PURDUE UNIVERSITY
GRADUATE SCHOOL
Thesis/Dissertation Acceptance

This is to certify that the thesis/dissertation prepared

By Kenny A. Karyadi

Entitled

THE EFFECTS OF ALCOHOL ODOR CUES ON FOOD AND ALCOHOL ATTENTIONAL BIAS, CRAVINGS, AND CONSUMPTION

For the degree of Doctor of Philosophy

Is approved by the final examining committee:

Melissa A. Cyders, PhD
Chair

Jesse Stewart, PhD

Catherine Mosher, PhD

Nicholas J. Grahame, PhD

To the best of my knowledge and as understood by the student in the Thesis/Dissertation Agreement, Publication Delay, and Certification Disclaimer (Graduate School Form 32), this thesis/dissertation adheres to the provisions of Purdue University's "Policy of Integrity in Research" and the use of copyright material.

Approved by Major Professor(s): Melissa A. Cyders, PhD

Approved by: Nicholas J. Grahame, PhD 6/9/2015
Head of the Departmental Graduate Program Date

THE EFFECTS OF ALCOHOL ODOR CUES ON FOOD AND ALCOHOL ATTENTIONAL BIAS, CRAVINGS, AND CONSUMPTION

A Dissertation

Submitted to the Faculty

of

Purdue University

by

Kenny A. Karyadi

In Partial Fulfillment of the

Requirements for the Degree

of

Doctor of Philosophy

August 2016

Purdue University

Indianapolis, Indiana

ACKNOWLEDGEMENTS

I would like to thank my mentor, Dr. Melissa A. Cyders, for her guidance throughout the entire process. I would also like to thank you my committee members—Drs. Jesse Stewart, Catherine Mosher, and Nicholas Grahame—for their expertise and feedback.

TABLE OF CONTENTS

ABSTRACT

Karyadi, Kenny A. Ph.D., Purdue University, August 2016. The Effects of Alcohol Odor Cues on Food and Alcohol Attentional Bias, Cravings, and Consumption. Major Professor: Melissa A. Cyders.

In order to elucidate the role of classical conditioning in food and alcohol co-consumption, the present study examined: (1) the effects of alcohol odor cues on alcohol and food cravings and attentional bias (bias in selective attention toward either food or alcohol pictures relative to neutral pictures); and (2) the role of alcohol odor cue elicited cravings and attentional biases on subsequent consumption. Participants (n = 77; mean age = 30.84, SD = 9.46; 51.9% female, 83.1% Caucasian) first completed the lab portion of the study. In this portion, they were exposed to alcohol and neutral odorants, after which their food and alcohol cravings and attentional bias were assessed. Participants then received an online survey the next day, on which they reported their level of food and alcohol consumption following the lab portion of the study. Using repeated measures analysis of covariance, alcohol odor cues were differentially effective in increasing food and alcohol attentional bias and cravings (Fs= 0.06 to 2.72, ps= 0.03 to 0.81). Using logistic and multiple regressions, alcohol odor cue elicited alcohol attentional bias, food attentional bias, and food cravings were associated with later alcohol consumption, but not with later food consumption or concurrent consumption (βs = -0.28 to 0.48, ps = 0.02

to 0.99; Exp(B)s = 0.95 to 1.83, *ps* = 0.33 to 0.91). Overall, alcohol odor cues can become conditioned stimuli that elicit conditioned food-related and alcohol-related responses, both of which persist long enough to motivate later alcohol consumption; however, these conditioned responses might not persist long enough to motivate later food or concurrent consumption. These findings serve as a first step in clarifying the role of classical conditioning in concurrent consumption. In particular, they suggest that additional empirical investigations are needed to: (1) clarify the classical conditioning mechanisms underlying concurrent consumption; and (2) examine whether interventions targeting classical conditioning mechanisms are effective for reducing alcohol use.

INTRODUCTION

Food and alcohol consumption frequently co-occur at both non-clinical and clinical levels (Heatherton, Polivy, Herman, & Baumeister, 1991; Polivy & Herman, 1976; Ward & Mann, 2000). For instance, food consumption is greater when simultaneously paired with or preceded by alcohol consumption among male subjects (Caton, Ball, Ahern, & Hetherington, 2004; Hetherington, Cameron, Wallis, & Pirie, 2001) and among overweight female college students (Hoffman & Friese, 2008). Additionally, problematic alcohol users engage in more problematic eating behaviors compared to non-problematic alcohol users (Stewart, Brown, Devoulyte, Theakston, & Larsen, 2006; Taylor, Peveler, Hibbert, & Fairburn, 1993) and problematic eaters engage in more problematic alcohol use behaviors compared to non-problematic eaters (Braun, Sunday & Halmi, 1994; Dunn, Larimer, & Neighbors, 2002; Grilo, White, & Masheb, 2009; Lundholm, 1989). Considering that concurrent consumption at higher levels can potentially result in harmful consequences—including physical health problems and higher mortality risk (Hingson, Heeren, Winter, & Wechsler, 2005; Hudson, Hiripi, Pope, & Kessler, 2007), there is a need to better understand the mechanisms underlying food and alcohol co-consumption, and to better identify effective treatment and prevention targets (Sinha & O'Malley, 2000).

Possible Mechanisms

Multiple possible mechanisms could account for co-occurring food and alcohol consumption. Food and alcohol consumption might co-occur due to: (1) third factor predictors; (2) the disinhibiting effects of alcohol on food consumption; and (3) classical conditioning processes. These mechanisms have received differing levels of empirical support, with the first two mechanisms having received wider empirical support than classical conditioning processes. However, it should be noted that this discrepancy in empirical support apply primarily to studies utilizing human participants rather than non-human animal subjects (Cooper & Turkish, 1989; Mineka, 1975), which will not be the focus of the present study. Furthermore, because these mechanisms might not be mutually exclusive in their influence on concurrent food and alcohol consumption, determining the independent role of each mechanism in concurrent consumption can be challenging (Karyadi, Coskunpinar, Entezari, Long, & Cyders, 2014). In particular, although third factor predictors have been examined independent of the pharmacological effects of alcohol on food intake and independent of classical conditioning processes (Sinha & O'Malley, 2000), studies demonstrating the disinhibiting pharmacological effects of alcohol on food consumption have not taken into consideration the influence of conditioned stimuli on food and alcohol consumption (Caton et al., 2004; Hetherington et al., 2001).

In particular, there is a paucity of studies that have elucidated how classical conditioning processes might influence co-occurring food and alcohol consumption. Indeed, only one pilot study to date has provided preliminary findings regarding the effects of conditioned food-related and alcohol-related stimuli on conditioned food-

related and alcohol-related responses, and on subsequent food and alcohol consumption (Karyadi & Cyders, 2014). This lack of research is unfortunate, especially considering that a better understanding of classical conditioning processes can potentially guide research that aims to develop more effective treatment and prevention approaches (e.g. interventions focused on attentional training and cue exposure) for comorbid problematic eating and alcohol use behaviors (Castellanos et al., 2009; Drummond, Cooper, & Glautier, 1990; Jansen, 1998; Schoenmakers et al., 2010). Building upon the results of the aforementioned pilot study, the present study examined: (1) whether alcohol-related stimuli can serve as conditioned stimuli that elicit conditioned food-related and alcohol-related responses, including food and alcohol attentional bias and cravings; and (2) whether those cued conditioned responses would influence food and alcohol consumption.

Disinhibiting Effects of Alcohol on Food Consumption.

Alcohol consumption has been thought to increase food consumption through its disinhibiting pharmacological effects (Yeomans, Caton, & Hetherington, 2003). Multiple prior studies have provided support for this. For instance, among college students, higher doses of alcohol increase food intake (Caton et al., 2004; Caton, Marks, & Hetherington, 2005; Caton, Bate, & Hetherington, 2007; Polivy & Herman, 1976). Additionally, consumption of alcoholic beverages increases food consumption to a greater degree compared to consumption of non-alcoholic beverages (Hetherington et al., 2001; Poppitt & Prentice, 1996; Yeomans, 2010; Yeomans, Hails, & Nesic, 1999).

There are multiple pathways through which the disinhibiting pharmacological effects of alcohol might facilitate increased food consumption. First, consumption of alcohol increases impulsiveness and reduces behavioral control (Reed, Levin, & Evans, 2012; Quay, 1997), both of which might cause individuals to be less likely to consider negative experiences that arise from overeating and more likely to engage in increased food consumption (Guerrieri, Nederkoorn, & Jansen, 2007; Jasinska et al., 2012; Svaldi, Brand, & Tuschen-Caffier, 2010). Second, intoxicated individuals might have a more difficult time retrieving and maintaining standards that regulate eating behaviors from memory (Baumeister, Heatherton, & Tice, 1994) and might become more likely to engage in increased food consumption due to a lack of regulating standards. Third, alcohol consumption might reduce self-awareness of the amount of food that one consumes (Hull & Bond, 1986; Wolfe & Maisto, 2000), which lead to increased food consumption (Heatherton et al., 1991; Ward & Mann, 2000). Fourth, alcohol consumption stimulates appetite, which facilitates increased likelihood of and need for food consumption (Caton et al., 2007; Yeomans, Hails, & Nesic, 1999).

