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Leveraging Rewards to Reduce Health-Compromising Behavior

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Leveraging Rewards to Reduce Health-Compromising Behavior

A dissertation submitted in partial satisfaction of the requirements for the degree Doctor of Philosophy in Psychology

by

Jenna Rosemarie Cummings

2018

ABSTRACT OF THE DISSERTATION

Leveraging Rewards to Reduce Health-Compromising Behavior

by

Jenna Rosemarie Cummings

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2018

Professor Ayako Janet Tomiyama, Chair

People engage in multiple behaviors that affect their health. There is little research on how these behaviors intersect within a person's life. In this dissertation, I hypothesized that behaviors that activate the neural reward system can "compete." In Study 1, I experimentally tested if eating sweet high-fat foods acutely reduced alcohol cravings for heavy drinking adults. I found that eating sweet high-fat foods acutely reduced alcohol cravings no more than watching a neutral video. In Study 2, I observed multiple reward-related behaviors (e.g., self-affirmation, social interactions, exercise) and health-compromising behaviors (e.g., unhealthy eating, alcohol use) within the everyday lives of young adults via a 4-day ambulatory electronic diary. I found that when young adults engaged in multiple reward-related behaviors they were more likely to eat unhealthy foods in the next hour. However, when young adults engaged in reward-related behaviors that provided a sense of accomplishment they were less likely to drink alcohol in the next hour. These studies fill scientific gaps in understanding intersections between reward-related behaviors and provide insight for health behavior change. Namely, that "replacing" health-compromising behavior with other reward-related behaviors may not consistently work.

The dissertation of Jenna Rosemarie Cummings is approved.

Adriana Galván

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Jennifer Ashley Silvers

Ayako Janet Tomiyama, Committee Chair

University of California, Los Angeles

2018

DEDICATION

I dedicate this dissertation to all the people who have ever struggled with changing their behavior to better their health. Thanks to science I have learned just how hard behavior change is to achieve; I hope that someday through my research I can help make it easier for you.

TABLE OF CONTENTS

INTRODUCTION 1

STUDY 1 3

LITERATURE REVIEW 3

AIMS & HYPOTHESES 11

METHODS 12

RESULTS 25

DISCUSSION 51

STUDY 2 57

LITERATURE REVIEW 57

AIMS & HYPOTHESES 66

METHODS 67

RESULTS 82

DISCUSSION 103

CONCLUSION 108

APPENDIX I 111

APPENDIX II 115

REFERENCES 119

LIST OF TABLES

TABLE 1 – Study 1 Demographics……..……14

TABLE 2 – Study 1 Descriptives Manipulation Check & Control Variables……..…….26

TABLE 3 – Study 1 Descriptives Dependent Variables……...…….27

TABLE 4 – Study 1 Descriptives Moderator Variables……...…….29

TABLE 5 – Study 1 Correlations Between Moderator Variables…….……30

TABLE 6 – Study 1 Hypothesis 1 Estimates…….……37

TABLE 7 – Study 1 Hypothesis 2 Estimates…….……39

TABLE 8 – Study 1 False Discovery Rate……..…..48

TABLE 9 – Study 2 Literature Review…….…...58

TABLE 10 – Study 2 Demographics……...……..…....70

TABLE 11 – Study 2 Descriptives Dichotomous Outcomes...……..…....83

TABLE 12 – Study 2 Descriptives Continuous Outcomes……..……..…....86

TABLE 13 – Study 2 Descriptives Moderator Variable.……...……..……..…....87

TABLE 14 – Study 2 Hypothesis 1 Estimates Sweet High-Fat Food Intake…….....90

TABLE 15 – Study 2 Hypothesis 1 Estimates Palatable Food Intake…….....92

TABLE 16 – Study 2 Hypothesis 1 Estimates Sugary Drink Intake……..…….....94

TABLE 17 – Study 2 Hypothesis 1 Estimates All Unhealthy Food Intake…….....96

TABLE 18 – Study 2 Hypothesis 1 Estimates Alcohol Use……...….....97

TABLE 19 – Study 2 Hypothesis 3 Estimates Dichotomous Outcomes…….....100

TABLE 20 – Study 2 Hypothesis 3 Estimates Continuous Outcomes……...….....102

LIST OF FIGURES

FIGURE 1 – Study 1 Self-Reported Alcohol Craving by Experimental Condition……..33

FIGURE 2 – Study 1 Heart Rate by Experimental Condition.……..34

FIGURE 3 – Study 1 Galvanic Skin Response by Experimental Condition…...……..35

FIGURE 4 – Study 1 Hypothesis 3 Sensitivity to Stimulating Alcohol Effects…….……..41

FIGURE 5 – Study 1 Hypothesis 3 G Allele of OPRM1 Gene……..……..43

FIGURE 6 – Study 1 Hypothesis 3 Family History of Alcoholism…….……..……..45

FIGURE 7 – Study 1 Hypothesis 3 Family History of Alcoholism…….…..……..46

FIGURE 8 – Study 2 Screenshot of PACO Reward-Related Behavior Question……..……..74

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Introduction

Reward-Related Behavior

A reward is a stimulus, object, event, activity, or situation that motivates human behavior (Schultz, 2015). People interact with a diversity of rewards that motivate a diversity of behaviors. For example, an individual can eat a slice of cake, drink a glass of wine, inhale a puff of a cigarette, purchase a new item of clothing, kiss an attractive person, or gamble to win money. These actions can be labeled as reward-related behaviors. Although scientific study of reward illuminates the basic processes involved in reward-related behavior (Schultz, 2015), scholars have understudied how different reward-related behaviors impact each other.

Health Behavior

A health behavior is any activity undertaken by humans that affects their health; if the behavior improves health it can be labeled as health-enhancing (e.g., eating vegetables) and if the behavior harms health it can be labeled as health-compromising (e.g., eating ultra-processed food; Taylor, 2012). Humans engage in a diversity of health behaviors including eating, drinking, smoking, sleeping, and exercising, and patterns of these behaviors are robustly associated with chronic disease risk (Meng, Maskarinec, Lee, & Kolonel, 1999), mortality risk (Mokdad, Marks, Stroup, & Gerberding, 2004), and health-related quality of life (Blanchard, Courneya, & Stein, 2008). Although scientific study of health behavior has led to the development of seminal health behavior theories that may guide efforts to change behavior (Schwarzer, 2011), for many years scholars ignored the study of how one health behavior may impact others (Spring, King, Pagoto, Fisher, & Spring, 2015). Research of this kind is critical to the development of theory that may guide efforts to simultaneously change multiple behaviors,

which ultimately may increase health benefits, maximize health promotion, and reduce health care costs (Prochaska & Prochaska, 2011).

Dissertation Research

In this dissertation, I examined the intersection of multiple reward-related behaviors with relevance to health behavior change. The proposed research focused on two particular health behaviors—eating and alcohol use—primarily because the scientific literature robustly documents these behaviors as also being reward-related (Conger, 1956; Rogers & Hardman, 2015). Moreover, multiple aspects of these behaviors are similar (e.g., reward-related, oral ingestion route, engage gustatory and olfactory systems), which becomes less surprising when one considers that alcohol is chemically derived from food sources (i.e., fermenting sugar). Importantly, studying these behaviors may have large implications for public health; scholars estimate that improving eating and alcohol use could prevent 20% of U.S. deaths per year (Mokdad et al., 2004).

In Study 1, I used an experimental design to test if eating sweet high-fat foods acutely reduced alcohol cravings in adults at risk for developing alcohol use disorders. Results may have implications for changing the drinking behavior of heavy drinking adults. In Study 2, I used a 4-day ambulatory electronic diary design to test if multiple reward-related behaviors (e.g., self-affirmation, social interactions, exercise) reduced the likelihood of unhealthy eating and alcohol use within the everyday lives of young adults. Results may have implications for changing the eating and drinking behavior of young adults. Ultimately, these studies fill scientific gaps in understanding intersections between reward-related behaviors and provide insight for health behavior change.

Study 1

Literature Review

Food/alcohol & the mesolimbic dopamine pathway. When individuals consume food/alcohol, their neural reward systems react, chiefly their mesolimbic dopamine pathways (Volkow, Wang, Fowler, & Telang, 2008). The mesolimbic dopamine pathway connects the ventral tegmental area (VTA) and substantial nigra (SN) to the nucleus accumbens (NAc) and dorsal striatum (DSTR). When individuals consume food/alcohol, neurons in the VTA and SN release dopamine to the NAc and DSTR, which contain dopamine receptors. In the mesolimbic dopamine pathway, dopamine (a) codes for pleasure, (b) enhances incentive salience, or the desire for food/alcohol, and (c) reinforces eating/alcohol use (Volkow et al., 2013).

Shared reward system & biological vulnerability. Given that eating and alcohol use both activate the neural reward system, does that affect how eating and alcohol use intersect? Gearhardt and Corbin (2009) hypothesized that if the neural reward system, "is occupied by one of the behaviors (i.e., eating or alcohol use), it would block the other" (pg. 217). As a result, someone who ate food frequently or in large amounts would not drink alcohol frequently or in large amounts and vice versa. The authors indirectly supported their hypothesis by finding that, in a sample of 37,259 adults (Mean Age = 46.55) from the U.S., individuals with a greater Body Mass Index (BMI; a proxy for increased eating) drank alcohol less frequently and had lower typical Blood Alcohol Concentrations (BAC) compared to those with a lower BMI.

To strengthen support for their hypothesis, Gearhardt and Corbin (2009) also examined the associations between BMI and alcohol use in those with a biological vulnerability related to the neural reward system's functioning. The authors identified those with a family history of alcoholism as those with a biological vulnerability. They found that BMI moderated the impact of a family history of alcoholism on frequency of alcohol use and typical BAC; (a) individuals

with a family history of alcoholism drank alcohol more frequently, but only if they had a lower BMI and (b) individuals with a family history of alcoholism had higher typical BACs, but this link was three times stronger for those with a lower BMI. In other words, family history of alcoholism conferred risk for alcoholism only when an individual did not already have obesity. These authors concluded that, "Although these results do not provide direct evidence for shared neurobiological pathways, they are quite consistent with the hypothesis that food occupies neurobiological pathways related to the reinforcement value of alcohol" (pg. 223).

Food-alcohol competition hypothesis. Extending from this prior work, I proposed a food-alcohol competition hypothesis wherein eating rewarding foods (e.g., processed foods, sweet high-fat foods) stimulates, occupies, and blocks the mesolimbic dopamine pathway, reducing the likelihood of alcohol cravings and consumption (Cummings, Ray, & Tomiyama, 2017). This hypothesis predicts that eating reduces alcohol use and does not predict that alcohol use reduces eating. I proposed this directionality because when individuals drink alcohol not only do their neural reward systems react but also their brains metabolize the alcohol. This is why alcohol depresses the Central Nervous System and causes effects such as disinhibition (Lustig, 2009). When individuals are disinhibited from alcohol use, they may eat *more* (Christiansen, Rose, Randall-Smith, & Hardman, 2016); indeed this may explain why a number of research studies indicate that small to moderate alcohol doses stimulate food intake (Caton, Marks, & Hetherington, 2005; Christiansen et al., 2016; Eiler et al., 2015; Hetherington, Cameron, Wallis, & Pirie, 2001; Hofmann, 2008; Schrieks et al., 2015; Yeomans, 2010)—although a number of studies indicate that small to moderate alcohol doses do not affect food intake (Caton, Ball, Ahern, & Hetherington, 2004; de Castro & Orozco, 1990; Mattes, 1996; Poppitt, Eckhardt, McGonagle, Murgatroyd, & Prentice, 1996; Rose et al., 2015; Yeomans & Phillips, 2002).

On the other hand, the brain does not metabolize food so—although the neural reward system reacts when someone eats—the Central Nervous System is not also depressed by food (Lustig, 2009), and individuals are not disinhibited from eating the same way they are disinhibited from alcohol use. Thus, although both eating and alcohol use may stimulate, occupy, and block the mesolimbic dopamine pathway, it is more plausible eating reduces the likelihood of alcohol cravings and use rather than the converse. Below, I summarize support for the food-alcohol competition hypothesis from anecdotal claims and observations, rodent studies, cross-sectional studies, and my preliminary work with longitudinal data.

Anecdotal claims & observations. Alcoholics Anonymous (1975) published a passage in the book Living Sober about the effectiveness of eating sweets for reducing alcohol cravings: "This booklet is based on our own personal experience, rather than on scientific reports. So we cannot explain precisely, in technical terms, why this should be so. We can only pass on the word that thousands of us—even many who said they had never liked sweets—have found that eating or drinking something sweet allays the urge to drink" (pg. 22). Unfortunately, Alcoholics Anonymous has not empirically tested the effectiveness of this practice. However, Yung, Gordis, and Holt (1983) observed diets for 30 days of 64 newly sober outpatients with alcoholism and reported that outpatients who remained sober had (a) twice as much added sugar in beverages and (b) greater overall carbohydrates in their diets compared to outpatients who relapsed.

Rodent studies. I conducted a systematic review identifying all empirical research studies at the intersection of eating and alcohol use (Cummings et al., under review). In my systematic review, I identified four rodent studies with results that support the food-alcohol competition hypothesis (Kampov-Polevoy, Overstreet, Rezvani, & Janowsky, 1995; Samson, Roehrs, & Tolliver, 1982; Sirohi, Van Cleef, & Davis, 2017a, 2017b). First, Samson et al. (1982) offered

rats a sucrose solution in varying doses along with an alcohol solution. Results indicated that with a dose of 1.00-1.25% (w/v) sucrose solution rats drank equivalent amounts of sucrose and alcohol, but with a dose of > 1.25% (w/v) sucrose solution rats drank less alcohol than sucrose [with the largest decrease in alcohol at the largest administered dose, which was 4% (w/v) sucrose solution]. Second, Kampov-Polevoy et al. (1995) found that, compared to rats that had access to water and chow only for five days, rats that also had access to as little as 0.10% (w/v) saccharin solution during the five days drank less alcohol up to ten days later. The authors remarked that the degree of suppression on ethanol intake was comparable to the effect of a five-day administration of fenfluramine (1.0 g/kg), a drug known for its anti-alcohol effects. Third, Sirohi et al. (2017a) found that, compared to rats that regularly had chow for five weeks, rats that either had a high-fat diet (Crisco) every three days or all-day everyday during the five weeks drank less alcohol two weeks later. Sirohi et al. (2017b) additionally found that, compared to rats that had chow for six weeks, rats that had intermittent access (24 hours twice per week) to a high-fat diet (Crisco) during the six weeks drank less alcohol up to three days later. Together these rodent studies suggest that eating rewarding food (e.g., high-fat, high-sugar) may reduce alcohol use, and that the food dose and the timing/pattern of eating may be important in modifying the effect.

Cross-sectional data. In my systematic review, I found there were no existing experimental studies in humans on the effect of eating on alcohol use (Cummings et al., under review). However—relevant to the food-alcohol competition hypothesis—I identified ten cross-sectional studies that reported negative associations between eating and alcohol use, wherein greater eating was associated with less alcohol use and vice versa (Butler, Popkin, & Poti, 2017; Colditz et al., 1991; Gruchow, Scheller, Sobocinski, & Barboriak, 1985; Hillers & Massey, 1985;

Ishikawa, Yokoyama, & Murayama, 2017; Kesse, Clavel-chapelon, Slimani, Liere, & Group, 2001; Ma, Betts, & Hampl, 2000; Ruf, Nagel, Altenburg, Miller, & Thorand, 2005; Ruidavets et al., 2004; Walmsley, Bates, Prentice, & Cole, 1998). To begin, Butler et al. (2017) found that those who ate the most calories, fat, carbohydrates, and sugar drank the least amount of alcohol. Colditz et al. (1991) found those who ate more carbohydrates including sucrose and added sugar as well as chocolates and candies drank less alcohol. Gruchow et al. (1985) found that those who ate the most nonalcoholic calories drank the least amount of alcohol. Hillers & Massey (1985) and Ishikawa et al. (2017) found that those who ate more protein, fat, and carbohydrates drank less alcohol. Kesse et al. (2001) found that those who ate more soup, yogurts, vegetables, and fruit drank less alcohol. Ma et al. (2000) found that those who ate more fruit, vegetables, and grains drank less alcohol. Ruf et al. (2005) found that those who ate more fruits, dairy products, and cereal products drank less alcohol. Ruidavets et al. (2004) found that those who ate the most fruits, cereal, milk/cottage cheese, and added sugar drank the least amount of alcohol. Walmsley et al. (1998) found that those who ate the most fat drank the least amount of alcohol. In sum, there are several cross-sectional studies suggesting that greater eating (most often greater intake of fat, sugar, and carbohydrates) is associated with less alcohol use.

Longitudinal data. In a sample of 2,379 adolescent girls assessed yearly from age 15 to 19, I used Latent Growth Modeling to capture how alcohol use might develop relative to certain types of eating behavior (Cummings et al., 2017). Adolescent girls who ate fast food more frequently at age 15 were less likely to drink more alcohol from age 15 to 19. Also, adolescent girls who ate more fat and sugar from age 15 to 19 were less likely to drink more alcohol from age 15 to 19. Thus, these data provide preliminary support that food-alcohol competition might unfold over time, specifically between sweet high-fat/processed food and alcohol.

Weaknesses of current evidence for food-alcohol competition hypothesis. Despite the anecdotal, observational, rodent, cross-sectional, and longitudinal support for the food-alcohol competition hypothesis, there are weaknesses in the literature. Foremost, there is no experimental test in humans, which means there is no evidence in humans that can support causality. Moreover, there are countless varieties of food and alcohol and these varieties of food and alcohol may intersect in different ways. The evidence cited above suggests that eating processed foods high in sugar and fat (e.g., sugar x fat, fast food, chocolate, carbohydrates) in particular may compete with alcohol use. Likewise, as seen in the rodent studies, eating may only compete with alcohol use at specific doses; for instance, an initial small food dose at one time point may not reduce alcohol use but a larger food dose may.

An additional weakness of the current evidence for the food-alcohol competition hypothesis is a lack of tested mediators. Although I hypothesized that downstream behavioral competition occurs because of upstream mesolimbic dopamine pathway competition, none of the current evidence in humans tests this and thus leaves open the possibility of other mechanistic explanations. For instance, food may compete with alcohol because both substances are calorie dense; eating may simply fill up the stomach. Indeed, one study found that drinking 1000ml of mineral water in 10 minutes versus control (no water) reduced self-reported alcohol craving in the laboratory. The authors speculated this occurred because water forces gastric distension, which can reduce ghrelin levels thereby reducing motivations to drink alcohol (and water did indeed reduce ghrelin levels in this sample; Koopmann et al., 2017). Another alternative is that food may compete with alcohol simply because it distracts an individual from alcohol.

Relatedly, different individuals may have different susceptibility to the effect, depending on what causes it. Therefore, a weakness of the current evidence for the food-alcohol

competition hypothesis is a lack of tested moderators. For example, if foods compete with alcohol because of upstream mesolimbic dopamine pathway competition, perhaps only those with a biological vulnerability related to the neural reward system's functioning experience food-alcohol competition. This would follow from Gearhardt and Corbin's (2009) finding that BMI attenuated the link between family history of alcoholism and increased alcohol use.

Study 1 contributions. I designed Study 1 in direct response to the aforementioned weaknesses. First, Study 1 was a randomized experiment of the effect of eating on alcohol cravings. This is a significant contribution because it is the first causal test in humans. Second, Study 1 manipulated type of food (sweet high-fat food or calorie equivalent bland food) and food dose (1 serving or 3 servings). This is a significant contribution because it provides the opportunity to reveal precisely which foods in which amount may compete with alcohol. I selected sweet high-fat food based on prior evidence but also because individuals report these foods as having higher addictive potential (Schulte, Avena, & Gearhardt, 2015). I selected 1 (~150 calories) or 3 (~450 calories) servings because it provided substantial variability in food dose while limiting health risk (see Health Significance section below).

Third, Study 1 ruled out other mechanisms by controlling for calories and distraction in comparison groups. This is a significant contribution because it provided the opportunity to falsify mesolimbic dopamine pathway competition as a mechanism. Fourth, Study 1 measured and tested for potential moderators of any observed effect. The potential moderators included endophenotypic markers associated with a biological vulnerability related to the neural reward system's functioning: the G allele of the OPRM1 gene (Ray & Hutchison, 2004), sensitivity to the stimulating effects of alcohol (Ray, Mackillop, & Monti, 2010), and family history of alcoholism (Gearhardt & Corbin, 2009). This is a significant contribution because, if these

variables moderated the hypothesized effect, it provided the opportunity to further build support for mesolimbic dopamine pathway competition as a mechanism (see Specific Aim 3 below).

Health significance. Alcohol use has been appraised as the third actual cause of death in the U.S. (Mokdad et al., 2004). Theories on alcohol use and prevention and treatment strategies for problematic alcohol use most often isolate this behavior from other health behaviors. Yet, in an individual's everyday life alcohol use is not isolated from other health behaviors. Research identifying how eating might change alcohol use is critically needed.

In applied settings, this research will shed light on multiple points in the efficacy of current prevention and treatment strategies for problematic alcohol use that use behavior modification. First, support for the food-alcohol competition hypothesis may foster the refinement of prevention and treatment strategies for problematic alcohol use to acknowledge eating behavior. In particular, supportive results would add credibility to an already existing community practice backed by Alcoholics Anonymous (1975). In that case, clinicians might also want to consider developing and evaluating interventions that use food to help reduce alcohol use. This aligns with the ideology of a harm reduction approach, wherein the main goal of intervention is to reduce the most harmful consequences of addictive behavior in an individual's life (Marlatt, 1996). In other words, alcohol is considered an acute toxin but sweet high-fat food (even 3 servings) is not considered an acute toxin (Lustig, 2009). For individuals who are severely harming themselves via their alcohol use, occasionally eating 1 or 3 servings of sweet high-fat food may have less detrimental acute health effects than continuing to drink alcohol.

Second, the results from the proposed research might also have broader implications for how to approach health behavior modification. If eating certain foods allays alcohol cravings, it may be worthwhile, for instance, to always modify eating and drinking behaviors simultaneously

while being cognizant of potential interplay. Third, identification of moderators in food-alcohol competition allows for maximum efficacy in behavioral modification strategies amongst diverse populations. This has implications for personalized prevention and treatment plans by directing behavioral modification strategies to individuals most fit for them. Indeed, this is congruent with the precision medicine initiative that the National Institutes of Health currently supports and recommends (Collins & Varmus, 2015).

Aims & Hypotheses

Specific Aim 1: To examine alcohol cravings after individuals eat sweet high-fat food. I hypothesized that individuals who consumed sweet high-fat food would have dampened alcohol cravings compared to individuals who consumed bland food or no food.

Specific Aim 2: To examine a dose-response relationship between eating sweet-high fat food and alcohol cravings. I hypothesized with an increased dose of sweet high-fat food there would be greater dampening of alcohol cravings. I hypothesized with an increased dose of bland food there would be no change in alcohol cravings.

Specific Aim 3: To test between-subjects moderators. I hypothesized alcohol craving, after individuals eat sweet high-fat food, would diminish most strongly for individuals who:

- **3a. Report sensitivity to the stimulating effects of alcohol.** Certain individuals consistently report greater stimulation from alcohol (see Ray et al., 2010 for a review). Given the commonalities between sweet high-fat food and alcohol (e.g., high in sugar, oral ingestion route), I expected that, for individuals who report greater stimulation from alcohol, sweet high-fat food would stimulate the mesolimbic dopamine pathway and thereby reduce alcohol cravings.

- **3b. Carry the G allele of the OPRM1 gene.** Scholars propose that the endogenous opioid system modulates activation of the mesolimbic dopamine pathway (Herz, 1997). Specifically, activation of mu opioid receptors in the VTA enhances dopamine release to the NAc (Tanda & Di Chiara, 1998). The OPRM1 gene codes for mu opioid receptor binding; the G allele may be associated with increased binding affinity and, indeed, carriers of this polymorphism report greater stimulation from alcohol compared to those who do not carry it (Ray & Hutchison, 2004; Ray et al., 2013). I expected that, for individuals with this polymorphism, sweet high-fat food (another reward) would stimulate the mesolimbic dopamine pathway and thereby reduce alcohol cravings.
- **3c. Have a family history of alcoholism.** In Gearhardt and Corbin's (2009) study, BMI attenuated the link between family history of alcoholism and increased alcohol use. Thus, I expected that, for individuals with a family history of alcoholism, sweet high-fat food could reduce alcohol cravings.

