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Invited review

Converging vulnerability factors for compulsive food and drug use

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ABSTRACT

Highly palatable foods and substance of abuse have intersecting neurobiological, metabolic and behavioral effects relevant for understanding vulnerability to conditions related to food (e.g., obesity, binge eating disorder) and drug (e.g., substance use disorder) misuse. Here, we review data from animal models, clinical populations and epidemiological evidence in behavioral, genetic, pathophysiologic and therapeutic domains. Results suggest that consumption of highly palatable food and drugs of abuse both impact and conversely are regulated by metabolic hormones and metabolic status. Palatable foods high in fat and/or sugar can elicit adaptation in brain reward and withdrawal circuitry akin to substances of abuse. Intake of or withdrawal from palatable food can impact behavioral sensitivity to drugs of abuse and vice versa. A robust literature suggests common substrates and roles for negative reinforcement, negative affect, negative urgency, and impulse control deficits, with both highly palatable foods and substances of abuse. Candidate genetic risk loci shared by obesity and alcohol use disorders have been identified in molecules classically associated with both metabolic and motivational functions. Finally, certain drugs may have overlapping therapeutic potential to treat obesity, diabetes, binge-related eating disorders and substance use disorders. Taken together, data are consistent with the hypotheses that compulsive food and substance use share overlapping, interacting substrates at neurobiological and metabolic levels and that motivated behavior associated with feeding or substance use might constitute vulnerability factors for one another.

This article is part of the special issue on 'Vulnerabilities to Substance Abuse'.

1. Introduction

Both highly palatable foods and drugs of abuse can impact the brain and behavior in ways that are relevant for understanding disorders ranging from binge eating disorder (BED) to obesity, type II diabetes and substance and alcohol use disorders (SUD, AUD). In this review, we focus on the overlapping neuroscience shared between highly palatable foods and drugs of abuse and highlight preclinical and clinical data to assess the case that there are converging vulnerability factors that can predispose an individual to compulsive food or drug use. First, we highlight how both highly palatable foods and drugs of abuse impact metabolic and neural systems in similar ways, heuristically exemplified in Table 1. Second, we describe a robust literature demonstrating commonalities in the effects of palatable food and substances of abuse on behavior, focusing on reinforcement, negative affect, negative urgency, and impulse control, as well as how food itself can impact sensitivity to drugs. We then consider how food or metabolic status may relate to vulnerability for SUD or AUD based on human data. We finish by exploring potential therapeutic approaches that might leverage the commonalities in neuroscience and behavior described throughout this review.

SUD is diagnosed (according to the Diagnostic Statistical Manual [DSM]-V) among individuals that experience at least 2–3 symptoms, including (but not limited to) craving, withdrawal, tolerance, continuing to use the substance despite interpersonal, professional or health problems (https://www.drugabuse.gov/publications/media-guide/science-drug-use-addiction-basics; accessed October 20, 2020). In contrast to SUD being the consensus diagnosis associated with drugs of abuse, compulsive eating (or "food addiction" or the somewhat preferred "eating addiction"; Burrows et al., 2017a,b) is not a specific diagnosis in the DSM-V (see Meule and Gearhardt 2014). Instead several quite different mental health conditions include diagnostic criteria that relate

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to overconsumption or "misuse" of highly palatable foods, including binge-related eating disorders (e.g., BED, bulimia nervosa [BN], anorexia nervosa [AN]-binge purge type) and obesity. As a result, the clinical populations that exhibit compulsive eating are much more heterogeneous than those identified with compulsive substance use (i.e., SUD). This mish-mash likely has hindered the acceptance and research and clinical utility of the concept of compulsive eating.

Importantly, binge-related eating disorders and obesity are not interchangeable, and several key features, including the diagnostic process, distinguish these conditions. For example, AN and BN both fall in the DSM-V classification of "Feeding and Eating Disorders", along with BED (American Psychiatric Association, 2013). BED is diagnosed among individuals that experience a minimum of 1–3 binge-eating episodes per week, defined as either 1) "eating, in a discrete period of time, an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances" or 2) "the sense of a lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control how much one is eating)" (Berkman et al., 2015). In contrast to AN, BN and BED, obesity was excluded as a psychiatric disorder from the DSM-V due to its heterogeneity and incomplete understanding of whether some forms of obesity have psychiatric components (Marcus and Wildes, 2012). Obesity instead has been considered a multifactorial disease, with genetic, metabolic, endocrinological, physical activity, nutritional and medical side effect components. Obesity is therefore primarily diagnosed using a different diagnostic process, often by different types of healthcare professionals. For example, most standard definitions of obesity used in the healthcare system include some index of body weight and/or presence of certain biomarkers. Thus, body mass index (BMI) or indices of body fat composition are used alone or in combination to diagnose obesity (https://www.nichd.nih.gov/health/topics/obesity/conditioninfo/diagnosed; accessed October 14, 2020). The DSM-V is more commonly used by the broad mental health community (including counselors, psychologists and psychiatrists), whereas other diagnostic methods and processes (e.g., biomarkers, cholesterol, BMI) are used mainly by physicians to diagnose obesity and associated diseases. This fundamental difference in diagnostic process leads to differences in how the conditions are conceptualized for treatment, the stigma surrounding patients and the conditions, and the likelihood that individuals will seek out treatment or preventative strategies. Relatedly, many medications currently used to treat BED versus obesity differ and focus on different aspects of each condition. Beyond these key diagnostic and therapeutic differences, BED and obesity also differ in national prevalence, with BED affecting approximately 2% of the global population (Kessler et al., 2013) as compared to obesity which affects 36–38% of the global population (Ng et al., 2014). Because of these distinctions, the possibility that highly palatable foods might play different roles in the development or maintenance of binge-related eating disorders vs. obesity is kept in mind throughout this review.

Several human disorders related to food (e.g., obesity and BED) often show lifetime comorbidity with SUD, suggesting potential shared vulnerability factors or pathophysiologic mechanisms. For example,

Table 1
Selected hormones and neurotransmitter systems similarly impacted by chronic palatable food or substances of abuse.

Neurotransmitter hormone	Impact of chronic highly palatable food		Impact of chronic substance use or in SUD patients	
Striatal Dopamine D2 receptors	↓	Dunn et al. (2012); Haltia et al. (2007); Johnson & Kenney et al. (2010); Lindgren et al. (2018); Stice et al. (2011); Tomasi and Volkow (2013); Bello et al. (2002) (though see also South and Huang 2008)	↓	Koob and Le Moal (2005)
Striatal Dopamine release	↓	Geiger et al. (2009); Rada et al. (2010)	↓	Koob and Le Moal (2005)
Striatal DA transporters	↓	Speed et al. (2011); Cone et al. (2013)	↓	Kuhar and Pilotte (1996)
Central amygdala CRF-CRF1 activity × effects are different depending on if examined during withdrawal or after chronic exposure. Arrows in this row refer to following withdrawal and not ongoing chronic exposure.	↑	Cottone et al. (2009a); Iemolo et al. (2012); Micioni Di Bonaventura et al. (2014); Micioni Di Bonaventura et al. (2017b)	↑	Bruijnzeel et al. (2009); Bruijnzeel et al. (2010); Knapp et al. (2004); Overstreet et al. (2004); Sommer et al. (2008); Skelton et al. (2007); Stinus et al. (2005); Basso et al. (1999); Sarnyai et al. (1995); George et al. (2007); Chu et al. (2007); Funk et al. (2007); Gehlert et al. (2007); Gilpin et al. (2008); Richardson et al. (2008); Sabino et al. (2006); Valdez et al. (2002); Specio et al. (2008); Greenwell et al. (2009); Roberto et al. (2010); Zorrilla et al. (2001); Heinrichs et al. (1995); Maj et al. (2003); McNally and Akil (2002); Weiss et al. (2001); Richter and Weiss (1999); Rodriguez de Fonseca et al. (1997); Marcinkiewcz et al. (2009); Iemolo et al. (2013); Roberto et al. (2017)
NAc preproenkephalin mRNA	↓	Spangler et al. (2004); Kelley et al. (2003)	↓	Spangler et al. (2004)
Naloxone/naltrexone withdrawal	↑	Colantuoni et al. (2002); Avena et al. (2008); Mason et al. (2015)	↑	Pothos et al. (1991); Skelton et al. (2007); Zhang et al. (2016a)
NAc Ach	↑	Colantuoni et al. (2002); Avena et al. (2008); Hoebel et al. (2007)	↑	Pothos et al. (1991); Rada et al. (1991);
Metabolic Hormones, inflammatory cytokines and insulin status	**Impact of highly palatable food**		**Impact by Drugs or in models of SUD/AUD or in SUD/AUD patients**	
Ghrelin	↓	Spierling et al. (2020); Cottone et al. (2008)	↓	Sustkova-Fiserova et al. (2020) (though see also Wittekind et al., 2019; Farokhnia et al., 2019)
Interleukin-6	↑	Cottone et al. (2009b)	↑	Wei et al. (2020)
Insulin resistance	↑	Clawson et al. (2019); Woods et al. (2004)	↑	Daws et al. (2011); see also Richardson et al. (2014); Ibias et al. (2018)

SUD and BED symptoms develop around similar age ranges (see Schreiber et al., 2013), and 24–27% of patients with BED also meet the diagnostic criteria for SUD at some point in their lifetime (Becker and Grilo, 2015; Grilo et al., 2009; see also Schreiber et al., 2013). Similarly, SUD is also highly comorbid among patients diagnosed with obesity (Chen et al., 2018; Stokes et al., 2019; Muller et al., 2018; Lanza et al., 2015 see also Gearhardt et al., 2018). In fact, a significant subgroup of patients that have had bariatric or gastric bypass surgery, subsequently show recurrence or *de novo* incidence of SUD (Kanji et al., 2019; Sarwar et al., 2019; Conason et al., 2013; Ivazaj et al., 2014, 2017). Although the substances used by individuals with BED or obesity may vary, data for use and abuse of opioids (Canan et al., 2017; Stokes et al., 2019; Smith et al., 2019), alcohol (Grucza et al., 2010; Conason et al., 2013; Smith et al., 2018; see also Traversy and Chaput, 2015; Sayon-Orea et al., 2011), nicotine (Gearhardt et al., 2018), stimulants (Ricca et al., 2009; Dutta et al., 2006; deLima et al., 1998), and polydrug use (Hu et al., 2018) suggest that the overlapping prevalence is not specific for a particular class of drugs.

2. Overlap between highly palatable foods and substances of abuse

2.1. Shared metabolic systems

Metabolic hormones and status represent a possible causal link between feeding and SUDs. Extensive data show that eating highly palatable foods can impact feeding-regulatory peptides, metabolism and energy expenditure, as well as insulin status. In animal models, for example, continuous access to a Western diet can increase adiposity in the offspring not only of obesity-prone dams, but also obesity-resistant dams, suggesting an effect of diet dissociable from obesity (Frihauf et al., 2016). The deleterious impact of diet is evident in increased plasma levels of endocrine risk markers, such as leptin, insulin, and adiponectin in weanlings, as well as decreased basal metabolic rate (Frihauf et al., 2016). Chronic intermittent access to a palatable diet, which elicits binge-like food intake, also produces major adaptations in metabolism, including increased fat accumulation and feed efficiency (Cottone et al., 2008b; Cottone et al., 2009; Parylak et al., 2012; Kreisler et a., 2017; Kreisler et al., 2018), and cyclic changes in fuel substrate utilization and energy expenditure. Of interest vis-à-vis motivational systems, a profile of lipid-sparing fuel substrate utilization correlated directly with the development of increased progressive ratio breakpoints for the palatable diet (Spierling et al., 2018). As another metabolism-motivation relation, an endocrine profile of heightened glucagon-like peptide-1 (GLP-1) and pancreatic polypeptide along with decreased ghrelin levels discriminated those rats that developed the most compulsive-like feeding behavior following intermittent access to palatable high-sucrose food (Spierling et al., 2020).

