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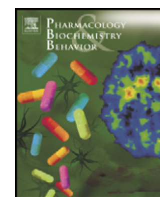

Intermittent access to a nutritionally complete high-fat diet attenuates alcohol drinking in rats

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A B S T R A C T

Binge eating disorder and alcohol use disorder (AUD) frequently co-occur in the presence of other psychiatric conditions. Data suggest that binge eating engages similar behavioral and neurochemical processes common to AUD, which might contribute to the etiology or maintenance of alcoholism. However, it is unclear how binge feeding behavior and alcohol intake interact to promote initiation or maintenance of AUD. We investigated the impact of binge-like feeding on alcohol intake and anxiety-like behavior in male Long Evans rats. Rats received chow (controls) or extended intermittent access (24 h twice a week; Int-HFD) to a nutritionally complete high-fat diet for six weeks. Standard rodent chow was available ad-libitum to all groups and food intake was measured. Following HFD exposure, 20.0% ethanol, 2.0% sucrose intake and endocrine peptide levels were evaluated. Anxiety-like behavior was measured using a light-dark (LD) box apparatus. Rats in the Int-HFD group displayed a binge-like pattern of feeding (alternations between caloric overconsumption and voluntary caloric restriction). Surprisingly, alcohol intake was significantly attenuated in the Int-HFD group whereas sugar consumption was unaffected. Plasma acyl-ghrelin levels were significantly elevated in the Int-HFD group, whereas glucagon-like peptide-1 levels did not change. Moreover, rats in the Int-HFD group spent more time in the light side of the LD box compared to controls, indicating that binge-like feeding induced anxiolytic effects. Collectively, these data suggest that intermittent access to HFD attenuates alcohol intake through reducing anxiety-like behavior, a process potentially controlled by elevated plasma ghrelin levels.

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1. Introduction

Alcohol use disorder (AUD) poses substantial fiscal, physical and mental health risks in the US (Bouchery et al., 2011; Grant et al., 2004). Importantly, AUD and disordered eating frequently co-occur in the presence of other psychiatric disorders (Bulik et al., 2004; Dunn et al., 2002). Specific to this topic is the observation that a significant proportion of the college-aged population engages in episodes of binge drinking and binge eating (Ferriter and Ray, 2011; Kelly-Weeder, 2011). Importantly, binge feeding behavior is further associated with emotional distress, obesity and metabolic risks (Gearhardt et al., 2014; Sonneville et al., 2013). Thus, these frequently co-occurring problems suggest greater psychiatric disturbance and medical risk (Catterson et al., 1997; Harrell et al., 2009; Johnston et al., 2005) for patients with AUD.

Binge eating, characterized by eating a large amount of food in a short period of time, as well as a sense of lack of control over food intake, is the most common behavioral manifestation present in a variety of eating disorders (Attia et al., 2013; Kessler et al., 2013). Data suggest that individuals who engage in binge eating behavior develop AUD, overweight/obesity, and worsening depressive symptoms over the course of time (Franko et al., 2005; Sonneville et al., 2013). Highly palatable foods, rich in sugar and fat, are the typically preferred foods consumed during binge episodes in humans and laboratory animals, and it is hypothesized that hedonic-based feeding (i.e., feeding in the absence of caloric need) regulates this phenomenon (Cottone et al., 2008; Yeomans et al., 2004). This is based on observations that like drugs of abuse, both sweet and fatty foods are capable of activating brain reward circuitry (Tomasi and Volkow, 2013; Volkow et al., 2012). In this context, feeding peptides that control appetite and energy metabolism also regulate the intake and reinforcing properties of alcohol (Vadnie et al., 2014). These feeding peptides are released during anticipation of scheduled meals, ingestion and storage of calories, calorie restriction and learned responses that facilitate approach behavior to food (Drazen et al., 2006; Kanoski et al., 2013; Vahl et al., 2010). Thus, any or all of these variables could mediate feeding-induced changes in

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alcohol intake. Both human and animal studies suggest a bidirectional positive relation between alcohol intake and consumption of sugar or fat (Avena et al., 2004; Carrillo et al., 2004; Herbeth et al., 1988; Mitchell et al., 1985). However, it is unclear how binge feeding behavior impacts alcohol consumption.

Based on DSM criteria, various rodent models of binge eating have emerged (Corwin and Babbs, 2012; Wolfe et al., 2009). Although, it is challenging to mimic all aspects of human binge eating episodes in rodent models, it has been suggested that modeling time-limited access to palatable food that induces repeated hyperphagic events could be helpful to study binge eating behavior (Perello et al., 2014). This is only possible if a palatable diet is provided in an intermittent fashion (Avena et al., 2009; Corwin and Babbs, 2012; Davis et al., 2007; Tong and D'Alessio, 2011). Utilizing this limited access (2 h) paradigm, we have recently shown that binge-like intake of high fat diet (HFD) induced anxiolytic effects and attenuated alcohol consumption in non-dependent rats (Sirohi et al., 2016). This finding could be explained by the length and exposure history of a calorie-rich food, which has fundamentally different behavioral outcomes (Krishna et al., 2016; Tracy et al., 2015) or changes in the feeding peptides, which control behavioral constructs such as motivation and anxiety that also contribute to excess alcohol intake (Barson and Leibowitz, 2016; Morganstern et al., 2011). Therefore, in the present study, we hypothesized that extended intermittent access to HFD would lead to increased alcohol consumption in non-dependent rodents. To do this, we investigated the impact of extended (24 h) intermittent access to HFD on alcohol intake, plasma levels of feeding peptides and anxiety-like behavior in rats.