Methods

Study Design. This study was a 2 (sweet high-fat food or bland food) x 2 (1 serving or 3 servings) + 1 (no food/control) randomized mixed factorial experiment.

Participants. I recruited 150 individuals age 21 or older from the Los Angeles community. I calculated this sample size based on a power analysis conducted in G*Power Version 3.1.7 (Faul et al., 2007). I selected a repeated measures, within-between interaction design and specified power of .95, five conditions, two time points, a correlation of $r = .50$ between time points, and an expected effect size of Cohen's $d = 0.40$ (medium). This expected effect size was based on a meta-analysis of cue reactivity to drug-related stimuli (Carter & Tiffany, 1999).

To ensure that there was sufficient variance in cue reactivity to alcohol beverage cues, I recruited non-dependent heavy drinkers (scored 8-15 on the Alcohol Use Disorder Identification Test; Babor, Higgins-biddle, Saunders, & Monteiro, 2001). This type of sample was successfully recruited for multiple cue reactivity studies at the University of California, Los Angeles (Ray et al., 2015; 2017). Full inclusion criteria were: (1) age 21-55 (above legal drinking age but not older adults), (2) fluency in English (in order to understand study procedure), and (3) score of 8-15 on the Alcohol Use Disorder Identification Test (AUDIT; Babor et al., 2001). Exclusion criteria were: (1) age greater than 55, (2) score less than 8 or greater than 15 on the AUDIT, (3) current treatment for alcohol use or a history of treatment or treatment seeking in the 30 days before enrollment, (4) current (last 12 months) diagnosis of a substance use disorder for psychoactive substances other than nicotine, (5) current (last 12 months) diagnosis of an eating disorder, (6) current diagnosis of food addiction [based on Yale Food Addiction Scale (Gearhardt, Corbin, & Brownell, 2009)], (7) following a strict diet that would prevent eating sweet high-fat food, and (8) food allergies to all five sweet high-fat food options. I chose exclusion criteria 3-8 to reduce the likelihood of adverse events for the study because participants are asked to smell alcohol and eat sweet high-fat food.

Participants were not excluded based upon sex or ethnicity. However, I over-recruited for Asian participants because the G allele of the OPRM1 gene has higher representativeness in this ethnicity (Chamorro et al., 2012). Each time I circulated a general flyer or online advertisement for the study, I also circulated a flyer or online advertisement that targeted Asian participants with text such as, “Are you Asian?” and, “The DiSH Lab in the Department of Psychology at UCLA is looking for Asians who regularly drink alcohol to participate in the Cravings in Everyday Life Study.” Full demographics for the final sample are presented in Table 1.

Table 1

Study 1 Demographics

	Mean	*SD*
	%	
Age	25.15	7.39
Sex (% Male)	53.3%	
Ethnicity		
Caucasian	34.0%	
Asian American	32.7%	
Hispanic/Latinx	16.7%	
African American	7.3%	
Multi-racial/Other	7.3%	
Arabic/Middle Eastern	2.0%	
Subjective SES		
1st Rung (Lowest)	0.0%	
2nd Rung	1.3%	
3rd Rung	2.0%	
4th Rung	12.7%	
5th Rung	12.7%	
6th Rung	18.7%	
7th Rung	28.7%	
8th Rung	18.0%	
9th Rung	5.3%	
10th Rung (Highest)	0.7%	
AUDIT Score	10.72	2.13
YFAS Score	1.04	1.26
Body Mass Index	24.15	3.37
Underweight	9.5%	
Normal	56.1%	
Overweight	28.4%	
Obese I	5.4%	
Obese II	0.7%	

Notes: AUDIT = Alcohol Use Disorders Identification Test Score, YFAS = Yale Food Addiction Scale Score; Symptom counts for food addiction range from 0-7 and ≥ 3 symptoms plus clinically significant impairment or distress indicates food addiction.

Procedure. All participants were scheduled for one laboratory session between 4 and 8 P.M. to dovetail with time-of-day drinking norms. The cover story for this experiment was that participants were joining the "UCLA Cravings in Everyday Life Study," where researchers were studying how alcohol cravings function in everyday settings like restaurants and movie theaters. Thus, the experiment was blinded to participants, which reduced risk of performance bias (The Cochrane Collaboration, 2016). Research assistants were not blinded to the randomly assigned experimental condition since they were serving food. However, to limit the risk of detection bias, research assistants were not informed of the experimental hypothesis until after the study was completed and the data were analyzed (The Cochrane Collaboration, 2016).

Participants were instructed to not consume caffeine during the four hours prior to the lab session and to not exercise or smoke during the three hours prior to the lab session. This was so that—prior to introducing the experimental manipulation—physiological arousal most likely reflected a true baseline. Participants were also instructed to not consume food during the hour prior to the lab and to not drink alcohol the day of the lab session. This was so that eating and drinking behavior that occurred prior in the day would not impact the experimental protocol. For instance, if participants ate a large amount of food right prior to the experiment and then were instructed to eat more food, they may have been too full. Or, if participants drank alcohol on the day of the experiment, it might have affected their ability to follow instructions. Participants completed a pre-questionnaire via Qualtrics Online Survey Software prior to their scheduled session; this included measurement of potential moderators (see Study 1 Measures below).

At the lab session, participants were led into a private testing room where they were seated and asked if they would provide informed consent. They provided a saliva sample via the Oragene kit (DNA Genotek, Kanata, Ontario, Canada). Then participants were attached to

physiological monitoring equipment (BIOPAC Systems, Inc., Goleta, California, U.S.A.). This equipment was non-invasive and did not require any clothing to be removed. Participants had electrode sensors adhered to their fingers on their non-dominant hands and their chests. These electrodes were attached to wireless transmitters, which sent feedback to the processor model (MP150; BIOPAC Systems, Inc., Goleta, California, U.S.A.). Participants were reminded to avoid touching the sensors, to sit still while allowing themselves to feel comfortable, and to keep their legs uncrossed and relaxed with both feet on the floor. Participants then underwent a 3-minute relaxation period to adjust to the physiological equipment (Leggio et al., 2014).

To determine the sweet high-fat food item participants ate, and the alcohol cue for the alcohol craving paradigm, I used an idiosyncratic method so that each participants would find the item and cue rewarding (Giuliani, Mann, Tomiyama, & Berkman, 2014). Participants were provided with a five-item list of sweet high-fat foods including ice cream, cookies, cupcakes, chocolate, and brownies and a five-item list of alcoholic beverages including beer, wine, champagne, vodka, and rum. Pilot study raters determined the items on the food list (n = 73) by ranking different kinds of sweet high-fat foods from most to least comforting and determined the items on the alcohol list (n = 385) by ranking different kinds of alcohol from most to least pleasurable. Participants selected one sweet high-fat food item and one alcoholic beverage from these lists that they personally considered the "most palatable and rewarding."

Next, participants completed additional self-reports on potential moderators (see Study 1 Measures below). During this time, a research assistant prepared the alcohol-craving paradigm, bringing two covered trays into the testing room (adapted from Leggio et al., 2014). When the participant finished the questionnaire, the research assistant stated the following script:

> "Next, I will be revealing to you a series of different types of beverages, alcoholic and non-alcoholic. However, I am not going to tell you in what order. I am going to play audio recordings that instruct you on smelling these beverages. You will not be drinking the beverages. Afterwards, I am going to ask you some questions. I will need to stay in the room, however, to provide you with privacy during the task, I will sit on the other side of the room and face the other direction. Do you have any questions?"

The research assistant then removed the cover off one tray to reveal a water bottle and an empty glass (neutral cue used to acquire baseline alcohol craving measurements). The research assistant opened the water bottle, poured the water into the glass, placed the glass in front of the participant, and started playing the audio recording. The audio recording instructed the participants to sniff the glass when they heard high tones and stop sniffing when they heard low tones. This procedure was 3-minutes and included 13 5-second olfactory exposures (Leggio et al., 2014). Immediately after the audio recording ended, the research assistant instructed the participant to respond to a visual analog scale measuring alcohol craving. Next, the research assistant repeated the procedure by removing the cover off the second tray to reveal a bottle of the participant's selected alcohol beverage and an empty glass. The research assistant opened the alcohol bottle, poured the alcohol into the glass, placed the glass in front of the participant, started playing the audio recording, and, at the end of the recording, instructed the participant to respond to a second visual analog scale measuring alcohol craving. During the alcohol-craving paradigm, the participant's Galvanic Skin Response and Heart Rate were continuously recorded using AcqKnowledge 4.2 software (BIOPAC Systems, Inc., Goleta, California, U.S.A.). The research assistant time stamped cue presentations in this software.

After the alcohol-craving paradigm, the participant completed additional self-reports on potential moderators (see Study 1 Measures below) while a research assistant prepared the experimental manipulation. I randomly assigned participants to consume no food, bland food, or their selected sweet high-fat foods. The bland food (plain corn tortilla) was determined by pilot study taste testers (n = 8) and was rated as lowest in palatability and reward value compared to four other bland foods (bread, pita bread, cereal, unsalted corn tortilla chips). All foods were selected to be isocaloric; 1 serving size for each food was roughly equivalent (150 +/- 10) in calories. I randomly assigned those in food conditions to consume 1 or 3 servings. This experimental manipulation was 15 minutes for all participants. For participants in food conditions, they had 5 minutes (1 serving) or 15 minutes (3 servings) to consume and during any remaining time watched a neutral video. This previously used time protocol provided the appropriate time for sweet high-fat food and bland food to be absorbed in the participants' body (Schoenmakers, Wiers, & Field, 2008). Participants in the no food/control condition spent the complete 15 minutes watching a neutral video. The neutral video served as an active control condition, such that participants were still engaging in a potentially distracting activity during the 15 minutes but not consuming food. All participants were also given 6 oz. of water (~177 ml) in all conditions to control for water intake (Koopmann et al., 2017).

Once the experimental manipulation was complete, the participant answered demographic and control questions (see Study 1 Measures below) while a research assistant prepared the second alcohol-craving paradigm. The alcohol-craving paradigm was then repeated (starting with the same script read by the research assistant) but with only the participant's selected alcohol beverage and an empty glass. The research assistant opened the alcohol bottle, poured the alcohol into the glass, placed the glass in front of the participant, started playing the

audio recording, and, at the end of the recording, instructed the participant to respond to a third visual analog scale measuring alcohol craving. Again, Galvanic Skin Response and Heart Rate were continuously recorded using AcqKnowledge 4.2 software (BIOPAC Systems, Inc., Goleta, California, U.S.A.) and the research assistant time stamped the cue presentation in this software. Finally, the research assistant funnel debriefed and compensated the participant.

Measures. Complete lists of items for questionnaires are presented in Appendix I.

Alcohol craving. In reviewing the craving literature, Sayette (2000) noted limitations in using single measures. Thus, the current research used a multi-method measurement approach with self-report and physiological measures.

Visual Analog Scale. Participants responded to the question "How much do you crave alcohol right now?" on a sliding scale ranging from 0-100. The scale was anchored with "Not at all" at 0 and "Extremely" at 100. Participants responded to the question after the water cue/baseline, after the first alcohol cue, and after the second alcohol cue. In response to the alcohol cues, I expected alcohol cravings to increase from baseline.

Galvanic Skin Response. Galvanic Skin Response was continuously measured throughout the study via physiological monitoring equipment (BIOPAC Systems, Inc., Goleta, California, U.S.A.). I reduced the Galvanic Skin Response data with AcqKnowledge 4.2 software (BIOPAC Systems, Inc., Goleta, California, U.S.A.). Using the time stamps marked into the recordings, I reduced the Galvanic Skin Response data into nine 1-minute segments: three 1-minute segments following water cue/baseline, first alcohol cue, and second alcohol cue. I calculated the mean for each of these nine 1-minute segments. Typical Galvanic Skin Responses range from 2-20 microsiemens (Dawson, Schell, & Filion, 2007); any Galvanic Skin Response outside of the bounds of 0-20 was recoded as missing.

In response to the alcohol cues, I expected Galvanic Skin Response to increase from baseline. This is because the alcohol-craving paradigm is conceptualized as triggering an excitatory pathway that activates the sympathetic nervous system (Leggio et al., 2014). When the sympathetic nervous system is activated, there is increased sweat gland activity, which increases the skin's potential to conduct electricity (Darrow, 1927). Thus, greater Galvanic Skin Response represents greater craving.

Heart Rate. Heart Rate was continuously measured throughout the study via BioPac Systems. I reduced the Heart Rate data with MindWare software (MindWare Technologies, Ltd., Gahanna, Ohio, U.S.A.). Using the time stamps marked into the recordings, I reduced the Heart Rates into nine 1-minute segments: three 1-minute segments following water cue/baseline, first alcohol cue, and second alcohol cue. I calculated the mean for each of these nine 1-minute segments. Typical Heart Rates ranged from 60-100 beats per minute (American Heart Association, 2016); any Heart Rate outside of the bounds of 50-120 was recorded as missing.

In response to the alcohol cues, I expected Heart Rate to increase from baseline. This is because the alcohol-craving paradigm is conceptualized as triggering an excitatory pathway that activates the sympathetic nervous system (Leggio et al., 2014). When the sympathetic nervous system is activated, there is increased Heart Rate (although Heart Rate may also be influenced by the parasympathetic nervous system too; Robinson, Epstein, Beiser, & Braunwald, 1966). Thus, greater Heart Rate represents greater craving.

Moderators. I measured my three hypothesized moderators based on prior work.

Biphasic Alcohol Effects Scale (Martin, Earleywine, Musty, Perrine, & Swift, 1993). The Biphasic Alcohol Effects Scale is a self-report questionnaire designed to measure an individual's sensitivity to the stimulating and sedating effects of alcohol. Participants rated 14-items

capturing the extent to which drinking alcohol has personally produced feelings like "energized" and "sedated" on an 11-point Likert scale (0 = "Not at all" to 10 = "Extremely"). Internal consistency among items was high for sensitivity to the stimulating effects of alcohol (Cronbach's $\alpha = .91$) and to the sedating effects of alcohol (Cronbach's $\alpha = .86$). For the current study, I averaged across items regarding the stimulating effects of alcohol.

Oragene Kit (DNA Genotek, Kanata, Ontario, Canada). Saliva samples were collected for DNA analyses using Oragene kits. Genotyping was performed to identify presence of the G allele of the OPRM1 gene. For the current study, I dummy coded presence of the G allele (0 = No G allele, 1 = G allele).

Family History of Alcoholism (Collaborative Study on the Genetics of Alcoholism; Rice et al., 1995). Participants were provided with a thorough description of the signs of a significant drinking problem, defined as drinking that did or should have led to treatment. Signs of problem drinking included legal problems (e.g., drunk driving violations), health problems (e.g., cirrhosis of the liver, alcohol withdrawal, symptoms), relationship problems (e.g., arguments about alcohol with family members), work/school problems (e.g., poor performance, absenteeism resulting from alcohol use), or actual treatment (e.g., detox or rehab, AA meeting attendance). Then, participants were asked for the number of biological relatives [grandfather, grandmother, father, mother, sister(s), and brother(s)], both living and dead, who in the past had or who currently have a significant drinking problem. For the current study, I coded a positive family history of alcoholism if the participant indicated either a paternal or maternal alcohol problem defined using the above description (Gearhardt & Corbin, 2009). I coded all other participants with a negative family history of alcoholism.

Covariates. Participants reported demographics including age, sex, and ethnicity. Participants also self-reported subjective socioeconomic status on the MacArthur Scale (Adler & Stewart, 2007). I selected this socioeconomic status measure because subjective socioeconomic status is more consistently and more strongly related with psychological functioning and health-related factors compared to objective indicators (Adler, Epel, Castellazzo, & Ickovics, 2000). The research assistant measured participant height and weight to calculate BMI. I used all these variables to identify any potential covariates to examine in statistical analyses. Indeed, in my prior longitudinal work I found that controlling for ethnicity and socioeconomic status weakened the negative longitudinal associations between eating and alcohol use (Cummings et al., 2017).

Manipulation check & control variables. After the experimental manipulation, participants self-reported on feelings of fullness, distraction, and reward before the final alcohol cue paradigm [self-reported feelings of reward was amended into the study after it began so only a subset of participants answered (n = 55)]. Participants responded to all items on 5-point Likert scales. At the very end of the study (during the funnel debrief), participants were asked if they believed sweet high-fat food reduced alcohol craving (to test for placebo effects).

Data Analysis. I preregistered my analytic plan via the Open Science Framework and the preregistration can be accessed at https://osf.io/cugw2/ (Cummings, 2017). Initial descriptive examination of all variables of interest indicated that only self-reported alcohol craving at baseline evidenced skew (>1) and kurtosis (>3). I performed natural log transformations for this variable and repeated measures of this variable to correct for this and to keep repeated measures on the same scale. A pairwise deletion approach was used for missing data in all analyses.

I first conducted a manipulation check and tested differences in control variables by experimental condition via One-Way Analysis of Variances (ANOVA) followed up with Least

Significant Difference (LSD) *post hoc* comparisons. I next conducted bivariate correlations between demographic variables and baseline dependent variables to test for potential covariates. Self-reported alcohol craving and Heart Rate at baseline did not correlate with any demographic variable. Galvanic Skin Response at baseline did negatively correlate with age, $r(133) = -.18$, $p = .03$. I therefore tested age as a time invariant covariate in all Galvanic Skin Response analyses. All Galvanic Skin Response results are presented with and without this covariate.

To test the validity of the alcohol-craving paradigm and the craving measures, I conducted growth modeling via multilevel modeling. The dependent variable was alcohol craving; I tested each different measure of alcohol craving separately (i.e., self-report, Galvanic Skin Response, Heart Rate). I did not center these dependent variables because the raw metrics had meaningful zero points. I entered time as the independent variable at Level 1. I centered time such that the intercept of this model represented expected alcohol cravings immediately after the water cue/baseline. The slope of this model represented changes in alcohol cravings from after the water cue/baseline to after the first alcohol cue.

To test my hypotheses, I conducted growth modeling via multilevel modeling. The dependent variable was alcohol craving (with each different measure of alcohol craving tested separately) again, and I did not center the dependent variable. In order to test how the experimental conditions impacted alcohol cravings over time, I first entered time as an independent variable at Level 1. Here, I centered time such that the intercept of this model represented expected alcohol cravings immediately after the first alcohol cue. The slope of this model represented changes in alcohol cravings from after the first to after the second alcohol cue. Alcohol craving in response to the water cue (baseline) was entered as a time invariant covariate.

I conducted Likelihood Ratio Tests between models that did and did not include random effects for the time intercept. The models including random effects for the time intercept fit better as indicated by a significantly smaller *-2LL*, Self-report: Δ*-2LL* = -178.50, $p < .001$; Galvanic Skin Response: Δ*-2LL* = -251.70, $p < .001$; Heart Rate: Δ*-2LL* = -98.00, $p < .001$. This indicated that there were significant differences between individuals on alcohol craving after the first alcohol cue. I also conducted Likelihood Ratio Tests between models that did and did not include random effects for the time slope. The models including random effects for the time slope fit better as indicated by a significantly smaller *-2LL*, Self-report: Δ*-2LL* = -7.70, $p = .006$; Galvanic Skin Response: Δ*-2LL* = -433.80, $p < .001$; Heart Rate: Δ*-2LL* = -85.40, $p < .001$. This indicated that there were significant differences between individuals on the rates of change in alcohol craving from after the first to after the second alcohol cue. The selected growth models thus included random effects for the time intercept and time slope.

To test Hypothesis 1, I independently entered between-subjects factors at Level 2 of the selected growth models. The between-subjects factors included two dummy-coded planned comparisons: (1) food type (Bland food = 0, Sweet high-fat food = 1) and (2) food (No food = 0, Sweet high-fat food = 1). To test Hypothesis 2, I independently entered new between-subjects factors at Level 2. The between-subjects factors included two dummy-coded planned comparisons and their interaction: (1) food type (Bland food = 0, Sweet high-fat food = 1), (2) dose (1 serving = 0, 3 servings = 1), and (3) the cross-product interaction term. If there was a significant interaction, I followed up with tests of simple effects to identify the level of the variable that conferred a significant effect. To test Hypothesis 3, I expanded models from Hypothesis 2 to include each moderator as a covariate at Level 2. This included interaction terms with the dummy-coded planned comparisons. For any significant interaction, I followed up to

identify the level of the variable that conferred a significant effect. An example equation is presented below (for Hypothesis 1, Dummy code 1, self-reported alcohol craving):

Level 1: $\text{Alcohol craving}_{ij} = \beta_{0j} + \beta_1(\text{Time}_j) + r_j$

Level 2: $\beta_{0j} = \gamma_{00} + \gamma_{01}(\text{Food type}_{0j}) + u_{0i}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Foodtype}_{1j}) + u_{1i}$

Given the high number of statistical tests, I corrected for multiple testing using the false discovery rate (Glickman, Rao, & Schultz, 2014). I chose this method because, unlike the Bonferroni adjustment, the false discovery rate correction distinguishes between exploratory and/or data-driven testing versus hypothesis-driven testing. I carried out the following steps to correct for multiple testing using the false discovery rate: (1) list the variables of interest within a set of analyses in rank order of *p*-value significance, (2) multiply the rank order by 0.05 and divide by the number of variables of interest in the set of analyses, and (3) accept the rejection of the null hypothesis if the *p*-value from the analysis is lower than the correction value (Glickman et al., 2014). In multilevel modeling, there are multiple statistical tests that are not based in *a priori* hypotheses (e.g., test for variance in intercepts, test for significant slopes). I only corrected with the false discovery rate for the statistical tests within Hypothesis 1, 2, and 3.

Results

Descriptives. Means, standard deviations, and ranges of the manipulation check and control variables are presented overall and by study condition in Table 2. Means, standard deviations, and ranges of the dependent variables are presented overall and by study condition in Table 3. Descriptives for the moderator variables overall are presented in Table 4, and correlations between moderator variables are presented in Table 5.