Intermittent palatable food access also decreases circulating ghrelin and growth hormone levels (Cottone et al., 2008b), and increases fasting insulinemia, glycemia (Kreisler et al., 2018), leptinemia (Cottone et al., 2008b) and levels of proinflammatory cytokines, like interleukin-1β and interleukin-6 (Cottone et al., 2009b). Plasma levels of the proinflammatory cytokine, interleukin-18 (IL-18), also were increased by chronic high fat feeding in mice (Zorrilla et al., 2007). While each of these molecules is well-known to be involved in the control of appetite and metabolism (Zorrilla et al., 2007; Zorilla and Conti, 2014), more recent data have linked each of them to SUD or AUD phenotypes (Sustkova-Fiserova et al., 2020; Wittekind et al., 2019; Farokhnia et al., 2019; Jerlhag, 2020; Pastor et al., 2017; Walter et al., 2017; Grönbladh et al., 2016; Labarthe et al., 2014; Wei et al., 2020; Bach et al., 2020; Xu, 2014; Aguiar-Nemer et al., 2013; Kebir et al., 2011).

For example, Ser^3-acylated ghrelin, a post-translationally activated gastric peptide hormone, is known to promote food intake via "anticipatory" increases prior to expected access to palatable food or mealtime as well as during energy insufficiency. Increased acyl:*des*-acyl ghrelin ratios are reported in young obese individuals (Kuppens et al., 2015; Fittipaldi et al., 2020). Manipulations that oppose actions of acylated ghrelin, including small molecule antagonists, immunoneutralization, catalytic antibodies, *des*-acyl ghrelin, and liver enriched antimicrobial peptide-2 (LEAP-2), an endogenous GHRS1A inverse agonist, have anorectic effects under many conditions (Zorrilla et al., 2006; Mayarov et al., 2008; Zakhari et al., 2012; Ge et al., 2018; Fittipaldi et al., 2020). They also are putative therapeutic approaches to address the food craving and compulsive overeating behavior seen in Prader-Willi syndrome (Allas et al., 2018; Carias et al., 2019), a developmental disorder with hyperghrelinemia. In parallel to these findings, mounting data link increased acylated-ghrelin activity to different aspects of drug or alcohol-craving behavior. For example, ghelin receptor antagonists reduced morphine-induced behavioral stimulation and sensitization, conditioned place preference and nucleus accumbens (Acb) dopamine release (Sustkova-Fiserova et al., 2014; Jerabek et al., 2017). They also reduced the acquisition and expression of methamphetamine- or fentanyl-conditioned place preference as well as methamphetamine or fentanyl self-administration (Havlickova et al., 2018; Sustkova-Fiserova et al., 2020). Higher fasting ghrelin levels also were seen in former smokers (Wittekind et al., 2019) and associate with increased craving and relapse (al'Absi et al., 2014; Lemieux and al'Absi, 2018). A burgeoning literature also links alcohol use disorder and alcohol craving to increased acylated ghrelin activity (Farokhnia et al., 2019). For example, acylated ghrelin levels increased during ethanol abstinence in relation to craving and ethanol cue-induced insula activation (Koopmann et al., 2012; Bach et al., 2019). Infusion of ghrelin to alcohol-dependent men increased intravenous ethanol self-administration on a progressive-ratio schedule and altered alcohol-associated fMRI signal (Farokhnia et al., 2018). Conversely, the ghrelin receptor inverse agonist PF-5190457 reduced alcohol craving and attention to alcohol cues in people with AUD (Lee et al., 2020).

An incongruity in this regard is, as reviewed, animal models of diet-induced obesity and most forms of adult human obesity typically are associated with decreased total ghrelin levels, in contrast with the apparent evidence of a role for increased ghrelin activity in various addictive disorders. A potential understanding of this discrepancy is that the decrease in ghrelin levels seen in older obese individuals may reflect a homeostatic mechanism to prevent further weight gain, a change not present in addictive disorders.

Heritable metabolic differences also have been linked to appetite-regulatory peptides implicated in substance use. For example, obesity-prone rats show heritable deficits in central anorectic sensitivity to feeding-regulatory corticotropin-releasing factor $(CRF)_2$ receptor agonists (Cottone et al., 2007), a phenotype accompanied by deficits in postmeal satiety (Cottone et al., 2007, 2013). In mouse knockout models, the same type 2 urocortin/CRF_2 receptor molecules have been linked to altered ethanol intake (Sharpe et al., 2005; Smith et al., 2015b) as well as changes in stress recovery (Neufeld-Cohen et al., 2010) and depressive-like behavior (Chen A et al., 2006).

Another metabolic hormone implicated in modulating addiction risk is fibroblast growth factor 21 (FGF21), a protein that regulates energy homeostasis (Ji et al., 2019) and that is induced by metabolic stresses, including ketogenic and high carbohydrate diets. Gene variants in the FGF21 gene and in the KLB gene (which encodes β-klotho) are associated with alcohol use phenotypes in people (Schumann et al., 2016; Søberg et al., 2017; Clarke et al., 2017; Zhou et al., 2020). In both mice (Staiger et al., 2017) and humans (Desai et al., 2017; Søberg et al., 2018), alcohol intake increases circulating FGF21 levels. Overexpression (Schumann et al., 2016) or administration (Talukdar et al., 2016; Søberg et al., 2018) of FGF21 markedly reduced alcohol intake and preference in mice in a manner dependent on the FGF21 co-receptor β-klotho. A β-klotho/FGFR21 complex-activating antibody also decreased alcohol intake (Chen et al., 2017).

In addition to genes that encode FGF21 and β-klotho, other gene loci commonly thought of as metabolism-related genes also are linked to

Table 2
Selected genetic loci jointly associated with risk for obesity and substance- or alcohol use-related disorders.

Hormones and Neurotransmitters	Relation to obesity, feeding or metabolism	Relation to SUD/ AUD
Fibroblast growth factor 21/KLB (β-klotho)	Ji et al. (2019)	Staiger et al. (2017); Desai et al. (2017); Soberg et al. (2018); Zhou et al. (2020); Schumann et al. (2016), Søberg et al. (2017); Clarke et al. (2017); Thompson et al. (2020)
GCKR	Kraja et al., 2014; Veiga-da-Cunha et al., 2003	Zhou et al. (2020); Thompson et al. (2020)
FTO	Chang et al. (2018); Yang et al. (2017)	Zhou et al. (2020)
ADH1B	Winner et al. (2015); Yokoyama et al. (2020)	Zhou et al. (2020); Thompson et al. (2020)
ISL1	Loid et al. (2020); Clément et al. (1999)	Zhou et al. (2020)
IGF2BP1	Rodrigues et al. (2010); Lu et al. (2016); Zhou et al. (2020)	Zhou et al. (2020)
DRD2	Lancaster et al. (2018); Jenkinson et al. (2000); Col Araz et al. (2012); Kvaløy et al. (2015); Aliasghari et al. (2020)	Lancaster et al. (2018); Jenkinson et al. (2000); Col Araz et al. (2012); Kvaløy et al. (2015) Zhou et al. (2020); Thompson et al. (2020)
ANKK1	Bauer (2014); Yamada et al. (2017); Aliasghari et al. (2020)	Zhou et al. (2020)
CRHR1	Curtis and UK10K Consortium (2016); Lu et al. (2015)	Zhou et al. (2020)
BDNF	Rios et al. (2001); Akbarian et al. (2018)	Zhou et al. (2020)
PDE4B	Zhang et al. (2009); Lee et al. (2011)	Zhou et al. (2020)
CADM2	Morris et al. (2019); Lamiquiz-Moneo et al. (2019); Yan et al. (2018); Miranda-Lora et al. (2017); Dorajoo et al. (2012)	Zhou et al. (2020)
BRAP	Imaizumi et al. (2018); Wu et al. (2013)	Zhou et al. (2020)
PSMC3	Zhuang et al. (2017)	Zhou et al. (2020)
SP1	Li et al. (2012); Liu et al. (2012); Li et al. (2014); Zhuang et al. (2017)	Zhou et al. (2020)
KRTCAP3	Keele et al. (2018)	Zhou et al. (2020)
SLC39A13/ZIP13	Fukunaka et al. (2017)	Zhou et al. (2020)
SLC39A8	Williams MJ et al., 2012; van Vliet-Ostaptchouk et al. (2013); Kraja et al., 2014	Zhou et al. (2020); Thompson et al. (2020).
FNDC4	Frühbeck G et al. (2020)	Zhou et al. (2020)
TBL2	Kim et al., 2011; Kraja et al., 2014	Zhou et al. (2020)

excess alcohol use phenotypes, as highlighted in Table 2. These include glucokinase regulatory protein (***GCKR***), a key enzyme in glucose homeostasis that regulates the activity and intracellular localization of glucokinase (Kraja et al., 2014; Veiga-da-Cunha et al., 2003); fat mass and obesity-associated protein/alpha-ketoglutarate dependent dioxygenase (***FTO***), a nuclear dioxygenase that influences global metabolism and energy homeostasis and its associated regulatory loci (Chang et al., 2018; Yang et al., 2017); alcohol dehydrogenase 1B (class I; ***ADH1B***), an enzyme that promotes metabolism of energy from alcohol, whether the alcohol is imbibed or derived endogenously from gut microbes or anaerobic respiration (Winner et al., 2015; Yokoyama et al., 2020); ISL LIM Homeobox 1 (***ISL1***), a transcription factor that binds the enhancer region of the insulin gene (Loid et al., 2020; Clément et al., 1999); and insulin like growth factor 2 MRNA binding protein 1 (***IGF2BP1***), a binding protein that regulates the translation of insulin-like growth factor 2 (Rodrigues et al., 2010; Lu et al., 2016; Zhou et al., 2020). An area of future research is to determine how exposure to and abstinence from palatable food impact the expression and function of products of these genes.

Both drugs of abuse and highly palatable foods can influence similar peptides, exemplified in the role of insulin in the rewarding effects of nicotine. It has been suggested that enhanced rewarding effects of nicotine promote tobacco use among patients diagnosed with diabetes (see O'Dell and Nazarian, 2016). This hypothesis was based on work demonstrating that a disruption in insulin signaling enhanced the rewarding effects of nicotine in diabetic rats (O'Dell et al., 2014; Richardson et al., 2014; Pipkin et al., 2017; O'Dell and Nazarian, 2016). These latter studies provide evidence that insulin disruptions reliably enhance the reinforcing and rewarding effects of nicotine in self-administration and place conditioning procedures in rat models of Type 1 and Type 2 diabetes. Importantly, disruptions in insulin signaling suppress dopamine transmission and alter dopamine receptor sensitivity (O'Dell et al., 2014).

Recently, a potential link between pancreatic glucose regulation and the reinforcing effects of nicotine has been identified in the medial habenula action of Transcription factor 7-like 2 (TCF7L2; Duncan et al., 2019). This diabetes-associated transcription factor, in bipartite association with β-catenin, activates Wnt target genes, thereby regulating glucose metabolism in enteroendocrine cells of the gut and pancreas as well as in liver. Several finding suggest a novel role for TCF7L2 within a habenula-pancreas axis to regulate nicotine's reinforcing and diabetes-promoting actions. First, TCF7L2 also is highly expressed in rodent medial habenula (mHb), where it regulates nicotinic acetylcholine receptor function. Second, inhibition of medial habenula TCF7L2 action reduced nicotine intake. Third, nicotine conversely increased circulating glucose levels via TCF7L2-dependent stimulation of the medial habenula, which is polysynaptically connected to the pancreas. Finally, the ability of nicotine intake to elicit diabetes-like changes in glucose homeostasis is blunted in mutant Tcf7l2 rats. The collective work supports a relationship between insulin resistance, glucose intolerance, metabolic markers, dopamine and nicotinic acetylcholine receptor sensitivity, and the escalation of nicotine intake in diabetic-like rodent models.