Taken together, previous empirical findings and theoretical pathways have provided support for a mechanism, wherein the disinhibiting pharmacological effects of alcohol facilitate increased food consumption (Hetherington et al., 2001; Sinha & O'Malley, 2000; Stewart et al., 2006; Yeomans et al., 1999). At the same time, the disinhibiting pharmacological effects of alcohol on food consumption might also be influenced and partially explained by classical conditioning processes. In particular, participants in these prior studies orally consumed alcohol, and were exposed to the sight and smell of alcohol (e.g. visual, odor, and taste stimuli). Because food and alcohol

consumption are frequently paired (Caton et al., 2004), these alcohol-related stimuli might become both conditioned food-related stimuli and conditioned alcohol-related stimuli. In turn, conditioned food-related stimuli might elicit conditioned food-related responses, while conditioned alcohol-related stimuli might elicit conditioned alcohol-related responses. Finally, these conditioned food-related and alcohol-related responses might influence food and alcohol consumption, respectively.

In this way, exposure to alcohol-related stimuli has the potential to influence both food and alcohol consumption through classical conditioning mechanisms and independent of the disinhibiting pharmacological effects of alcohol on food consumption (Hetherington et al., 2004; Sinha & O'Malley, 2000). If this is truly the case, intervening based on only the disinhibiting pharmacological effects of alcohol (e.g. limiting amount of alcohol consumed during eating or abstaining from alcohol consumption completely) might be effective for reducing alcohol intake, but might not necessarily be effective for reducing food intake. Due to classical conditioning processes, even if alcohol is not consumed and does not exert a disinhibiting effect on food consumption, individuals would still be at risk for overeating if they are around alcohol-related stimuli (e.g. sight and smell of alcohol). Relatedly, through the same classical conditioning processes, these individuals would also be at risk for drinking excessively if they are around alcohol-related stimuli. As such, in addressing alcohol and food co-consumption, interventions should focus not only on (1) the disinhibiting pharmacological effects of alcohol consumption on food intake but also on (2) classical conditioning processes.

Classical Conditioning as a Mechanism

Classical conditioning is a form of associative learning, wherein a meaningful stimulus (unconditioned stimulus) that elicits a natural response (unconditioned response) becomes associated with a neutral stimulus. After repeated pairings between neutral and unconditioned stimuli, the neutral stimulus becomes powerful enough (conditioned stimulus) to elicit the same response by itself (conditioned response). This process of association was most famously demonstrated in Ivan Pavlov's classic experiments (Pavlov, 1927). Pavlov noticed that dogs naturally produced the salivation reflex (unconditioned response) when presented with food (unconditioned stimulus), but not when presented with the noise of a buzzer (neutral stimulus). However, after the buzzer was repeatedly paired with the food, the buzzer became powerful enough (conditioned stimulus) to elicit the salivation reflex by itself (conditioned response). In this way, the buzzer (conditioned stimulus) elicited a consumptive response (conditioned response) that prepares the dog for eventual food presentation and consumption.

Following Pavlov's experiment, numerous studies have demonstrated that classical conditioning processes apply to a wide variety of consumptive behaviors—including food and alcohol consumption (Childress, Ehrman, Rohsenow, Robbins, & O'Brien, 1992; Kennedy, Katz, Neitzert, Ralevsky, & Mendlowitz, 1995). However, relative to the simple example outlined above, classical conditioning processes apply differently to food and alcohol consumption (see Appendix B, Figure B1). For instance, neutral environmental stimuli present during consumption (e.g. sight and smell of food or alcohol) become associated with both consumption (unconditioned stimuli) and the effects of consumption (unconditioned responses) (Jansen, 1998; O'Brien, Childress,

McLellan, & Ehrman, 1992). After repeated pairings, those neutral stimuli become conditioned food-related or conditioned alcohol-related stimuli that elicit multiple different conditioned responses, all of which can potentially prepare individuals for and motivate alcohol or food consumption (Jansen, 1998; O'Brien et al., 1992).

In particular, alcohol consumption produces numerous physiological (e.g. cardiovascular and digestive changes) and psychological effects (e.g. euphoria and excitement) (O'Brien et al., 1992). Both alcohol consumption and its effects tend to occur repeatedly within the context of multiple neutral environmental stimuli (O'Brien et al., 1992). These environmental stimuli (e.g. environment of use, sight and smell of alcohol) become repeatedly paired with: (1) pleasant and euphoric effects of alcohol consumption (Townshend & Duka, 2001); (2) physiological changes following alcohol consumption (e.g. decreased heart rate and body temperature) (Siegel, 1983); and (3) withdrawal effects produced by declining alcohol plasma level (Wikler, 1973). Through repeated associations with these differing effects, formerly neutral environmental stimuli become conditioned alcohol-related stimuli that produce conditioned responses (Drummond et al., 1990; O'Brien et al., 1992). There are multiple forms of these conditioned responses (e.g. conditioned drug-like responses, conditioned attentional bias, conditioned compensatory responses, and conditioned tolerance), all of which prepare individuals for the effects of alcohol consumption and all of which motivate alcohol consumption (Drummond et al., 1990).

Similar to alcohol consumption, food consumption also produces numerous psychological (e.g. satiety and euphoria) and physiological effects (e.g. increased gut motility and salivation). These psychological and physiological effects tend to occur

within the context of multiple neutral environmental stimuli (Jansen, 1998; Wardle, 1990). The environmental stimuli present during food consumption (e.g. environment of food intake, smell and sight of food) become paired with (1) physiological changes associated with food consumption (Wardle, 1990) and (2) pleasant and euphoric effects associated with food consumption (Castellanos et al., 2009), but not necessarily with (3) withdrawal effects or hunger following food deprivation (Cravens & Renner, 1969; Mineka, 1975; Jansen, 1998; Wardle, 1990). Through repeated pairings, these environmental stimuli become conditioned food-related stimuli that elicit multiple different forms of conditioned responses (e.g. conditioned compensatory responses and conditioned attentional bias), all of which prepare eaters for the effects of food consumption and all of which motivate food consumption (Wardle, 1990).

In general, the role of classical conditioning in food consumption (Jansen, 1998; Wardle, 1990) and alcohol consumption (O'Brien, Childress, McLellan, & Ehrman, 1990) is well documented. However, it is unclear whether classically conditioned stimuli (e.g. the smell of alcohol or food) might contribute to co-occurring food and alcohol consumption independent of the disinhibiting pharmacological effects of alcohol on food consumption (Caton et al., 2007; Hetherington et al., 2001; Yeomans et al., 1999). Because food and alcohol consumption are often paired (Caton et al., 2004; Hetherington et al., 2001), food-related stimuli and alcohol-related stimuli might become associated with both food-related and alcohol-related effects. Food-related stimuli and alcohol-related stimuli then both become conditioned alcohol-related and conditioned food-related stimuli. These conditioned stimuli might in turn elicit both conditioned alcohol-related responses and conditioned food-related responses—both of which motivate

alcohol use and eating behaviors, respectively. In this way, alcohol-related and food-related stimuli can lead to concurrent food and alcohol consumption through classical conditioning mechanisms and independent of the pharmacological effects of alcohol on food consumption.

There is some support for the role of classical conditioned processes in concurrent food and alcohol consumption. For instance, alcohol-related stimuli can serve as conditioned stimuli capable of eliciting conditioned smoking related responses, which in turn influence smoking behaviors (Burton & Tiffany, 1997; Drobes, 2002; Glautier, Clements, White, Taylor, & Stolerman, 1996; Sayette, Martin, Wertz, Perrott, & Peters, 2005). Furthermore, food-related and alcohol-related stimuli are similar because they are associated with consumptive behaviors and because of their characteristics (e.g. sight, smell, and taste). Due to their similarities, the conditioned responses and consumptive behaviors elicited by alcohol-related stimuli should also be elicited by food-related stimuli and vice versa (Pearce, 1987; Till & Priluck, 2000). Collectively, food-related and alcohol-related stimuli should have comparable effects on both conditioned food-related and alcohol-related responses, and on subsequent food and alcohol consumption. However, preliminary findings from a recent pilot study suggested that food-related stimuli and alcohol-related stimuli might have differential effects on both conditioned food-related and alcohol-related responses, and on subsequent food and alcohol consumption (Karyadi & Cyders, 2014).