Table 2

Study 1 Descriptives of Manipulation Check & Control Variables

		Overall (N = 150)	No food (n = 30)	1 serving bland food (n = 28)	3 servings bland food (n = 32)	1 serving sweet high-fat food (n = 29)	3 servings sweet high-fat food (n = 31)
Reward	Mean	2.11	1.75	1.80	1.43	2.36	3.17
	SD	0.96	0.89	0.80	0.65	0.67	0.72
	Range	1-4	1-3	1-3	1-3	1-3	1-4
Fullness	Mean	2.43	1.47	2.54	3.00	2.07	3.03
	SD	1.08	0.78	0.88	1.02	0.92	0.95
	Range	1-5	1-3	1-5	1-5	1-4	1-4
Distraction	Mean	2.71	2.47	3.11	2.50	3.00	2.55
	SD	1.13	1.20	1.20	1.08	1.07	1.03
	Range	1-5	1-5	1-5	1-5	1-4	1-4

Table 3

Study 1 Descriptives of Dependent Variables

		Overall (N = 150)	No food (n = 30)	1 serving bland food (n = 28)	3 servings bland food (n = 32)	1 serving sweet high-fat food (n = 29)	3 servings sweet high-fat food (n = 31)
Baseline							
Self-report[1]	Mean	11.78	9.13	9.86	8.66	13.62	17.58
	SD	17.82	16.49	17.09	13.87	21.06	19.50
	Range	0.00-80.00	0.00-64.00	0.00-66.00	0.00-60.00	0.00-80.00	0.00-70.00
GSR	Mean	8.83	7.24	8.30	8.95	8.90	10.64
	SD	5.06	5.19	5.53	4.98	5.33	3.95
	Range	0.18-20.00	0.52-20.00	0.18-18.62	0.66-19.57	1.56-18.89	2.71-19.67
HR	Mean	71.84	76.21	68.81	73.04	73.06	67.88
	SD	11.24	10.77	12.26	11.87	8.71	10.75
	Range	51.31-107.48	61.58-97.20	51.31-94.46	52.76-107.48	54.02-87.31	52.93-93.81
Alcohol Cue 1							
Self-report[1]	Mean	48.29	48.37	45.14	45.13	45.38	57.06
	SD	28.92	28.36	28.35	34.16	25.56	27.01
	Range	0.00-100.00	1.00-100.00	0.00-100.00	0.00-100.00	0.00-90.00	0.00-100.00
GSR	Mean	8.40	7.40	7.80	8.64	8.53	9.55
	SD	4.92	4.99	5.48	4.92	4.90	4.36
	Range	0.13-20.00	0.13-18.91	0.46-19.07	0.87-18.58	1.24-16.75	0.93-20.00
HR	Mean	73.74	76.80	71.09	75.02	75.26	70.39
	SD	10.67	9.99	11.57	11.59	8.91	10.15
	Range	50.66-107.74	58.73-96.19	51.60-94.95	54.35-107.74	57.53-92.05	50.66-97.93
Alcohol Cue 2							
Self-report[1]	Mean	39.81	42.67	36.71	40.00	38.24	41.10
	SD	28.82	29.96	28.90	31.63	27.25	27.53
	Range	0.00-100.00	1.00-100.00	0.00-100.00	0.00-92.00	0.00-100.00	0.00-100.00
GSR	Mean	7.45	6.18	6.85	7.24	8.20	8.86
	SD	4.67	4.85	3.92	4.54	4.94	4.94

	Range	0.07-20.00	0.07-15.00	0.58-13.61	0.36-16.15	1.12-19.10	0.86-20.00
HR	Mean	71.30	74.90	67.91	72.89	71.79	68.83
	SD	10.63	9.84	11.33	11.61	9.68	9.63
	Range	51.87-107.45	60.36-99.50	51.87-99.04	52.03-107.45	53.14-89.18	52.69-95.94

Notes: Means, standard deviations, and ranges are presented for the first minute after the cue. [1]Untransformed values presented. Self-report = Self-reported alcohol craving, GSR = Galvanic Skin Response, HR = Heart Rate

Table 4

Study 1 Descriptives of Moderator Variables

	Mean	SD
	%	
BAES Stimulant	7.39	1.71
OPRM1 gene		
AA alleles	64.0%	
AG alleles	28.7%	
GG alleles	7.3%	
Family History of Alcoholism		
Positive	23.1%	
Negative	76.9%	

Note: BAES = Biphasic Alcohol Effects Scale

Table 5

Study 1 Correlations Between Moderator Variables

	BAES Stimulant	G allele of OPRM1 gene	Family History of Alcoholism
BAES Stimulant		-0.10	-0.05
G allele of OPRM1 gene			-0.02
Family History of Alcoholism			

Notes: G allele of OPRM1 gene was dummy coded (0 = No G allele, 1 = G allele). Family History of Alcoholism was dummy coded (0 = Negative, 1 = Positive). BAES = Biphasic Alcohol Effects Scale

Manipulation check & control variables. As expected, there were differences by experimental condition in how rewarded participants felt after the manipulation, $F(4, 50) = 10.567$, $p < .001$, $d = 1.84$. *Post hoc* comparisons confirmed that participants felt more rewarded after eating three servings of sweet high-fat food compared to eating one serving of sweet high-fat food ($p = .01$, 95% CI [0.19, 1.42]), and compared to all other conditions (ps < .001, 95% CIs [>0.74, <2.32]). Also, participants felt more rewarded after eating one serving of sweet high-fat food compared to eating three servings of bland food ($p = .003$, 95% CI [0.34, 1.53]), but only marginally so compared to eating one serving of bland food ($p = .084$, 95% CI [-0.08, 1.21]) or no food ($p = .077$, 95% CI [-0.07, 1.30]).

There were also differences by experimental condition in how full participants felt after the manipulation, $F(4, 145) = 16.02$, $p < .001$, $d = 1.33$. *Post hoc* comparisons indicated that participants felt more full in all food conditions compared to the no food condition (ps < .05, 95%CIs [>-1.07, <-0.13]). Importantly, participants felt equally full after eating three servings of sweet high-fat food compared to three servings of bland food ($p = 0.89$, 95% CI [-0.42, 0.49]), and more full after eating three servings of compared to one serving of any food (ps < .04, 95% CIs [>0.03, <1.43]; although marginal for eating three servings of compared to one serving of bland food, $p = .052$, 95% CI [-.003, 0.93]). Also, participants felt marginally less full after eating one serving of sweet high-fat food compared to one serving of bland food ($p = .056$, 95%CI [-0.95, 0.01]).

In addition, there were marginal differences by experimental condition in how distracted participants felt after the manipulation, $F(4, 145) = 2.19$, $p = .073$, $d = 0.49$. *Post hoc* comparisons indicated that participants felt more distracted after eating one serving of bland food compared to no food ($p = .03$, 95% CI [0.06, 1.22]), three servings of bland food ($p = .04$, 95%

CI [0.04, 1.18]), and three servings of sweet high-fat food (p = .056, 95% CI [-0.02, 1.13]; marginal). Participants also felt marginally more distracted after eating one serving of sweet high-fat food compared to no food (p = .068, 95% CI [-0.04, 1.11]) and three servings of bland food (p = .082, 95% CI [-0.06, 1.06]).

Validity of the alcohol craving paradigm. As expected, alcohol craving increased from baseline to after the first alcohol cue for self-report, β = 1.92, SE_β = 0.11, p < .001, 95% CI [1.70, 2.14], and Heart Rate, β = 0.48, SE_β = 0.07, p < .001, 95% CI [0.34, 0.62]. This indicated that participants did indeed increase in alcohol craving in response to the alcohol-craving paradigm. Alcohol craving then decreased from after the first alcohol cue to after the second alcohol cue for self-report, β = -0.30, SE_β = 0.05, p < .001, 95% CI [-0.40, -0.20], and Heart Rate, β = -0.32, SE_β = 0.09, p < .001, 95% CI [-0.49, -0.15]. These growth patterns are presented in Figures 1 and 2. Unexpectedly, Galvanic Skin Response decreased from baseline to after the first alcohol cue, β = -0.17, SE_β = 0.06, p = .002, 95% CI [-0.28, -0.06]. Then, alcohol craving further decreased from the first alcohol cue to after the second alcohol cue, β = -0.33, SE_β = 0.10, p = .001, 95% CI [-0.52, -0.14]. This growth pattern is presented in Figure 3. For the purpose of this dissertation, I still tested all hypotheses with Galvanic Skin Response. However, the lack of increase from baseline to after the first alcohol cue calls into question if Galvanic Skin Response was a valid measure of alcohol craving in this study.

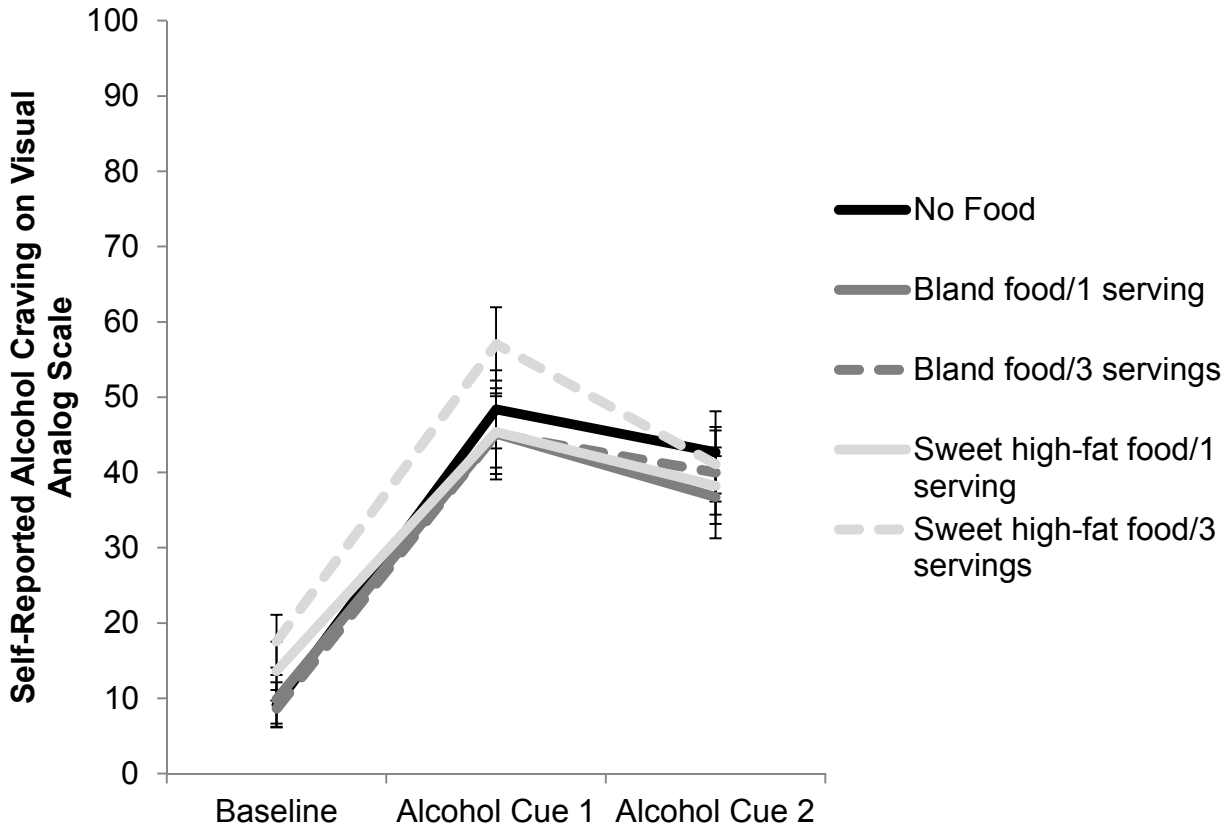


Figure 1. Study 1 Self-reported alcohol craving by experimental condition. *Note:* Vertical bars indicate standard errors.

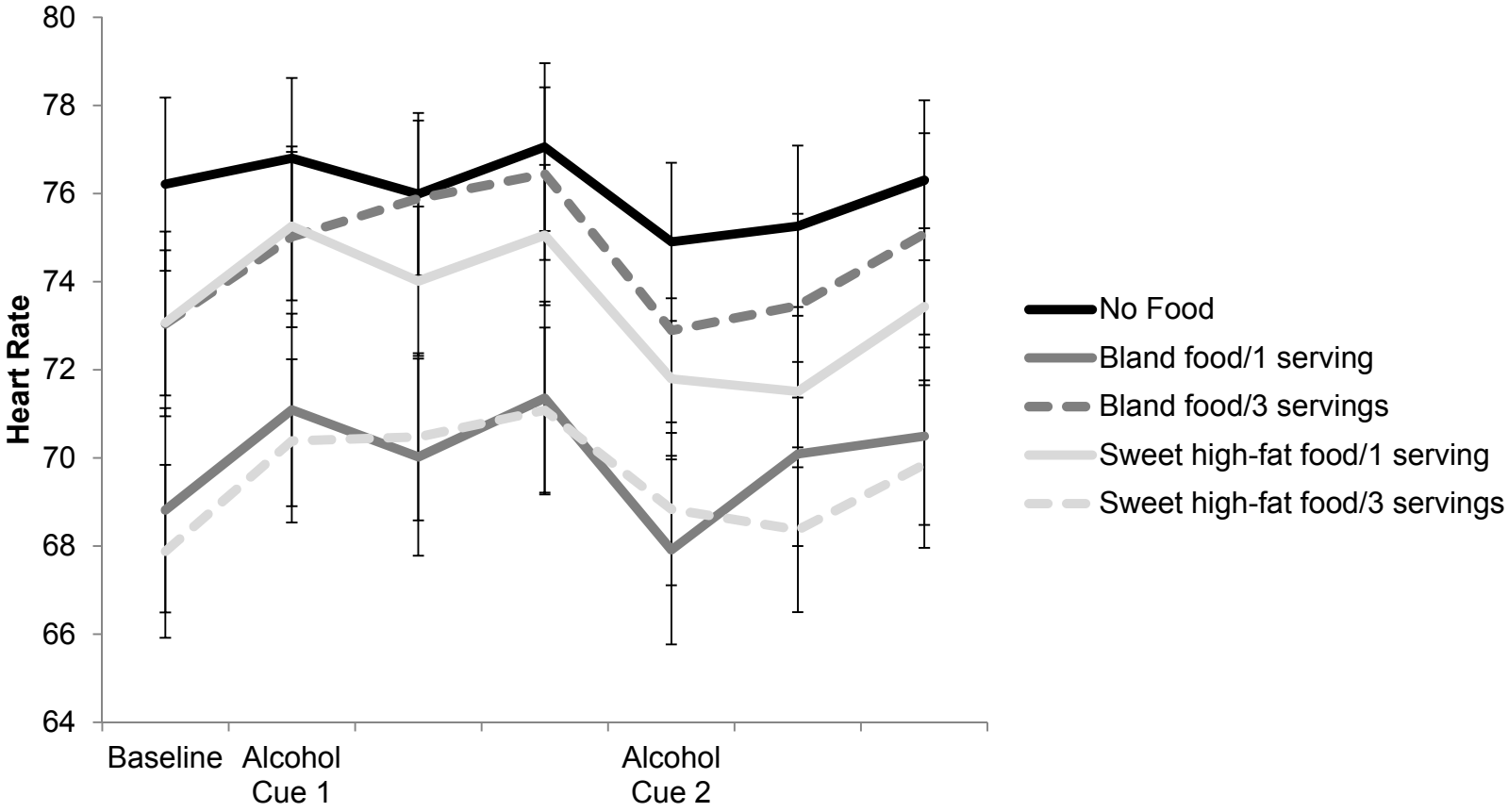


Figure 2. Study 1 Heart Rate by experimental condition. *Note:* Vertical bars indicate standard errors.

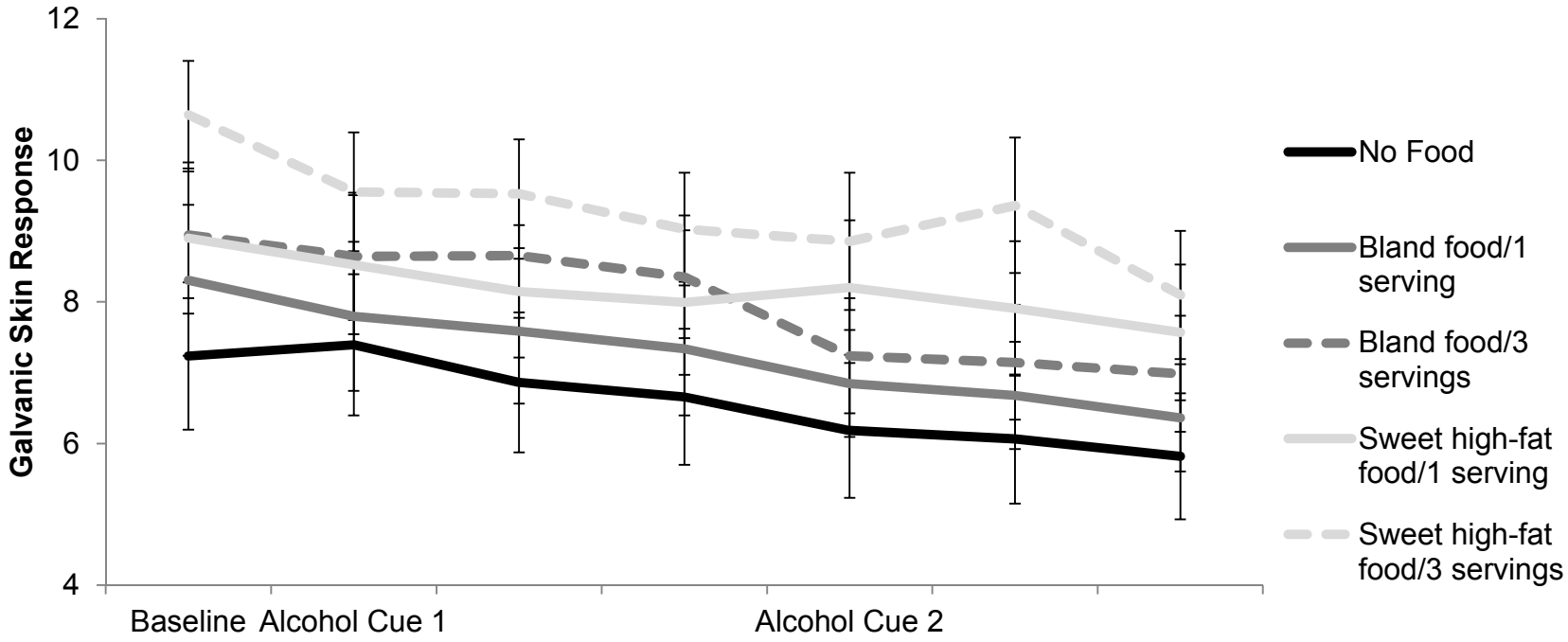


Figure 3. Study 1 Galvanic Skin Response by experimental condition. *Note:* Vertical bars indicate standard errors

Hypothesis 1. Results indicated that—and as can be seen in Figures 1, 2, and 3—there were no significant differences in changes in alcohol craving from after the first to after the second alcohol cue between (1) participants who ate sweet high-fat food versus bland food and (2) participants who ate sweet high-fat food versus no food. These results were consistent across self-report, Galvanic Skin Response, and Heart Rate. Multilevel estimates of fixed effects are presented in Table 6.

Table 6

Study 1 Hypothesis 1 Estimates of Fixed Effects

		γ	SE_γ	p	95% CI	
					Lower	Upper
Self-report	Time*Food Type	-0.14	0.12	.27	-0.39	0.11
	Time*Food	-0.20	0.14	.16	-0.47	0.08
GSR	Time*Food Type	0.07	0.23	.77	-0.39	0.52
		[0.07]	[0.23]	[.77]	[-0.39]	[0.52]
	Time*Food	-0.06	0.24	.82	-0.54	0.43
		[-0.06]	[0.24]	[.82]	[-0.54]	[0.43]
HR	Time*Food Type	-0.13	0.20	.53	-0.52	0.27
	Time*Food	-0.22	0.24	.36	-0.70	0.26

Notes: Bracketed values are estimates controlling for age. Food Type was dummy coded (0 = Bland food, 1 = Sweet high-fat food). Food was dummy coded (0 = No food, 1 = Sweet high-fat food). Self-report = Self-reported alcohol craving, GSR = Galvanic Skin Response, HR = Heart Rate

Hypothesis 2. Results indicated that there were no significant differences in changes in alcohol craving from after the first to after the second alcohol cue between eating sweet high-fat food and bland food by dose. These results were consistent across self-report, Galvanic Skin Response, and Heart Rate. To confirm that there were no significant differences in changes in alcohol craving from after the first to after the second alcohol cue by dose (regardless of food type), I dropped the interaction term from the model for *post hoc* analyses. Results confirmed there were no significant differences in changes in alcohol craving from after the first to after the second alcohol cue by dose (irrespective of food type) for self-report, Galvanic Skin Response, and Heart Rate. Multilevel estimates of fixed effects are presented in Table 7.

Table 7

Study 1 Hypothesis 2 Estimates of Fixed Effects

		γ	SE_γ	p	95% CI	
					Lower	Upper
Self-report	Time*Dose*Food Type	-0.36	0.25	.15	-0.85	0.14
	Time*Dose	-0.02	0.13	.86	-0.27	0.23
GSR	Time*Dose*Food Type	0.20	0.46	.67	-0.72	1.12
		[0.20]	[0.46]	[.67]	[-0.72]	[1.12]
	Time*Dose	-0.05	0.23	.82	-0.51	0.40
		[-0.05]	[0.23]	[.82]	[-0.51]	[0.40]
HR	Time*Dose*Food Type	0.26	0.40	.52	-0.54	1.05
	Time*Dose	0.21	0.20	.29	-0.18	0.61

Notes: Bracketed values are estimates controlling for age. Dose was dummy coded (0 = 1 serving, 1 = 3 servings). Food Type was dummy coded (0 = Bland food, 1 = Sweet high-fat food). Self-report = Self-reported alcohol craving, GSR = Galvanic Skin Response, HR = Heart Rate

Hypothesis 3.

3a. Sensitivity to the stimulating effects of alcohol. Results indicated there was a significant interaction between eating sweet high-fat versus no food and reporting greater sensitivity to the stimulating effects of alcohol on changes in Heart Rate, $\gamma = 0.32$, $SE_\gamma = 0.15$, $p = .041$, 95% CI [0.01, 0.61]. Tests of simple effects indicated that sensitivity to the stimulating effects of alcohol did not influence changes in Heart Rate if individuals ate sweet high-fat food, $\gamma = 0.11$, $SE_\gamma = 0.10$, $p = .30$, 95% CI [-0.10, 0.31]. However, those scoring lower compared to higher in sensitivity to the stimulating effects of alcohol had marginal increases in Heart Rate if they did not eat food, $\gamma = -0.21$, $SE_\gamma = 0.10$, $p = .052$, 95% CI [-0.42, 0.002]. This interaction is presented in Figure 4, and was not found with self-report and Galvanic Skin Response. No other interactive effects emerged between study conditions and sensitivity to the stimulating effects of alcohol on alcohol craving.

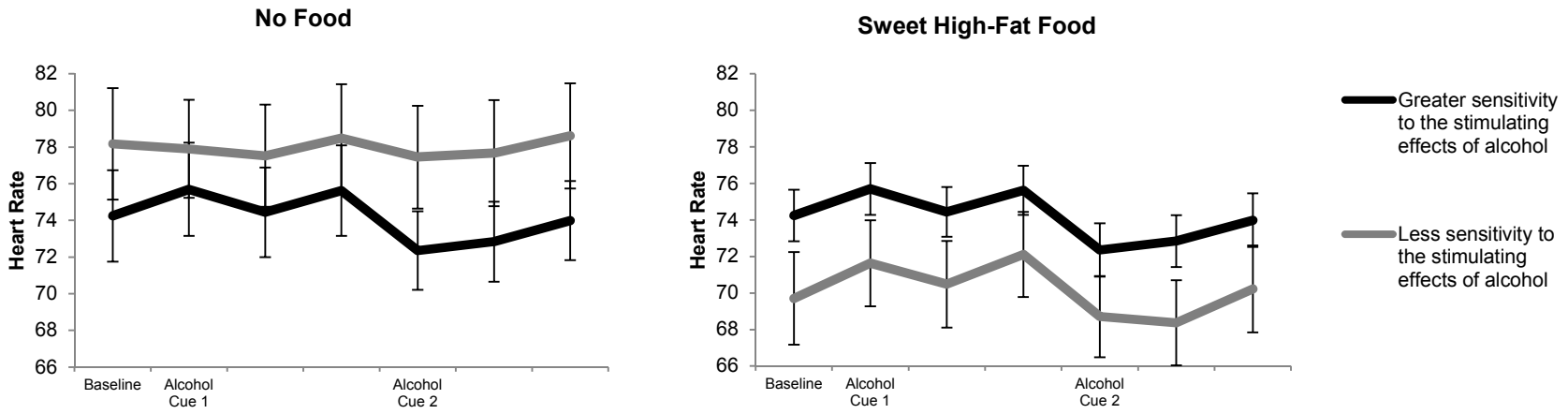


Figure 4. Study 1 Hypothesis 3 Heart rate differs in the no food and sweet high-fat food conditions as a function of sensitivity to the stimulating effects of alcohol. For ease of presentation, means and standard errors are presented based on a median split of sensitivity to the stimulating effects of alcohol score from the Biphasic Alcohol Effect Scale; however, the analyses examined this score as a continuous predictor. The black line indicates greater sensitivity to the stimulating effects of alcohol and the grey line indicates less sensitivity to the stimulating effects of alcohol.

3b. G allele of the OPRM1 Gene. Results indicated there was a significant interaction between the effect of eating sweet high-fat versus no food and the presence of the G allele of the OPRM1 gene on changes in Heart Rate, $\gamma = 1.26$, $SE_\gamma = 0.52$, $p = .017$, 95% CI [0.23, 2.30]. Tests of simple effects indicated those who carry compared to not carry the G allele of the OPRM1 gene had increases in Heart Rate if individuals ate sweet high-fat food, $\gamma = 0.69$, $SE_\gamma = 0.30$, $p = .027$, 95% CI [0.08, 1.29]. However, presence of the G allele of the OPRM1 gene did not influence changes in Heart Rate if individuals ate no food, $\gamma = -0.57$, $SE_\gamma = 0.40$, $p = .16$, 95% CI [-1.39, 0.24]. This interaction is presented in Figure 5, and was not found with self-report and Galvanic Skin Response. No other interactive effects emerged between study conditions and presence of the G allele of the OPRM1 gene on alcohol craving.