Conversely, prior work also has revealed that drugs of abuse can alter an array of metabolic indices. For example, nicotine prevents the distribution of abdominal fat produced by a high fat diet in male mice (Mangubat et al., 2012). Also, self-administered nicotine increases fat metabolism and suppresses weight gain in rats (O'Dell et al., 2007; Rupprecht et al., 2018) and mice (Calarco et al., 2017). Epidemiological work has shown that chronic cannabis smoking was associated with visceral adiposity and adipose tissue insulin resistance, suggesting another interaction between drug use and metabolic systems that regulate food intake (Muniyappa et al., 2013).

2.2. Shared addiction constructs

2.2.1. Reward and mesolimbic dopamine

In addition to impacting and being conversely regulated by metabolic systems, intake of drugs and highly palatable foods can have similar acute and chronic effects on brain reward pathways. For example, under acute conditions, highly palatable food (Hernandez and

Hoebel, 1988) and drugs of abuse (Di Chiara and Imperato, 1988) both result in an initial increase in release of dopamine. The neurochemical mechanism of action by which many drugs of abuse produce their rewarding effects involves activation of a primarily dopaminergic pathway in the brain, the mesolimbic pathway (Wise and Bozarth, 1985). This pathway is normally activated by so called "natural" rewards, such as food and sex, and the fact that drugs of abuse also activate this pathway has been the subject of scientific exploration for decades (Volkow et al., 2007; Volkow et al., 2011; Taber et al., 2012; see also Nutt et al., 2015).

Similarly, palatable foods likewise initially promote increased intake and weight gain in people through positive reinforcement (Bray et al., 2017; Cobb et al., 2015; Pereira-Lancha et al., 2010). Thus, neuroimaging studies of healthy weight adolescents without disordered eating find that greater reward region activation in response to palatable foods predicts short-term weight gain (Shearrer et al., 2018; Stice et al., 2015; Stice and Yokum, 2016a, 2016b; Winter et al., 2017). In normal weight individuals with healthy eating patterns, palatable carbohydrate-rich food and drink also increase emotional well-being and calmness within 1–2 h of consumption (Reid and Hammersley, 1999; Gibson, 2006; Strahler and Nater, 2018). Reward enhancement, defined by intake of palatable food for "pleasure, excitement, or increased fun" (Boggiano, 2016; Boggiano et al., 2015) is 1 of 4 motives identified by Boggiano and colleagues for eating palatable food.

2.2.2. *Within-system, opponent process adaptation*

While drug use initially elicit pleasurable states, such as contentment or well-being, both human and preclinical studies show these are followed in opponent-process fashion by worse mood and increased vigilance/tension (Ettenberg et al., 1999; Jhou et al., 2013; Knackstedt et al., 2002; Radke et al., 2011; Vargas-Perez et al., 2007, 2009; Wenzel et al., 2011). With repeated drug use, this counter-regulatory opponent process predominates, such that more rewarding substance is needed to maintain euthymia. Negative emotional signs (e.g., irritability, anxiety, dysphoria and subjective feelings of need) result when use stops. This deficit emotional state persists with repeated use, leading to negative emotional behavior, hyperarousal, and increased stress responses despite prolonged abstinence. Behaviorally, the anhedonia and negative affective state that result from the opponent-process drive continued drug use via negative reinforcement (Koob and Le Moal, 2005). Anhedonia (Hatzigiakoumis et al., 2011) and other aspects of this "dark side" of addiction (Koob and Le Moal, 2005) have been associated with drugs of abuse from every major drug class, including stimulants (Leventhal et al., 2010), opiates (Garfield et al., 2017), nicotine (Cook et al., 2015), alcohol (Martinotti et al., 2008), and cannabinoids (Leventhal et al., 2017). Within-system impairment of normal mesolimbic pathway function is one putative mechanism underlying the long-term, drug use-induced opponent process. Thus, chronic drug use is characterized by downregulation of dopamine D_2 receptors; decreases in production of tyrosine hydroxylase, the rate limiting factor for dopamine synthesis; decreases in dopamine release; and upregulation of dopamine D_1 receptors (Koob and Le Moal, 2005).

Similarly, long term consumption of high fat diets can disrupt mesolimbic dopamine systems. Several human and animal studies demonstrate decreases in basal or food-stimulated striatal dopamine release; dopamine D_2 receptor binding and expression (Dunn et al., 2012; Haltia et al., 2007; Johnson and Kenny, 2010; Lindgren et al., 2018; Stice et al., 2011; Tomasi and Volkow, 2013; although see also South and Huang, 2008); and decreased function and membrane expression of dopamine transporters (Speed et al., 2011; Cone et al., 2013). Daily access to sucrose can similarly reduce striatal dopamine D_2 receptor binding (Bello et al., 2002) and mRNA expression (Spangler et al., 2004; Bello et al., 2003), and prolonged access to a high-sucrose/high-fat "junk food" mash reduced Acb D_2 mRNA expression (Robinson et al., 2015). Long term access to cafeteria diets (which typically contain both high fat and high sugar/carbohydrate items) also decreases basal levels of extracellular dopamine in the nucleus accumbens, and leads to lower stimulation-evoked dopamine release in the nucleus accumbens and dorsal striatum (Geiger et al., 2009). During cyclic access to palatable, high-sucrose food, rats show blunted locomotor, reward-potentiating, place preference conditioning, and Acb dopamine responses to D-amphetamine, consistent with opponent-process changes in mesolimbic function. Similar to reviewed models, they also show reduced basal extracellular dopamine in no-net flux *in vivo* microdialysis analysis (Moore et al., 2020).

While these studies demonstrate largely consistent changes in dopamine systems following long term exposure to highly palatable foods and drugs, there may be differences in their impact on dopamine D_1 receptor expression. Opposite to chronic drug use (Koob and Le Moal, 2005), long term access to highly palatable foods may result in a decrease in dopamine D_1 receptor gene expression (Alsiö et al., 2010; though see also Ramos et al., 2020), a possible difference worth future study.

The above-described effects of chronic palatable food on brain reward neurocircuitry, similar to drugs of abuse (see Table 1), have been hypothesized to promote compulsive eating via opponent-process affective dysregulation and negative urgency (Cottone et al., 2009; Cottone et al., 2008a; 2008b 2009; Kreisler et al., 2017; Parylak et al., 2012; Parylak et al., 2011; Spierling et al., 2018; Zorrilla and Koob, 2019; Zorrilla and Koob, 2020). Below, we separately consider human and animal findings in key behavioral domains to evaluate support of this opponent-process model for palatable food and whether food-induced addiction-like adaptations might increase risk for substance use and relapse behavior.

2.2.3. *Tolerance*

2.2.3.1. Human findings. Similar to substances of abuse, chronic intake of palatable foods may result in food reward tolerance due to opponent-process adaptation. Thus, 2 items on the Yale Food Addiction Scale (YFAS) 2.0 measure hypohedonic tolerance ("did not give me as much enjoyment", "needed to eat more … to get the feelings I wanted"; Gearhardt et al., 2016). Consistent with this view, the ability of a preferred beverage to improve mood diminishes with repeated use (Spring et al., 2008).

This putative food reward tolerance is hypothesized to reflect reviewed adaptations in mesolimbic dopaminergic reward circuitry that drive compensatory overeating of palatable food in a vicious circle (Bello and Hajnal, 2010; Blum et al., 2014, 2018; Gold et al., 2015, 2018; Volkow et al., 2008a). Consistent with this opponent-process "reward deficiency" hypothesis and similar to chronic palatable fed animal models, obese subjects show addiction-like reductions in striatal dopamine D_2 receptors and decreased basal or food-stimulated dorsal striatal extracellular dopamine responses (Dunn et al., 2012; Haltia et al., 2007; Lindgren et al., 2018; Stice et al., 2011; Volkow et al., 2007, 2008a, 2009, 2013; Wang et al., 2009, 2011) as compared with lean subjects (Volkow et al., 2002). The degree of reduction in striatal dopamine D_2 receptor levels correlates directly with greater BMI (Volkow et al., 2008a; Wang et al., 2001). Furthermore, the degree to which caudate activation responses of obese subjects to a milkshake are reduced (Stice et al., 2008) also predicts greater subsequent increases in BMI.

In SUD, tolerance is typically accompanied by adaptive escalation of intake of the substance of abuse to achieve the same level of hedonic reward. Findings in this regard have been mixed in humans with palatable food. On the one hand, several findings suggest a similar tolerance-motivated increase in palatable food intake in humans. Indeed, a YFAS item explicitly measures this ("needed to eat more … to get the feelings I wanted") (Gearhardt et al., 2016), Similarly, volunteers who showed blunted fMRI BOLD caudate activation responses to an ice-cream milkshake drank milkshakes more frequently (Burger and

Stice, 2012) and showed greater subsequent increases in BMI (Stice et al., 2010). On the other hand, women who received a macaroni-and-cheese meal daily for 5 weeks decreased their intake more than did those with weekly access (Avena and Gold, 2011; Epstein et al., 2011).

2.2.3.2. Animal models. Animal models also support the hypothesis that palatable food elicits allostatic decrements in reward function that manifest as tolerance. Intracranial lateral hypothalamic self-stimulation thresholds increase in rats provided extended access to a palatable cafeteria diet (Johnson and Kenny, 2010; but see Iemolo et al., 2012) that reduced striatal dopamine D_2 receptor levels. Elevated self-stimulation thresholds, an index of reduced brain reward function, developed with obesity and persisted despite forced abstinence from the cafeteria diet for two weeks (Johnson and Kenny, 2010). Mimicking the dopamine D_2 receptor downregulation by knocking down dopamine D_2 receptor expression with a lentiviral construct accelerated diet-induced increases in reward thresholds, supporting a causal role for striatal dopamine D_2 receptor deficiency in impaired brain reward function (Johnson and Kenny, 2010). As in humans, dampened mesolimbic dopaminergic transmission may promote weight gain, because obesity-prone rats have lower basal and lipid-stimulated extracellular dopamine levels in the nucleus accumbens than do obesity-resistant rats (Geiger et al., 2008; Rada et al., 2010).

Similar to substances of abuse, palatable food-induced changes in mesolimbic circuitry also blunt neurochemical and behavioral responses to alternative reinforcers. For example, cafeteria diet eliminated the normal ability of standard chow to increase dopamine efflux (Geiger et al., 2009). Rats that received chronic, intermittent extended access to a chocolate-flavored, sucrose-rich diet showed reductions in progressive ratio break points for a less preferred, but otherwise palatable, diet (Cottone et al., 2009a, 2009b). Likewise, access to highly preferred diets led to underconsumption of otherwise acceptable chow even when it was the only food available, leading to voluntary weight loss (Cottone et al., 2008a, 2008b, 2009a, 2009b; Iemolo et al., 2013; Johnson and Kenny, 2010; Pickering et al., 2009; Rossetti et al., 2014). The chow hypophagia increased with longer durations of access to palatable food (24 h vs. 30 min/day) and is persistent (Kreisler et al., 2017); rats that received chronic *ad lib* access to a highly preferred diet continued to undereat standard, grain-based chow for at least 2 weeks after it was the only diet available, despite having returned to normal body weight and adiposity (Kreisler et al., 2017). Intake of and breakpoints for moderately sweet solutions also decreased in rats with a history of access to sweet foods or solutions (Iemolo et al., 2012; Vendruscolo et al., 2010). Both the chow hypophagia and the motivational deficits to obtain less preferred food are mitigated by pretreatment with a CRF_1 receptor antagonist (Cottone et al., 2009a), perhaps analogous to the ability of a CRF receptor antagonist to reverse blunted reward function during drug withdrawal (Bruijnzeel et al., 2009, 2010).