Using a sample of college students who regularly co-consume alcohol and food, this pilot study examined: (1) whether food-related stimuli and alcohol-related stimuli (e.g. alcohol and food odor cues) serve as conditioned stimuli that would elicit

conditioned food-related and alcohol-related responses; and (2) whether those cued conditioned responses would influence both food and alcohol consumption. Although results were generally non-significant (see Appendix E), examination of the pattern of results indicated meaningful trends and associations that should be more fully examined in a larger sample that is more properly powered to find effects. In particular, alcohol-related stimuli were more consistent than food-related stimuli in eliciting conditioned food-related responses and conditioned alcohol-related responses. In turn, cued conditioned food-related and alcohol-related responses might be more consistent in increasing alcohol consumption compared to food consumption. Overall, these findings suggest that alcohol-related stimuli might be more effective than food-related stimuli in eliciting conditioned food-related and alcohol-related responses, both of which might be inconsistent in influencing food and alcohol consumption. However, there are factors that might have influenced the results of the pilot study—including (1) the small sample size (n = 20) and (2) the use of only two questions to measure consumption.

The present study addressed these limitations by recruiting a larger sample of participants and by including a more comprehensive set of consumption measurements. However, the present study only examined the effects of alcohol-related stimuli rather than both alcohol-related stimuli and food-related stimuli. There are three main reasons for this change: (1) previous studies have established that alcohol consumption can disinhibit food consumption (Hetherington et al., 2001; Sinha & O'Malley, 2000; Stewart et al., 2006; Yeomans et al., 1999), but no studies have examined whether food consumption can increase alcohol consumption; (2) alcohol-related stimuli have been shown to influence other consumptive responses and behaviors, including conditioned

smoking responses and subsequent smoking behaviors (Burton & Tiffany, 1997; Drobes, 2002); and (3) pilot study results indicated that alcohol-related stimuli might more consistently influence conditioned food-related and alcohol-related responses compared to food-related stimuli. Collectively, these findings suggest that alcohol-related stimuli might more effectively serve as conditioned stimuli that can elicit conditioned food-related and alcohol-related responses, both of which might influence food and alcohol consumption, respectively. As such, the present study examined whether: (1) alcohol-related stimuli are capable of eliciting both conditioned alcohol-related and food-related responses, including food and alcohol attentional bias and cravings; and (2) whether those cued conditioned responses can influence both alcohol and food consumption.

Third Factor Predictors

Independent of classical conditioning mechanisms and the disinhibiting pharmacological effects of alcohol on food intake, third factor predictors also influence concurrent food and alcohol consumption (Sinha & O'Malley, 2000). For instance, distress coping (tendency to cope with distressing emotions through risky behaviors) is a common third factor predictor that drives concurrent food and alcohol consumption through multiple pathways (Fischer, Settles, Collins, Gunn, & Smith, 2012; Fischer, Smith, Annus, & Hendricks, 2007; Rush, Becker, & Curry, 2009; Stewart et al., 2006). First, alcohol and food are effective in providing emotional relief. Reliance on both food and alcohol for providing emotional relief from distress might over time facilitate the development of concurrent food and alcohol consumption (Agras & Telch, 1998; Grant, Stewart, O'Connor, Blackwell, & Conrod, 2007; Hohlstein, Smith, & Atlas, 1998).

Second, both food and alcohol are perceived and expected to be effective for reducing distress (Cooper, Frone, Russell, & Mudar, 1995; Hohlstein et al., 1998). Expectations about the effectiveness of food and alcohol for reducing distress might over time facilitate engagement in concurrent food and alcohol consumption, especially in times of distress. Third, some individuals experience impairments in impulse control when faced with distressing emotional states, which might in turn drive them to engage in food and alcohol consumption either concurrently or sequentially at excessive levels and without forethought in order to immediately alleviate those distressing emotional states (Fischer et al., 2007; Fischer et al., 2012; Rush et al., 2009).

These findings indicate that distress coping is a third factor predictor for concurrent food and alcohol consumption; at the same time, it should be noted that other findings also suggest multiple other possible third factor predictors—such as learned expectancies, other dispositional traits, family history, dieting severity and food deprivation, responsiveness to endogenous opioid peptide, and dysfunctions in neurotransmitter systems (Sinha & O'Malley, 2000). Overall, prior findings provide support for a mechanism, wherein third factor predictors might influence concurrent food and alcohol consumption. More importantly, these prior studies have also examined the role of third factor predictors on concurrent food and alcohol consumption independent of the pharmacological effects of alcohol on food consumption and independent of classical conditioning processes (Agras & Telch, 1998; Cooper et al., 1995; Fischer et al., 2012; Rush et al., 2009). Because the independent role of third factor predictors in concurrent consumption is well understood, the present study will not focus on third factor predictors.

Cravings as a Conditioned Response

Alcohol-related stimuli can serve as conditioned stimuli that elicit conditioned alcohol and food cravings and consumption through at least two pathways. First, declining alcohol level following alcohol consumption (unconditioned stimulus) elicits withdrawal like responses (unconditioned responses) that can be subjectively perceived as alcohol cravings (Cooney, Litt, Morse, Bauer, & Gaupp, 1997; Drummond et al., 1990; Siegel, 1983; McCusker & Brown, 1990). According to the withdrawal model, formerly neutral alcohol-related stimuli (e.g. sight and smell of alcohol) become associated with declining alcohol level and with subsequent withdrawal-like responses, and consequently become conditioned alcohol-related stimuli capable of eliciting conditioned pharmacological withdrawal (Ludwig & Wikler, 1974; Wikler, 1973). The need to relieve or avoid aversive conditioned withdrawal responses, which are subjectively perceived alcohol cravings, might then motivate alcohol consumption (Drummond et al., 1990; O'Brien et al., 1990). Indeed, prior studies have provided support for this (Cooney et al., 1997; Edwards, 1990; Siegel, 1983; McCusker & Brown, 1990; Tiffany, 1990). Similarly, food deprivation might elicit withdrawal like responses, which can be perceived as hunger and food cravings (Wardle, 1990). Because alcohol and food consumption are frequently paired (Caton et al., 2004), formerly neutral alcohol-related stimuli (e.g. sight and taste of alcohol) might become paired with these withdrawal like responses. These formerly neutral alcohol-related stimuli then become conditioned food-related stimuli capable of eliciting conditioned food withdrawal like responses. These conditioned withdrawal like responses, which are subjectively perceived as food cravings, then motivate food consumption (Wardle, 1990). However, it

should be noted that findings in support of this pathway have been mixed (Cravens & Renner, 1969; Mineka, 1975; Sahakian, Lean, Robbins, & James, 1981; Wooley & Wooley, 1981).

Second, food and alcohol consumption (unconditioned stimulus) produce a variety of physiological effects (unconditioned responses)—such as increased salivation, heart rate, and pulse rate (Cox & Klinger, 1988; Wardle, 1990). According to compensatory response models (Powley, 1977; Siegel, 1983), formerly neutral alcohol-related stimuli (e.g. sight and smell of alcohol) become repeatedly associated with these physiological effects. These formerly neutral alcohol-related stimuli then become both (1) conditioned alcohol-related stimuli and (2) conditioned food-related stimuli, both of which are capable of eliciting conditioned physiological responses opposite to the physiological effects produced by consumption (Jansen, 1998; O'Brien et al, 1992; Siegel, 1983; Wardle, 1990). For instance, exposure to these conditioned stimuli produce changes in heart rate, salivation, blood pressure, skin conductance, and gastric activity that are opposite in direction to the physiological effects of food consumption (Nederkoorn, Smulders, & Jansen, 2000; Powley, 1977; Wardle, 1990) and alcohol consumption (Newlin, 1985; Newlin, 1986; Staiger & White, 1988). In turn, these physiologically compensatory conditioned responses prepare individuals for the physiological effects of food or alcohol consumption (Laibson, 2001). However, when alcohol or food is not consumed following the induction of these conditioned compensatory responses, these conditioned responses are subjectively perceived as cravings (Jansen, 1998; Powley, 1977; Siegel, 1983). In turn, food and alcohol cravings

might motivate individuals to consume food and alcohol, respectively, for the purpose of reducing cravings (Cox & Klinger, 1988; Jansen, 1998; Laibson, 2001).