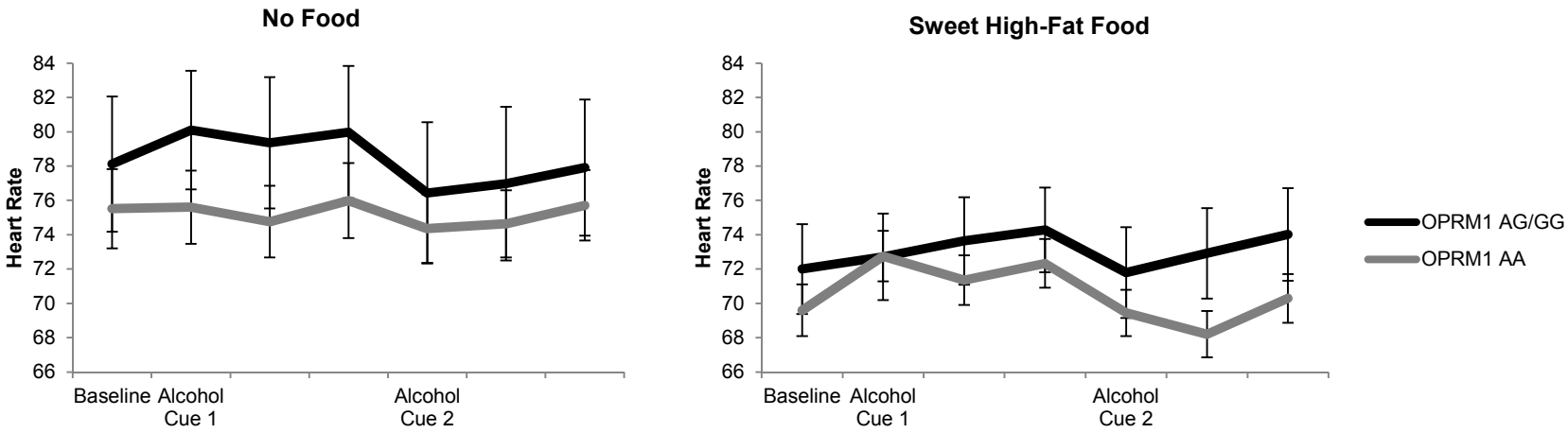


Figure 5. Study 1 Hypothesis 3 Heart rate differs in the no food and sweet high-fat food conditions as a function of carrying the G allele of the OPRM1 Gene. The black line indicates those with the GG or AG alleles and the grey line indicates those with the AA Alleles.

3c. Family History of Alcoholism. Results indicated there was a significant interaction between the effect of eating a large versus small amount of food and positive family history of alcoholism on changes in self-reported alcohol craving, γ = -0.73, SE_γ = 0.30, p = .018, 95% CI [-1.34, -0.13], and Heart Rate, γ = -1.39, SE_γ = 0.47, p = .004, 95% CI [-2.31, -0.46]. Tests of simple effects indicated that those with a positive compared to negative family history of alcoholism had decreases in self-reported alcohol craving if individuals ate a large amount of food, γ = -0.44, SE_γ = 0.21, p = .039, 95% CI [-0.85, -0.02]. However, positive family history of alcoholism did not influence self-reported alcohol craving if individuals ate a small amount of food, γ = 0.29, SE_γ = 0.22, p = .20, 95% CI [-0.16, 0.73]. Tests of simple effects indicated that positive family history of alcoholism did not influence Heart Rate if individuals ate a large amount of food, γ = -0.48, SE_γ = 0.35, p = .17, 95% CI [-1.17, 0.22]. However, those with a positive compared to negative family history of alcoholism had increases in Heart Rate if individuals ate a small amount of food, γ = 0.91, SE_γ = 0.31, p = .005, 95% CI [0.29, 1.54]. These findings are presented in Figures 6 and 7, respectively, and were not found with Galvanic Skin Response. No other interactive effects emerged between study conditions and positive family history of alcoholism on alcohol craving.

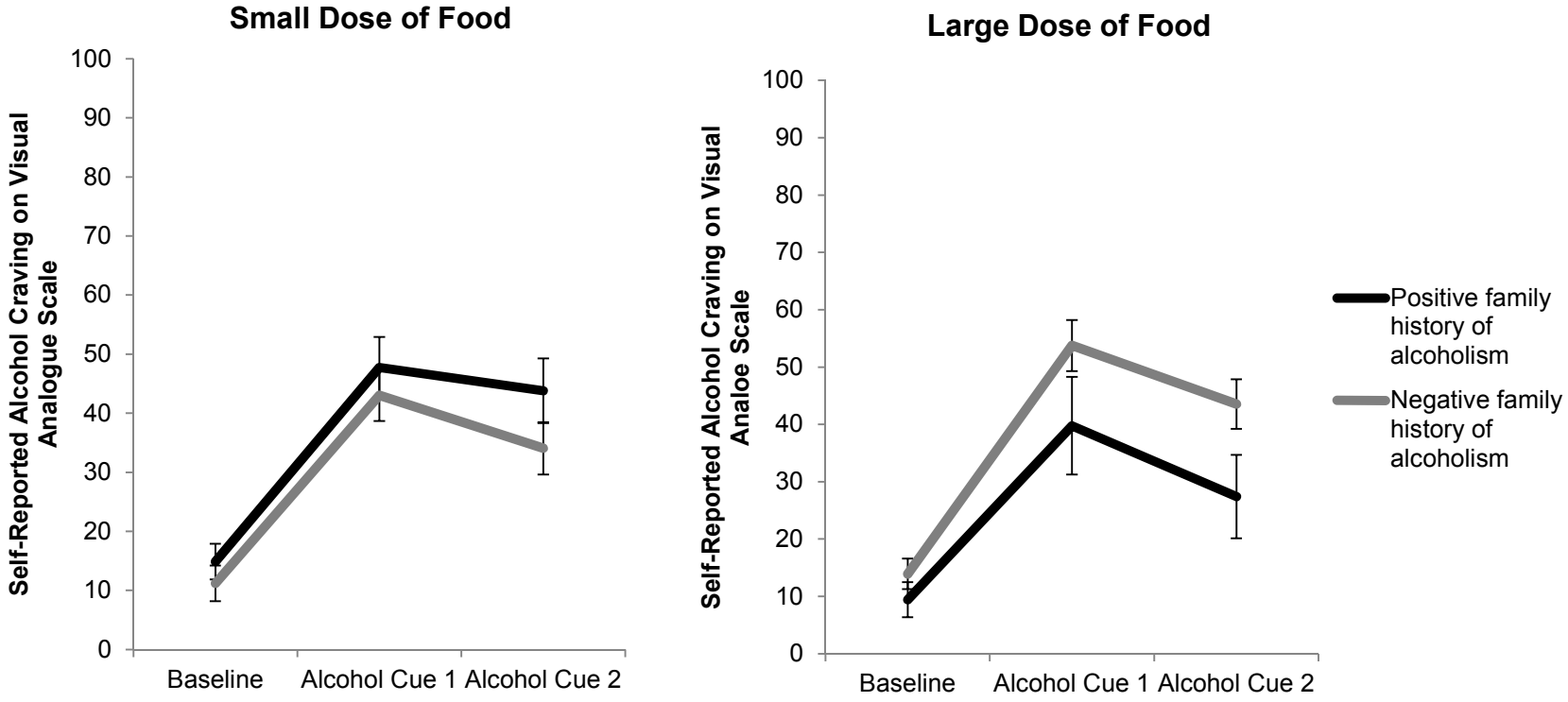


Figure 6. Study 1 Hypothesis 3 Self-reported alcohol craving differs in the small dose and high dose of food conditions as a function of family history of alcoholism. The black line indicates those with a positive family history of alcoholism and the grey line indicates those with a negative family history of alcoholism.

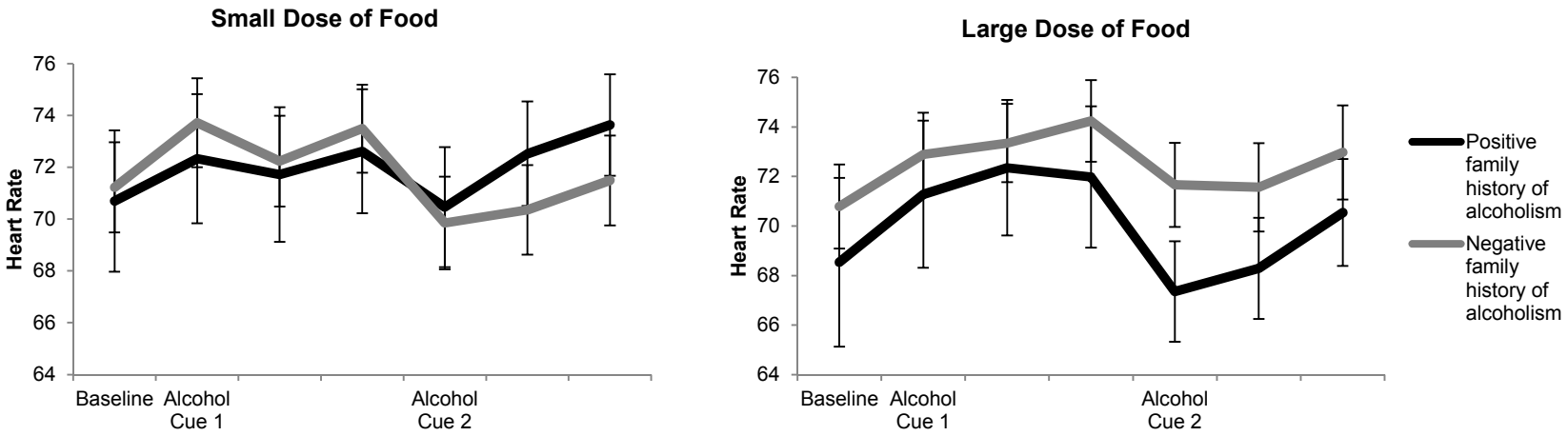


Figure 7. Study 1 Hypothesis 3 Heart Rate differs in the small dose and high dose of food conditions as a function of family history of alcoholism. The black line indicates those with a positive family history of alcoholism and the grey line indicates those with a negative family history of alcoholism.

False Discovery Rate. Using the false discovery rate to correct for multiple testing, only one finding from Study 1 Results would be accepted as significant. This is presented in Table 8.

Table 8

Study 1 False Discovery Rate

Rank of *p*-value	Hypothesis of Interest	Model	*p*-value from Analysis	Correction	Accept as Significant?
Hypothesis 1					
1	Food	Self-report	.16	.0125	No
2	Food type	Self-report	.27	.025	No
3	Food	HR	.36	.0375	No
4	Food type	HR	.53	.05	No
Hypothesis 2					
1	Interaction	Self-report	.15	.0125	No
2	Dose	HR	.29	.025	No
3	Interaction	HR	.52	.0375	No
4	Dose	Self-report	.86	.05	No
Hypothesis 3					
3a. Biphasic Alcohol Effects Scale (BAES)					
1	BAES x Food	HR	.041	.00625	No
2	BAES x Food type	Self-report	.25	.0125	No
3	BAES x Interaction	Self-report	.29	.01875	No
4	BAES x Food type	HR	.52	.025	No
5	BAES x Dose	HR	.69	.03125	No
6	BAES x Interaction	HR	.73	.0375	No
7	BAES x Food	Self-report	.80	.04375	No
8	BAES x Dose	Self-report	.96	.05	No
3b. G Allele of OPRM1 Gene					
1	G Allele x Food	HR	.017	.00625	No
2	G Allele x Food type	HR	.45	.0125	No
3	G Allele x Interaction	Self-report	.65	.01875	No
4	G Allele x Dose	HR	.68	.025	No
5	G Allele x Dose	Self-report	.84	.03125	No
6	G Allele x Food type	Self-report	.84	.0375	No
7	G Allele x Food	Self-report	.86	.04375	No
8	G Allele x Interaction	HR	.86	.05	No
3c. Family History of Alcoholism (FHA)					
1	FHA x Dose	HR	.004	.00625	Yes
2	FHA x Dose	Self-report	.018	.0125	No
3	FHA x Interaction	HR	.15	.01875	No
4	FHA x Food type	HR	.35	.025	No
5	FHA x Food	HR	.43	.03125	No
6	FHA x Food	Self-report	.44	.0375	No

7	FHA x Food type	Self-report	.60	.04375	No
8	FHA x Interaction	Self-report	.73	.05	No

Exploratory Analyses. In addition to the planned analyses for Study 1 of this dissertation, I preregistered multiple *a priori* exploratory analyses (which were discussed at my preliminary exam). First, results indicated that craving responsiveness to the alcohol cue did not moderate effects. These results were consistent across self-report, Heart Rate, and Galvanic Skin Response. I calculated craving responsiveness by computing differences between alcohol craving at baseline and Time 1 for each individual, and I dummy coded based on a median split of these difference scores (< Median = 0 = Non-Craver, ≥ Median = 1 = Craver; Higley et al., 2011). Second, results indicated that type of alcohol that was used as the alcohol cue did moderate effects for Heart Rate and Galvanic Skin Response but did not moderate effects for self-reported alcohol craving. Specifically, I dummy coded type of alcohol based on whether or not the alcohol was heavy in sugar (0 = Beer, 1 = Liquor/Wine/Champagne). Results indicated there was a marginally significant interaction between the effect of eating sweet high-fat versus no food and dummy coded type of alcohol on changes in Heart Rate, γ = -1.03, SE_γ = 0.52, p = .051, 95% CI [-2.06, 0.004], and a significant interaction for changes in Galvanic Skin Response, γ = 1.12, SE_γ = 0.53, p = .039, 95% CI [0.06, 2.18]. For Heart Rate, tests of simple effects indicated that smelling alcohol heavy in sugar increased Heart Rate if individuals ate no food, γ = 0.87, SE_γ = 0.38, p = .029, 95% CI [0.10, 1.64], but did not change Heart Rate if individuals ate sweet high-fat food, γ = -0.16, SE_γ = 0.30, p = .60, 95% CI [-0.75, 0.44]. For Galvanic Skin Response, tests of simple effects yielded no significant results (ps = .14-.37).

In addition to these preregistered exploratory analyses, I ran *post hoc* exploratory analyses to test if self-reported feelings of reward, distraction, and fullness reported after the manipulations predicted changes in alcohol craving across conditions. I also ran *post hoc* exploratory analyses to test if reporting the belief (at the end of the study) that sweet high-fat

foods can reduce alcohol cravings predicted changes in alcohol craving across conditions. Results indicated that feelings of reward did not predict changes in self-reported alcohol craving across conditions, γ = -0.15, SE_γ = 0.10, p = .14, 95% CI [-0.35, 0.05]. However, feelings of distraction, γ = -0.10, SE_γ = 0.05, p = .038, 95% CI [-0.19, -0.005], and fullness, γ = -0.11, SE_γ = 0.05, p = .028, 95% CI [-0.20, -0.01], both predicted decreases in self-reported alcohol craving across conditions. Also, reporting a belief that sweet high-fat foods can reduce alcohol cravings (at the end of the study) predicted decreases in self-reported alcohol craving across conditions, γ = -0.46, SE_γ = 0.10, p < .001, 95% CI [-0.66, -0.27]. These effects were not observed for Heart Rate and Galvanic Skin Response.

Discussion

Many drinkers believe that eating sweet high-fat food will combat their alcohol cravings. However—in the first experiment testing this belief—eating sweet high-fat food was no more effective at reducing alcohol cravings than eating calorie-equivalent bland food or watching a neutral video. This suggests that eating sweet high-fat food is not a viable strategy to reduce drinking. Confidence in this result is bolstered by many study strengths. First, participants idiosyncratically selected sweet high-fat foods such that each participant had a sweet high-fat food they personally considered rewarding. Participants similarly selected alcohol they personally considered rewarding to induce their cravings. This provided ecological validity. Second, there were multiple measurement methods for alcohol cravings including self-report and physiological indices. This allowed for understanding of what happens psychologically and physiologically in response to eating foods to alleviate alcohol cravings. Third, the study was well powered to capture a medium effect size, which was expected based on prior studies examining cue reactivity to drug-related stimuli (Carter & Tiffany, 1999).

The current experiment's results challenge the food-alcohol competition hypothesis, and challenge prior rodent studies and cross-sectional and longitudinal research in humans. Specifically, prior rodent studies show that when rats are offered or fed alternatives to alcohol such as saccharin or fat they will reduce their alcohol use. Yet, in all but one of the rodent studies that found this effect, the authors suggest that it was a large dose of food or a binge-eating pattern that was needed to reduce alcohol use. It is hard to make a direct comparison in dose between rodent studies and the current research in humans, but perhaps the two findings are inconsistent because participants ate up to only 450 calories of sweet high-fat food in the current research. Future work could test if eating more than 450 calories of sweet high-fat food reduces alcohol cravings. Although from a clinical and public health standpoint, recommending that someone eat greater than 450 calories of sweet high-fat food to reduce the urge to drink after each occurrence of alcohol cravings in their everyday life could increase the risk for metabolic health issues and weight gain.

There are a number of cross-sectional studies and one longitudinal study in humans that find that individuals who eat more food drink less alcohol. However, even well-controlled cross-sectional and longitudinal studies do not perfectly rule out confounds. Given that my experiment tested the causal effect of sweet high-fat food on alcohol cravings, it is possible that a third variable or reverse causation explains prior research findings in humans. Indeed, there are a number of potential third variables that might explain the inverse association between eating and alcohol use. For example, individuals who eat more food may drink less alcohol because the calories from food physiologically displace calories from alcohol use (i.e., "fill up" an individual and reduce motivation to drink more calories); indeed, feeling full predicted decreases in alcohol

craving in the current study. Alternatively, individuals who eat more food may spend more money on food and have less money to spend on alcohol.

In regards to reverse causation, there is a substantial literature suggesting bidirectional associations between eating and alcohol use during restricted intake, and that restricting alcohol use can selectively increase intake of sweet, starchy, and salty/spicy foods (Cummings & Tomiyama, 2018). Therefore it is also possible that inverse associations appear in prior literature not because greater eating reduces alcohol use but because less alcohol use increases eating. Certainly, the Alcoholics Anonymous testimonials were given in the context of alcohol abstinence (Alcoholics Anonymous, 1975). Thus, it is plausible that those who quit drinking ate more sweets—and while that may have been a pleasurable and rewarding experience—greater intake of sweets was actually not serving to reduce their alcohol cravings or alcohol use.

Nevertheless, although across the sample eating sweet high-fat food did not yield distinct effects on alcohol cravings, I also tested the possibility that this strategy might only work for certain individuals. Moderated effects, however, were contrary (and in some cases irrelevant) to hypotheses, and most of these effects would not be accepted as significant using the false discovery rate to correct for multiple testing. To begin, results indicated that those who scored lower compared to higher in sensitivity to the stimulating effects of alcohol had increases in Heart Rate in response to the alcohol cues if they did not eat food, but not if they ate sweet high-fat food. This suggests that watching a neutral video may increase Heart Rate reactivity to alcohol cues for those scoring lower in sensitivity to the stimulating effects of alcohol, yet it should be noted this effect would not be accepted as significant according to the false discovery rate. Since sensitivity to the stimulating effects of alcohol did not moderate any other effects, overall no finding suggested that eating sweet high-fat food reduces alcohol cravings specifically

for those with higher sensitivity to the stimulating effects of alcohol. Notably, in this experiment compared to prior work Biphasic Alcohol Effects Scale was measured outside of the lab context versus in the lab after alcohol administration (Ray et al., 2010). This may have limited the sensitivity of the measure.

Next, results indicated that those who carry compared to not carry the G allele of the OPRM1 gene had increases in Heart Rate in response to the alcohol cues if individuals ate sweet high-fat food, but not if they ate no food. Unexpectedly, this suggests that eating sweet high-fat food may backfire and actually increase alcohol cravings for those who carry the G allele of the OPRM1 gene. The association between eating sweet high-fat food and carrying the G allele of the OPRM1 gene is critical to understand because carrying the G allele of the OPRM1 gene confers risk for increased alcohol use (Ray & Hutchison, 2004; Ray et al., 2013). Thus, not only did eating sweet high-fat food fail to reduce alcohol cravings any more than eating calorie-equivalent food or watching a neutral video, but it additionally may have caused some harm for those who were at greater risk for alcohol use disorder. However, this result should be interpreted as preliminary because the effect was not accepted as significant according to the false discovery rate.

On the other hand, results indicated that for those who had a parental family history of alcoholism, eating a large dose of food—regardless of food type (i.e., 450 calories of sweet high-fat or bland food)—reduced self-reports of alcohol craving. These self-report findings synergized with findings from physiological indices of alcohol craving; for those who had a family history of alcoholism, eating a small dose of food—regardless of food type—increased Heart Rate but eating a large dose of any food did not. Taken together, these findings suggest that while eating 150 calories of any food may stimulate drinking for those with a family history of alcoholism,

eating 450 calories of any food may reduce alcohol cravings. Although only one of these effects would be accepted as significant according to the false discovery rate, these findings also corroborate with prior work that found that, for individuals with a family history of alcoholism, having a greater BMI was associated with a lower frequency of alcohol use and a lower amount of alcohol consumed at a drinking occasion (Gearhardt et al., 2009). Thus, parental family history of alcoholism may be a relevant individual difference factor to consider when understanding the association between eating and alcohol use, and when making clinical recommendations.

However, there are two important points to consider in regards to the family history of alcoholism findings. First, eating 450 calories of any food type interacted with parental family history of alcoholism to produce these effects. This suggests a potential intervention strategy without health drawbacks; that is, those with a parental family history of alcoholism might consider eating a calorie-dense, nutritious snack or a meal (one that could be healthy such as whole grain bread, rice, etc.) to regulate their alcohol cravings. Second, parental family history of alcoholism was not correlated with having the G allele of the OPRM1 gene nor correlated with reporting sensitivity to the stimulating effects of alcohol. It seems then that parental family history of alcoholism reflects a unique biological vulnerability (potentially related to the neural reward system's functioning) that is relevant to eating, alcohol use, and the intersection of these behaviors. Future research on eating and alcohol use should continue measuring family history of alcoholism as a potential moderator, and also identify if this risk factor is associated with differential mesolimbic dopamine pathway function after eating food. For example, does family history of alcoholism associate with greater levels of striatal dopamine release after eating?

The current results should be interpreted in light of study limitations. First, this study did not employ brain imaging methods, which is critical because it was hypothesized that food may reduce alcohol cravings via mesolimbic dopamine pathway blocking. Although the current results overall suggest that eating does not reduce alcohol cravings, brain imaging would have provided insight into what was happening at the neural level. Second, this study was conducted in the short term (i.e., the eating manipulation and alcohol craving paradigm took a total of about 25 minutes). It is possible that longer, repeated eating behavior manipulations might produce different effects on drinking patterns. Third, this study measured alcohol cravings rather than actual alcohol use in response to eating behavior. It is thus possible that eating might differentially impact actual alcohol use, yet responses to alcohol craving paradigms in the lab and self-reports of alcohol cravings robustly predict alcohol use in the real world (Flannery et al., 2003; Higley et al., 2011; King et al., 2014; Rohsenow et al., 1994). Fourth, this study tested effects in a sample of non-dependent heavy drinking adults not seeking treatment. Different effects may be observed in samples with alcohol use disorder, in samples seeking treatment, or in samples practicing alcohol abstinence. Indeed, research shows that when individuals decrease drinking or practice abstinence from alcohol they often increase eating (Cummings et al., 2018), and research has highlighted important differences between treatment and non-treatment seekers with alcohol use disorder (Ray et al., 2017; Rohn et al., 2017). Nevertheless, this experiment provides strong evidence that eating sweet high-fat food does not reduce alcohol cravings any more than distracting oneself. And critically, these results suggest that Alcoholics Anonymous should not be recommending this strategy to those who are trying to quit drinking. Even in the current sample, 44% of participants believed that eating sweets could reduce alcohol cravings, which demonstrates just how widespread this inaccurate belief may be.