Continuous access to palatable sucrose solutions diminishes striatal dopamine responses to sucrose, but intermittent access does not (Rada et al., 2005). The latter neurochemical finding may explain why rats with intermittent, extended access to a palatable diet show profoundly elevated daily intake and operant self-administration of palatable food, whereas those with *ad libitum* access decrease their palatable food intake to that of chow controls (Kreisler et al., 2017; Spierling et al., 2018, 2020). This finding resembles the above-described pattern in humans wherein daily access to macaroni and cheese reduces intake unlike weekly access and seems to contrast from findings that extended access to substances of abuse typically leads to compensatory increases in intake with tolerance. However, it also has been observed that escalation of nicotine self-administration only develops with intermittent, extended access to nicotine and not with daily extended access (Cohen et al., 2012), which can even decrease self-administration at some unit doses (O'Dell et al., 2007).

Similarly, Hoebel, Avena and colleagues observed dramatic increases in glucose intake over successive days of intermittent access to sugar, including increased consumption during the first hour of access (Colantuoni et al., 2002). Such binge-like escalation of intake and operant self-administration has been seen across many laboratories using diverse palatable diets and schedules of intermittent access (Bello et al., 2009; Cooper, 2005; Corwin and Babbs, 2012; Cottone et al., 2008b, 2009a, 2009b; Kreisler et al., 2017; Parylak et al., 2012; Rossetti et al., 2014; Spierling et al., 2018). Rats with intermittent, extended access (24 h/day) to a palatable diet developed increased binge-like intake and first 30-min operant self-administration to a similar degree as highly time-restricted rats (30 min/day). Thus, similar to substances of abuse, intermittent access to, and not only restrictedness, of palatable diet can escalate consumption (Kreisler et al., 2017; Spierling et al., 2018).

The uncertain escalation of intake with the development of tolerance upon continuous access to palatable food differs from what is seen for most substances of abuse. Perhaps, as with nicotine, intermittency, such as occurs with dieting or dietary restraint, is key in the development of reward-compensatory increases in intake of palatable food as tolerance develops. Alternatively, feeding-elicited orosensory- or energy-related satiety factors may oppose further food intake eve though tolerance develops. Such factors would not impede the reward-compensatory intake of most substances of abuse. Future studies may assess these hypotheses by testing 1) whether tolerance to palatable food is associated with cross-escalation of intake of dissimilar rewarding food (Treesukosol et al., 2015) or substances of abuse, 2) whether tolerance to palatable, low-energy tastants (e.g., sugar-saccharin mixtures; Valenstein et al., 1967; Spierling et al., 2017) leads to escalated self-administration of those and other palatable tastants, and 3) whether sham-fed subjects continuously fed a palatable diet escalate their self administration to a greater degree than real-fed subjects (Weingarten, 1982; Nissenbaum and Sclafani, 1987; Treesukosol et al., 2015).

2.2.4. Opponent-process affective dysregulation

2.2.4.1. Human findings. Several items on the YFAS measure negative emotional and somatic symptoms during abstinence from palatable food (Zorrilla and Koob, 2019), akin to opponent-process symptoms seen during drug withdrawal. Headache, irritability and flu-like symptoms have been described in clinical accounts of acute food abstinence (Davis and Carter, 2009). Dieting, which often involves reduced intake of palatable food, predicts increases in self-reported 'stress' (Rosen et al., 1990) and depressive symptoms (Stice and Bearman, 2001) in both overweight and non-overweight individuals (Crow et al., 2006; Goldschmidt et al., 2016; Hinchliff et al., 2016; Stice, 2001). Increased depressive symptoms also are seen in some weight-management programs (Smoller et al., 1987). Individuals who habitually skip breakfast also have increased distress, depressive symptoms and suicidal ideation (Kelly et al., 2016; Khan et al., 2020; Kwak and Kim, 2018; Lee et al., 2017a, 2017b; Lien, 2007; Tanihata et al., 2015).

People also become irritable ("hangry") during abstinence from food. For example, women with lower glucose levels more frequently delivered aversive noise blasts to their spouse and stabbed pins into a voodoo doll that symbolized them than did women with normal glucose levels (Bushman et al., 2014). Further, after eating a high fat diet for one month, men and women who were switched to a lower-fat diet had greater anger, anxiety and hostility during the next month than did volunteers who were kept on the high fat diet (Wells et al., 1998).

As a human analog of precipitated opioid withdrawal, a placebo-controlled trial found that oral naltrexone elicited cortisol stress responses and aversive nausea in obese women. These effects were greater in volunteers who had greater food addiction symptoms and reward-driven eating (Mason et al., 2015).

Supporting the view that these abstinence symptoms may reflect opponent-process affective dysregulation, intake of palatable food has

been linked to subsequent increases in self-reports of negative emotions. Indeed, 5 items on the YFAS 2.0 (Gearhardt et al., 2016) measure aversive visceroemotional outcomes after overeating palatable food (e.g., "caused emotional problems", "distress", "felt so bad", "sluggish", "tired", "physically ill"). Higher YFAS scores in turn relate not only to greater binge eating, night eating, caloric and fat intake, and BMI (Ayaz et al., 2018; Brunault et al., 2017; Burrows et al., 2017a,b; Hauck et al., 2017; Masheb et al., 2018; Meule et al., 2017; Nolan and Geliebter, 2017; Richmond et al., 2017; Schulte et al., 2018), but also to "dark side" symptoms such as depression, anxiety, post-traumatic stress symptoms; emotional reactivity; and insomnia (Berenson et al., 2015; Brewerton, 2017; Burmeister et al., 2013; Burrows et al., 2017a,b, 2018; Ceccarini et al., 2015; Chao et al., 2017; Davis et al., 2011; de Vries and Meule, 2016; Gearhardt et al., 2012; Granero et al., 2014; Koball et al., 2016; Masheb et al., 2018; Meule et al., 2014, 2015).

Likewise, high intake of processed, palatable foods, such as "crisps or savoury snacks; sweets … or chocolate; biscuits; fried food, chips, samosas or bhajis … and soft drinks" is associated with clinically significant anxiety and depression not only cross-sectionally (Jacka et al., 2010a; Jacka 2010b; Oddy et al., 2009), but also prospectively (Akbaraly et al., 2009; Baskin et al., 2017; Jacka et al., 2011, 2014; Sánchez-Villegas et al., 2009; Sarris et al., 2015). While the role of 3rd variables in these epidemiologic relations remain under debate (Lai et al., 2016; Winpenny et al., 2018), causally-oriented studies found that dietary interventions that promote long-term abstinence from such foods ultimately improve mental health (Adjibade et al., 2018) and reduce major depression more than social support controls (Jacka et al., 2017).

Binge-like eating, long linked to negative emotional states and traits (Womble et al., 2001), also involves eating of palatable foods (Singh, 2014), including breads/pasta, sweets, high-fat meat items, and salty snacks (Allison and Timmerman, 2007). Similarly, the Nurses' Health Studies (n = 123,688 women) found that YFAS-defined food addiction, which associates strongly with binge eating, was associated with an increased frequency of eating hamburgers and other red/processed meats, French fries, pizza, and other palatable foods, such as snacks and candy bars (Lemeshow et al., 2018). Consistent with opponent-process theory, ecological momentary assessment studies have observed that high levels of negative affect progressively develop post-binge (Haedt-Matt and Keel, 2011; Berg et al., 2017). Data also suggest that binge eating may prospectively increase subsequent depression (Spoor et al., 2006; Stice, 1998, 2001). Consistent with a transdiagnostic, opponent-process view, individuals with BED and BN have increased rates of major depression, bipolar disorder, anxiety disorders, and alcohol or drug abuse (Hudson et al., 2007; Mitchell and Mussell, 1995; Swanson et al., 2011). About 30–80% of BED patients have comorbid depression and anxiety disorders over their lifetime (Herzog et al., 1992), and both current (27.3% vs. 4.9%) and lifetime rates (52.3% vs. 23.0%) of mood disorders are greater in obese patients with BED vs. obese patients without BED (Sheehan and Herman, 2015).

Suicidality also commonly associates with binge eating. Over half of teenagers diagnosed with BN and approximately one-third of those with BED report suicidal ideation; and one-third of those diagnosed with BN report attempting suicide (Brown et al., 2018; Carano et al., 2012; Swanson et al., 2011). Suicide attempts and completion are also high in BED (Runfola et al., 2014), especially with comorbid depression (Pisetsky et al., 2013).

Depression and anxiety likewise are well-reviewed comorbidities of human obesity (Singh, 2014). The high depression symptomatology is not secondary to obesity because non-obese BED patients show depressive symptoms at levels similar to those of obese BED patients (Dingemans and van Furth, 2012). Obesity also has been linked to increased suicidality in a large (n = 14,497), diverse representative sample from the Collaborative Psychiatric Epidemiologic Surveys both independent of and synergistically with binge eating (Brown et al., 2018).

In sum, human data suggest that overconsuming palatable food ultimately may causally contribute in opponent-process fashion to the negative emotional disturbance seen in BED, BN and obesity. These negative emotional symptoms, in turn, are conceptualized as driving further intake, similar to negative reinforcement use of substances of abuse (Leon et al., 1999; Pearson et al., 2015; Hughes et al., 2013; Zorrilla and Koob, 2019).

2.2.4.2. Animal models. Behavioral signs of a negative emotional state also are documented in animals following palatable food access (Sharma et al., 2013). An early report described "nippiness" during withdrawal from sucrose solutions (Galic and Persinger, 2002). Hoebel and colleagues found that rats with intermittent daily access to high sugar solutions alternated with food deprivation developed somatic and anxiogenic-like signs of opiate withdrawal when challenged with the opioid receptor antagonist naloxone (Colantuoni et al., 2002). They also showed decreased preproenkephalin mRNA expression in the nucleus accumbens (Spangler et al., 2004), as do rats with limited daily access to a sweet-fat diet (Kelley et al., 2003). Opioid-like withdrawal signs also occurred after a 24–36 h fast (Avena et al., 2008). Hoebel and colleagues hypothesized that these effects may result from an altered balance of striatal dopaminergic vs. acetylcholinergic (ACh) signaling. Thus, similar to morphine withdrawal (Pothos et al., 1991; Rada et al., 1991), naloxone or an extended fast elicited greater Acb ACh release in rats with a cyclic glucose + chow/deprivation history than in *ad lib* chow rats (Colantuoni et al., 2002; Avena et al., 2008), as well as reduced extracellular Acb dopamine. The shift towards greater ACh release vs. decreased dopamine putatively underlies a shift towards harm avoidance and away from approach behaviors (Hoebel et al., 2007).

Diet-cycled rats with alternating 2-day access to a highly-preferred high-sucrose, chocolate-flavored diet vs. 5-day access to standard chow similarly showed increased anxiety-like behavior in the elevated plus-maze and defensive withdrawal tests when tested during the chow phase of their diet cycle (Cottone et al., 2009a; Cottone et al., 2009b; but see Rossetti et al., 2014). Increased forced swim immobility (Iemolo et al., 2012), a depressive-related behavior, and reduced locomotor activity in a novel open field (Rossetti et al., 2014), a measure of increased emotionality, also were reported in withdrawn diet-cycled rats. Increased aggressive-like irritability also was seen in rats withdrawn from intermittent (MWF), extended (24-hr/day) access to the same diet (Spierling et al., 2019). At least some behavioral effects of abstinence from palatable food are not unique to extended access, because female rats with more limited (10–120 min/day) access to the same diet exhibited an anxiogenic-like reduction in plus-maze open arm time (Cottone et al., 2008b; Kreisler et al., 2018) and greater bottle-brush irritability (Spierling et al., 2020) during abstinence.

During palatable food withdrawal, rats in the 5-day/2-day alternating access model showed increased expression of the stress-related neuropeptide CRF in the (central nucleus of the amygdala (CeA; Cottone et al., 2009a), a nucleus also activated in mice acutely withdrawn from high-fat diet (Teegarden and Bale, 2007). This finding is notable because CeA CRF systems also are activated during withdrawal from alcohol (Funk et al., 2007; Roberto et al., 2010; Sommer et al., 2008; Zorrilla et al., 2001), opiates (Heinrichs et al., 1995; Maj et al., 2003; McNally and Akil, 2002; Weiss et al., 2001), cocaine (Richter and Weiss, 1999), cannabinoids (Rodriguez de Fonseca et al., 1997), and nicotine (George et al., 2007; Marcinkiewcz et al., 2009). When diet-cycled animals were studied during access to the palatable diet, plus-maze behavior, forced swim immobility, and CeA CRF levels normalized, supporting the hypothesis that increased activation of the amygdala CRF system and negative emotional behavior reflected an acute abstinence-related state (Cottone et al., 2009a, 2009b; Iemolo et al., 2012).