2. Material and methods

2.1. Animals

Male Long-Evans rats (Harlan, IN) were housed in an environmentally controlled vivarium on a reverse light cycle (lights off at 7 a.m.) with food and water available ad libitum in standard shoebox cages. All work adhered to the National Research Council's Guide for the Care and Use of Laboratory and Institutional Animal Care and Use Committee guidelines at Washington State University, WA.

2.2. Diets

All rats were maintained on chow (Teklad, 3.41 kcal/g, 0.51 kcal/g from fat) throughout. Rats in the experimental group received intermittent access to HFD (Research Diets, New Brunswick, NJ, 4.41 kcal/g, 1.71 kcal/g from fat). Dietary composition of standard rodent chow and HFD has been described previously (Davis et al., 2007).

2.3. General procedure

Rats (n = 5–6/group) matched for body weight and food intake received chow (controls) or intermittent access (24 h, every Tuesday and Thursday, only) to HFD (Int-HFD) for six weeks (Fig. 1A). Following six weeks of intermittent cycling on HFD, all rats underwent a series of tests while still maintained on the intermittent HFD schedule, as outlined in the (Fig. 1B and C). Standard rodent chow was available to all groups at all times. Food intake was measured daily, unless otherwise noted. First, we evaluated alcohol intake on the following days (Mon, Wed and Fri) of HFD exposure on 6 separate occasions. In order to examine if this could be due to decrease in overall caloric need following HFD intake, 2.0% sucrose consumption was examined next in the same groups under identical conditions over two separate days. HFD, alcohol and sucrose were introduced in the rat cages at the onset of dark cycle and represent a measurement over a period of 24 h, unless noted otherwise. Next, we examined anxiety-like behavior, a behavioral evaluation that required one day. To determine if the observed attenuation of alcohol drinking was a result of altered alcohol metabolism or changes in feeding peptides, both groups of rats received a final alcohol drinking session, that occurred after all other tests. Since, we observed differences in intake specifically 4–12 h after alcohol intake had ensued, rats were allowed to drink alcohol and 11 h later tail blood samples were collected at three time points, 15 min apart, to measure blood alcohol, ghrelin, and GLP-1 levels. Body weight was recorded every fourth day, except on alcohol and sucrose drinking days when rats were weighed daily.

2.4. Alcohol and sucrose intake

On testing days, rats were provided with an unsweetened alcohol solution (20% v/v) using a two-bottle choice paradigm (Simms et al., 2008). The position of alcohol and water bottles were alternated between sessions to account for conditioning effects on alcohol intake. Bottles were weighed, gently placed in the cages and re-weighed manually following each session to evaluate alcohol intake (g/kg). In addition, we also evaluated alcohol consumption by manually weighing bottles at multiple time points during one separate alcohol drinking session. Therefore, data represent six separate alcohol drinking sessions, where 24 h alcohol intake was measured in the first five sessions and one separate session evaluated alcohol consumption at multiple time points. Sucrose preference was also examined under identical conditions. To do this, all rats were exposed to a 2% sucrose solution using the same two-bottle choice paradigm.

2.5. Anxiety-like behavior

Anxiety-like behavior was measured using a light dark box apparatus, which occurred 12 days following sucrose drinking days, while rats were still on intermittent HFD cycling. Rats in each group were gently introduced in the light side (600 lx) facing dark side (4 lx) of the box and allowed to freely explore between compartments for 10 min. The total number of entries and time spent in the light side were counted by two independent investigators.

2.6. Blood alcohol levels & analysis of feeding peptides

Blood alcohol levels were determined from tail blood samples and analyzed using Analox microstat GL5 (Analox Instruments Ltd., Lunenberg, MA). Plasma levels of acyl-ghrelin and glucagon-like peptide-1 (GLP-1) were detected using a MILLIPLEX MAP Rat Metabolic Hormone Magnetic Bead Panel - Metabolism Multiplex Assay kit (RMHMAG-84K, EMD Millipore Corporation). Blood samples (0.2 ml) were collected in tubes containing EDTA, dipeptidyl peptidase 4 (DPP-4) inhibitor and Pefabloc (an irreversible serine protease inhibitor) at concentration 1.86 mg/ml, 18.8 μg/ml and 1.0 mg/ml of blood, respectively, on ice. All samples were centrifuged at 21000*g* for 20 min and plasma was transferred into a separate tube on ice and stored at −20 °C until further analysis. On the day of analysis, samples were thawed on ice and assayed in duplicates according to the kit manufacturer's instructions.

2.7. Body composition analysis

Body composition analysis (total, lean and fat body mass) was measured using a Bruker Minispec LF110 NMR Body Composition Rat Analyzer (Bruker Biospin, Rheinstetten, Germany).

2.8. Gene expression analysis

Following completion of the study, rats were sacrificed and total RNA was isolated from ventral striatum using Qiagen RNeasy Micro Kit (Qiagen, CA) and quantified using a Nano drop 2000c spectrophotometer. Complementary DNA (cDNA) was revere transcribed using High Capacity cDNA Reverse Transcription Kit with RNase Inhibitor

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