Study 2

Literature Review

Rewards & the mesolimbic dopamine pathway. In addition to eating and alcohol use, when individuals engage in a number of alternative reward-related behaviors, their neural reward systems react. Unlike with eating and alcohol use, however, Positron Emission Tomography scans in humans have not confirmed that all of these behaviors stimulate neurons in the VTA and the SN to release dopamine to the NAc and DSTR. Nonetheless, other human brain imaging research confirms that the ventral striatum (where the NAc is located) activates in response to a number of alternative reward-related behaviors as seen in Table 9 below.

Table 9

Study 2 Literature Review of Ventral Striatum Activation in Reward-Related Behaviors

Reward-related behavior	Citation(s)
Actively or passively listening to music	Blood & Zatorre, 2001; Brown, Martinez, & Parsons, 2004; Salimpoor, Benovoy, Larcher, Dagher, & Zatorre, 2011
Affirming values	Dutcher et al., 2016
Cooperating with others to achieve the same goal	Rilling et al., 2002
Disclosing self-relevant information	Tamir & Mitchell, 2012
Donating money to a charity	Harbaugh, Mayr, & Burghart, 2007; Moll et al., 2006
Earning more money compared to another while completing the same task	Fliessbach et al., 2007
Exercising	MacRae, Spirduso, Cartee, Farrar, & Wilcox, 1987[1]
Experiencing novelty	Guitart-Masip, Bunzeck, Stephan, Dolan, & Düzel, 2010
Expressing compassion to someone who was suffering	Kim et al., 2009
Forming and maintaining a monogamous relationship	Aragona et al., 2006[2]
Obtaining a good reputation from peers	Korn, Prehn, Park, Walter, & Heekeren, 2012
Obtaining good feedback from someone you like	Hughes, Zaki, & Ambady, 2017
Playing a video game	Koepp et al., 1998
Reading a funny cartoon	Mobbs, Greicius, Abdel-Azim, Menon, & Reiss, 2003
Receiving a lot of "likes" for a post on social media	Sherman, Payton, Hernandez, Greenfield, & Dapretto, 2016
Receiving gaze from an attractive person	Kampe, Frith, Dolan, & Frith, 2001

Seeing a person who has a beautiful face	Aharon et al., 2001
Selecting the correct response to a difficult cognitive task	Satterthwaite et al., 2012
Viewing sexual stimuli	Hamann, Herman, Nolan, & Wallen, 2004
Watching someone similar to you win money	Mobbs et al., 2009
Winning money	Zink, Pagnoni, Martin-Skurski, Chappelow, & Berns, 2004

Notes: [1]Dopamine receptor binding observed in rodents. [2]Dopamine receptor binding observed in voles.

Addiction transfer & reward deficiency syndrome. Given that engaging in many reward-related behaviors activates the neural reward system, Blum et al. (2011) proposed the construct "addiction transfer" or that individuals may adopt a new behavior in exchange for another behavior (pg. 1). This addiction transfer may occur specifically with individuals who have a biological vulnerability related to the neural reward system's functioning. For these individuals, reward-related behavior helps to increase neural reward activity. When individuals quit one reward-related behavior (which may characterize their addiction), they may need another reward-related behavior to keep the neural reward activity elevated. Blum, Cull, Braverman, & Comings (1996) labeled this biological vulnerability related to the neural reward system's functioning the "reward deficiency syndrome" (pg. 132).

Reward competition. Extending from this prior work and my work testing the food-alcohol competition hypothesis, I proposed a domain-general, reward competition hypothesis. I proposed that engaging in any reward-related behavior could stimulate, occupy, and block the mesolimbic dopamine pathway, reducing the likelihood of another reward-related behavior. This domain-general hypothesis may explain competition between multiple types of reward-related behavior. Below I summarize support for this domain-general hypothesis from anecdotal claims, rodent models, and cross-sectional and longitudinal data.

Anecdotal claims. Clinicians treating patients with addictions describe the phenomenon of individuals using one reward-related behavior to replace another reward-related behavior, and how this may have relevance for health. For instance, Blum et al. (2011) provides a case report of an individual who used exercise to reduce the likelihood of overeating: "He has transferred his food addiction to exercise. He runs jogs and exercises religiously five times per week. He has already run 2 half marathons and plans to run a full marathon (26 miles) in a few months. This is

an example of a positive transfer addiction" (pg. 7). Likewise, Glasner-Edwards (2015) states that, "People with the most success in staying sober tend to get involved in a range of pleasurable activities and do them frequently. These activities can replace the time and energy that they had been spending on addictive behaviors, enabling them to experience pleasure without the devastating consequences of alcohol or drug use. Just like the rewarding feelings that follow the use of drugs or alcohol in the early stages lead to forming a damaging habit, rewarding healthy behaviors can establish positive habits" (quote in Sullivan, 2015). Although these clinicians provide compelling anecdotal claims and suggestions, the effectiveness of these strategies remains empirically understudied.

Rodent models. The conditioned place preference paradigm is a protocol where researchers create two distinct contexts (e.g., a red box and a black box) in which an animal spends equal time (Bardo & Bevins, 2000). Only one context provides access to a stimulus (e.g., cocaine in the red box but nothing in the black box). After the conditioning occurs, the animal will choose between contexts in the absence of the stimulus. If the animal chooses the context that was paired with the stimulus (e.g., the red box), researchers conclude that the stimulus (e.g., cocaine) is rewarding.

Relevant to a reward competition hypothesis, researchers have adapted the conditioned place preference paradigm in rodent models to test if the introduction of another stimulus could change a rodent's preference for the original stimulus. That is, after the first conditioning occurs, the rodent would additionally be conditioned to a different stimulus in the other context (e.g., cocaine in the red box but food in the black box). After the first and second conditionings occur, the animal is given the opportunity to choose between contexts in the absence of both stimuli.

Results from the latter studies indicate that certain stimuli may change rodent preferences for other stimuli. For instance, when one context was paired with access to 10mg/kg injection of cocaine but the other context was paired with access to three of a rodent's pups, early (+8 days) postpartum rodents chose the context paired with three of their pups (Mattson, Williams, Rosenblatt, & Morrell, 2001). Also, when one context was paired with access to 7.5mg/kg of cocaine but the other context was paired with access to a novel object, rodents chose the context paired with a novel object (Reichel & Bevins, 2008). This effect was replicated wherein rodents preferred contexts paired with a novel object versus contexts paired with up to 20mg/kg of cocaine but not 30mg/kg of cocaine (Reichel & Bevins, 2011). In sum, these studies suggest that alternative rewards (mother-pup interactions and novel objects) could offset reward-related behavior (cocaine use) up to a specific dose.

Cross-sectional and longitudinal data. In human research, a few studies have tested cross-sectional and longitudinal associations among reward-related behaviors. First, Carr and Epstein (2017) tested associations between BMI and engagement in cognitive-enriching and social activities in a sample of 276 adults (Mean age = 36.4 years). Results indicated that adults who engaged in more cognitive-enriching and social activities had lower BMIs. Moreover, participants in this study made hypothetical choices between receiving a preferred food item or engaging in their favorite cognitive-enriching/social activity; these hypothetical choices were made in the context of different mouse click requirements for the food choice. That is, participants would first choose between 40 clicks for food or 80 clicks for the alternative, and then the clicks for food would progressively increase from 40 to 650 (over 18 increments) while the clicks for the alternative would remain constant. Results indicated that the "breakpoint," or the number of clicks for food at which participants chose the alternative instead, was lowest

when participants chose between their favorite food item and their favorite social activity. From these results, the authors concluded, "alternatives to food can reduce the motivation to eat" (p. 4). In particular, this study suggests that rewarding social activities may compete with eating.

Another study of 41 adolescents (Mean age = 16.30 years at baseline) suggests that neural sensitivity to eudaimonic rewards—defined as prosocial behavior such as donating money to those in need—may offset neural sensitivity to hedonic rewards—defined as self-focused behavior such as winning money (Telzer, Fuligni, & Galvan, 2015). Specifically, adolescents completed a fMRI session that included a family assistance task—wherein they donated money under various conditions (e.g., donated to family at a cost to themself)—and a risk-taking task—wherein they chose when to stop pumping a balloon, and larger balloons resulted in greater money but greater risk of balloon popping. Results indicated that there was a negative association between ventral striatum response during the family assistance task (contrast between making a donation to the family that involves self-sacrifice and pure cash gains for oneself) and ventral striatum response during the risk-taking task (parameter estimates of signal intensity during increasing risky decisions). That is, adolescents with higher neural sensitivity to eudaimonic rewards had lower neural sensitivity to hedonic rewards, and vice versa.

At the baseline assessment and a one-year follow-up, the adolescents also completed the Youth Self-Report form of the Child Behavior Checklist, which has items on internalizing and externalizing symptoms. Results indicated that adolescents with the higher ventral striatum response during the family assistance task decreased in externalizing symptoms such as associating with deviant peers, drinking alcohol without parental approval, or using drugs from baseline to one-year follow-up (Telzer, Fuligni, Lieberman, & Galván, 2013). This suggests that sensitivity to one type of reward may offset other reward-related behaviors over time.

Weaknesses of current evidence for a reward competition hypothesis. Despite the anecdotal, rodent, cross-sectional, and longitudinal support for the reward competition hypothesis, there are limited studies on the topic and many weaknesses. First, it is not clear which reward-related behaviors may potentially compete with one another. Anecdotal claims refer to a variety of pleasurable activities (e.g., exercising, playing sports, planning parties, eating chocolate) competing with eating and drug use; rodent models specifically test the ability of novelty and social interactions to compete with drugs; and cross-sectional and longitudinal studies in humans test the ability of social activities, cognitively-enriching activities, and eudaimonic reward-related behavior (donating money) in competing with eating and drug use.

Another weakness of the current evidence for the reward competition hypothesis is the lack of tested mediators and moderators. Similar to Study 1, I hypothesize that mesolimbic dopamine pathway competition is the mechanism responsible for any reward competition but this has not directly been tested. Reward-related behaviors could evidence inverse associations simply because of time trade-offs; for example, if someone spends their evenings exercising those same hours cannot simultaneously be spent drinking alcohol. Furthermore, similar to Study 1, different individuals may have different susceptibility to reward competition if mesolimbic dopamine pathway competition is the mechanism responsible for the effect (e.g., only those with reward deficiency syndrome experience reward competition). A final weakness is that (unlike with the food-alcohol competition hypothesis) it is not clear to whom reward competition may be relevant for health behavior change. Anecdotal claims refer to the benefits of reward-related behavior for adults who have sought treatment for addiction but cross-sectional and longitudinal data test associations in generally healthy adolescents and adults.

Study 2 contributions. I designed Study 2 in direct response to the aforementioned weaknesses. First, Study 2 was an ambulatory electronic daily diary study of the effect of reward-related behavior on eating sweet high-fat foods and alcohol use. This is a significant contribution because the study identified an array of reward-related behaviors individuals engaged in everyday life and tested which of these rewards may compete with health-compromising behaviors. Second, Study 2 measured and tested for a potential moderator of any observed effect. The potential moderator is an endophenotypic marker associated with a biological vulnerability related to the neural reward system's functioning, the G allele of the OPRM1 gene (Ray & Hutchison, 2004). This is a significant contribution because, as mentioned in Study 1, if this variable does moderate the hypothesized effect, it further builds support for (or falsifies) mesolimbic dopamine pathway competition as a mechanism.

Third, Study 2 measured and tested for a potential mediator of any observed effect (self-reported feelings of reward) and used time-lagged analysis to eliminate time as a third variable. This is a significant contribution because it is a more conservative test of associations between reward-related behavior and health-compromising behavior. Fourth, Study 2 recruited a young adult sample. This advances from prior work and is a significant contribution because it captured a unique developmental stage. Specifically, entry into college results in increased access to food and alcohol, increased autonomy in behavior, changes in living environments, and refined psychological identities; these factors collectively create a time period wherein health-compromising behaviors—especially eating and alcohol use—may be more amenable to change compared to other time periods in adulthood (Borsari, Murphy, & Barnett, 2009; Nelson, Story, Larson, Neumark-Sztainer, & Lytle, 2008).

Health significance. One drawback of eating sweet high-fat foods as a strategy to reduce alcohol use (Study 1) is that eating sweet high-fat foods may have negative consequences too. Indeed, eating sweet high-fat foods might increase addictive-like eating behavior (Schulte et al., 2015) and repeatedly eating sweet high-fat foods may increase cancer risk and metabolic dysregulation (Monteiro, Levy, Claro, de Castro, & Cannon, 2011; Moubarac et al., 2012). Research identifying how other, non-consumptive reward-related behaviors—particularly social and eudaimonic reward-related behavior—might cause changes in eating or alcohol use may uncover reward-related behaviors that offset health-compromising behaviors without creating additional negative consequences. Although results from this research are preliminary, results may have broader implications for how to approach health behavior modification. If, for example, increased social and eudaimonic reward-related behavior can offset health-compromising behaviors, it may be important for interventions to encourage individuals to seek out alternative pleasurable activities. Identifying a moderator would provide insight on the individuals that would be most fit for these kinds of interventions, which (as mentioned in Study 1) is congruent with the precision medicine initiative (Collins & Varmus, 2015).

Aims & Hypotheses

Specific Aim 1: To examine reward-related eating and alcohol use one hour after participation in other reward-related behavior (within-subjects). I hypothesized that one hour after engaging in other reward-related behavior individuals would eat less sweet high-fat foods, palatable foods, and fast foods and drink less sugary drinks and alcohol.

Specific Aim 2: To test a between-subjects moderator. I hypothesized that reward-related eating or alcohol use, following engagement in other reward-related behavior, would

diminish most strongly for individuals who carry the G allele of the OPRM1 gene (see Study 1 Aims & Hypotheses for justification of this moderator).

Specific Aim 3: To test the mediating role of feelings of reward. I hypothesized that increased feelings of reward (in the same hour as engagement in other reward-related behavior) would mediate any association between engagement in other reward-related behavior and reward-related eating or alcohol use one hour later.

Methods

Study Design. I preregistered study plans via the Open Science Framework at https://osf.io/wn69h/ (Cummings, 2017). The study was an ambulatory electronic diary study in which participants reported on their reward-related behavior for 15-hours that they were awake each day over a 4-day period. I selected an hourly sampling density because it is not clear how long neural reward activity persists in response to reward. However, research on emotion suggests that emotions persist as short as a few seconds to as long as several hours (Verduyn, Van Mechelen, & Tuerlinckx, 2011). Thus, I weighed the theoretical and practical considerations and decided one hour was sensitive enough to capture most of the aforementioned range while limiting recall bias and participant burden (Stone & Shiffman, 2002).

A prior daily diary study showed that participants engaged in a moderate amount of pleasurable activities in one week (1-5 times reported per week; Steger, Kashdan, & Oishi, 2008). This study had a single measurement per day, which is in contrast to the current study's intensive design. Thus, I weighed the theoretical and practical considerations and decided four days provided a sizeable amount of time points to capture these behaviors while providing enough statistical power and limiting participant burden (Stone & Shiffman, 2002). Two days were weekend days and two were weekdays given research indicating there are substantial

differences in eating/alcohol use on weekdays versus weekends (Reich, Cummings, Greenbaum, Moltisanti, & Goldman, 2015; Thompson, Larkin, & Brown, 1986).

Participants. I recruited 85 young adults from the University of California, Los Angeles campus. I calculated this sample size based on a power analysis conducted in G*Power Version 3.1.7 (Faul et al., 2007). I selected a repeated measures design and specified power of .95, 60 time points, a correlation of $r = .50$ between time points, and an expected effect size of Cohen's $d = 0.20$ (small). I selected this expected effect size because this was the first diary study on this topic and I wanted to optimize the likelihood of capturing any effect. The power analysis resulted in a sample size of 39 but I ran 85 participants for two reasons. First, simulation multilevel modeling studies demonstrate that sample sizes of 50 or less at the highest level of analysis usually lead to biased estimates of standard errors (Maas & Hox, 2005). In contrast, sample sizes greater than 50 at the highest level of analysis usually lead to unbiased estimates of standard errors as well as accurate fixed and random effects (Maas & Hox, 2005). Second, I received additional money for this study via the APA Dissertation Research Award, which allowed me to recruit more participants than originally planned.

Inclusion criteria were: (1) age 18-24 (young adults amenable to eating and drinking behavior change; Borsari et al., 2009; Nelson et al., 2008), (2) fluency in English (in order to understand study procedure), and (3) owning an electronic device compatible with Personal Analytics Companion (PACO; in order to carry out the study procedure). Exclusion criteria were: (1) following a strict diet that would prevent them from eating sweet high-fat foods and (2) remaining abstinent from drinking alcohol. I chose these exclusion criteria so that confounding factors (e.g., dieting) would not create floor effects of low reports of eating and alcohol use. Participants were not excluded based upon ethnicity; however, I over-recruited for Asian

participants because the G allele of the OPRM1 gene has higher representativeness in this ethnicity (Chamorro et al., 2012). I used a similar approach as in Study 1 (see Study 1 Methods).

For data analysis, I excluded any participant who (1) scored greater than a 25 (Mean + 2SD, rounded) on the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960; see below) or (2) missed any of the three quality control questions I designed to identify participants who responded without reading the questions (e.g., "For this question, please mark the answer, 'Often.'"). This was an *a priori* decision; I chose to exclude any participant who met these criteria to optimize response accuracy and limit reporting bias. In particular, the Marlowe-Crowne Social Desirability Scale was used to assess social desirability bias because this bias may cause floor effects for undesirable behaviors (e.g., sweet high-fat/palatable food consumption, alcohol consumption) and ceiling effects for desirable behaviors (e.g., donating to charity). No participant incorrectly answered any quality control question. However, one participant scored > 25 on the Marlowe-Crowne Social Desirability Scale and was excluded from all analyses. Therefore, the final sample included 84 participants. Full demographics for this final sample are presented in Table 10.

Table 10

Study 2 Demographics

	Mean	SD
	%	
Age	20.06	1.65
Sex (% Female)	76.2%	
Ethnicity		
Asian American	41.7%	
Caucasian	22.6%	
Hispanic/Latinx	13.1%	
Multi-racial/Other	11.9%	
Arabic/Middle Eastern	7.1%	
African American	3.6%	
Subjective SES		
1st Rung (Lowest)	1.2%	
2nd Rung	0.0%	
3rd Rung	3.6%	
4th Rung	11.9%	
5th Rung	8.3%	
6th Rung	17.9%	
7th Rung	38.1%	
8th Rung	14.3%	
9th Rung	2.4%	
10th Rung (Highest)	2.4%	
Objective SES		
<$5,000	3.6%	
$5,000-$30,000	13.2%	
$30,001-$75,000	29.7%	
$75,001-$150,000	34.5%	
>$150,001	17.9%	
AUDIT Score	5.88	4.50
YFAS 2.0 Score	2.52	2.65
Body Mass Index	22.84	3.72
Underweight	21.7%	
Normal	55.4%	
Overweight	19.3%	
Obese I	2.4%	
Obese II	1.2%	

Notes: AUDIT = Alcohol Use Disorders Identification Test Score, YFAS 2.0 = Yale Food Addiction Scale 2.0 Score; Symptom counts for food addiction range from 0-7 and ≥ 3 symptoms plus clinically significant impairment or distress indicates food addiction.

Procedure. Participants were recruited for the "UCLA Rewards in Everyday Life Study." The study description mentioned that researchers were studying how people experience reward in everyday life and did not mention that researchers were studying eating and alcohol use to reduce risk of performance bias (The Cochrane Collaboration, 2016). To limit the risk of detection bias, research assistants were not informed of the study hypothesis until after the study was completed and the data were analyzed (The Cochrane Collaboration, 2016).

All participants were scheduled for a baseline laboratory session from 9AM-8PM on Wednesday, Thursday, or Friday; the ambulatory electronic diary procedure began the next day one hour after the participant's waking time. By starting on these particular weekdays, each participant reported on two weekend and two weekday days. PACO allowed for participants to set their own unique waking time. PACO also time stamped data collection so that I could test for any differences in reward-related behavior due to day of the week or time of day (Reich et al., 2015). Using this time stamp, I also coded for the week of the academic quarter so that I could test for any differences in reward-related behavior due to academic climate (e.g., finals week).

At the baseline laboratory session, participants provided informed consent, completed baseline questionnaires, provided a saliva sample via the Oragene kit, and learned and practiced the ambulatory electronic diary procedure. The baseline questionnaire included the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960), the Snaith-Hamilton Pleasure Scale (Snaith, Hamilton, Morley, Humayan, & Trigwell, 1995), the Alcohol Use Disorders Identification Test (Babor et al., 2001), the Yale Food Addiction Scale 2.0 (Gearhardt, Corbin, & Brownell, 2016), attention check questions, and a demographics questionnaire.

Participants downloaded PACO onto their personal electronic devices and practiced one diary entry under supervision. Participants were instructed to engage in normal activities and

complete the diaries each time they were alerted. However, participants were instructed to skip any entry that occurred during an incompatible event such as an exam or while driving (Tomiyama, Mann, & Comer, 2009). Participants were also trained for comprehension of each diary question and to estimate standard drinks of alcohol.

Over the following four days, PACO alerted participants once each hour (minus sleep times), from an hour after waking time to sleeping time, to complete a diary entry. This included 15 alerts per day per participant based on participants sleeping 7-9 hours a day [the sleep duration the National Sleep Foundation recommends for young adults (Hirshkowitz et al., 2015)]. The diary entries included questions on which reward-related behavior participants engaged within the last hour (since they were last alerted), current feelings of affect and reward, and if they consumed sweet high-fat/palatable food and alcohol in the last hour (since they were last alerted). After participants completed four days of the requested diary entries, the research assistant emailed the participants to notify them of study completion and schedule them for a final laboratory visit. At the final laboratory visit, the research assistant provided the participant with 1 point of course credit for each full day of study participation (up to 5 points) and $2.50 for each full day of ambulatory electronic diary study participation (up to $10). For participants recruited outside of the department's subject pool, the research assistant provided the participant with $10 for each full day of ambulatory electronic diary study participation (up to $40).

Measures. Complete lists of items for questionnaires (that are unique to Study 2) are presented in Appendix II.

Ambulatory electronic diary questions. The independent, potential mediator, and dependent variables were measured during each ambulatory electronic diary assessment.

Reward-related behavior. Each hour, participants were asked to report engagement in a list of rewarding activities in the last hour. I determined the items on this list based on the existing neural research identifying behavior that activates the ventral striatum (see Table 9 on pg. 58). I decided to define reward-related behavior this way because, if the mechanism responsible for reward-related behavior competition were mesolimbic dopamine pathway competition, only behaviors that activate the mesolimbic dopamine pathway would induce competition. PACO prompted participants with the question, "In the last hour, did you," followed by a list of these reward-related behaviors as seen in the screenshots in Figure 8 below. Participants selected as many responses as applicable. Participants also had the opportunity to enter any other reward-related behaviors they engaged in which were not included on this list.