In sum, because food and alcohol consumption are often paired (Caton et al., 2004; Hetherington et al., 2001), formerly neutral alcohol-related stimuli can become conditioned stimuli that are capable of eliciting food and alcohol cravings and consumption through the aforementioned pathways (Cox & Klinger, 1988; Jansen, 1998; Wardle, 1990). In this way, alcohol cue elicited conditioned food and alcohol cravings increase the likelihood of food and alcohol co-consumption independent of the pharmacological effects of alcohol consumption on food consumption (Caton et al., 2007; Hetherington et al., 2001; Yeomans et al., 1999). Previous studies have provided partial support of this by demonstrating the effectiveness of alcohol-related stimuli in influencing cigarette cravings and consumption (Burton & Tiffany,1997; Drobes, 2002; Glautier et al., 1996; Sayette et al., 2005), and in influencing both food and alcohol cravings (Karyadi & Cyders, 2014). A better understanding of whether alcohol-related stimuli serve as conditioned stimuli that elicit conditioned alcohol and food cravings, as well as subsequent food and alcohol consumption, can elucidate more effective approaches for intervening on concurrent problematic alcohol and food consumption (Conklin & Tiffany, 2002). For instance, cue exposure treatments can be effective for reducing problematic food and alcohol consumption (Drummond & Glautier, 1994; Toro et al., 2003). Elucidating the role of alcohol-related stimuli in concurrent food and alcohol consumption can guide the development of effective cue exposure treatments that operate by targeting conditioned food and alcohol cravings elicited by alcohol-related stimuli (Drummond et al., 1990; Jansen, 1998).

Attentional Bias as a Conditioned Response

Alcohol-related stimuli can also serve as conditioned stimuli that elicit alcohol and food attentional bias and consumption. Alcohol and food consumption (unconditioned stimulus) produce pleasant effects (unconditioned responses)—such as relaxation, euphoria, and satiety. Formerly neutral alcohol-related stimuli (e.g. sight and smell of alcohol) that are present during alcohol or food consumption become paired with these pleasant effects. Through repeated pairings with the pleasant effects of consumption, these formerly neutral alcohol-related stimuli become both (1) conditioned food-related stimuli and (2) conditioned alcohol-related stimuli, both of which are imbued with enhanced reinforcing properties (Dobson & Dozois, 2004; Field et al., 2007). According to the incentive sensitization model (Castellanos et al., 2009; Dobson & Dozoiz, 2004; Townshend & Duka, 2001; Yokum, Ng, & Stice, 2011), these imbued conditioned stimuli elicit both conditioned food and alcohol attentional bias—defined as the tendency to more selectively attend to cues predictive of consumption and the pleasant effects of consumption (e.g. food and alcohol pictorial cues) over non-predictive cues (e.g. non-food and non-alcohol pictorial cues). In turn, conditioned food attentional bias and alcohol attentional bias incentivize and facilitate food seeking and alcohol seeking behaviors, respectively (Field et al., 2007; Schoenmakers et al., 2010; Shafran, Lee, Cooper, Palmer, & Fairburn, 2007; Smeets, Roefs, van Furth, & Jansen, 2008).

In sum, exposure to alcohol-related stimuli can potentially increase food and alcohol consumption through conditioned food and alcohol attentional bias. Previous findings have provided some support for this. Exposure to alcohol-related stimuli increase alcohol attentional bias among college students (Coskunpinar, Dir, Karyadi,

Koo, & Cyders, 2013; Cox, Brown, & Rowlands, 2003; Duka & Townshend, 2004), problematic drinkers (Stormark, Laberg, Hordby, &Hugdahl, 2000), and heavy drinkers (Kareken et al., 2012). In addition, preliminary findings suggest that exposure to alcohol-related stimuli also increase food and alcohol attentional bias among college students who regularly co-consume alcohol and food (Karyadi & Cyders, 2014). Finally, food attentional bias and alcohol attentional bias influence food consumption and alcohol consumption, respectively (Calitri, Pothos, Tapper, Brunstrom, & Rogers, 2010; Cox et al., 2002; Field & Eastwood, 2005; Hepworth, Mogg, Brignell, & Bradley, 2010; Nederkoorn et al., 2000). Collectively, these findings suggest that alcohol-related stimuli can serve as conditioned stimuli that elicit conditioned alcohol and food attentional biases, which then increase alcohol consumption and food consumption, respectively (Calitri et al., 2010; Cox, Hogan, Kristian, & Race, 2002; Nederkoorn et al., 2000).

Attentional bias is most commonly measured through the assessments of eye movements (Castellanos et al., 2009; Field, Mogg, Zetteler, & Bradley, 2004; Mogg, Bradley, Field, & De Houwer, 2003; Schoenmakers et al., 2010) and through reaction time tasks (Castellanos et al., 2009; Field & Eastwood, 2005). Using reaction time tasks (e.g. visual probe tasks), cognitive attentional bias is inferred through participants' timed performance on a primary task, during which food or alcohol pictorial cues and matched control pictorial cues are presented. Faster reaction times toward food or alcohol pictorial cues relative to neutral pictorial cues are thought to reflect greater food or alcohol cognitive attentional bias. Assessments of eye movements measure visuospatial selective attention toward food or alcohol pictorial cues relative to neutral pictorial cues, with greater attention toward food and alcohol pictorial cues compared to neutral pictorial cues

being indicative of food and alcohol attentional bias. Two forms of attentional bias are assessed using measurements of eye movements: (1) duration attentional bias reflects a bias in maintained attention and (2) direction attentional bias reflects a bias in initial orienting of attention (Catellanos et al., 2009; Field et al., 2004; Field, Munafo, & Braken, 2009). In sum, different measurements reflect different aspects of attentional bias, which are likely differentially associated with food and alcohol consumption (Castellanos et al., 2009; Field et al., 2004; Field et al., 2009). As such, the present study utilized multiple measurements to tap into different aspects of attentional bias (cognitive, duration, and direction attentional bias).

Overall, independent of the pharmacological effects of alcohol (Caton et al., 2007; Hetherington et al., 2001; Yeomans et al., 1999), alcohol-related stimuli might become conditioned stimuli that elicit both conditioned food and alcohol attentional bias, as well as subsequent food and alcohol consumption (Calitri et al., 2010; Cox et al., 2002; Karyadi & Cyders, 2014; Papsies et al., 2008). Previous studies have provided support of this (Calitri et al., 2010; Cox et al., 2002; Karyadi & Cyders, 2014). A better understanding of such classical conditioning process can elucidate effective intervention and treatment targets (Chambers, Lo, & Allen, 2008). For instance, attentional training interventions are effective for reducing attentional bias and consumption (Hardman, Rogers, Etchells, Houstoun, &Munaro, 2013; Schoenmakers et al., 2010; Werthmann, Field, Roefs, Nederkoorn, & Jansen, 2014). Elucidating the role of alcohol-related stimuli in food and alcohol attentional bias and consumption will guide the development of more effective attentional training interventions that operate by re-training alcohol cue elicited attentional bias (Castellanos et al., 2009; Schoenmakers et al., 2010).

Study Aims

Alcohol odor cues were used as the stimuli for eliciting food and alcohol cravings; however, participants were also exposed to food or alcohol pictorial cues and matched control pictorial cues to assess their attentional bias. Exposure to odor and pictorial cues has been shown to increase food cravings (Fedoroff, Polivy, & Herman, 2003; Hawk, Baschnagel, Ashare, & Epstein, 2004) and alcohol cravings (Laberg, 1990; Litt & Cooney, 1999; Litt, Cooney, Kadden, & Gaupp, 1990); however, odor cue exposure might be more powerful in eliciting alcohol cravings (Litt & Cooney, 1999) and food cravings (Drobes et al., 2001; Hawk et al., 2004) compared to pictorial cue exposure. Similarly, alcohol odor cues were used as the stimuli for eliciting food and alcohol attentional bias and sympathetic arousal. Alcohol odor cues are effective in increasing alcohol attentional bias (Coskunpinar et al., 2013; Cox et al., 2003; Duka & Townshend, 2004; Field & Eastwood, 2005) and food attentional bias (Castellanos et al., 2009; Eiler, Dzemidzic, Case, Considine, & Kareken, 2012). The present study assessed three forms of attentional bias: cognitive, duration, and direction attentional bias.

The first aim of the present study was to examine the effects of odor cues (alcohol vs. water) on food and alcohol cravings and attentional bias. The second aim of the study was to examine the effects of alcohol odor cue elicited food and alcohol cravings and attentional bias on post-lab food and alcohol consumption—defined as consumption immediately following dismissal from the lab and on the day of participation. In exploratory analyses, the present study also examined the effects of odor cues on food and alcohol sympathetic arousal (as measured by pupillary response), and the effects of cued sympathetic arousal on post-lab alcohol and food consumption. I hypothesized that:

(1) alcohol odor cues would elicit increased alcohol and food cravings (hypotheses 1 and 2); (2) alcohol and food cravings elicited by alcohol odor cues would predict greater post-lab alcohol and food consumption, respectively (hypotheses 3 and 4); (3) alcohol odor cues would elicit increased alcohol and food attentional bias—including cognitive, direction, and duration attentional bias (hypotheses 5 and 6); and (4) alcohol and food attentional bias elicited by alcohol odor cues would predict greater post-lab alcohol and food consumption, respectively (hypotheses7 and 8).