Pharmacological and electrophysiologic data also implicate the CRF_1 receptor system. Systemic pretreatment with the CRF_1 receptor

antagonist R121919 blocked food withdrawal-associated anxiety at doses that did not alter behavior of chow controls (Cottone et al., 2009a). This result resembles findings that CRF_1 receptor antagonists reduce aversive- and anxiety-like states during withdrawal from alcohol (Knapp et al., 2004; Overstreet et al., 2004; Sommer et al., 2008), opiates (Skelton et al., 2007; Stinus et al., 2005), benzodiazepines (Skelton et al., 2007), cocaine (Basso et al., 1999; Sarnyai et al., 1995), and nicotine (George et al., 2007). CRF_1 receptor antagonist pretreatment also reduced the degree to which diet-cycled animals overate the preferred diet when access was renewed. This finding resembles findings that CRF_1 receptor antagonists can reduce excessive intake of alcohol (Chu et al., 2007; Funk et al., 2007; Gehlert et al., 2007; Gilpin et al., 2008; Richardson et al., 2008; Sabino et al., 2006; Valdez et al., 2002), cocaine (Specio et al., 2008), opiates (Greenwell et al., 2009), and nicotine (George et al., 2007) in animal models of dependence, while having less effect on self-administration of non-dependent animals. Also, similar to findings during alcohol withdrawal (Roberto et al., 2010), diet-cycled rats showed increased sensitivity of CeA GABAergic neurons to modulation by CRF_1 antagonism; thus, R121919 reduced evoked inhibitory postsynaptic potentials to a greater degree in diet-cycled rats. Finally, intra-CeA R121919 reduced the anxiogenic-like behavior and palatable diet intake of withdrawn diet-cycled rats (Iemolo et al., 2013), similar to the ability of intra-CeA CRF antagonist administration to reduce withdrawal-associated negative emotional symptoms and self-administration in models of substance dependence (Roberto et al., 2017). In sum, many findings in diet-cycled rats resemble the between-system adaptation of central extended amygdala CRF systems seen in animal models of addiction (see Table 1). The results also raising the possibility that the adaptations in stress-related amygdala circuitry may increase relapse or incidence risk for the other condition.

Evidence of adaptation in the endocannabinoid (eCB) system also has been seen during abstinence from palatable food. For example, the CB_1 receptor inverse agonist surinabant (SR147778) less potently reduced binge-like intake of rats with highly-limited access to sweet-fat diet than it did in *ad lib*-fed chow or palatable diet controls (Parylak et al., 2012). Withdrawal from cyclic palatable food was associated with increased levels of the eCB 2-arachidonoylglycerol and its cannabinoid receptor 1 (CB_1) in the CeA (Blasio et al., 2013). These findings have been interpreted as signs of a compensatory stress response because amygdala eCB-CB_1 signaling has been proposed to act as an anti-stress buffer (Lutz et al., 2015; Parsons and Hurd, 2015). Also suggesting adaptation, systemic or intra-CeA infusion of rimonabant, a CB_1 inverse agonist, more potently precipitated anxiogenic-like behavior and anorexia in rats withdrawn from cyclic palatable food than in chow controls (Blasio et al., 2013, 2014a). The food induced-changes in eCB function may have implications for SUD and AUD vulnerability (D'Addario et al., 2014).

2.2.5. Negative cue reactivity

2.2.5.1. Human findings. The neurobiological basis for palatable food effects on negative emotionality is unclear, but the above results suggest that drug-like between-system adaptation in the central amygdala may be involved (Zorrilla and Koob, 2019) (see Table 1). Consistent with this hypothesis, similar to drug and alcohol cue reactivity studies of people with SUDs (Engelmann et al., 2012; Goudriaan et al., 2010; Heinz et al., 2009; Jasinska et al., 2014; Mainz et al., 2012), obesity and food addiction symptoms are each associated with increased amygdala reactivity to pictures of palatable, high-calorie foods (e.g., cheesecake, milkshake; Stoeckel et al., 2008; Gearhardt et al., 2011; Ng et al., 2011). Women with BN also show increased functional connectivity of the amygdala to the insula and putamen during a milkshake cue as compared to healthy volunteers (Bohon and Stice, 2012). Such cue-induced activation is often aversive (Carelli and West, 2014; Colechio et al., 2014, 2018; Colechio and Grigson, 2014; Nyland and Grigson, 2013), and may reflect amygdala-subserved reward omission (Calu et al., 2010; Iordanova et al., 2016; Kawasaki et al., 2015, 2017; Tye et al., 2010), negative contrast, or conditioned opponent processes (Childress et al., 1988; Childress et al., 1986; Colechio and Grigson, 2014; Colechio et al., 2014; Grigson, 2008; McLellan et al., 1986; Topp et al., 1998; see also Siegel and Ramos, 2002; Koob, 2015; Koob and Le Moal, 2008; Roberto et al., 2017; Wenzel et al., 2011). Women with a history of BN (Ely et al., 2017) and obese children (Boutelle et al., 2015) also show greater amygdala responses to tastes of sucrose than do healthy controls.

2.2.5.2. Animal models. Consistent with the human findings of stress-like responding to "frustrative" food or drug cues (defined as previously reward-predictive, but now non-rewarded, cues), rats with cyclic palatable diet histories that were presented with the sight and smell of unobtainable palatable food showed Hypothalamic-Pituitary-Adrenal (HPA)-axis activation (Cifani et al., 2009) and more cells expressing phosphorylated extracellular signal-regulated kinases in the CeA, paraventricular nucleus of hypothalamus (PVN), and dorsal and ventral bed nuclei of the stria terminalis (BNST; Micioni Di Bonaventura et al., 2017a). When access was ultimately provided, "frustratively cued" rats ate twice as much as on days when no cues were presented (Cifani et al., 2009b; Micioni Di Bonaventura et al., 2012). The frustratively non-rewarded cue increased extended amygdala CRF_1 receptor mRNA. Antagonizing CRF_1 receptors in the CeA and BNST reduced frustrative cue-induced binge-like intake (Micioni Di Bonaventura et al., 2014, 2017b). Other reports also demonstrate increases in responding for cues previously paired with high fat foods, sucrose and saccharin after abstinence (Aoyama et al., 2014; Darling et al., 2016; Dingess et al., 2017; McCue et al., 2019) mirroring similar increases in responding after abstinence when cues previously paired with drugs of abuse are presented (e.g., cue-induced reinstatement; Epstein et al., 2006; Shalev et al., 2002; see Grimm 2020 for a review). Thus, while many theorists have emphasized the role of past predictive appetitive associations in the incentive actions of these cues, the present findings support models that emphasize the role of unexpected non-reward and/or the aversive, arousing effects that occurs with unexpected omission of incentives after previously reward-predictive cues (Amsel, 1958, 1994; Papini and Dudley, 1997; Pearce and Hall, 1980; Schultz, 2016; Sutton and Barto, 1981).

Cyclic overeating of vs. restriction from palatable food also leads to binge-like ingestive responses to stress in rats. Thus, intermittent restriction alternated with access to palatable food (Nutella chow or cookies) led to increased consumption in response to a stress trigger (footshock or frustrative food cue nonreward; Boggiano and Chandler, 2006; Cifani, Polidori, et al., 2009a; Hagan et al., 2002). These adaptations to cyclic palatable food access involve changes in feeding responses to mu/kappa, nociceptin/orphanin FQ, CRF_1 and orexin receptor ligands (Boggiano et al., 2005; Piccoli et al., 2012).

2.2.6. Negative reinforcement motivational factors

2.2.6.1. Human findings. In an addiction framework, the reviewed negative emotional effects of abstinence from palatable food are viewed as motivating use via negative reinforcement ("coping-like") mechanisms, a shift away from positive reinforcement motive. As in substance use, negative affect is recognized to precede binge eating (Berg et al., 2015, 2017; Dingemans et al., 2017; Fischer et al., 2018; Haedt-Matt and Keel, 2011) and is a putative trigger of overeating (Carels et al., 2004; Razzoli et al., 2017) and selecting (Wallis and Hetherington, 2009; Zellner et al., 2006) palatable food in vulnerable populations. Several human laboratory studies have found that negative mood and stressful inductions increase palatable food intake in vulnerable populations (Stice, 2002; Fay and Finlayson, 2011) (e.g., obese binge eaters; Chua et al., 2004).

These relations may reflect that palatable carbohydrate-rich food and drink reduce anger and tension in negative reinforcement fashion within 1–2 h of intake (Benton and Owens, 1993; DeWall et al., 2011). Sweets reduce "feelings of being stressed out" in emotional eaters (Strahler and Nater, 2018). A palatable carbohydrate-rich food also prevented stress-induced increases in depressive symptoms in stress-prone subjects (Markus et al., 1998). Finally, a preferred high-carbohydrate beverage reduced mood induction-induced dysphoria to a greater degree than did isocaloric intake of a less-preferred, high-protein drink (Corsica and Spring 2008; Spring et al., 2008). The collective results indicate negative reinforcing actions of palatable food (Agras and Telch, 1998). Accordingly, global negative affect initially decreases after binges in obese adults (Berg et al., 2015) and patients with AN (Engel et al., 2013) and BN (Smyth et al., 2007).

Consistent with the negative reinforcement perspective, people high in YFAS scores more often reported using food to self-soothe (Berenson et al., 2015; Brewerton, 2017; Burmeister et al., 2013; Burrows et al., 2017a,b, 2018; Ceccarini et al., 2015; Chao et al., 2017; Davis et al., 2011; de Vries and Meule, 2016; Gearhardt et al., 2012; Granero et al., 2014; Koball et al., 2016; Masheb et al., 2018; Meule et al., 2014, 2015) and anticipated less positive reinforcement from eating (Meule and Kübler, 2012). Individuals who self-identify as having food addiction also reported using food to self-medicate their feelings of being tired, anxious, depressed or irritable (Ifland et al., 2009). The Palatable Eating Motives Scale documents that eating palatable food to "cope" with problems, worries and negative feelings is a primary motive for why people eat palatable food (Burgess et al., 2014; Boggiano, 2016; Boggiano et al., 2015b). Eating palatable food "to cope" predicts binge eating (Boggiano et al., 2014, 2015a), BMI, and class 3 obesity (Boggiano et al., 2015b; Burgess et al., 2014).

2.2.6.2. Animal findings. A large body of preclinical data also support the hypothesis that palatable food has "comforting" effects that ultimately may promote its intake and relapse (Avena et al., 2008; Cottone et al., 2009a; Dallman et al., 2003; Ulrich-Lai et al., 2010). For example, palatable food attenuated the exogenous activation of behavioral, autonomic, neuroendocrine and neurochemical stress responses (Christiansen et al., 2011; Dallman et al., 2005; Fachin et al., 2008; Kinzig et al., 2008; Krolow et al., 2010; Maniam et al., 2016; Maniam and Morris, 2010a, 2010b, 2010c; Nanni et al., 2003; Pecoraro et al., 2004; Teegarden and Bale, 2008; Ulrich-Lai et al., 2007, 2010, 2011; Warne, 2009).

2.2.7. Negative urgency

2.2.7.1. Human findings. Palatable food intake also interacts with and may promote negative urgency, the tendency to act impulsively and rashly when in extreme distress that has been implicated in pathological food, alcohol and substance use (Cyders and Smith, 2008; Zorrilla and Koob, 2019). Neurobiologically, negative urgency involves impaired "top-down" cortical-amygdala/striatal processing, yielding reduced inhibitory control over potentially detrimental actions, as well as heightened "bottom-up" amygdala-cortical/striatal processing, yielding greater attention to, incentive salience of and cognitive resource interference by emotion-evoking stimuli. These biases are thought to reflect altered structure, function and connectivity of bidirectional amygdala–orbitofrontal (OFC)/ventromedial prefrontal cortical (vmPFC) projections (Cyders and Smith, 2008; Smith and Cyders, 2016; Robbins et al., 2012; Zorrilla and Koob, 2019).