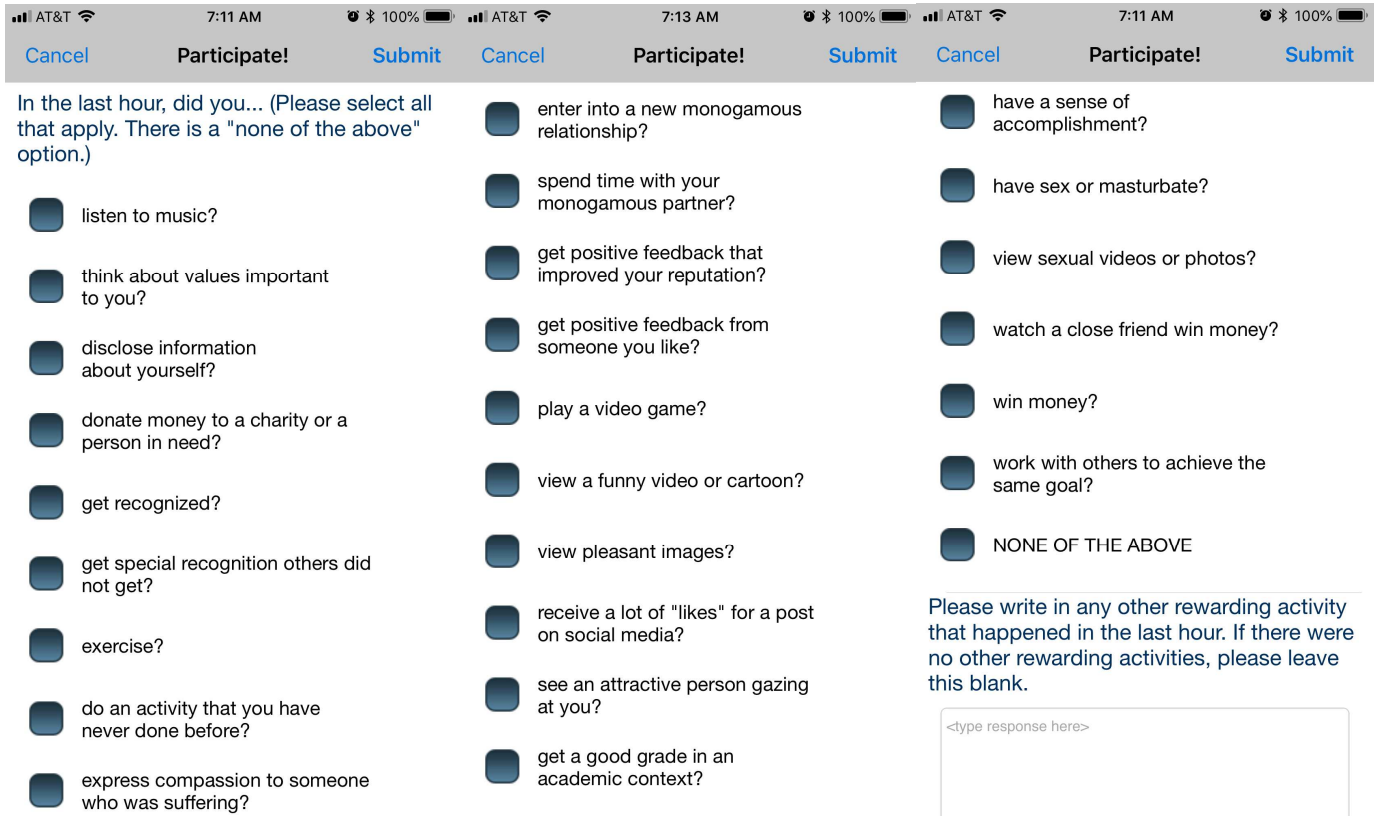


Figure 8. Study 2 Screenshot of PACO reward-related behavior question.

Feelings of reward. Each hour, participants were asked to self-report on feelings of reward. Since (to my knowledge) there are no existing measures that capture state-like feelings of reward, I used the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988), which is the gold standard measure for capturing state-like feelings of positive and negative affect. Scholars postulate that positive affect is a construct closely related to feelings of reward but that there are dissociable aspects (Chiew & Braver, 2011). As such, I created two new items to add to the measure: "rewarded," and "pleasured." I added these items because of their prevalence in the scientific nomenclature of reward (Berridge & Kringelbach, 2008).

PACO prompted individuals with the Positive and Negative Affect Schedule prompt: "Read each item and then indicate to what extent you feel this way right now at the present moment, on a scale from 1-5." By asking the participants to rate their feelings "right now," I conceptualized this variable as a mediator because the feelings will temporally follow reward-related behavior in the last hour. I performed a principal component analysis of the Positive and Negative Affect Schedule in two UCLA student samples from our laboratory's previous work. The two positive affect words with the highest factor loadings across samples were "enthusiastic" (factor loadings > .75) and "happy" (factor loadings > .71). The two negative affect words with the highest factor loadings across samples were "afraid" (factor loadings > .68) and "distressed" (factor loadings > .64). To reduce participant burden, I used only these four words in addition to "rewarded" and "pleasured." I also included the word "stressed." For the current study, I created the feelings of reward variable by taking the average across the ratings for the words "rewarded" and "pleasured."

Eating and alcohol use. Each hour, participants were asked to report if they ate sweet rewarding foods in the last hour. PACO prompted participants with the question, "In the last

hour, did you…" for each of the following behaviors: "eat sweet high fat foods (e.g., brownies, ice cream, cookies, cake, chocolate)?" "eat fast foods (e.g., food from a place like McDonald's, Kentucky Fried Chicken, Pizza Hut)?" "eat palatable foods (e.g., food that is most pleasurable to you)?" and, "drink non-alcoholic sugary drinks (e.g., cokes, diet cokes, other soda drinks, sweet tea, milkshakes, and sweet coffee drinks)?" Participants answered yes or no. In prior ambulatory electronic diary studies, similar eating questions have been used (Boggiano et al., 2015; Strahler & Nater, 2017; Tomiyama et al., 2009), and one study compared results for dichotomous eating questions with results for continuous eating questions (i.e., number of servings) and found the same results across these outcomes (Tomiyama et al., 2009).

I selected measurement of these particular food groups because of their relevance to my hypothesis and I did not include other food groups so to limit participant burden (Stone & Shiffman, 2002). I created the first item based on my finding that sugar x fat intake was implicated in food-alcohol competition (Cummings et al., 2017). In this item, I included examples of sweet high-fat foods that were top rated by pilot study raters for Study 1. The second item was drawn from the fast food intake item in the National Heart, Lung, and Blood Institute Growth and Health Study; using that data item I previously observed food-alcohol competition (Cummings et al., 2017). I created the third item based on research that suggests that a complex interaction of psychological, biological, and cultural factors affects food preferences so pleasant foods can be unique to each individual (Rozin & Vollmecke, 1986). In the current study, participants reported what they considered to be palatable foods at baseline and common reports included chocolate, ice cream, pasta, pizza, sandwiches, salmon, sushi, and noodles. Lastly, the fourth item's wording was copied from the prompt of the Palatable Eating Motives Scale (Burgess, Turan, Lokken, Morse, & Boggiano, 2014) and was included because fMRI

studies show that sugary beverages activate the ventral striatum (Stice, Yokum, Blum, & Bohon, 2010). For my analysis, I also created a composite of all unhealthy food intake in the last hour by summing across these items.

Each hour, participants were asked to report on alcohol use in the last hour with the question: "In the last hour, how many standard drinks of alcohol did you drink? (1 standard drink = 1 12 oz. beer, 1 5 oz. wine, 1 1.5 oz. liquor)?" Participants estimated the number of standardized drinks consumed. During PACO training, participants practiced estimating the number of standard drinks for various alcoholic beverages (e.g., beer, wine, liquor) poured into various glasses (e.g., shot glass, wine glass, beer mug). This practice is common in studies that ask participants to self report on alcohol use (Sobell & Sobell, 1992), as it may improve reliability. Participants also were provided with an example sheet that included standard drink images to take home. This example sheet is presented in Appendix II.

Moderator. I measured my hypothesized moderator based on prior work.

Oragene Kit (DNA Genotek, Kanata, Ontario, Canada). Saliva samples were collected for DNA analyses using Oragene kits. Genotyping was performed to identify presence of the G allele of the OPRM1 gene. I dummy coded presence of the G allele (0 = No G allele, 1 = G allele) for the current study.

Covariates. At baseline, participants reported age, sex, ethnicity, and subjective socioeconomic status on the MacArthur Scale (Adler & Stewart, 2007), and completed the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960), which assesses social desirability bias. The research assistant also measured participant height and weight to calculate BMI. I used these variables to identify any potential covariates (see below). In addition, it is a widely held belief that cigarette smoking can acutely suppress eating (albeit non-human and

human studies do not find this result; Perkins, Sexton, DiMarco, & Fonte, 1994) so in PACO I measured cigarette smoking in the last hour to identify if it was a potential covariate for the eating outcomes. Given that working, being in class, or having an obligation (e.g., doctor's appointment) may influence the likelihood that someone eats or drinks, in PACO I also measured the occurrence of these events in the last hour to identify if they were potential covariates.

Finally, PACO provided date and time stamps for each diary entry. Thus, I used those time stamps to identify if the day of week, the time of day, or the week of the academic quarter for diary entries were potential covariates. I additionally used those time stamps to identify if day in the study for diary entries was a potential covariate (to test for reactivity to the questions).

Data Analysis. Initial descriptive examination of all variables of interest indicated that all reward-related behavior composites, the eating behavior composite, and alcohol use evidenced skew (>1) and kurtosis (>3). I performed natural log transformations for these variables to correct for this. No other continuous variables were non-normally distributed. A pairwise deletion approach was used for missing data in all analyses.

To test each of my hypotheses, I conducted analysis via multilevel modeling to account for repeated measurement. Dependent variables included eating and alcohol use in the following hour; I tested each dependent variable separately (e.g., sweet high-fat food, fast food, palatable food, sugary drinks, all unhealthy food, alcohol). I tested the individual eating variables dichotomously (consumed or did not consume). I tested the eating behavior composite (number of unhealthy food groups eaten) and alcohol use (number of standard drinks) continuously. I did not center the independent or dependent variables because they were dichotomous or had meaningful zero points. Models including variables with too few positive occurrences (see Table 11) would not converge. Therefore, results are not reported or not consistently reported for

models including the following variables (although these variables are still included in their respective composites): fast food intake, entered into a new monogamous relationship, watched close friend win money, and won money.

For dichotomous outcomes, I included random effects for the intercept in each model but not random effects for the slope because Level 1 variance for dichotomous outcomes is non-constant and dependent on success probability. For continuous outcomes, I conducted Likelihood Ratio Tests between models that did and did not include random effects for the intercept and slope for each predictor. If including random effects for the intercept and/or slope improved model fit, the respective random effects were included. For example, for the models with all reward-related behavior as the predictor, including random effects for the intercept improved model fit as indicated by a significantly smaller *-2LL*, All unhealthy food intake: Δ*-2LL* = -275.20, $p < .001$; Alcohol use: Δ*-2LL* = -81.60, $p < .001$. This indicated that there were significant differences between individuals on these continuous outcomes. Including random effects for the slope improved the model fit as indicated by a significantly smaller *-2LL*, All unhealthy food intake: Δ*-2LL* = -303.90, $p < .001$; Alcohol use: Δ*-2LL* = -114.60, $p < .001$. This indicated that there were significant differences between individuals on the associations between all reward-related behavior and these continuous outcomes.

To test for covariates, I entered potential covariates as between-subjects variables at Level 2 (i.e., age, sex, ethnicity, subjective socioeconomic status, social desirability bias, BMI) or within-subjects variables at Level 1 (i.e., cigarette smoking, working, studying, obligation, day of the week, time of day, week in quarter, day in study). Results indicated that age significantly predicted all eating outcomes: sweet high-fat food intake, OR = 0.85, $p = .018$, 95% CI [0.75, 0.97], palatable food intake, OR = 0.87, $p = .032$, 95% CI [0.77, 0.99], sugary drink

intake, OR = 0.73, $p < .001$, 95% CI [0.61, 0.87], and all unhealthy food intake, $\gamma = -0.03$, $SE_\gamma = 0.01$, $p = .003$, 95% CI [-0.06, -0.01]. Subjective socioeconomic status significantly predicted sugary drink intake, OR = 0.84, $p = .045$, 95% CI [0.71, 0.99], and ethnicity significantly predicted alcohol use, $\gamma = -0.005$, $SE_\gamma = 0.002$, $p = .049$, 95% CI [-0.01, -0.00002] such that those who identified as White were most likely to drink alcohol.

In addition, having class in the current hour significantly predicted all unhealthy food intake, $\gamma = -0.05$, $SE_\gamma = 0.02$, $p = .023$, 95% CI [-0.10, -0.01], and having work in the current hour significantly predicted alcohol use, $\gamma = -0.02$, $SE_\gamma = 0.01$, $p = .014$, 95% CI [-0.04, -0.01]. Time of the day significantly predicted alcohol use and all eating outcomes but sugary drink intake such that there were greater odds of drinking and eating later in the day: alcohol use, $\gamma = 0.005$, $SE_\gamma = 0.0005$, $p < .001$, 95% CI [0.004, 0.006], sweet high-fat food intake, OR = 1.06, $p < .001$, 95% CI [1.04, 1.09], palatable food intake, OR = 1.03, $p = .003$, 95% CI [1.01, 1.05], and all unhealthy food intake, $\gamma = 0.008$, $SE_\gamma = 0.002$, $p < .001$, 95% CI [0.005, 0.01]. Day of the week significantly predicted palatable food and all unhealthy food intake such that there were greater odds of eating later in the week and on the weekend: palatable food intake, OR = 1.04, $p = .031$, 95% CI [1.004, 1.09], and all unhealthy food intake, $\gamma = 0.006$, $SE_\gamma = 0.003$, $p = .040$, 95% CI [0.0003, 0.01]. I therefore tested all the aforementioned covariates in their respective analyses. All results are presented with and without these covariates. There were no other covariates.

To test Hypothesis 1, I entered reward-related behavior in the current hour as a within-subjects variable at Level 1. Models with all reward-related behavior entered simultaneously would not converge. However, models with reward-related behavior entered independently or entered as *a priori* composites did converge. *A priori* composites included the following:

- All reward-related behavior = Sum of all reward-related behavior

- Eudaimonic reward-related behavior = Sum of affirming values, donating, expressing compassion, starting a new monogamous relationship, spending time with a monogamous partner, feeling a sense of accomplishment
- Hedonic reward-related behavior = Sum of playing video games, viewing funny videos, viewing pleasant images, getting a lot of likes on social media, seeing an attractive person gazing, having sex, viewing sexual videos, winning money
- Social reward-related behavior = Sum of disclosing self-relevant information, getting recognized, getting special recognition, expressing compassion, spending time with a monogamous partner, starting a new monogamous relationship, getting positive feedback that improved reputation, getting positive feedback from a liked person, seeing an attractive person gazing, having sex, cooperating with others
- Primary reward-related behavior = Sum of exercising and having sex/masturbating
- Secondary reward-related behavior = Sum of all reward-related behavior but exercising and having sex

Since models with reward-related behavior entered independently and entered as *a priori* composites evidenced similar model fits, results are reported for all models that converged. A sample equation is presented below (for Hypothesis 1, all unhealthy food intake predicted by a composite of all reward-related behavior without covariates):

Level 1: $\text{All unhealthy food intake}_{ij} = \beta_{0j} + \beta_1(\text{All reward-related behavior}_j) + r_j$

Level 2: $\beta_{0j} = \gamma_{00} + u_{0i}$

$\beta_{1j} = \gamma_{10} + u_{1i}$

To test Hypothesis 2, I expanded the models from Hypothesis 1 to include the presence of the G allele of the OPRM1 gene (Dummy coded: 0 = No, 1 = Yes) as a between-subjects

covariate at Level 2. I also included an interaction term between this dummy code and reward-related behavior in the current hour. For any significant interaction, I followed up with tests of simple effects to identify the level of the variable that conferred a significant effect.

To test Hypothesis 3, I first identified any significant results from Hypothesis 1. For any significant results from Hypothesis 1, I tested my hypothesized pathway using standard guidelines for a Level 1 → Level 1 → Level 1 mediation (Krull & MacKinnon, 2001). First, I tested whether reward-related behavior in the current hour predicted feelings of reward at the assessment (*a* path). Next, I tested whether feelings of reward at the assessment predicted eating and alcohol use in the following hour (*b* path), and whether the associations between reward-related behavior in the current hour and eating and alcohol use in the following hour weakened with inclusion of feelings of reward at the assessment in the model (*c'* path). I used the Sobel test formula to obtain estimates of the mediated effect and the standard error of the mediated effect, and to examine significance of the mediated effect. The Sobel test yields a critical Z-value; any critical Z-value higher than 1.645 indicated a statistically significant mediated effect at $p < .05$.

Results

Descriptives. Frequencies of participants' responses to ambulatory electronic diary reward-related behavior, individual eating, alcohol use (dichotomized), mediator, and covariate questions are presented in Table 11. Means, standard deviations, and ranges of composited reward-related behavior, all unhealthy food intake, alcohol use, and feelings of reward are presented in Table 12. Descriptives for the moderator variable are presented in Table 13.

Table 11

Study 2 Frequencies of Dichotomous Variables from Ambulatory Electronic Diary

	Yes *N* (%) [Valid]	No *N* (%) [Valid]	Missing *N* (%)
Reward-related behavior			
Listened to music	1441 (28.6%) [36.0%]	2557 (50.7%) [64.0%]	1042 (20.7%)
Thought about values important to them	574 (11.4%) [14.4%]	3424 (67.9%) [85.6%]	1042 (20.7%)
Disclosed self-relevant information	340 (6.7%) [8.5%]	3658 (72.6%) [91.5%]	1042 (20.7%)
Donated money to a charity or person in need	12 (0.2%) [0.3%]	3986 (79.1%) [99.7%]	1042 (20.7%)
Got recognized	180 (3.6%) [4.5%]	3818 (75.8%) [95.5%]	1042 (20.7%)
Got special recognition	64 (1.3%) [1.6%]	3934 (78.1%) [98.4%]	1042 (20.7%)
Exercised	261 (5.2%) [74.1%]	3737 (74.1%) [93.5%]	1042 (20.7%)
Did something new	156 (3.1%) [3.9%]	3842 (76.2%) [96.1%]	1042 (20.7%)
Expressed compassion	86 (1.7%) [2.2%]	3912 (77.6%) [97.8%]	1042 (20.7%)
Entered into a new monogamous relationship	1 (<0.1%) [<0.1%]	3997 (79.3%) [100.0%]	1042 (20.7%)
Spent time with a monogamous partner	329 (6.5%) [8.2%]	3669 (72.8%) [91.8%]	1042 (20.7%)
Got positive feedback that improved their reputation	61 (1.2%) [1.5%]	3937 (78.1%) [98.5%]	1042 (20.7%)
Got positive feedback from a person they liked	173 (3.4%)	3825 (75.9%)	1042 (20.7%)

	[4.3%]	[95.7%]	
Played a video game	93	3905	1042
	(1.8%)	(77.5%)	(20.7%)
	[2.3%]	[97.7%]	
Watched a funny video or cartoon	438	3560	1042
	(8.7%)	(70.6%)	(20.7%)
	[11.0%]	[89.0%]	
Viewed pleasant images	368	3630	1042
	(7.3%)	(72.0%)	(20.7%)
	[9.2%]	[90.8%]	
Received a lot of "likes" on a social media post	92	3906	1042
	(1.8%)	(77.5%)	(20.7%)
	[2.3%]	[97.7%]	
Saw an attractive person gazing at them	60	3938	1042
	(1.2%)	(78.1%)	(20.7%)
	[1.5%]	[98.5%]	
Got a good grade in an academic context	29	3969	1042
	(0.60%)	(78.8%)	(20.7%)
	[0.70%]	[99.3%]	
Had a sense of accomplishment	288	3710	1042
	(5.7%)	(73.6%)	(20.7%)
	[7.2%]	[92.8%]	
Had sex or masturbated	83	3915	1042
	(1.6%)	(77.7%)	(20.7%)
	[2.1%]	[97.9%]	
Viewed sexual videos or photos	28	3970	1042
	(0.6%)	(78.8%)	(20.7%)
	[0.7%]	[99.3%]	
Watched a close friend win money	4	3994	1042
	(0.1%)	(79.2%)	(20.7%)
	[0.1%]	[99.9%]	
Won money	12	3986	1042
	(0.2%)	(79.1%)	(20.7%)
	[0.3%]	[99.7%]	
Worked with others to achieve the same goal	127	3871	1042
	(2.5%)	(76.8%)	(20.7%)
	[3.2%]	[96.8%]	
Other	359	3639	1042
	(7.1%)	(65.2%)	(20.7%)
	[9.0%]	[91.0%]	
Eating			
Sweet high-fat food	545	3622	873
	(10.8%)	(71.9%)	(17.3%)
	[13.1%]	[86.9%]	
Fast food	180	3993	867
	(3.6%)	(79.2%)	(17.2%)

	[4.3%]	[95.7%]	
Palatable food	824 (16.3%) [19.8%]	3345 (66.4%) [80.2%)	871 (17.3%)
Sugary non-alcoholic drink	375 (7.4%) [9.0%]	3796 (75.3%) [91.0%]	869 (17.2%)
Alcohol use			
	88 (1.7%) [2.1%]	4076 (80.9%) [97.9%]	876 (17.4%)
Covariates			
Cigarette smoking	12 (0.2%) [0.3%]	4161 (82.6%) [99.7%]	867 (17.2%)
Class	410 (8.1%) [10.3%]	3573 (70.9%) [89.7%]	1057 (21.0%)
Work	341 (6.8%) [8.6%]	3642 (72.3%) [91.4%]	1057 (21.0%)
Obligation	382 (7.6%) [9.6%]	3601 (71.4%) [90.4%]	1057 (21.0%)

Notes: N = Number of reports of behavior. (%) = Percentage of reports of behavior. [Valid] = Percentage of reports of behavior adjusting for missing data.

Table 12

Study 2 Means, Standard Deviations, & Ranges of Continuous Variables from Ambulatory Electronic Diary

	Mean	*SD*	Range
Reward-related behavior			
Total composite	1.33	1.45	0-12
Social composite	0.40	0.76	0-7
Eudaimonic composite	0.32	0.55	0-3
Hedonic composite	0.30	0.64	0-4
Primary composite	0.09	0.28	0-2
Secondary composite	1.23	1.38	0-11
Health-compromising behavior			
Unhealthy food intake composite	0.46	0.82	0-4
Alcohol use	0.04	0.25	0-5
Potential Mediator			
Feelings of reward composite	1.82	0.94	1-5

Note: Untransformed values presented.

Table 13

Study 2 Descriptives of Moderator Variable

	%
OPRM1 gene	
AA	59.75%
AG	34.15%
GG	6.10%

Hypothesis 1. Results indicated that multiple reward-related behaviors in the current hour predicted sweet high-fat food intake, palatable food intake, sugary drink intake, and all unhealthy food intake in the next hour. In detail, a composite of all reward-related behavior, a composite of hedonic reward-related behavior, a composite of secondary reward-related behavior, listening to music, donating to a charity or a person in need, getting special recognition, doing something new, getting positive feedback that improved their reputation, playing a video game, viewing pleasant images, seeing an attractive person gazing at them, and viewing sexual images or videos in the current hour significantly predicted eating in the following hour. By and large, the associations were such that greater engagement in reward-related behavior predicted *greater* food intake. For the dichotomous outcomes, Odds Ratios ranged from 1.26 to 6.32 (with covariates in the model); this indicates that, over and above the effects of any covariates, for each instance of reward-related behavior, the expected odds of eating were increased by 26% to 532%. For the composite of all unhealthy food intake, β coefficients ranged from 0.05 to 0.08 (with covariates in model); this indicates that, over and above the effects of any covariates, for each instance of reward-related behavior all unhealthy food intake increased by 0.05 to 0.08 instances (unhealthy food intake ranged from 0 to 4 instances of eating). Multilevel estimates of all fixed effects are presented in Tables 14, 15, 16, and 17, and estimates of significant effects are bolded (note that effects were only considered significant if they held with covariates included in the model).

In addition, results indicated that some reward-related behaviors in the current hour predicted alcohol use in the next hour. Specifically, a composite of eudaimonic reward-related behavior and having a sense of accomplishment significantly predicted alcohol use in the next hour. The associations were such that greater engagement in reward-related behavior predicted

less food intake. β coefficients ranged from -0.03 to -0.02 (with covariates in model); this indicates that, over and above the effects of any covariates, for each instance of reward-related behavior alcohol use changed by -0.03 to -0.02 standard drinks (alcohol use ranged from 0 to 5 standard drinks). Multilevel estimates of all fixed effects are presented in Table 18, and estimates of significant effects are bolded (again, note that effects were only considered significant if they held with covariates included in the model).