METHOD

General Design

In the lab portion of the study, all participants engaged in four randomized experimental trials: (1) trial 1 assessed the effects of alcohol odor cues on alcohol attentional bias; (2) trial 2 assessed the effects of alcohol odor cues on food attentional bias; (3) trial 3 assessed the effects of neutral odor cues on alcohol attentional bias; and (4) trial 4 assessed the effects of neutral odor cues on food attentional bias. Alcohol and food cravings were assessed before these four trials and after each of the four trials. During each of the four trials, alcohol and food attentional bias were assessed using the visual probe tasks and using eye-tracking measurements, and alcohol and food sympathetic arousal were assessed using eye-tracking measurements. The day after the lab portion of the study, all participants received an online survey, on which they reported their level of alcohol and food consumption after their dismissal from the lab and on the day of their participation (post-lab alcohol and food consumption).

Participants

Participants were recruited from advertisements posted on a college classifieds section, online classifieds section (e.g. Craigslist), and on public advertisement spaces. On the advertisement, all potential participants were informed that they might be eligible for a study that examines eating and alcohol use behaviors. They were also informed that

participation in the study involved exposure to alcohol odors, as well as exposure to food and alcohol pictures. The advertisement also noted that they must provide their phone number for a phone interview to assess eligibility and would receive $20 for participating in the study. During the phone interview, participants who indicated that they (1) consume beer at least once a week, (2) enjoy co-consuming beer and pizza, (3) are fluent in English, and (4) are at least 18 years old were considered eligible and were recruited for participation in the study. These eligibility criteria ensured that participants have a history of concurrent alcohol and food consumption.

Analyses with G*Power 3.1.7 (Buchner, Erdfelder, Faul, & Lang, 2009) indicated that approximately 80 eligible participants were needed to have adequate power (95%) to detect medium effect sizes; therefore, the present study recruited 80 participants (50% men and 50% women). In recruiting these participants, 199 participants were contacted for a phone interview, with 129 participants being considered eligible for the study. However, 49 participants scheduled for participation did not show up for their scheduled study session. Out of the 80 participants who completed the study, 3 were excluded due to random responding (e.g. quick and mindless clicking to survey questions) and/or inattention during experimental trials (e.g. inconsistent clicking on visual probe tasks, closing eyes, moving odor tube). The final sample consisted of 77 participants (see Appendix A, Table A1 for participant characteristics; final $n = 77$). Comparison of excluded ($n = 3$) and included ($n = 77$) participants revealed a number of differences across key study variables (see the Results section); therefore, analyses were run both with and without these participants. Because the pattern of results did not differ significantly, these participants were excluded from study analyses.

Measurements and Materials

Table A1 (Appendix A) presents descriptive information for post-lab food and alcohol consumption variables, as well as descriptive information on covariates and past consumption. Table A2 (Appendix A) presents descriptive information for food and alcohol cravings, attentional bias, sympathetic arousal, and odor ratings across the four experimental trials.

Alcohol and Food Cravings

Alcohol cravings were measured using the Alcohol Urge Questionnaire (AUQ; Bohn, Krahn, & Staehler, 1995). The AUQ consists of 8 items, which assess a participant's urge for an alcoholic drink at the time the questionnaire is completed (sample item: I crave a drink right now). All items of the AUQ were scored along a 7-point Likert scale, with response options ranging from (1) "Strongly disagree" to (7) "Strongly agree." The AUQ was calculated as a summed value, with higher values indicating greater levels of alcohol cravings. In the present study, alcohol cravings were measured prior to odor exposure (baseline alcohol cravings) and after each of the four experimental trials. Internal consistency coefficients were comparable across baseline and the four experimental trials: (1) 0.86 at baseline; (2) 0.91 following exposure to neutral odors and alcohol pictures; (3) 0.90 following exposure to neutral odors and food pictures; (4) 0.93 following exposure to alcohol odors and alcohol pictures; and (5) 0.93 following exposure to alcohol odors and food pictures. Drummond and Phillips (2002) provided validity evidence for the AUQ.

Food cravings were measured using the Food Cravings Questionnaire-State (FCQ-S; Cepeda-Benito, Gleaves, Williams, & Erath, 2001). The FCQ-S consists of 15 items assessing participant's cravings for food at the time the questionnaire is completed (sample item: I'm craving one or more specific foods). Response options ranged from (1) "Strongly disagree" to (4) "Strongly agree". The FCQ-S was calculated as a mean value, with higher mean values indicative of greater levels of food cravings. Food cravings were also measured prior to odor exposure (baseline food cravings) and after each of the four experimental trials. Internal consistency coefficients were comparable across baseline and the four experimental trials: (1) 0.92 at baseline; (2) 0.94 following exposure to neutral odors and alcohol pictures; (3) 0.94 following exposure to neutral odors and food pictures; (4) 0.95 following exposure to alcohol odors and alcohol pictures; and (5) 0.94 following exposure to alcohol odors and food pictures. Cepeda-Benito et al. (2001) provided validity evidence for the FCQ-S.

Cognitive Attentional Bias

The visual probe task (see Appendix B, Figure B2) was used to measure cognitive attentional bias (Castellanos et al., 2009; Field & Eastwood, 2005). The visual probe task was presented on a computer screen using the Eprime software (Psychology Software Tools, Inc.). More specifically, participants faced a computer screen, on which a pair of alcohol or food pictures and matched control pictures was presented side-by-side for 1000 milliseconds (see Appendices F and G). All pictures have been used in previous studies of attentional bias (Castellanos et al., 2009; Field et al., 2004). After picture offset, a visual probe (e.g. either a right or a left arrow) appeared where one of the

pictures had previously been presented. Participants were instructed to identify the visual probe as quickly as possible by pressing either the left mouse button when they see a left arrow or the right mouse button when they see a right arrow. Faster reaction times to visual probes replacing alcohol-related or food-related pictures versus matched control pictures are indicative of greater alcohol or food cognitive attentional bias, respectively (Castellanos et al., 2009; Field et al., 2004). In particular, cognitive attentional bias was calculated by subtracting the average time (in milliseconds) to respond to control pictures from the average time (in milliseconds) to respond to either alcohol or food pictures, with greater positive values being indicative of greater cognitive attentional bias toward food or alcohol pictures. Because pictorial cues were presented for 1000 milliseconds during visual probe tasks, cognitive attentional bias reflects a bias in maintained attention (Field & Cox, 2008). Across the four experimental trials, four cognitive attentional bias variables were calculated: (1) alcohol cognitive attentional bias following neutral odors; (2) alcohol cognitive attentional bias following alcohol odors; (3) food cognitive attentional bias following neutral odors; and (4) food cognitive attentional bias following alcohol odors.

Duration and Direction Attentional Bias

Attentional bias was also measured using an eye-tracking device (Applied Science Laboratories, Bedford, MA). Using this device, the eye movements of each participant were assessed while participants completed the visual probe task. The eye-tracker device contains the eye camera and the eye illuminator, as well as an automatic tracking mirror that moves the camera and the illuminator to follow the motion of a subject's eye.

Measurement of eye movements was recorded digitally on the Eye-Tracker Interface PC. Using the eye-tracking device, two forms of attentional bias were calculated: duration attentional bias and direction attentional bias. Across the four experimental trials, four duration attentional bias and four direction attentional bias variables were calculated: (1) alcohol duration and direction attentional bias following neutral odors; (2) alcohol duration and direction attentional bias following alcohol odors; (3) food duration and direction attentional bias following neutral odors; and (4) food duration and direction attentional bias following alcohol odors.

Across each of the four experimental trials, average gaze duration (in milliseconds) on food-related or alcohol-related pictures and control pictures was measured via the eye-tracking device. Average gaze duration data were then used to calculate food and alcohol duration attentional bias. Specifically, using the ASL Results software (Applied Science Laboratories, Bedford, MA), average gaze duration was computed by averaging the total amount of time that eye fixations were directed at the regions of the screen occupied by alcohol or food pictures and control pictures. Overall average gaze duration was calculated by subtracting average gaze duration on control pictures (in milliseconds) from average gaze duration on food or alcohol pictures (in milliseconds), with greater positive values being indicative of greater average gaze duration on alcohol or food pictures relative to neutral pictures. Because average gaze duration has been thought to assess a bias in maintained attention (Catellanos et al., 2009; Field et al., 2004; Field & Cox, 2008), greater positive average gaze duration is indicative of greater food or alcohol duration attentional bias.