People with BN and YFAS-defined food addiction are high in negative urgency (Murphy et al., 2014; Pivarunas and Conner, 2015; Rose et al., 2018; VanderBroek-Stice et al., 2017). Negative urgency more strongly associates with binge eating and uncontrolled eating than do other facets of impulsivity, such as sensation seeking, lack of planning, or lack of persistence (Booth et al., 2018; Fischer et al., 2008; Pearson et al., 2014). It prospectively predicts binge eating symptoms in elementary, middle school and college students (Pearson et al., 2015b) and relates to increased snacking in adolescents (Coumans et al., 2018; G. T. Smith and Cyders, 2016). Transdiagnostically, negative urgency also is implicated in compulsive smoking (Billieux et al., 2007a), alcohol use (Fischer et al., 2007; Stautz and Cooper, 2013), cell phone use, shopping and gambling (Billieux et al., 2007b, 2008; Maclaren et al., 2011).

While negative urgency is often thought of as a stable antecedent (e.g., Engel et al., 2007; Fischer et al., 2018), feeding status impacts its underlying circuitry (Silbersweig et al., 2007). For example, skipping breakfast, which increases the appeal of high (vs. low) calorie foods, increases fMRI BOLD activation in the amygdala, orbitofrontal cortex (OFC), and anterior insula (Ely et al., 2017; Goldstone et al., 2009). Conversely, a high-protein intervention in "breakfast skippers" reduced post-meal craving for sweet and savory foods (Hoertel et al., 2014) and reduced pre-dinner amygdala and insula activation (Leidy et al., 2013). Palatable food (or its metabolic consequences) also may alter urgency circuits, because decreased striatal dopamine D_2 receptor availability in obese subjects, but not non-obese subjects, correlates with reduced glucose metabolism in frontal cortical regions that subserve inhibitory control, including dorsolateral prefrontal, orbitofrontal, and anterior cingulate cortices (Michaelides et al., 2012; Tomasi and Volkow, 2013; Volkow and Baler, 2015; Volkow et al., 2008b). Similar change have been observed in individuals with alcohol use disorder (Volkow et al., 2007). Reduced striatal dopamine D_2 receptor availability also relates to increased negative urgency in pathological gamblers. Within-subject increases in negative urgency predict increased symptoms of BN (Anestis et al., 2007) and may similarly predict substance use.

The increased food cue reactivity seen in the amygdala, OFC, cingulate cortex, vmPFC and dlPFC, and anterior insula of people with compulsive eating may reflect adaptations within negative urgency circuits, and not (only) reward processing as is often interpreted (e.g., Schulte et al., 2016; Stice et al., 2015; Winter et al., 2017). YFAS scores correlate directly with milkshake picture-induced activation of the amygdala, medial OFC and anterior cingulate; individuals with categorically higher YFAS scores also showed greater dlPFC activation (Gearhardt et al., 2011). As compared to lean women, obese women showed greater activation of the amygdala, vmPFC and inhibitory frontal operculum (Higo et al., 2011) in response to pictures or taste of a palatable milkshake (Ng et al., 2011). They also showed increased amygdala, OFC, anterior cingulate, insula, and mPFC responses to pictures of palatable, high-calorie, but not less preferred, low-calorie foods (Stoeckel et al., 2008). Resting-state functional connectivity of the amygdala to the insula also is increased in obese patients (Lips et al., 2014; Wijngaarden et al., 2015). Graph theory analysis found reduced nodal-degree/efficiency in the amygdala, medial OFC, rostral anterior cingulate, and insula of obese subjects. Further, greater BMI correlated with decreased global efficiency (Eglob) and decreased nodal-degree/efficiency of the medial OFC (Meng et al., 2018). Structurally, obese patients had decreased gray matter densities in the OFC, inferior and superior frontal gyri, rostral anterior cingulate, insula and dmPFC. The reduced OFC gray matter/white matter ratios correlated with greater BMI and YFAS scores. Many cortical structural differences normalized after bariatric surgery, suggesting maintenance by overeating or metabolic factors (Zhang et al., 2016b).

2.2.7.2. Animal findings. As alluded to earlier, animal models based on intermittent access to palatable food show adaptations in the amygdala, frontal cortex and insula (Blasio et al., 2014; Cottone et al., 2009a; Iemolo et al., 2013; Spierling et al., 2020). This plasticity may increase negative urgency and contribute to the risky eating-directed behaviors that develop in these models. For example, some rodents in these models exhibit palatable food-seeking and self-administration despite threat or contingent receipt of footshock punishment. They also rapidly emerge

into unsheltered open spaces when palatable food is present and persistently (high progressive ratio [PR] breakpoints) and urgently (very short latencies and rapid eating) seek palatable food despite decreasing reinforcement (Johnson and Kenny, 2010; Moore et al., 2017; Oswald et al., 2011; Parylak et al., 2011, 2012; Rossetti et al., 2014; Spierling et al., 2018; Teegarden and Bale, 2007). Supporting a functional role, optogenetic inhibition of a projection from the insula to the ventral striatum reduced the elevated PR breakpoints for palatable food of rats with punishment- and PR-resistant self-administration (Spierling et al., 2020). For future study is whether food-related increases in negative urgency and impulsivity also increase relapse and incidence of compulsive substance and alcohol use.

3. Shared vulnerability and sex differences

3.1. Cross-vulnerability to alcohol and drug addiction

3.1.1. Human findings

Given the reviewed neurobiological and motivational effects of exposure to and abstinence from palatable food as well as palatable food's ability to moderate effects of substances of abuse, it is unsurprising that feeding status and metabolic consequences of palatable food have been explored as possible vulnerability factors for addiction. Indeed, adolescents with YFAS-defined food addiction symptoms are more likely also to have used alcohol, cannabis and cigarettes (Mies et al., 2017), and conversely, food addiction is more prevalent in men with heroin use disorder (Canan et al., 2017). These comorbidities may reflect transdiagnostic substrates for food and substance use disorders (Gold et al., 2015; Parylak et al., 2011; Zhang et al., 2011).

Drug treatment programs recognize that hunger, which occurs during abstinence or dieting from palatable food, is a cross-relapse risk factor for substance use, including smoking relapse (Leeman et al., 2010). Indeed, the H.A.L.T. mnemonic in 12-step addiction recovery programs recognizes the role of Hunger as a relapse trigger (Nowinski et al., 1999). Consequently, palatable food, especially sweets, are often provided at Alcoholic Anonymous meetings (Edge and Gold, 2011) and recommended to in the Big Book to keep handy to relieve relapse craving ("Many … have noticed a tendency to eat sweets and have found this practice beneficial; One of the many doctors … told us that the use of sweets was often helpful; occasionally … a vague craving arose which would be satisfied by candy"; Alcoholics Anonymous, 2001). Perhaps accordingly, rats withdrawn from high fat diet show increased alcohol intake, an effect that appears to relate to an imbalance in dopamine transmission in the mesolimbic pathway (Martins de Carvalho et al., 2019).

These effects of abstinence from palatable food may explain why some bariatric surgery patients subsequently show increases in addictive behaviors, including gambling, spending, exercise, sexuality, smoking, and alcohol, narcotic or psychostimulant use (Azam et al., 2018; Bak et al., 2016; Conason et al., 2013; Dutta et al., 2006; Steffen et al., 2015; Wendling and Wudyka, 2011). They also may account for why there is an inverse cross-sectional, but not lifetime, relationship of obesity (Warren and Gold, 2007) and BMI (Gearhardt et al., 2018) on the one hand with SUDs on the other. Significant inverse genetic correlations also have been reported between AUDIT-C scores for alcohol use, which identify at-risk drinkers (e.g., binge drinking), and BMI ($r_g = -0.350$, $p = 3.25 \times 10E\text{-}19$) (Kranzler et al., 2019a, 2019b, 2019c).

The findings have led to the substance of abuse-food competition hypothesis (Gearhardt and Corbin, 2009; Cummings et al., 2017) whereby substances of abuse and food are proposed to compete within the same neurobiological substrate to elicit positive reinforcing and rewarding effects. Others have similarly, but conversely, proposed that the findings suggest a shared negative reinforcement substrate through which foods or substances of abuse can comfort relief craving (Zorrilla and Koob, 2019). In both models, food and substances of abuse would be expected to be misused successively (e.g., alternatively or as substitutes), rather than concurrently, consistent with the reviewed lifetime comorbidities seen between eating disorders or obesity on the one hand with SUD on the other.

Also consistent with shared vulnerability factors, human genome-wide association study (GWAS) data suggest shared genes in obesity and addiction risk (see Table 2) (Clarke et al., 2017; Kranzler et al., 2019; Schumann et al., 2016; Xu et al., 2015; Zhou et al., 2020). For example, many genes jointly implicated in problematic alcohol use and obesity phenotypes encode key molecules commonly studied in brain reward, stress and executive control circuitry, including genes for the dopamine D_2 receptor (***DRD2***; Lancaster et al., 2018; Jenkinson et al., 2000; Col Araz et al., 2012; Kvaløy et al., 2015); ankyrin repeat and kinase domain containing 1 (***ANKK1;*** Bauer 2014; Yamada et al., 2017); corticotropin-releasing hormone type 1 receptor (***CRHR1***; Curtis and UK10K Consortium, 2016; Lu et al., 2015); brain-derived neurotrophic factor (***BDNF***; Rios et al., 2001; Akbarian et al., 2018); phosphodiesterase 4b (***PDE4B***; Zhang et al., 2009; Lee et al., 2011); and cell adhesion molecule 2 (***CADM2***; Morris et al., 2019). Future research should further determine how exposure to and abstinence from palatable food impact the expression of these genes as well as others that jointly associate with risk for eating-related disorders and addictive disorders, including BRCA1 associated protein (***BRAP***); proteasome 26S subunit, ATPase 3 (***PSMC3***); Spi-1 Proto-Oncogene (***SPI1***); keratinocyte associated protein 3 (***KRTCAP3***); solute carrier family 39 member 13 (***SLC39A13***); solute carrier family 39 member 8 (***SLC39A8***); fibronectin type III domain containing 4 (***FNDC4***); and transducin beta like 2 (***TBL2***) (Zhou et al., 2020).

3.1.2. Animal findings

In addition to the aforementioned similarities shared by highly palatable foods and substances of abuse, preclinical studies also show that feeding condition (e.g., type and amount of food consumed) influences drug sensitivity. For example, food restriction (i.e., access to a restricted amount of food, such that weight loss is promoted) enhances sensitivity of rodents to drugs of abuse (Carroll et al., 1979; Branch et al., 2013; de la Garza et al., 1981; Deroche et al., 1993; Stamp et al., 2008; Carroll and Meisch, 1984; Carroll 1985; Shalev et al., 2000, 2003) and other drugs that act on dopamine systems (Carr et al., 2003; Collins et al., 2008).