Table 14

Study 2 Hypothesis 1 Estimates of Fixed Effects for Sweet High-Fat Food Intake

	Without covariates				With covariates				
			95% CI				95% CI		
	OR	*p*	Lower	Upper	OR	*p*	Lower	Upper	-2LL
Composited reward-related behaviors									
All	**1.38**	**.003**	**1.12**	**1.71**	**1.30**	**.016**	**1.05**	**1.62**	**17236.89**
Eudaimonic	0.90	.535	0.65	1.26	0.88	.456	0.63	1.23	17218.19
Hedonic	1.08	.614	0.80	1.46	1.04	.794	0.77	1.41	17213.58
Social	1.37	.020	1.05	1.78	1.28	.071	0.98	1.67	17219.81
Primary	1.19	.509	0.71	2.00	1.19	.512	0.71	2.01	17205.50
Secondary	**1.40**	**.002**	**1.13**	**1.74**	**1.32**	**.014**	**1.06**	**1.64**	**17241.46**
Independent reward-related behaviors									
Listened to music	1.37	.011	1.07	1.74	1.28	.050	1.00	1.63	17225.43
Thought about values important to them	0.87	.430	0.63	1.22	0.86	.385	0.62	1.21	17221.25
Disclosed self-relevant information	1.15	.466	0.79	1.66	1.07	.735	0.73	1.55	17211.38
Donated money to a charity or person in need	**6.36**	**.033**	**1.16**	**34.92**	**6.32**	**.037**	**1.12**	**35.69**	**17217.80**
Got recognized	1.12	.632	0.70	1.82	1.06	.822	0.65	1.71	17213.03
Got special recognition	**2.22**	**.025**	**1.11**	**4.43**	**2.15**	**.032**	**1.07**	**4.34**	**17223.89**
Exercised	1.26	.258	0.85	1.87	1.27	.242	0.85	1.89	17204.85
Did something new	**2.06**	**.002**	**1.32**	**3.22**	**2.03**	**.002**	**1.30**	**3.18**	**17238.03**
Expressed compassion	0.93	.822	0.48	1.80	0.93	.819	0.48	1.80	17211.10
Spent time with a monogamous partner	0.99	.946	0.63	1.54	0.95	.828	0.61	1.49	17212.53
Got positive feedback that improved their reputation	**2.18**	**.021**	**0.12**	**1.44**	**2.07**	**.032**	**1.07**	**4.02**	**17220.05**
Got positive feedback from a person they liked	1.06	.820	0.64	1.76	1.00	.989	0.60	1.67	17211.91
Played a video game	1.06	.890	0.49	2.28	1.01	.982	0.46	2.19	17211.08
Watched a funny video or cartoon	0.88	.466	0.62	1.25	0.85	.358	0.60	1.21	17212.43
Viewed pleasant images	0.95	.762	0.65	1.37	0.96	.821	0.66	1.39	17212.18
Received a lot of "likes" on a social media post	1.70	.084	0.93	3.11	1.68	.096	0.91	3.09	17209.30
Saw an attractive person gazing at them	**2.36**	**.020**	**1.15**	**4.85**	**2.10**	**.046**	**1.02**	**4.35**	**17224.24**
Got a good grade in an academic context	1.52	.437	0.53	4.34	1.71	.317	0.60	4.87	17212.59

Had a sense of accomplishment	0.94	.753	0.62	1.42	0.93	.747	0.62	1.42	17213.52
Had sex or masturbated	0.70	.428	0.29	1.70	0.67	.384	0.27	1.65	17220.86
Viewed sexual videos or photos	0.82	.756	0.23	2.94	0.70	.586	0.19	2.55	17211.97
Won money	0.71	.748	0.09	5.89	0.61	.648	0.07	5.16	17209.39
Worked with others to achieve the same goal	1.57	.094	0.93	2.67	1.50	.137	0.88	2.56	17203.95

Table 15

Study 2 Hypothesis 1 Estimates of Fixed Effects for Palatable Food Intake

	Without covariates				With covariates				
			95% CI				95% CI		
	OR	*p*	Lower	Upper	OR	*p*	Lower	Upper	-2LL
Composited reward-related behaviors									
All	**1.38**	**< .001**	**1.15**	**1.66**	**1.34**	**.002**	**1.11**	**1.61**	**16137.59**
Eudaimonic	1.06	.147	0.80	1.41	1.05	.759	0.79	1.40	16131.29
Hedonic	**1.46**	**.003**	**1.13**	**1.88**	**1.41**	**.008**	**1.10**	**1.82**	**16139.95**
Social	1.25	.064	0.99	1.58	1.21	.115	0.96	1.53	16134.25
Primary	1.18	.479	0.74	1.88	1.17	.497	0.74	1.87	16131.20
Secondary	**1.39**	**< .001**	**1.15**	**1.67**	**1.35**	**.002**	**1.11**	**1.63**	**16136.03**
Independent reward-related behaviors									
Listened to music	**1.30**	**.012**	**1.06**	**1.60**	**1.26**	**.028**	**1.03**	**1.56**	**16129.75**
Thought about values important to them	0.88	.381	0.66	1.18	0.88	.399	0.66	1.18	16134.94
Disclosed self-relevant information	1.07	.705	0.76	1.49	0.76	.728	0.76	1.49	16131.55
Donated money to a charity or person in need	4.42	.086	0.81	24.04	4.21	.102	0.75	23.64	16129.21
Got recognized	1.17	.446	0.78	1.77	1.15	.495	0.77	1.74	16131.69
Got special recognition	1.70	.096	0.91	3.17	1.67	.108	0.89	3.12	16134.18
Exercised	1.11	.573	0.77	1.60	1.12	.545	0.78	1.62	16130.34
Did something new	1.49	.072	0.97	2.30	1.48	.078	0.96	2.28	16138.20
Expressed compassion	1.15	.637	0.65	2.02	1.14	.655	0.65	2.00	16129.85
Spent time with a monogamous partner	1.26	.213	0.88	1.82	1.21	.317	0.84	1.75	16129.12
Got positive feedback that improved their reputation	1.46	.250	0.77	2.77	1.42	.285	0.75	2.70	16133.39
Got positive feedback from a person they liked	1.23	.341	0.80	1.88	1.18	.449	0.77	1.80	16132.04
Played a video game	**1.84**	**.036**	**1.04**	**3.25**	**1.79**	**.046**	**1.01**	**3.17**	**16130.85**
Watched a funny video or cartoon	1.23	.166	0.92	1.63	1.19	.236	0.89	1.59	16133.85
Viewed pleasant images	**1.60**	**.002**	**1.19**	**2.16**	**1.58**	**.003**	**1.17**	**2.13**	**16143.55**
Received a lot of "likes" on a social media post	0.84	.561	0.46	1.53	0.80	.476	0.44	1.47	16133.43
Saw an attractive person gazing at them	0.69	.045	1.02	3.90	1.90	.062	0.97	3.74	16134.95
Got a good grade in an academic context	0.65	.409	0.23	1.81	0.68	.462	0.24	1.90	16130.08

Had a sense of accomplishment	1.09	.621	0.77	1.54	1.09	.609	0.78	1.55	16132.80
Had sex or masturbated	1.12	.723	0.59	2.13	1.08	.823	0.57	2.04	16131.34
Viewed sexual videos or photos	0.59	.427	0.16	2.15	0.56	.377	0.15	2.03	16131.58
Worked with others to achieve the same goal	0.93	.799	0.56	1.57	0.91	.733	0.54	1.54	16130.22

Table 16

Study 2 Hypothesis 1 Estimates of Fixed Effects for Sugary Drink Intake

	Without covariates				With covariates				
			95% CI				95% CI		
	OR	*p*	Lower	Upper	OR	*p*	Lower	Upper	-2LL
Composited reward-related behaviors									
All	**1.53**	**.001**	**1.19**	**1.97**	**1.50**	**.002**	**1.17**	**1.93**	**18453.70**
Eudaimonic	1.29	.192	0.88	1.88	1.31	.168	0.89	1.91	18407.51
Hedonic	**1.43**	**.034**	**1.03**	**1.98**	**1.41**	**.041**	**1.02**	**1.95**	**18399.14**
Social	1.19	.268	0.87	1.63	1.20	.261	0.88	1.63	18400.15
Primary	1.16	.642	0.63	2.12	1.18	.642	0.64	2.16	18395.82
Secondary	**1.54**	**.001**	**1.19**	**1.99**	**1.50**	**.002**	**1.16**	**1.94**	**18457.11**
Independent reward-related behaviors									
Listened to music	**1.38**	**.026**	**1.04**	**1.84**	**1.33**	**.049**	**1.001**	**1.77**	**18428.94**
Thought about values important to them	1.32	.130	0.92	1.90	1.32	.137	0.92	1.89	18393.28
Disclosed self-relevant information	1.17	.485	0.76	1.79	1.14	.539	0.75	1.76	18393.62
Donated money to a charity or person in need	1.39	.746	0.19	10.40	1.43	.733	0.19	10.93	18393.77
Got recognized	1.08	.800	0.61	1.88	1.08	.787	0.62	1.89	18397.56
Got special recognition	1.81	.152	0.80	4.05	1.83	.144	0.81	4.12	18409.55
Exercised	1.01	.984	0.62	1.63	1.01	.956	0.62	1.65	18396.41
Did something new	1.16	.608	0.66	2.01	1.14	.645	0.65	1.98	18396.39
Expressed compassion	0.77	.543	0.33	1.79	0.76	.525	0.33	1.77	18397.09
Spent time with a monogamous partner	1.40	.189	0.85	2.30	1.45	.145	0.88	2.39	18419.85
Got positive feedback that improved their reputation	0.73	.544	0.27	2.00	0.74	.553	0.27	2.01	18395.08
Got positive feedback from a person they liked	1.38	.228	0.82	2.35	1.38	.238	0.81	2.33	18400.68
Played a video game	1.13	.759	0.52	2.43	1.14	.743	0.53	2.45	18395.28
Watched a funny video or cartoon	1.26	.217	0.87	1.83	1.23	.270	0.85	1.79	18296.89
Viewed pleasant images	**1.57**	**.023**	**1.06**	**2.33**	**1.55**	**.028**	**1.05**	**2.29**	**18407.54**
Received a lot of "likes" on a social media post	0.62	.245	0.28	1.39	0.62	.241	0.28	1.38	18422.52
Saw an attractive person gazing at them	1.11	.821	0.44	2.85	1.10	.837	0.43	2.82	18394.30
Got a good grade in an academic context	0.53	.385	0.13	2.23	0.53	.390	0.13	2.26	18395.40

Had a sense of accomplishment	0.87	.580	0.52	1.44	0.88	.611	0.53	1.46	18393.63
Had sex or masturbated	1.46	.363	0.65	3.28	1.50	.325	0.67	3.39	18402.63
Viewed sexual videos or photos	**4.33**	**.003**	**1.63**	**11.48**	**4.56**	**.003**	**1.71**	**12.18**	**18423.89**
Won money	1.16	.889	0.14	9.76	1.19	.873	0.14	10.04	18393.39
Worked with others to achieve the same goal	0.68	.302	0.32	1.42	0.66	.270	0.31	1.39	18400.38

Table 17

Study 2 Hypothesis 1 Estimates of Fixed Effects for All Unhealthy Food Intake

	Without covariates					With covariates					
				95% CI					95% CI		
	β	SE_{β}	*p*	Lower	Upper	*β*	SE_{β}	*p*	Lower	Upper	-2LL
Composited reward-related behaviors											
All	**0.07**	**0.02**	**< .001**	**0.03**	**0.10**	**0.06**	**0.02**	**< .001**	**0.03**	**0.10**	**3360.5**
Eudaimonic	0.02	0.02	.443	-0.03	0.06	0.01	0.02	.694	-0.04	0.06	3386.9
Hedonic	**0.05**	**0.02**	**.015**	**0.01**	**0.10**	**0.05**	**0.02**	**.035**	**0.003**	**0.09**	**3379.9**
Social	0.05	0.02	.008	0.01	0.09	0.03	0.02	.083	-0.01	0.07	3383.5
Primary	0.05	0.04	.175	-0.02	0.12	0.05	0.04	.221	-0.03	0.12	3384.5
Secondary	**0.07**	**0.02**	**< .001**	**0.03**	**0.10**	**0.06**	**0.02**	**< .001**	**0.03**	**0.10**	**3361.2**
Independent reward-related behaviors											
Listened to music	**0.06**	**0.02**	**.003**	**0.02**	**0.10**	**0.06**	**0.02**	**.004**	**0.02**	**0.09**	**3370.7**
Thought about values important to them	-0.01	0.02	.768	-0.05	0.04	-0.01	0.02	.684	-0.06	0.04	3386.6
Disclosed self-relevant information	0.03	0.03	.265	-0.03	0.09	0.02	0.03	.456	-0.04	0.08	3386.0
Donated money to a charity or person in need	0.21	0.14	.152	-0.08	0.49	0.19	0.14	.189	-0.09	0.47	3377.5
Got recognized	0.004	0.04	.901	-0.06	0.07	0.01	0.04	.808	-0.06	0.08	3383.8
Got special recognition	0.15	0.07	.057	-0.01	0.30	0.13	0.08	.092	-0.02	0.29	3375.9
Exercised	0.03	0.03	.244	-0.02	0.09	0.04	0.03	.206	-0.02	0.10	3385.1
Did something new	0.09	0.05	.087	-0.01	0.19	0.10	0.05	.052	-0.001	0.20	3368.9
Expressed compassion	0.03	0.06	.629	-0.09	0.15	0.02	0.06	.711	-0.10	0.14	3384.9
Spent time with a monogamous partner	0.02	0.02	.296	-0.03	0.08	0.01	0.02	.708	-0.04	0.06	3381.8
Got positive feedback that improved their reputation	0.10	0.06	.078	-0.01	0.21	0.07	0.06	.263	-0.05	0.18	3379.4
Got positive feedback from a person they liked	0.03	0.04	.424	-0.05	0.11	0.02	0.04	.626	-0.05	0.09	3385.8
Played a video game	0.05	0.05	.291	-0.04	0.15	0.04	0.05	.535	-0.10	0.17	3385.1
Watched a funny video or cartoon	0.01	0.03	.789	-0.05	0.06	0.004	0.03	.898	-0.05	0.06	3386.0
Viewed pleasant images	**0.07**	**0.03**	**.006**	**0.02**	**0.12**	**0.08**	**0.03**	**.007**	**0.02**	**0.13**	**3376.5**
Received a lot of "likes" on a social media post	0.003	0.05	.956	-0.09	0.10	-0.002	0.05	.965	-0.10	0.09	3385.5
Saw an attractive person gazing at them	0.14	0.08	.093	-0.03	0.31	0.12	0.09	.184	-0.06	0.31	3375.1
Got a good grade in an academic context	-0.17	0.08	.036	-0.33	-0.01	-0.15	0.08	.069	-0.31	0.01	3379.6
Had a sense of accomplishment	0.01	0.03	.828	-0.05	0.07	0.01	0.03	.841	-0.06	0.07	3386.3
Had sex or masturbated	0.05	0.05	.293	-0.05	0.15	0.03	0.05	.597	-0.08	0.13	3382.4
Viewed sexual videos or photos	0.09	0.08	.277	-0.09	0.27	0.09	0.09	.312	-0.08	0.26	3382.8
Won money	-0.11	0.12	.348	-0.36	0.13	-0.11	0.13	.406	-0.36	0.15	3383.0
Worked with others to achieve the same goal	0.02	0.05	.716	-0.08	0.11	0.01	0.05	.782	-0.09	0.12	3383.5

Table 18

Study 2 Hypothesis 1 Estimates of Fixed Effects for Alcohol Use

	Without covariates					With covariates					
				95% CI					95% CI		
	β	SE_β	p	Lower	Upper	β	SE_β	p	Lower	Upper	-2LL
Composited reward-related behaviors											
All	0.01	0.01	.197	-0.003	0.02	0.002	0.01	.712	-0.01	0.01	-3470.6
Eudaimonic	**-0.02**	**0.01**	**.016**	**-0.03**	**-0.004**	**-0.02**	**0.01**	**.009**	**-0.04**	**-0.01**	**-3476.3**
Hedonic	0.01	0.01	.330	-0.01	0.04	0.01	0.01	.399	-0.01	0.03	-3493.3
Social	0.01	0.01	.338	-0.01	0.02	0.004	0.01	.649	-0.01	0.02	-3480.0
Primary	-0.002	0.01	.902	-0.03	0.03	-0.00001	0.01	.998	-0.03	0.03	-3473.2
Secondary	0.01	0.01	.188	-0.003	0.02	0.002	0.01	.723	-0.01	0.01	-3470.2
Independent reward-related behaviors											
Listened to music	0.01	0.01	.041	0.001	0.03	0.01	0.01	.188	-0.01	0.02	-3528.2
Thought about values important to them	-0.01	0.01	.460	-0.02	0.01	-0.01	0.01	.172	-0.02	0.004	-3482.0
Disclosed self-relevant information	-0.01	0.01	.589	-0.02	0.01	-0.01	0.01	.249	-0.03	0.01	-3473.2
Donated money to a charity or person in need	-0.01	0.05	.893	-0.10	0.09	-0.02	0.05	.744	-0.11	0.08	-3473.2
Got recognized	-0.003	0.02	.853	-0.04	0.03	-0.01	0.02	.660	-0.04	0.02	-3490.1
Got special recognition	0.02	0.03	.459	-0.04	0.08	0.02	0.03	.442	-0.04	0.08	-3478.3
Exercised	-0.02	0.01	.066	-0.04	0.001	-0.02	0.01	.082	-0.04	0.002	-3473.0
Did something new	0.004	0.01	.747	-0.02	0.03	-0.0001	0.01	.993	-0.02	0.02	-3473.3
Expressed compassion	-0.01	0.02	.372	-0.04	0.02	-0.01	0.02	.493	-0.04	0.02	-3475.7
Spent time with a monogamous partner	-0.004	0.01	.753	-0.03	0.02	-0.01	0.01	.509	-0.04	0.02	-3479.6
Got positive feedback that improved their reputation	0.02	0.02	.512	-0.03	0.06	0.02	0.03	.518	-0.04	0.07	-3478.8
Got positive feedback from a person they liked	0.04	0.02	.100	-0.01	0.09	0.04	0.02	.122	-0.01	0.08	-3517.7
Played a video game	0.01	0.02	.624	-0.03	0.04	0.0004	0.02	.980	-0.04	0.04	-3471.1
Watched a funny video or cartoon	-0.002	0.01	.783	-0.02	0.01	-0.01	0.01	.560	-0.02	0.01	-3470.6
Viewed pleasant images	-0.003	0.01	.701	-0.02	0.01	-0.002	0.01	.801	-0.02	0.02	-3479.2
Received a lot of "likes" on a social media post	-0.01	0.02	.572	-0.04	0.02	-0.01	0.02	.597	-0.04	0.02	-3471.2
Saw an attractive person gazing at them	0.01	0.02	.510	-0.03	0.05	0.001	0.02	.967	-0.04	0.04	-3471.4
Got a good grade in an academic context	-0.02	0.02	.480	-0.06	0.03	-0.01	0.02	.697	-0.22	0.20	-3478.9
Had a sense of accomplishment	**-0.04**	**0.01**	**< .001**	**-0.05**	**-0.02**	**-0.03**	**0.01**	**< .001**	**-0.05**	**-0.01**	**-3481.3**
Had sex or masturbated	0.06	0.05	.250	-0.04	0.16	0.07	0.05	.183	-0.03	0.17	-3480.4
Viewed sexual videos or photos	0.04	0.07	.521	-0.10	0.19	0.03	0.06	.613	-0.10	0.16	-3506.0
Won money	-0.01	0.04	.794	-0.10	0.07	-0.02	0.04	.688	-0.10	0.07	-3473.1
Worked with others to achieve the same goal	-0.003	0.01	.831	-0.03	0.02	-0.01	0.01	.614	-0.03	0.02	-3470.9

Hypothesis 2. In general, results indicated that the presence of the G allele of the OPRM1 gene did not moderate the associations between engaging in reward-related behavior and eating and alcohol use. However, results indicated that there were two (out of a potential of 144) significant interactions between independent reward-related behaviors and presence of the G allele of the OPRM1 gene on eating or alcohol use. Specifically, presence of the G allele of the OPRM1 gene significantly interacted with:

(1) viewing a funny video or cartoon to predict palatable food intake, OR = 0.93, p = .011, 95% CI [0.59, 1.48];

(2) viewing pleasant images to predict palatable food intake, OR = 0.93, p = .048, 95% CI [0.59, 1.47];

Tests of simple effects indicated that those with the G allele of the OPRM1 gene were *more likely* to eat the hour after engaging in reward-related behavior. In detail, those with the G allele of the OPRM1 gene were more likely to:

(1) eat palatable food the hour after viewing a funny video or cartoon, OR = 1.95, p = .004, 95% CI [1.23, 3.08];

(2) eat palatable food the hour after viewing pleasant images, OR = 2.42, $p < .001$, 95% CI [1.52, 3.85];

No other interactive effects emerged between reward-related behavior and presence of the G allele of the OPRM1 gene.

Hypothesis 3. In general, results indicated that engaging in reward-related behavior in the current hour was associated with increased feelings of reward at the time of the PACO assessment. These increased feelings of reward at the time of the PACO assessment predicted *greater* eating and *greater* alcohol use, and weakened the association between reward-related

behavior and greater eating as well as the association between reward-related behavior and less alcohol use. Yet, the mediated effects were not all significant. Specifically, Sobel tests indicated that 21 of the 26 tested mediated effects were significant. And although the Sobel tests indicated these mediated effects were significant, in many cases there still was a significant association between reward-related behavior and eating/alcohol use when controlling for feelings of reward. Therefore, these mediated effects can be interpreted as partial mediations. Estimates of *a*, *b*, and *c'* pathways in addition to the estimates of mediated effects, standard errors of mediated effects, and critical Z-values are presented in Table 19 for dichotomous outcomes and Table 20 for continuous outcomes. Whether the mediated effect was significant or not is indicated in the last column of each of these tables.

Table 19

Study 2 Hypothesis 3 Estimates of a, b, and c' Pathways & Estimates of Mediated effects, Standard Errors of Mediated Effects, and Critical Z-values for Dichotomous Outcomes

	a					*b*				*c'*							
				95% CI				95% CI				95% CI					
	β	SE_β	*p*	Lower	Upper	OR	*p*	Lower	Upper	OR	*p*	Lower	Upper	$\hat{\beta}_a\hat{\beta}_b$	$\widehat{SE}_{\beta a \beta b}$	*Z*	?
Sweet high-fat food intake																	
All reward-related behavior	**0.57**	**0.05**	**<.001**	**0.48**	**0.66**	**1.17**	**.019**	**1.03**	**1.34**	**1.20**	**.109**	**0.96**	**1.51**	**0.09**	**0.04**	**2.24**	**Y**
Secondary reward-related behavior	**0.50**	**0.04**	**<.001**	**0.41**	**0.59**	**1.18**	**.017**	**1.03**	**1.34**	**1.22**	**.084**	**0.97**	**1.54**	**0.08**	**0.04**	**2.25**	**Y**
Donated to a charity or a person in need	0.14	0.38	.734	-0.81	1.08	1.21	.004	1.06	1.37	5.81	.049	1.00	33.66	0.03	0.07	0.37	N
Got special recognition	**0.96**	**0.18**	**<.001**	**0.59**	**1.32**	**1.20**	**.006**	**1.05**	**1.36**	**1.87**	**.079**	**0.93**	**3.77**	**0.17**	**0.07**	**2.62**	**Y**
Did something new	**0.50**	**0.10**	**<.001**	**0.31**	**0.70**	**1.19**	**.008**	**1.05**	**1.35**	**1.87**	**.007**	**1.19**	**2.94**	**0.09**	**0.04**	**2.22**	**Y**
Got positive feedback that improved their reputation	**0.65**	**0.16**	**<.001**	**0.32**	**0.99**	**1.20**	**.006**	**1.05**	**1.36**	**1.95**	**.051**	**0.99**	**3.83**	**0.12**	**0.06**	**2.10**	**Y**
Saw an attractive person gazing at them	**0.42**	**0.13**	**.003**	**0.16**	**0.69**	**1.21**	**.003**	**1.07**	**1.37**	**2.15**	**.039**	**1.04**	**4.45**	**0.08**	**0.03**	**2.49**	**Y**
Palatable food intake																	
All reward-related behavior	**0.57**	**0.05**	**<.001**	**0.48**	**0.66**	**1.12**	**.070**	**0.99**	**1.26**	**1.28**	**.015**	**1.05**	**1.55**	**0.06**	**0.03**	**1.84**	**Y**
Hedonic reward-related behavior	**0.34**	**0.07**	**<.001**	**0.21**	**0.47**	**1.15**	**.014**	**1.03**	**1.29**	**1.38**	**.014**	**1.07**	**1.78**	**0.05**	**0.02**	**2.26**	**Y**
Secondary reward-related behavior	**0.50**	**0.04**	**<.001**	**0.41**	**0.59**	**1.12**	**.056**	**0.99**	**1.26**	**1.82**	**.012**	**1.06**	**1.57**	**0.06**	**0.03**	**1.81**	**Y**
Listened to music	**0.26**	**0.05**	**<.001**	**0.17**	**0.35**	**1.16**	**.012**	**1.03**	**1.29**	**1.23**	**.051**	**0.99**	**1.52**	**0.04**	**0.02**	**2.06**	**Y**
Played a video game	**0.40**	**0.14**	**.010**	**0.11**	**0.69**	**1.16**	**.008**	**1.04**	**1.30**	**1.71**	**.065**	**0.97**	**3.03**	**0.06**	**0.03**	**1.96**	**Y**
Viewed pleasant images	**0.16**	**0.07**	**.018**	**0.03**	**0.29**	**1.16**	**.009**	**1.04**	**1.30**	**1.57**	**.003**	**1.16**	**2.12**	**0.02**	**0.01**	**1.69**	**Y**
Sugary drink intake																	
All reward-related behavior	0.57	0.05	<.001	0.48	0.66	0.93	.822	0.52	1.69	3.43	.077	0.88	13.39	-0.04	0.17	-0.24	N
Hedonic reward-related behavior	**0.34**	**0.07**	**<.001**	**0.21**	**0.47**	**1.16**	**.043**	**1.01**	**1.35**	**1.38**	**.059**	**0.99**	**1.91**	**0.05**	**0.02**	**2.11**	**Y**
Secondary reward-related behavior	0.50	0.04	<.001	0.41	0.59	1.11	.167	0.96	1.30	1.45	.007	1.11	1.90	0.06	0.04	1.37	N
Listened to music	**0.26**	**0.05**	**<.001**	**0.17**	**0.35**	**1.16**	**.046**	**1.00**	**1.35**	**1.27**	**.109**	**0.95**	**1.69**	**0.04**	**0.02**	**1.71**	**Y**
Viewed pleasant	0.16	0.07	.018	0.03	0.29	1.17	.032	1.01	1.36	1.54	.032	1.04	2.27	0.03	0.02	1.62	N

images																
Viewed sexual videos or photos	0.32	0.17	.110	-0.10	0.73	1.17	.033	1.01	1.36	4.35	.004	1.61	11.76	0.05	0.04	1.29	N

Notes: The final column indicates whether or not the Sobel test indicated a significant mediated effect; Y = Yes, N = No.