Across each of the four experimental trials, number of fixations on food or alcohol pictures and control pictures was also assessed using the eye-tracking device. Data on fixations were then used to calculate food and alcohol direction attentional bias. Using the ASL Results software (Applied Science Laboratories, Bedford, MA), percentage of fixations for alcohol and food pictures was calculated by: (1) dividing the total number of fixations on alcohol or food pictures by the total number of fixations on both alcohol or food pictures and control pictures; and (2) subsequently multiplying the result of this division by 100 (Field & Cox, 2008; Field et al., 2004; Schoenmakers et al., 2010). Greater percentage values are indicative of a greater number of fixations toward alcohol or food pictures relative to matched control pictures. Because fixation count percentage has been thought to reflect a bias in initial orientation of attention toward food or alcohol pictures relative to neutral pictures (Castellanos et al., 2009; Field & Cox, 2008; Field et al., 2004), greater percentage values are indicative of greater food or alcohol direction attentional bias.

Post-Lab Alcohol and Food Consumption

Participants' level of post-lab food and alcohol consumption was assessed through a number of questions. Participants were asked whether they consumed (1) alcohol, (2) food, and (3) both alcohol and food concurrently (e.g. whether participants ate anything when they consumed alcohol) following their participation in the lab portion of the study (post-lab concurrent consumption). Response options for these items were dichotomous (e.g. yes or no). Participants were also asked to indicate: (1) how full they felt after eating (post-lab food satiety), with response options ranging from (0) "Extremely hungry" to

(100) “Extremely full” (Holt et al., 1995; Miller, Petocz, & Farnakalidis, 1995); (2) whether the amount of food they ate is typical (post-lab food typicality), with response options ranging from (0) “Less than usual” to (100) “More than usual”; (3) the number of alcoholic drinks they consumed after participating in the study (post-lab alcohol use), with response options ranging from (1) “No drinks” to (7) “Nine or more drinks” (Daily Drinking Questionnaire; Collins, Parks, & Marlatt, 1985); and (4) whether the amount of alcohol they consumed after participating in the study is typical (post-lab food typicality), with response options ranging from (0) “Less than usual” to (100) “More than usual.” With the exception of post-lab food satiety and post-lab alcohol use, which were adapted from State Hunger Index and Daily Drinking Questionnaire (Collins et al., 1985; Holt et al., 1995), post-lab consumption questions were developed by the lab.

Covariates

The present study also assessed a number of variables that could influence food and alcohol attentional bias, cravings, and consumption. These variables include time of day, day of the week, state hunger, and demographic characteristics (e.g. age, gender, and race). Time of day in military time and day of the week (1 = Monday and 7 = Sunday) were recorded when participants arrived at the lab. Furthermore, participants also reported on their current level of hunger (Holt et al., 1995), with response options ranging from (1) “Extremely full” to (7) “Extremely hungry,” and on their demographic characteristics on an online survey.

Past Alcohol and Food Consumption

Participants were interviewed using the Daily Drinking and Eating Questionnaire, which is a modified version of the Daily Drinking Questionnaire (DDQ; Collins et al., 1985). The Daily Drinking and Eating Questionnaire assesses both eating and alcohol use behaviors in the past two weeks. During the interview, participants were asked about: (1) the number of alcohol drinks consumed each day of the past two weeks; (2) the number of hours of alcohol use each day of the past two weeks; (3) the type of alcoholic beverage consumed each day of the past two weeks; and (4) the type of food consumed while drinking alcohol each day of the past two weeks. Using this interview information, four consumption frequency variables were calculated: (1) number of days of general alcohol consumption; (2) number of days of beer consumption; (3) number of days of concurrent food and alcohol consumption; and (4) number of days of concurrent beer and food consumption. Kivlahan, Marlatt, Fromme, Coppel, and Williams (1990) provided validity evidence for the Daily Drinking Questionnaire.

Problematic alcohol use was assessed using the Alcohol Use Disorder Identification Test (AUDIT; Babor, de la Fuente, Saunders, & Grant, 1992). The AUDIT consists of 10 items ($\alpha = 0.83$ from the current sample), which assess problematic patterns of alcohol use. The first eight items were rated on a 5-point Likert scale (e.g. 0 = Never to 4 = Daily). Items 9 and 10 were rated on a 3-point Likert scale, which was then converted into a 5-point Likert scale (e.g. 0 = No; 2 = Yes, but not during the last year; and 4 = Yes, during the last year). The AUDIT assesses multiple aspects of problematic alcohol use behaviors: (1) items 1-3 assess level of alcohol consumption; (2) items 4-6 assess alcohol dependence symptoms; and (3) items 7-10 assess negative alcohol

consequences. In the present study, the AUDIT was calculated as a summed value, ranging from 0 to 40, with higher summed values indicating greater levels of problematic alcohol use. Reinert and Allen (2002) discussed validity evidence of the AUDIT.

Problematic eating was assessed using the Three Factor Eating Questionnaire-R18 (TFEQ-R18; Karlsson, Persson, Sjostrom, & Sullivan, 2000). The first seventeen items were rated on a 4-point Likert scale. The last item was rated on an 8-point Likert scale, which was then converted to a 4-point Likert scale. The TFEQ-R18 ($\alpha = 0.84$ from the current sample) measures three different aspects of eating behaviors: (1) restrained eating, which refers to conscious restriction of food intake in order to control body weight or to promote weight loss; (2) uncontrolled eating, which refers to the tendency to eat more than usual due to a loss of control over food intake; and (3) emotional eating, which refers to the inability to resist emotional cues that lead to excessive eating. In order to get an overall index of problematic eating patterns, the TFEQ-R18 was calculated as a mean value in the present study, with mean values ranging from 1 to 4. Higher mean values indicate greater levels of problematic eating patterns. Karlsson et al. (2000) provided validity evidence for the TFEQ-R18.

Sympathetic Arousal

The eye-tracking device also allowed for the measurement of pupil diameter (in millimeters) in response to alcohol or food pictures relative to matched control pictures. Pupil diameter data can consequently be used to calculate food and alcohol sympathetic arousal. For each of the four trials, averaged pupil diameter for neutral pictures (in millimeters) was subtracted from averaged pupil diameter for food or alcohol pictures (in

millimeters), with greater positive values indicating greater pupil diameter in response to alcohol or food pictures relative to neutral pictures. Because pupil diameter size has been thought to reflect degree of interest and sympathetic arousal (Blaha, 1977; Monti, Rohsenow, Abrams, & Binkoff, 1988), greater positive pupil diameter values indicate greater levels of interest or sympathetic arousal in response to food pictures (food sympathetic arousal) or alcohol pictures (alcohol sympathetic arousal) compared to matched control pictures. Across the four experimental trials, four sympathetic arousal variables were calculated: (1) alcohol sympathetic arousal following neutral odors; (2) alcohol sympathetic arousal following alcohol odors; (3) food sympathetic arousal following neutral odors; and (4) food sympathetic arousal following alcohol odors.

Odor Cues

Two pilot surveys were conducted to determine the type of alcohol odor cue to utilize in the present study. In the first pilot survey, college students who were enrolled in introductory level psychology courses were asked to complete a screening survey. Participants were asked about the type of alcoholic beverages they most frequently consume. Out of the 1050 students who completed the survey, 33.9% declined to answer, 18.9% reported consuming mixed drinks most frequently, 18.7% reported consuming other types of drinks most frequently, 15% reported consuming beer most frequently, 7.1% reported consuming liquor shots most frequently, and 5.9% reported consuming wine most frequently. However, participants were not able to specify more specific types of alcoholic beverages on the screening survey. As such, there was no general consensus on which one specific type of alcoholic beverage is most frequently consumed.

A second pilot survey was conducted on a sample of college students (n = 305). On this survey, participants were able to list the types of alcoholic beverages they consume most frequently. Across participants, types of alcoholic beverages were separated and then summed to get counts of different types of alcoholic beverages that participants endorsed. In terms of most frequently consumed alcoholic beverage, 26.2% reported consuming mixed drinks most frequently, 24.3% reported consuming shots most frequently, 20% reported consuming beer most frequently, 12.1% reported consuming wine most frequently, 11.1% did not provide a response, and 6.2% reported consuming a variety of other drinks. Again, there was no general consensus on which one specific type of alcoholic beverage is most frequently consumed. However, because several participants indicated that they consumed beer (~15-20%) most often across both surveys, the present study utilized beer as the alcohol odorant.

Bud Light served as the beer odorant (4.20% alcohol by volume) because it is considered to be the most frequently consumed light beer in the United States (Barrow, 2014). Water served as the control odorant because it is considered to be a non-appetitive neutral odorant. Unlike appetitive odorants (e.g. light beer and grape juice), water can be considered a non-appetitive stimulus that is not linked with caloric-related consumption of food and/or alcohol (Kareken et al., 2010). In this way, exposure to water odorant is less likely to elicit food and alcohol responses (e.g. cravings and attentional bias) and consumption compared to exposure to appetitive odorants. Because of this, the effects of alcohol odor cues in the present study can be more easily differentiated from the effects of neutral odor cues.