Further, rats eating a high fat or high sugar diet are also more sensitive to some of the behavioral effects of drugs of abuse, including cocaine (Baladi et al., 2012; Blanco-Gandia et a., 2017a; 2017b; 2018; Clasen et al., 2020; Gosnell 2004; Serafine et al., 2015a; Serafine et al., 2015b; Puhl et al., 2011; though see also Wellman et al., 2007), amphetamine (Avena and Hoebel, 2003; Robinson et al., 2015; Fordahl et al., 2016; though see also Davis et al., 2008), methamphetamine (McGuire et al., 2011; Ramos et al., 2020), alcohol (Avena et al., 2004) and nicotine (Richardson et al., 2014, 2020). In some reports, the term "cross-sensitization" is used to describe that a prior history with highly palatable foods can increase sensitivity to the behavioral effects of drug of abuse upon subsequent exposure (Avena and Hoebel, 2003; Avena et al., 2004; see Avena et al., 2008 for a review) and in some cases, vice versa (Avena and Hoebel, 2003; Barson et al., 2009; see also Karatayev et al., 2009). Although many reports have demonstrated that eating highly palatable foods can enhance sensitivity to drug-induced locomotion or sensitization (Avena and Hoebel, 2003; Baladi et al., 2012; Serafine et al., 2015a, 2015b; McGuire et al., 2011; Ramos et al., 2020) others have shown increased cocaine self-administration (Clasen et al., 2020; Puhl et al., 2011; though see also Wellman et al., 2007) suggesting that a history of eating highly palatable foods might enhance vulnerability to addiction, at least in animal models. In addition to drugs of abuse, feeding condition has also been studied in the context of other dopaminergic drugs, including agonists like quinpirole and antagonists like raclopride. In general most of these assessments have demonstrated that high fat or high sugar diets can result in an increased sensitivity of rats and mice to drugs that act directly on dopamine receptors (Baladi

and France, 2010; Baladi et al., 2012; Foley et al., 2006; Serafine et al., 2015b; but see Geiger et al., 2009; van de Giessen et al., 2014).

Consistent with the notion of shared antecedent vulnerability factors, rats that are vulnerable to palatable diet-induced obesity also show drug use-relevant altered mesolimbic dopamine function and susceptibility to appetitive cues even before they have received obesogenic access to palatable food. This is evident as increased locomotor (Vollbrecht et al., 2015, 2016; Oginsky et al., 2016) and neurochemical (Vollbrecht et al., 2018) responses to cocaine; increased cue-induced food motivation and seeking behavior (Alonso-Caraballo and Ferrario, 2019; Derman and Ferrario, 2020) in relation to Acb core calcium permeable-AMPARs (Derman and Ferrario, 2018); increased excitability of Acb core medium spiny neurons (Oginsky et al., 2016; Alonso-Caraballo and Ferrario, 2019) in relation to a lower fast transient potassium current (I_A) (Oginsky and Ferrario, 2019); and increased sensitivity to the D_2/D_3 receptor agonist quinpirole (Vollbrecht et al., 2016).

3.2. Sex differences

3.2.1. Human findings

Epidemiological evidence suggests that the mechanisms that motivate food and drug intake vary in females versus males. Indeed, females are generally more susceptible to eating disorders; however, sex differences in SUD can vary depending on the drug that is being considered. Although more females meet the diagnostic criteria for BED than males, males with a confirmed BED diagnosis are more likely also to have a concurrent diagnosis of SUD than are females (Schrieber et al., 2013). Similarly, lifetime prevalence for SUD among patients diagnosed with BED is greater among males as compared to females (Grilo et al., 2009). However, greater use of certain types of drugs among female patients with BED (as compared to males) including cocaine and alcohol have been described (Merikangas and McClair, 2012).

Obesity also is more prevalent among women than men (Chooi et al., 2019; though see also Kim and Shin, 2020) and when mental health comorbidities are taken into consideration, females diagnosed with obesity appear to experience more depression (Luppino et al., 2010; Muhlig et al., 2016). However, sex difference data regarding drug and alcohol use among obese individuals have been more mixed (Yeomans, 2010; Traversy and Chaput, 2015; Barry and Petry, 2009). For example, light-to-moderate regular alcohol consumption was not associated with obesity among women; however, heavy or binge drinking was more strongly associated with obesity and weight gain regardless of sex (Trioneri et al., 2017). Fewer studies have evaluated sex differences in SUD vis-à-vis obesity, and none appear to show substantial sex differences (Barry and Petry, 2009; Mather et al., 2009; Simon et al., 2006; though see also Pickering et al., 2011).

3.2.2. Animal findings

Prior authors have suggested that females may be more susceptible to eating and/or drug addiction versus males, due to greater reinforcing effects and/or stronger withdrawal responses that motivate continued use and/or relapse behavior. A study examining sex differences in the reinforcing efficacy of food and compulsive eating revealed that female rats display higher levels of operant responding for palatable food (Spierling et al., 2018). Further, the latter report revealed that high-responding females with intermittent access to food had elevated respiratory exchange ratios, indicating a fat-sparing phenotype that was absent in males.

Few pre-clinical studies have assessed sex differences with regard to the impact of feeding condition or metabolic status on the behavioral effects of drugs of abuse (Collins et al., 2015; Ibias et al., 2018; Ramos et al., 2019). One report examining sex differences in the behavioral effects of nicotine, revealed that place preference produced by nicotine is enhanced in hypoinsulinemic male, but not female, rats (Ibias et al., 2018). This finding suggests that mechanisms by which diabetes enhances the reinforcing effects of nicotine are sex-dependent. Prior rodent studies also have revealed diet-induced changes in dopamine systems that are sex-dependent. For example, eating a high fat diet increased sensitivity of male rats to yawning induced by the dopamine D_2/D_3 receptor agonist, quinpirole (Ramos et al., 2019). However, other effects of this drug (hypothermia) remained unchanged. In contrast, female rats yawned significantly less than male rats, and eating a high fat diet had no effect in female rats. Eating a high fat diet also reduced pAkt (aka phosphorylated protein kinase B) levels in male, but not female, rats. Subsequent work revealed that eating a high fat diet enhanced the stimulant effects of a dopamine D_1 receptor agonist in female, but not male rats (Ramos et al., 2019; see also Speed et al., 2011). This suggests that a high fat diet enhances sensitivity to dopamine D_1 receptor agonist only among females. Together, these data demonstrate the importance of studying drug sensitivity in both male and female subjects. Importantly, this work has also revealed that certain behavioral measures in rats may be limited to a particular sex. For example, behavioral assessments of dopamine receptor sensitivity based on yawning behavior cannot be tested in female rats, as females do not display robust quinpirole-induced yawning, an androgen-mediated behavior (Berendsen and Nickolson, 1981). Moreover, chronic high fat diet consumption enhances quinpirole-induced yawning in male, but not female, rats making this screen for dopamine sensitivity useful in males only.

Prior work also has found sex differences in the efficacy of adiposity hormones to control food intake. For example, the brains of female rodents are more sensitive to the catabolic actions of low doses of leptin, whereas the brains of males are more sensitive to the catabolic action of low doses of insulin (Clegg et al., 2003). Also, female rats are less sensitive to insulin actions in the brain as compared to males (Clegg et al., 2003). The latter study also revealed that effect of insulin on food intake is observed in male, but not female rats. Future studies are needed to characterize the parameters of insulin resistance and changes in drug intake in both female and male rats. This will be important towards understanding the complex interplay of insulin, ovarian hormone systems, and drug abuse in females versus males. This pre-clinical work has important implications for understanding sex differences in the propensity for metabolic syndrome disorders, such as type-2 diabetes, cardiovascular disease, and certain cancers, in addition to SUD.

4. Treatment implications

Because highly palatable foods and drugs of abuse impact the same brain reward and stress pathways, recent research has sought to evaluate whether some treatments might be beneficial for both food-related conditions (e.g., BED, obesity, diabetes) and SUD. In this section and summarized in Table 3, we explore the literature on these medications, including current and previously approved medications for obesity (lorcaserin, buproprion/naltrexone, rimonabant; phentermine and topiramate) and BED (lisdexamfetamine dimesylate) and selected compounds in clinical trials.

4.1. Pharmacological interventions

In 2012, the FDA approved a serotonin $(5\text{-HT})_{2C}$ receptor agonist for the treatment of obesity, lorcaserin, which reduces feeding in animals and humans (Thomsen et al., 2008; Perez Diaz et al., 2019). In pre-clinical studies, lorcaserin also reduced drug intake in self-administration procedures (Anastasio et al., 2020; Collins et al., 2016; Collins and France, 2018; Gannon et al., 2018; Harvey-Lewis et al., 2016; Neelakantan et al., 2017; Perez Diaz et al., 2019; see also Howell and Cunningham, 2015; though see also Banks and Negus, 2017), attenuated reinstatement of responding for drug (Gerak et al., 2016; Gerak et al., 2019; for relevant review see Higgins et al., 2020), and blocked sensitization and withdrawal (Zhang et al., 2016a). Although these promising preclinical data prompted experiments in humans (Pirtle et al., 2019) as well as clinical trials (cocaine: https://clinicaltrials.gov/ct2/show/NCT03007394, accessed October 14, 2020;

Table 3
Selected FDA-approved treatments with therapeutic potential in both feeding- and substance/alcohol use-related disorders.

Treatment	Mechanism of action	Effect on feeding/ obesity		Effect on SUD/AUD or animal models of SUD/AUD		Clinical Trial
Lorcaserin	5-HT2C receptor agonist	↓	Thomsen et al. (2008); Perez Diaz et al. (2019)	↓	Anastasio et al. (2020); Collins et al. (2016); Collins and France (2018); Gannon et al. (2018); Harey-Lewis et al. (2016); Neelakantan et al. (2017); Perez Diaz et al. (2019) though see also Banks and Negus (2017)	NCT03007394 NCT02044874 NCT03637842 NCT03192995 NCT02906644 NCT03169816 NCT04396834 NCT04396847 NCT03253926 NCT03143543 NCT02537873 NCT03143855 NCT03266939
Rimonabant	CB1 receptor inverse agonist	↓	Nguyen et al. (2019)	↓	Galaj and Xi (2019)	NCT00464256 NCT00464165 NCT00358228 NCT00459173 NCT00458718 NCT00075205 NCT01041170 NCT00656487
Buproprion + naltrexone	Dual DAT/NET inhibition and noncompetitive nAchR antagonist + opioid receptor antagonist	↓	Sherman et al. (2016); Apovian (2016); Wang et al. (2014); Sinnayah et al. (2007); Guerdjikova et al. (2017); Halseth et al. (2018)	↓	*naltrexone alone (Martin et al., 1973; Anton 2008) *bupropion alone Howes et al. (2020)	NCT04553263 NCT00129246
Lisdexamfetamine dimesylate	Prodrug of D-amphetamine, monoamine reverse transport	↓	Citrome (2015); Gasior et al. (2017); Hudson et al. (2017); McElroy et al. (2016) & 2017	↓	Ezard et al. (2018)	NCT00573534 NCT02034201 NCT01490216 NCT00958282 NCT01486810 NCT00736255 NCT02144415
Phentermine + Topiramate	Stimulant & TAAR1 receptor agonist + GABA facilitator/glutamate inhibitor	↓	Munro et al. (1968)	↓	Phentermine alone (Glowa et al., 1997; Glatz et al., 2002; Wojnicki et al., 1999), Topiramate alone (Kampman et al., 2013; Kranzler et al., 2014; Johnson et al., 2013; Siniscalchi et al., 2015)	NCT02239913

nicotine: https://clinicaltrials.gov/ct2/show/NCT02044874, accessed October 14, 2020) to explore the potential of lorcaserin for the treatment of SUD, these trails were largely unsuccessful. Further, lorcaserin, previously FDA-approved for the treatment of obesity https://www.accessdata.fda.gov/drugsatfda_docs/nda/2016/208524Orig1s000TOC.cfm; accessed October 14, 2020) was recently removed from the market due to risks related to cancer (https://www.fda.gov/drugs/drug-safety-and-availability/fda-requests-withdrawal-weight-loss-drug-belviq-belviq-xr-lorcaserin-market, accessed October 14, 2020; https://www.fda.gov/drugs/drug-safety-and-availability/safety-clinical-trial-shows-possible-increased-risk-cancer-weight-loss-medicine-belviq-belviq-xr, accessed October 14, 2020).

