divergences may provide key insights into the mechanisms contributing to addictive disorders.

In this study, impulsive choice behavior was not affected by multiple weeks of exposure to a sugary, highly palatable diet and the development of binge-like eating. To our knowledge, no studies have shown binge-like eating to cause changes in impulsive choice behavior.

However, cocaine exposure was found to cause increases in impulsive choice (Simon et al., 2007; Mitchell et al., 2014), and greater cocaine use was associated with higher impulsive choice (Mitchell et al., 2014). Interestingly, in one study, cocaine-administering animals were matched to a yoked-sucrose control group, and amount of sucrose exposure (high vs. low) also had no effect on later impulsive choice (Mitchell et al., 2014). While this study has distinct differences from the present study (e.g. sucrose yoked-controls vs. uncapped palatable diet self-administration), these results also suggest that impulsive choice is not modified by sucrose exposure.

As the present study was conducted in male rats only, our findings cannot be extrapolated to females without further research. Other studies into sex differences in impulsive choice behavior have shown that female rats and humans typically display greater impulsive choice behavior compared to males, and this may be associated with greater vulnerability to addictive behaviors (e.g. cocaine self-administration and reinstatement; Perry et al., 2008; Weafer and de Wit, 2014). Most epidemiological studies report similar or higher rates of binge eating in women compared to men (Striegel-Moore and Franko, 2003; Lee-Winn et al., 2016), and the neurobiological etiology of these behaviors may differ by sex/gender. Thus, further investigation into sex differences in impulsive choice effects on binge eating behavior would be of interest.

Conclusions

The relationship between impulsivity components and binge eating has implications for the prevention, treatment, as well as neurobiological targets (Manasse et al., 2016). Impulsive action and impulsive choice have distinct neural substrates (Wang et al., 2016), and uncorrelated behavioral expression (Broos et al., 2012b). Prior experiments have indicated that impulsive action is a risk factor for binge- and addictive-like eating behavior, mirroring what observed with cocaine (Belin et al., 2008). This study shows that, similarly to drug addiction, impulsive choice is not a risk factor for the development of binge-like eating in rats. Furthermore, the exposure to a sugary, highly palatable diet does not affect impulsive choice behavior.

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Figure 1.

Impulsive choice does not predict binge-like eating. (A) Effects of daily 1-hour access to a palatable diet on food intake in Low- and High- impulsive rats. (B) Baseline Impulsivity Score (IS) is not associated with later food intake (kcal) in 1-hour self-administration sessions. N = 9–12/group. Data show Mean $\pm$ SEM.

Figure 2.

Exposure to a palatable diet does not alter impulsive choice behavior. (A) Effects of daily 1-hour access to a palatable diet on IS, (B) Mean Adjusted Delay (MAD), and (C) latency (log(sec)) in the Adjusting Delay Task (ADT). N = 9–12/group. Data show Mean $\pm$ SEM.