Odor Ratings

Across the four experimental trials, participants rated both beer and water odorants on three characteristics (see Appendix A, Table A2): (1) intensity (1 = weak, 2 = moderate, 3 = strong, 4 = very strong); (2) pleasantness (1 = very unpleasant, 2 = unpleasant, 3 = pleasant, 4 = very pleasant); and (3) representativeness (1 = not at all representative, 2 = not representative, 3 = representative, 4 = very representative). Across the two trials involving alcohol pictures during the visual probe task, participants indicated that beer odorant was more intense (Alcohol: $M = 2.87$, $SD = 0.80$; Water: $M = 1.35$, $SD = 0.66$; $p < 0.001$), pleasant (Alcohol: $M = 2.94$, $SD = 0.63$; Water: $M = 2.71$, $SD = 0.68$; $p = 0.03$), and representative (Alcohol: $M = 3.16$, $SD = 0.77$; Water: $M = 1.55$, $SD = 0.70$; $p < 0.001$) than water odorant. Across the two trials involving food pictures during the visual probe task, participants indicated that the beer odorant was more intense (Alcohol: $M = 2.90$, $SD = 0.83$; Water: $M = 1.38$, $SD = 0.72$; $p < 0.001$) and representative (Alcohol: $M = 3.20$, $SD = 0.65$; Water: $M = 1.85$, $SD = 1.56$; $p < 0.001$) than the water odorant; however, the difference in pleasantness rating fell short of significance (Alcohol: $M = 2.93$ $SD = 0.60$; Water: $M = 2.77$, $SD = 0.54$; $p = 0.07$). For the two trials involving beer odor cue exposure, participants rated the beer odorant as being moderate to strong in intensity, pleasant, and representative. Alcohol odor ratings did not significantly differ (ps= 0.26 to 0.84) between these two trials. For the two trials involving water odor cue exposure, participants rated the water odorant as being weak in intensity, pleasant, and not representative. Water odor ratings did not significantly differ (ps= 0.13 to 0.65) between these two trials.

Odor Cue Administration

Beer and water odorants were delivered via an 8-channel air dilution olfactometer (Bragulat et al., 2008; Kareken et al., 2004). Using the olfactometer, odors can be detected within 2 seconds of delivery (Bragulat et al., 2008; Kareken et al., 2004). The olfactometer generated airflow through the use of an oil-less pump, with the air being humidified and filtered with a charcoal filter. Odor delivery was controlled using the Dasylab software and a Personal Daq/56 module (IO-Tech, Inc., Cleveland, OH). Small polytetrafluoroethylene tubes were used to deliver air to the participants' nose at 2.0 liters per minute (lpm). Throughout the procedure, the airstream consisted of a constant 1.0 lpm stream and a second 1.0 lpm stream of one of the two odorants through one of the glass vials containing the odorant.

Computers and Programs

Three computers were used during the study session (see Appendix B, Figure B3): (1) the participants' computer; (2) the Eye-Tracker Interface computer; and (3) the olfactometer laptop. Participants completed the study procedures (e.g. online survey and the four experimental trials) while sitting at the participants' computer. The eye-tracking apparatus was positioned immediately beneath the participants' computer monitor. The participants' seating was placed 24 inches away from this apparatus, which is considered the optimal distance for pupil detection (EyeTracker Systems Manual, 2009). This computer also held the Eprime programs, which served multiple functions during the study session: (1) work in conjunction with the olfactometer laptop to control odor delivery; (2) administer the visual probe tasks; (3) contain target points for eye

calibration; and (4) collect odor ratings from participants. Because there were four experimental trials, four different Eprime programs were created for use in the study: (1) beer odor administration and (2) water odor administration with food and matched control pictures during the visual probe task; and (3) beer odor administration and (4) water odor administration with alcohol and matched control pictures during the visual probe task (see Appendix B, Figure B4). The Eye-Tracker Interface computer was the computer researchers used to set up the eye-tracker and collect eye movement data during the visual probe task. In particular, this computer contained the eye-tracker software, which was used to control the eye-tracking device and to collect eye movement data. The eye-tracker monitoring system, placed next to this computer, was used for monitoring and calibrating participants' eye movements. The olfactometer laptop contained the Dasylab software, which provided direct control over the olfactometer. In particular, the aforementioned Eprime programs were programmed to send commands to the Dasylab software, which in turn dictated when odors are delivered to participants.

Procedure

Preparation

First, prior to participants arriving in the lab, the order of the four experimental trials was randomized. Second, the two odorants used in the study were prepared in two separate test tubes. Third, the eye-tracker software was uploaded on the Eye-Tracker Interface computer, and the eye-tracking device and the eye-tracker monitoring system were turned on. Fourth, the Dasylab program on the olfactometer laptop was connected to

the participants' computer, which ensured that the Eprime programs on the participants' computer can control the olfactometer throughout the procedure. Fifth, an online survey and the Eprime programs were prepared on the participants' computer. The online survey contained measures of food and alcohol cravings (e.g. five measurements across baseline and the four trials), past food and alcohol consumption, state hunger, and demographic characteristics. Sixth, the pre-assigned participation number was inputted for the online survey and the E-prime programs for the four experimental trials. Finally, two informed consent forms were prepared for each participant, one for them to sign if they agree to participate in the study and one for them to keep for their records.

Lab and Post-Lab Sessions

When participants arrived at the lab, time of day in military time and day of the week were recorded. Participants were then seated in front of the participants' computer. On this computer, participants completed the online survey questionnaires and the visual probe task. In order to control for the effects of food and alcohol consumption on study results, participants were then asked about the last time they consumed food or alcohol; fortunately, none of the participants reported eating less than 2 hours prior to the study and/or consuming alcohol less than 8 hours prior to the study. Participants were then given a brief explanation of the study, and were instructed to review and sign the consent forms. Afterward, participants completed baseline measures of food and alcohol cravings, as well as a measure of their current level of hunger. Using the calibration targets in each Eprime program, participants then had their eyes calibrated on target points. Following successful calibration, participants then engaged in one of the four experimental trials.

During each experimental trial, participants were first exposed to an odor, after which they rated the intensity, pleasantness, and representativeness of this odor. After providing these odor ratings, they then completed the rest of the trial. Participants heard the "ready" "sniff" command, during which a 2-second odorant was delivered via an 8-channel air dilution olfactometer, followed by a tone indicating that they could exhale (Bragulat et al., 2008; Kareken et al., 2004). Following this, participants completed the visual probe task, which was repeated a total five times. Following the five visual probe tasks, the "ready" "sniff" tone was presented again with the odorant and was followed by another set of visual probe tasks. In total, this sequence was repeated a total of six times during each experimental trial, with participants being exposed to 30 visual probe tasks and 6 odorants. After completing one trial, participants re-assessed their alcohol or food cravings. Following this re-assessment, participants engaged the next experimental trial until all four trials were completed. The same instructions and sequence, including eye calibration, applied to all experimental trials (see Appendix B, Figure B4).

After completion of all four experimental trials, participants were interviewed about their eating and drinking habits in the past two weeks and completed the online survey. They were then informed that they would be emailed a short online survey the next day, which will ask them about health behaviors they have recently engaged in. On this survey, participants were asked their level of food and alcohol consumption after their dismissal from the lab and on the day of their participation. Participants had the day after they received the online survey to complete the online survey and were debriefed with more information following survey completion. The entire study procedure was approximately 70-80 minutes in length: (1) study explanation and consenting process

took approximately 5 minutes to complete; (2) each experimental trial lasted approximately 4 minutes and had a setup time of approximately 5 minutes, resulting in approximately 35-40 minutes for completing all four experimental trials; (3) the lab online survey and the interview took approximately 30 minutes to complete; and (4) the post-lab online survey took approximately 5 minutes to complete.

Data Preparation

Three types of data files were cleaned prior to analyses. First, all self-report data were compiled through: (1) a lab online survey, which contains questionnaires for cravings, covariates, and past consumption; (2) a post-lab online survey, which contains post-lab consumption questions; (3) an interview using Daily Drinking and Eating Questionnaire (Collins et al., 1985); and (4) a listing of time of day in military time and day of the week of participation for all participants. All data from were exported into a single Excel file. Because the four experimental trials were randomized, data cleaning included re-organizing alcohol and food cravings data. Data were then imported into SPSS 18. All data discussed below were also uploaded into the same SPSS file after cleaning was completed and after the appropriate variables were created to run the necessary statistical analyses to test study hypotheses.