Besides lorcaserin, a serotonergic drug, several other anti-obesity medications also have shown promise for the treatment of SUD. For example, rimonabant a CB_1 receptor inverse agonist was approved in the UK for the treatment of obesity since it suppresses feeding, and showed promise in animal models for other conditions, including SUD (Galaj and Xi, 2019). Unfortunately, rimonabant was associated with high rates of depression, anxiety (Blasio et al., 2013) and suicidal ideation, leading to its removal from the European market in 2008 (Sam et al., 2011). Current, related drug development efforts exploring CB_1 receptors as potential therapeutic mechanisms for both obesity and SUD are ongoing (Nguyen et al., 2019). A combination of buproprion and naltrexone is currently FDA-approved for the treatment of obesity (Sherman et al., 2016). Buproprion is a dual dopamine and norepinephrine transporter inhibitor, while naltrexone is an opioid receptor antagonist, and this combination has been shown to reduce feeding in animals and humans (Apovian, 2016; Wang et al., 2014; Sinnayah et al., 2007). Although the combination of naltrexone with buproprion is not currently on-label approved for SUD, naltrexone has a long history as a treatment for opioid use disorder (Martin et al., 1973) and AUD (Anton 2008), and bupropion is FDA-approved for the treatment of depression. Bupropion formulated with naltrexone has also been found to reduce the severity of binge eating and depressive symptoms in open-label trials of obese patients with suspected BED (Guerdjikova et al., 2017; Halseth et al., 2018), and clinical trials are ongoing (NCT03045341, NCT03047005, NCT03063606).

Tesofensine, like buproprion, also has monoamine reuptake inhibition as a main mechanism of action (Astrup et al., 2008a, 2008b; Nielsen et al., 2009; Sjodin et al., 2010; Appel et al., 2014; Axel et al., 2010; Hansen et al., 2013; van de Giessen et al., 2012), and is currently being investigated for the treatment of obesity (Bonamichi et al., 2018); however, this drug might have cardiac effects that could be problematic (George et al., 2014). Another monoaminergic drug, lisdexamfetamine dimesylate, which is FDA approved for the treatment of BED (Citrome, 2015; Gasior et al., 2017; Hudson et al., 2017; McElroy, Hudson, et al., 2016; McElroy et al., 2017; McElroy et al., 2015) and in Phase 2 trials for BN (NCT03397446; Keshen and Helson, 2017) is a prodrug of D-amphetamine, and therefore also inhibits reuptake of monoamines (Guerdjikova et al., 2016). Lisdexamfetamine elicits sustained increases

in striatal dopamine efflux (Rowley et al., 2012) that may remediate within-system deficits in mesolimbic function. Lisdexamfetamine elicits mild positive affective responses and appears to have less abuse potential than other stimulant drugs (Jasinski and Krishnan, 2009; Kaland and Klein-Schwartz, 2015). It has been (NCT00573534, NCT02034201; NCT01490216; NCT00958282) and continues to be explored as a possible substitution treatment for psychostimulant use disorders (Ezard et al., 2018; Levy, 2016).

A combination of phentermine plus topiramate was FDA approved in 2012 (https://www.fda.gov/consumers/consumer-updates/medications-target-long-term-weight-control; accessed October 14, 2020) for the treatment of obesity (in combination with a reduced calorie diet and exercise; https://www.accessdata.fda.gov/drugsatfda_docs/nda/2012/022580orig1s000_qsymia_toc.cfm; accessed October 14, 2020). Phentermine hydrochloride is also FDA approved in a low dose formulation for obesity (since 2016; https://www.healio.com/news/endocrinology/20161115/fda-approves-lowdose-phentermine-for-obesity#:~:text=The%20FDA%20has%20approved%20phentermine,announced%20in%20a%20press%20release; accessed October 14, 2020). Phentermine has a long and somewhat controversial history as part of Fen-Phen, an anti-obesity medication that included the combination of phentermine along with fenfluramine (a serotonin releaser) which was FDA approved in 1959 (Coulter et al., 2018) but was removed from the market in 1997 due to toxicity. Phentermine is a stimulant and an agonist at the TAAR1 receptor site, stimulating release of epinephrine and norepinephrine (Nguyen and Clements, 2017; Coulter et al., 2018). Phentermine suppresses appetite even when taken intermittently (Munro et al., 1968). In animal models, phentermine (typically in combination with other drugs) has been shown to decrease cocaine self-administration (Glowa et al., 1997; Glatz et al., 2002; Wojnicki et al., 1999; though see also Stafford et al., 2001) and ethanol intake (Yu et al., 1997; see also Halladay et al., 2006). Topiramate (prescribed in combination with phentermine for obesity) was originally FDA approved as an antiepileptic drug. The mechanism of action of topiramate includes facilitating GABA transmission, and inhibition of glutamate transmission via AMPA/kainate glutamate receptors (Siniscalchi et al., 2015). There are several reports demonstrating the potential of topiramate as a SUD treatment (Kampman et al., 2013; Kranzler et al., 2014; Johnson et al., 2013 see also Siniscalchi et al., 2015) as well as a recently completed clinical trial investigating topiramate-phentermine combinations for cocaine use disorder (https://clinicaltrials.gov/ct2/show/NCT02239913; accessed October 14, 2020).

Consistent with the abovementioned findings of between-system neuroadaptations related to CRF systems, a double-blind placebo-controlled trial in individuals with restrained eating found that the selective CRF_1 receptor antagonist pexacerfont reduced food craving and stress-induced eating in a laboratory setting (Epstein et al., 2016). Unfortunately, this study was halted by the NIH IRB due to reasons unrelated to adverse drug effects or efficacy (reinterpretation of the Common Rule for human subject protection under HHS, 45 CFR 46A). As such, it only had ~30% power to detect *a priori* effects of interest. Still, pexacerfont showed modest effect sizes to reduce stress-induced eating in a laboratory setting and craving for sweet foods. In bogus taste tests, pexacerfont reduced palatable food intake across all imagery scripts. Finally, YFAS food addiction symptoms were lower in subjects that received pexacerfont. A caveat to this last finding is that the reduction of YFAS scores within 24 h might be faster than pexacerfont's predicted time course of CNS action. Overall, the results provide rationale for well-powered trials of CRF_1 receptor antagonists to reduce compulsive eating (Epstein et al., 2016; Spierling and Zorrilla, 2017).

Negative emotional and sleep disturbance symptoms of alcohol withdrawal involve a hyperglutamatergic state for which acamprosate, a neuromodulator of glutamatergic tone, has been suggested (Higuchi and Japanese Acamprosate Study, 2015; Mason and Heyser, 2010; Mason and Lehert, 2012; Perney et al., 2012). Given reviewed withdrawal-like findings, anti-glutamatergic treatments also have been explored for binge eating. A small, placebo-controlled open-label study of outpatients with BED found that acamprosate yielded improvements in binge day frequency and measures of compulsiveness of binge eating and food craving in endpoint analysis; these effects were not significant in longitudinal analysis, however (McElroy et al., 2011). Memantine, a low-affinity, voltage-dependent, NMDA receptor antagonist also has shown evidence of reducing binge-type eating in open-label trials (Brennan et al., 2008; Hermanussen and Tresguerres, 2005) and animal models (Popik et al., 2011; Smith et al., 2015a,2015b).

Many promising psychotropics, including lisdexamfetamine, tesofensine, and dasotraline (NCT03107026, NCT02684279; Heal et al., 2016; Hopkins et al., 2016; Koblan et al., 2015; McElroy, Mitchell, et al., 2016b; Vickers et al., 2017) may improve outcomes not only by targeting hypohedonia and negative affect, but also by increasing inhibitory control during distress (Yarnell et al., 2013).

4.2. Metabolic interventions

Although diabetes and insulin function are not a main focus of this review, treatments that are focused on improving insulin status might also be effective for treating SUD, and are therefore relevant to describe. For example, GLP-1 receptor agonists, which have been primarily used as treatments for Type 2 diabetes (Drucker et al., 2017), might also be beneficial for the treatment for SUD (Brunchmann et al., 2019). GLP-1 activity is important within the mesolimbic dopamine system (Alhadeff et al., 2012; Cork et al., 2015; Heppner et al., 2015; Merchenthaler et al., 1999) and GLP-1 receptor agonists have been found to decrease ethanol (Abtahi et al., 2018; Thomsen et al., 2017; Sorensen et al., 2016; Sirohi et al., 2016) and drug self-administration in animal models (Hernandez et al., 2018, 2019; Sorensen et al., 2015; Schmidt et al., 2016; Tuesta et al., 2017). At least one investigation exploring the potential of GLP-1 receptor agonists in humans with AUD is currently ongoing (Antonsen et al., 2018; NCT03232112).

With regard to insulin, recent work suggests that insulin replacement normalizes the strong reinforcing effects of nicotine and dopamine deficits observed in diabetic rats (Cruz et al., 2020). These findings have led to the suggestion that glucose normalization is an important aspect to consider when considering drug cessation approaches in persons with diabetes (see O'Dell and Nazarian, 2016 for a review). The literature has not reached a consensus regarding the potential efficacy of diabetes medications for treating SUD; however, there are emerging concerns with the potential abuse liability of diabetic medications, such as metformin that appears to have abuse liability in persons suffering from eating disorders (Geer et al., 2019). Also, the dopamine receptor agonist (bromocriptine) is a drug that has been used to treat insulin resistance in persons with Type 2 diabetes; however, the literature does not support the use of dopamine receptor agonists, such as bromocriptine for treating cocaine misuse (Minozzi et al., 2015). Indeed, there is a need for research to better understand the efficacy of pharmacotherapeutic agents that may help reduce the vulnerability to drug use produced by diabetes. For example, the FDA approval of Cycloset, a dopamine receptor agonist, for the treatment of insulin resistance attests to the importance of understanding the role of dopamine in treating co-morbid conditions such as diabetes and drug use. A recent report illustrated that glucophage (i.e., Metformin) reduces nicotine withdrawal signs in mice (Brynildsen et al., 2018; see also Smith and George, 2020 for a review). Given that glucophage improves metabolic function, it is possible that this drug may also have the benefit of reducing withdrawal states that promote nicotine use and relapse behavior. Future studies are needed to address whether insulin regulation is a key aspect to improving substance use outcomes in diabetic persons with co-morbid drug use.

4.3. Non-pharmacologic interventions

Several non-pharmacologic approaches that target negative

emotions show promise to mitigate the impact of palatable food on eating behavior. Cognitive therapy to reduce emotional eating in response to negative emotions promoted weight loss and reduced relapse to obesity (Jacob et al., 2018; Werrij et al., 2009). Others with evidence of promoting healthy eating behaviors and improving weight management by improving mood include exercise, acupuncture, mindfulness, emotion reappraisal, and the relaxation response (Dunn et al., 2018; Katterman et al., 2014; Laraia et al., 2018; Masih et al., 2017; Peckmezian and Hay, 2017; Yeh et al., 2017). Finally, cognitive-behavior therapy, dialectical behavior therapy and integrated cognitive-affective therapy may improve eating behavior by increasing self-regulation as well (Cancian et al., 2019; Chen et al., 2008; Murray et al., 2015; Wallace et al., 2014; Wonderlich et al., 2014).

5. Conclusion

In this review, we have described the overlapping neuroscience shared between highly palatable foods and drugs of abuse, which demonstrates converging vulnerability factors that may predispose an individual to compulsive food or drug use. Specifically, we have described the similar impact of both highly palatable foods and drugs of abuse on metabolic and neural systems; highlighted commonalities in implicated genes and behavior, focusing on reinforcement, negative affect, negative urgency and impulse control; and summarized how food itself can impact sensitivity to drugs and potentially increase individual vulnerability for SUD. Given this overlap, we explored how therapeutic interventions that have been successful for disorders related to food might be effective for the treatment of SUD and vice versa. Taken together, this review highlights the variety of overlapping factors that may increase individual vulnerability to compulsive food or drug use and demonstrate a critical need for continued investigation on the mechanism(s) that underlie their singular and shared disease susceptibility.

Credit author statement

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Declaration of competing interest

EPZ is inventor on an on a patent for CRF_1 antagonists (US20100249138A1) and anti-ghrelin immunopharmacotherapies (US20100021487A1).

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