Table 20

Study 2 Hypothesis 3 Estimates of a, b, and c' Pathways & Estimates of Mediated effects, Standard Errors of Mediated Effects, and Critical Z-values for Continuous Outcomes

	a					*b*					*c'*								
				95% CI					95% CI					95% CI					
	β	SE_β	*p*	Lower	Upper	β	SE_β	*p*	Lower	Upper	β	SE_β	*p*	Lower	Upper	$\hat{\beta}_a\hat{\beta}_b$	$\widehat{SE}_{\beta a \beta b}$	*Z*	?
All unhealthy food intake																			
All reward-related behavior	**0.57**	**0.05**	**<.001**	**0.48**	**0.66**	**0.03**	**0.01**	**.009**	**0.01**	**0.04**	**0.05**	**0.02**	**.007**	**0.01**	**0.08**	**0.01**	**0.006**	**2.00**	**Y**
Hedonic reward-related behavior	**0.34**	**0.07**	**<.001**	**0.21**	**0.47**	**0.03**	**0.01**	**<.001**	**0.02**	**0.05**	**0.04**	**0.02**	**.095**	**-0.01**	**0.08**	**0.01**	**0.004**	**2.72**	**Y**
Secondary reward-related behavior	**0.50**	**0.04**	**<.001**	**0.41**	**0.59**	**0.03**	**0.01**	**.006**	**0.01**	**0.05**	**0.05**	**0.02**	**.008**	**0.01**	**0.09**	**0.02**	**0.005**	**2.92**	**Y**
Listened to music	**0.26**	**0.05**	**<.001**	**0.17**	**0.35**	**0.03**	**0.01**	**<.001**	**0.01**	**0.05**	**0.05**	**0.02**	**.012**	**0.01**	**0.09**	**0.01**	**0.003**	**2.84**	**Y**
Viewed pleasant images	**0.16**	**0.07**	**.018**	**0.03**	**0.29**	**0.03**	**0.01**	**<.001**	**0.02**	**0.05**	**0.07**	**0.03**	**.011**	**0.02**	**0.12**	**0.01**	**0.003**	**1.82**	**Y**
Alcohol intake																			
Eudaimonic reward-related behavior	**0.43**	**0.05**	**<.001**	**0.32**	**0.54**	**0.01**	**0.003**	**.004**	**0.003**	**0.01**	**-0.02**	**0.01**	**.002**	**-0.04**	**-0.01**	**0.004**	**0.001**	**3.05**	**Y**
Had a sense of accomplishment	**0.40**	**0.07**	**<.001**	**0.27**	**0.54**	**0.01**	**0.003**	**.005**	**0.002**	**0.01**	**-0.04**	**0.01**	**<.001**	**-0.06**	**-0.02**	**0.004**	**0.001**	**2.93**	**Y**

Notes: The final column indicates whether or not the Sobel test indicated a significant mediated effect; Y = Yes, N = No.

Discussion

Engaging in alternate reward-related behaviors to "replace" and reduce health-compromising behaviors may be an appealing strategy. However, findings from the current study suggest that in everyday life when individuals engage in reward-related behaviors—predominately hedonic and secondary reward-related behaviors—they may be more likely to eat rewarding, unhealthy foods. Yet, when individuals engage in eudaimonic reward-related behaviors or have a sense of accomplishment, individuals may be less likely to drink alcohol. The current research had a number of study strengths that fortify the veracity of these results. First, the study used a 4-day ambulatory electronic diary design that assessed behavior hour by hour within young adults in their everyday lives. This greatly enhanced the ecological validity of the results. Second, the study used lag-hour analyses to ensure that the reward-related behavior preceded the eating or alcohol use in time, and ruled out a number of confounds. Although causality cannot be determined by the current research, this type of analysis bolsters the notion that there is a direction of causality from reward-related behavior to eating and alcohol use. Third, the study measured a number of different types of reward-related behavior and eating behavior, which captured a breadth of behavior and allowed for observation of patterns across this breadth of behavior.

In addition, I tested if any effect may be more evident in certain individuals, namely individuals who possess the G allele of the OPRM1 gene. However, moderated effects were largely inconclusive because only a few (2 out of a potential of 144) significant moderated effects emerged, which suggests they may have appeared due to chance.

I also tested if any effect may be explained by increased self-reported feelings of reward, which may reflect increased mesolimbic dopamine reward pathway activity in response to

reward-related behavior. By and large, results indicated that engaging in reward-related behavior predicted increases in feelings of reward, and increased feelings of reward predicted increases in eating and alcohol use. This suggests that—contrary to hypothesis—engaging in reward-related behavior may not block the mesolimbic dopamine reward pathway and reduce the likelihood of other reward-related behaviors like eating and alcohol use. Instead, it is possible that engaging in reward-related behavior potentiates the mesolimbic dopamine reward pathway and makes it more sensitive to other reward-related behaviors like eating and alcohol use. The latter is consistent with the idea that dopamine in the mesolimbic dopamine reward pathway reinforces eating and alcohol use (Volkow et al., 2013) but also suggests that behaviors other than eating and alcohol use can initiate the reinforcement of eating and alcohol use.

Nevertheless, although Sobel tests indicated the significance of the meditated effects by self-reported feelings of reward, a number of the associations between engaging in reward-related behavior and eating/alcohol use remained when controlling for self-reported feelings of reward, and a few were not mediated. This indicates partial mediation and suggests that engaging in reward-related behavior may also affect eating/alcohol use through other mechanisms. For instance, many of the reward-related behaviors that predicted greater eating were behaviors that boost self-image (e.g., donating to those in need, getting special recognition, seeing an attractive person gazing at you). Perhaps individuals wanted to celebrate after these events by eating rewarding foods; this is consistent with research on motives for eating palatable foods (Burgess et al., 2014). For example, individuals report wanting palatable foods "to celebrate a special occasion," and this motive predicts eating outside of the laboratory (Boggiano et al., 2015). This explanation is speculative and future research should test this mechanism by including measures of motives for eating each hour as well as test other alternative mechanisms.

Overall, results from the current study are contrary to related prior work. To begin, rodent research has suggested that engaging with rewards such as social interaction and novel objects can reduce drug-seeking behavior up to certain doses of the drug (Mattson, Williams, Rosenblatt, & Morrell, 2001; Reichel & Bevins, 2008; 2011). There are a few factors that may explain the discrepancy between findings from rodent research and the current study findings. Foremost, the rodent research examined effects of reward-related behavior on cocaine use in particular whereas I tested associations between reward-related behavior and eating/alcohol use. Although my reward competition hypothesis posited domain-general effects among various reward-related behaviors, perhaps domain-specific effects better describe behavior intersections (e.g., reward-related behavior has a different effect on eating vs. alcohol use vs. cocaine use). This is plausible because, although all reward-related behaviors activate the neural reward systems, other unique neural and psychosocial pathways modulate eating versus alcohol use versus cocaine use. And even in the current study, reward-related behavior differentially related to eating and alcohol use.

Another factor that may explain the discrepancy in findings between the current study and the prior rodent research is timing and availability. That is, prior rodent research concurrently tested whether rodents preferred contexts where they could engage with an alternate reward-related behavior or contexts where they could seek cocaine (Mattson, Williams, Rosenblatt, & Morrell, 2001; Reichel & Bevins, 2008; 2011). This paradigm gave rodents only two options during one time period. In contrast, in the current study participants were outside any controlled laboratory context where they would be limited in choices, and instead the participants could potentially engage in multiple reward-related behaviors at once. Moreover, eating and alcohol use *the hour after* engaging in multiple reward-related behaviors were the

dependent variables for the current study. It is possible having a smaller time increment between reward-related behavior and eating/alcohol use would yield different results.

The current study findings that greater engagement in reward-related behavior in the current hour predicted eating in the following hour also challenge prior cross-sectional research in humans. Specifically, greater engagement in cognitive-enriching and social activities has been associated with lower Body Mass Indexes, and—when the effort needed to obtain favorite foods increased—individuals have chosen their favorite social activities over their favorite food (Carr & Epstein, 2017). Again, there are a couple of differences between the current and prior research that may explain the discrepant findings. Foremost, Carr and Epstein (2017) used Body Mass Index as a proxy for real world eating behavior and tested cross-sectional associations between this proxy and engagement in reward-related behavior reported retrospectively. The current study measured eating behavior and reward-related behavior each hour for four days in an individual's everyday life, which may better capture real world eating behavior and its antecedents.

In addition, Carr and Epstein (2017) gave individuals a hypothetical choice between only two behaviors (e.g., engaging in favorite social activity and getting favorite food) in the context of increasing efforts needed for getting their favorite food. Given that in the real world individuals are not limited to choosing just one behavior and can obtain food with minimal effort, the current study provides a more ecologically valid test of how reward-related behavior and eating intersect in the real world. However, again eating behavior *in the hour after* reward-related behavior was the dependent variable in the current study whereas Carr and Epstein (2017) examined a concurrent choice. Thus, it is plausible that measuring eating-related behavior after an event versus concurrently may explain discrepant results between the two studies.

It is important to note that not all of the current study findings challenge prior research in humans. In particular, the current study found that engaging in eudaimonic reward-related behaviors or having a sense of accomplishment in the current hour predicted less alcohol use in the next hour, which is consistent with some prior human research. That is, adolescents with greater neural sensitivity to eudaimonic rewards had less neural sensitivity to hedonic rewards (Telzer, Fuligni, & Galvan, 2015), and this neural sensitivity to eudaimonic rewards predicted decreases in externalizing symptoms like drinking alcohol (Telzer, Fuligni, Lieberman, & Galván, 2013). It is unclear why eudaimonic reward-related behaviors would "compete" with alcohol use whereas other reward-related behaviors would not (or would increase eating) but it is possible that idiosyncratic aspects of the behaviors matter. Prior research suggests that engaging in eudaimonic reward-related behaviors provides self-acceptance (Machado & Cantilino, 2017); this may reduce motivation for a psychoactive drug like alcohol, which alters one's self-awareness (Hull, 1981). This explanation is very speculative and future research should more directly test it by, for example, including measures of self-awareness each hour.

The current study results should be interpreted in light of study limitations. First, although the study used time-lag analysis and ruled out a number of confounds by including them as covariates, the methodology cannot completely rule out reverse causation or third variables that explain the results. It is possible that, for example, engaging in less eudaimonic reward-related behavior increased alcohol use. It is also possible the association between engaging in reward-related behavior and eating/alcohol use emerged because individuals who reported engaging in reward-related behavior were generally more likely to report any behavior. However, given that the current research identified differential associations among individual reward-related behaviors, eating, and alcohol use suggests that at least the latter explanation is

unlikely. A second weakness of the current study was that eating behavior and alcohol use were measured with self-report questions rather than more objective measures such as the Remote Food Photography Method (Martin et al., 2012) or wearable sensors (Bedri et al., 2015). Third, one may argue that reporting on eating and alcohol use may cause eating and alcohol use to change, which is also known as reactivity. This is a possibility in the current study but to mitigate this concern I tested if day in the study covaried with results and it did not. Moreover, several other ambulatory electronic diary studies on eating and alcohol use have not observed reactivity (le Grange, Gorin, Dymek, & Stone, 2002; Litt, Cooney, & Morse, 1998; Stein & Corte, 2003). Third, this study tested effects in a sample of healthy, young adults not seeking treatment. Different effects may be observed in clinical samples seeking behavioral treatment for addictive-like eating and/or alcohol use. Indeed, anecdotal support comes from clinicians who treat patients with addictions (Blum et al., 2011; Sullivan, 2015). These limitations notwithstanding, the current ambulatory electronic diary study challenges the notion that engaging in alternative reward-related behavior can offset unhealthy eating behavior and shows the boundaries of engaging in alternative reward-related behavior in offsetting alcohol use. Importantly, these results suggest that clinicians may not want to recommend a "replacement" strategy to those who are trying to change their eating behavior, and exercise caution in recommending "replacement" strategies for those who are trying to change their alcohol use.

Conclusion

Summary of Findings

In this dissertation, I tested if behaviors that activate the neural reward system—specifically, the mesolimbic dopamine reward pathway—can "compete." In Study 1, I found that eating sweet high fat foods acutely reduced alcohol cravings no more than watching a neutral

video for heavy drinking adults. I also found preliminary results that eating sweet high fat foods amplified physiological indices of alcohol craving for those with the G allele of the OPRM1 gene, which is an endophenotypic marker that confers greater risk for alcohol use disorder. Eating 150 calories of any food also amplified alcohol craving for those with a parental family history of alcoholism, a proxy for a biological vulnerability for greater eating and alcohol use. However, eating 450 calories of any food may attenuate alcohol craving for that group.

In Study 2, I found that engaging in multiple reward-related behaviors in one hour—especially hedonic and secondary reward-related behaviors—predicted greater likelihood of eating in the following hour for young adults. However, engaging in eudaimonic reward-related behaviors or having a sense of accomplishment in one hour predicted less likelihood of alcohol use in the following hour for young adults. A majority of these results were partially mediated by increases in self-reported feelings of reward. This is consistent with the notion that dopamine in the mesolimbic dopamine reward pathway reinforces eating and alcohol use (Volkow et al., 2013), and suggests dopamine activity from non-eating/drinking reward-related behaviors may reinforce eating and alcohol use. However, these results also suggest that other mechanisms may explain associations between reward-related behavior and eating/alcohol use, and that unique neural and psychosocial pathways that modulate eating versus alcohol use might explain how engaging in reward-related behavior differentially affects these behaviors.

Clinical & Public Health Significance

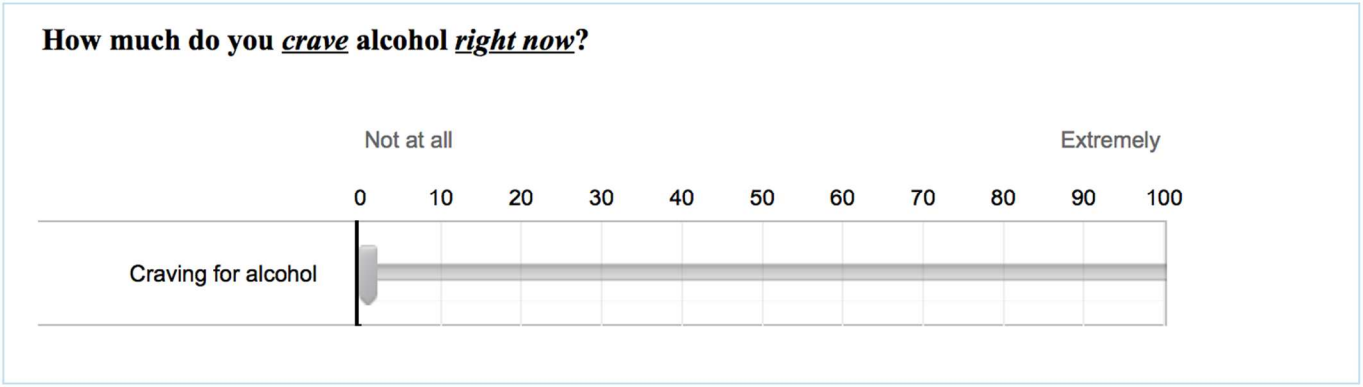
In sum, across two studies there was minimal support for the hypothesis that behaviors activating the neural reward system "compete." The supportive findings (e.g., large doses of any food dampening alcohol cravings for those with a family history of alcoholism, eudaimonic reward-related behavior reduced alcohol use) were only evident under specific contexts or for

specific people. Clinicians and the community should thus exercise caution when suggesting any "replacement" strategies. For example, it is possible that eating a larger amount of calorie-dense, nutritious foods may be an option for reducing alcohol cravings among those with a family history of alcoholism. It also possible that encouraging engagement in eudaimonic reward-related behaviors (e.g., affirming values, donating, expressing compassion, spending time with a monogamous partner, feeling a sense of accomplishment) may be an option for preventing heavier alcohol use in young adults. However, future experimental work testing these contextually specific treatment and prevention strategies is needed to support causality of effects.

In contrast, across two studies there was strong support for the notion that behaviors activating the neural reward system may encourage each other. In particular, eating sweet high-fat foods or a small amount of any food may encourage further alcohol use for specific individuals at risk for alcohol use disorder, and engaging in multiple reward-related behavior may encourage new events of eating for young adults. By and large, this suggests that clinicians and the community should not generally recommend "replacement" strategies. In particular, Alcoholics Anonymous should perhaps cease recommending this strategy to those who are trying to quit drinking. And although clinicians and the community may want to encourage increases in non-consumptive reward-related behaviors because of other potential benefits, it is important that clinicians and the community stay cognizant of how any changes in other reward-related behaviors may influence eating and alcohol use. Therefore, the current research strongly supports efforts to simultaneously change multiple behaviors at once (Prochaska & Prochaska, 2011), and strongly supports future research that will determine the most effective strategies for doing so.

Appendix I

Craving – Visual Analog Scale



Biphasic Alcohol Effects Scale

The following adjectives describe feelings that some people have after drinking alcohol. Please rate the extent to which drinking alcohol has produced these feelings in you at the present time.

1) Difficulty Concentrating

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

2) Down

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

3) Elated

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

4) Energized

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

5) Excited

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

6) Heavy head

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

7) Inactive

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

8) Sedated

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

9) Slow thoughts

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

10) Sluggish

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

11) Stimulated

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

12) Talkative

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

13) Up

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

14) Vigorous

0 1 2 3 4 5 6 7 8 9 10
(not at all) (extremely)

Family History Interview

Family Grid

This instrument is to be administered as a personal interview

This questionnaire concerns your family and experiences that family members have had with alcohol. Please begin by describing your family by indicating in *Column A* the total number of biological (i.e., related by blood) relatives (both living and dead) that you have in each category on each side of your family. For example, although you have only one biological grandmother on your mother's side (as shown in Column A), you may have several aunts (your mother's biological sisters) or none at all. If you have no relatives in a particular category, put the letter "N" (for "None") in Column A in the space next to the category. If you don't know how many relatives you have in a category, put "DK" (for "Don't Know") in the space.

Next, please indicate in *Column B* the number of biological relatives (both living and dead) in each category that had in the past, or currently have, what you would call a significant drinking problem, one that did, or should have, led to treatment. Some signs that drinking may be a problem include legal problems (e.g., drunk driving violations), health problems (e.g., cirrhosis of the liver, alcohol withdrawal symptoms), relationship problems (e.g., arguments about alcohol with family members), or work/school problems (e.g., poor performance, absenteeism resulting from alcohol use), or actual treatment (e.g., detox or rehab, AA meeting attendance). If you have no relatives with alcohol problems in a particular category, put the letter "N" (for "None") in Column A in the space next to the category. If you don't know how many relatives you have in a category, put "DK" (for "Don't Know") in the space.

Biological Relative	A	B
Mother' Side	Number of biological relatives	Number of relatives with alcohol problems
Grandmother	1	
Grandfather	1	
Mother	1	
Aunt(s)		
Uncle(s)		
Father's Side		
Grandmother	1	
Grandfather	1	
Father	1	
Aunt(s)		
Uncle(s)		
Siblings		
Brother(s)		
Sister(s)		

Appendix II

Standard Drinks

Standard Alcohol Drinks

Liquor	
1 **shot** of **80 proof** liquor = **1 drink** 1 **shot** of **151 proof** liquor = **2 drinks**	1 shot = 
Mixed drinks = 1 drink/every shot in the drink Typically 1 8oz mixed drink has 1 shot in it 1 **Long Island** iced tea = **4 drinks** (typically)	8oz Mixed Drink = 
1 **cosmopolitan, appletini,** etc. = **2 drinks** (typically)	Cosmopolitan = 
1 **"fifth"** or **"750"** of **80 proof** liquor = 26oz = **17 drinks**	1 "fifth" = 
1 **"liter"** of **80 proof** liquor = 1.14 liters = 38.5oz = **26 drinks**	1 "liter" = 
1 **"handle"** of **80 proof** liquor = 1.75 liters = 59oz = **39 drinks**	1 "handle" = 

Beer	
1 **beer** = 12oz = **1 drink**	12oz Beer = 
1 **quart** of beer = 32oz = **3 drinks**	Quart of Beer = 
1 **pitcher** of beer = 60 ounces = **5 drinks**	Pitcher of Beer = 
1 **pony keg** of beer = 8 gallons = **80 drinks**	Pony Keg = 
1 full-size **keg** of beer = 15 gallons = **160 drinks**	Full Sized Keg = 

Other	
1 **wine** = 5 to 7oz = **1 drink**	5-7oz Wine = 
1 **bottle of wine** = **5 drinks**	Bottle of Wine = 
1 flute of **champagne** = 6oz = **1 drinks**	Flute of Champagne = 
1 **malt beverage** (Schmirnoff Ice, etc.) = **1 drink**	Malt Beverage = 
1 **"forty"** of **malt liquor** = 40oz = **6 drinks**	1 "Forty" = 

Conversions		
1 Cup = 8oz	1 *Solo* cup = 16oz (typically)	1 liter = 33.8oz
1 Pint = 16oz	1 gallon = 128oz	

Positive Affect and Negative Affect Schedule (*Items included)

The following is a list of words that describe different feelings and emotions. Read each item and then indicate to what extent you feel this way right now at the present moment, on a scale from 1-5:

1 = very slightly or not at all
2 = a little
3 = moderate
4 = quite a bit
5 = extremely

___1. Interested
___2. Distressed*
___3. Excited
___4. Upset
___5. Strong
___6. Sad
___7. Guilty
___8. Scared
___9. Hostile
___10. Enthusiastic*
___11. Proud
___12. Irritable
___13. Alert
___14. Ashamed
___15. Inspired
___16. Nervous
___17. Determined
___18. Attentive
___19. Jittery
___20. Active
___21. Afraid*
___22. Happy*
___Rewarded*
___Pleasured*

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