Second, the Eprime programs generated individual Excel files for each of the four experimental trials. In this way, each participant had four Excel files—with each file containing information on (1) the type of odors presented (e.g. water or beer), (2) the visual probe picture pairs presented (e.g. food vs. alcohol pictures with matched control pictures), (3) the order of the visual probe picture pairs, (4) the side the visual probe

presented during each visual probe session, and (5) the participant's reaction time (in milliseconds) each time the visual probe was presented. Using information from these Excel files, cognitive attentional bias was calculated from visual probe reaction time data for the four experimental trials.

Third, the eye-trac software collected eye-movement data from each participant for each of the four experimental trials. Each participant had four eye-trac files containing information on (1) average gaze duration (in milliseconds), (2) number of fixations, and (3) pupil diameter size. Each file was opened with ASL Results. Each file was organized based on XDAT values, which marked the data set to determine when the participants were seeing the visual probe pictures as opposed to anything else in the program. Because each Eprime program utilized 30 visual probe tasks, there were 30 events for each experimental trial, each of which presented participants with two pictures side-by-side (e.g. alcohol or food pictures versus matched control pictures). These 30 events were analyzed for average gaze duration, number of fixations, and pupil diameter size toward these pictures—all of which were consequently used to calculate direction and duration attentional bias, as well as sympathetic arousal.

In organizing the eye-trac files, I configured two backgrounds, both of which were used in defining the areas of interest (AOIs). One of the backgrounds had an alcohol or food picture on the left side with a neutral picture on the right side and the other had an alcohol or food picture on the right side with a neutral picture on the left side. The appropriate background was configured to the corresponding visual probe session for each participant. After each event was configured with a background, I then created AOIs that defined the parameters of the alcohol or food and neutral pictures that the

participants saw during the visual probe tasks (see Appendix B, Figure B5 for an example). Following configuration, data on average gaze duration, number of fixations, and pupil diameter size toward visual probe pictures were then used to calculate attentional bias and sympathetic arousal.

Data Cleaning

For the visual probe reaction time data, reaction times less than 200ms or greater than 2000ms, and reaction times more than 2 standard deviations above each participant's mean were excluded to reduce the influence of outliers (Bradley, Mogg, Wright, & Field, 2003; Glinder, Beckjord, Kaise, & Compas, 2007; Mogg, Holmes, Garner, & Bradley, 2008). For eye-tracking data, because the proposed study only examined eye-movements in relation to food-related or alcohol-related pictures and control pictures (see Appendix B, Figure B5), eye-movement data outside of the pre-defined AOIs were not used in the analyses (Castellanos et al., 2009).

Data Analyses

I hypothesized that: (1) alcohol odor cues would elicit increased alcohol cravings and food cravings relative to neutral odor cues and baseline (hypotheses 1 and 2); and (2) alcohol odor cues would elicit alcohol and food cognitive, direction, and duration attentional bias relative to neutral odor cues (hypotheses 5 and 6). For these hypotheses, I utilized repeated measures analysis of covariance (ANCOVA) and compared main effects with a Sidak adjustment to examine whether alcohol and food cravings and attentional bias would differ across the experimental trials (e.g. neutral vs. alcohol odorants) and

baseline (e.g. for cravings). Compared to Bonferroni adjustments, Sidak adjustments are more mathematically accurate and are not based on approximation (Cardinal & Aitken, 2006). State hunger, time of day, day of the week, and demographic characteristics (e.g. age, gender, and race) were included as covariates in these analyses. Relatedly, I also conducted these analyses without covariates using analysis of variance (ANOVA) and main effects comparison with a Sidak adjustment. The pattern of results did not change; therefore, only covariate-corrected analyses were reported in the text (see Appendix H, Table H1, for a summary of the non-covariate corrected results).

I also hypothesized that: (1) alcohol and food cravings elicited by alcohol odor cues would predict increased alcohol and food consumption, respectively (hypotheses 3 and 4); and (2) alcohol and food attentional bias elicited by alcohol odor cues would predict increased alcohol and food consumption, respectively (hypotheses 7 and 8). I utilized hierarchical multiple regression analyses and logistic multiple regression analyses to test these hypotheses. In these analyses, covariates were entered in the first step and predictors (e.g. alcohol or food cravings and attentional bias) were entered in the second step. Cravings and attentional bias were entered in separate regression analyses. Because there are two cravings measurements following alcohol odorants, these two cravings variables were entered in the same step. State hunger, time of day, day of the week, and demographic characteristics (e.g. age, gender, and race) were included as covariates in these analyses. To examine these aforementioned associations without covariates correction, correlational analyses were also conducted. Because the pattern of results did not change, only covariate-corrected analyses were reported in the text (see Appendix H, Table H2, for a summary of the non-covariate corrected results).

I ran exploratory analyses to examine (1) whether exposure to alcohol odor cues relative to neutral odor cues would elicit increased food and alcohol sympathetic arousal, and (2) whether alcohol and food sympathetic arousal elicited by alcohol odor cues would influence post-lab food and alcohol consumption. I utilized the same analyses mentioned above (e.g. repeated measures ANCOVA with main effects comparison using Sidak adjustment, multiple regression analyses, and logistic regression analyses) to examine the effect of odor cues on food and alcohol sympathetic arousal, and to examine the effect of food and alcohol sympathetic arousal following alcohol odor cues on post-lab food and alcohol consumption. State hunger, time of day, day of the week, and demographic characteristics (e.g. age, gender, and race) were included as covariates in these analyses. These exploratory analyses were also conducted without covariates using ANOVA and main effects comparison with a Sidak adjustment, as well as correlational analyses. Because the pattern of results did not change, only covariate-corrected analyses were reported in the text (see Appendix H for summaries of non-covariate corrected results).

In total, multiple analyses were conducted: 10 repeated measures ANCOVAs, 40 multiple regressions, and 10 logistic regressions. Despite the multitude of analyses, corrections were not applied. In particular, I did not apply Bonferonni corrections, wherein alpha level (0.05) is divided by the number of analyses prior to data interpretations. Practically and empirically, although applying Bonferroni corrections would reduce the likelihood of a Type I error (incorrect rejections of true null hypotheses), such corrections would inevitably lead to: (1) a higher likelihood of a Type II error (failure to reject false null hypotheses) and (2) analyses that were not sufficiently

powered to find effects. Theoretically, there are no justifications for accepting a "universal null hypothesis," wherein all effects in a study can be considered manifestations of chance and should not be fully interpreted (Rothman, 1990). In particular, corrections based on such a hypothesis would lead investigators to: (1) conclude that potentially significant findings are unimportant and only due to chance; and (2) not critically evaluate and interpret potentially important findings (Rothman, 1990). Relatedly, the present study can be considered a pilot study, whose results can potentially guide future studies. Application of Bonferonni corrections might mask potentially important results that could guide future studies.

RESULTS

Excluded and Included Participants

As mentioned above, 3 participants were excluded from the final sample. Excluded participants (M = 26.00, SD = 3.00) were younger than included participants (M = 30.84, SD = 9.46), and were less diverse in terms of gender (100% male vs. 48.1% male) and race (33% African American and 67% other races for excluded participants). Excluded participants appeared to have lower alcohol odor ratings (Ms= 2.00 to 2.67, SDs = 0.00 to 1.53) compared to included participants (Ms = 2.87 to 3.21, SDs = 0.61 to 0.82), but similar neutral odor ratings (Ms = 1.33 to 2.67, SDs = 0.58 to 1.16) compared to included participants (Ms = 1.33 to 2.78, SDs = 0.53 to 1.54). Post-lab food and alcohol consumption levels were generally comparable between excluded (Ms = 1.00 to 73.33, SDs = 0.00 to 26.46) and included participants (Ms = 1.33 to 66.01, SDs = 0.50 to 23.97). Excluded participants appeared to have higher cravings (Ms = 21.96 to 59.33, SDs = 1.15 – 13.00) compared to included participants (Ms = 18.64 – 36.01, SDs = 8.09 to 12.46), but appeared to generally have lower attentional bias (Ms = -31.72 to 54.00, SDs = 0.07 to 71.11) compared to included participants (Ms = -3.61 to 56.20, SDs = 0.18 to 58.69). Therefore, I conducted study analyses with and without these excluded participants to examine whether excluding these participants would bias study results. Because results did not differ with and without these participants, these participants were excluded from the data set and from analyses of study data presented below.

Missing Data

All participants included in the final sample completed measures of food and alcohol cravings, attentional bias, sympathetic arousal, and post-lab consumption. For each participant, there should have been a total of 31 separate variables: (1) 10 cravings variables (food and alcohol cravings at baseline and across four trials); (2) 12 attentional bias variables (food and alcohol cognitive, duration, and direction attentional bias across four trials); (3) 4 sympathetic arousal variables (food and alcohol sympathetic arousal across four trials); and (4) 5 post-lab consumption variables (food and alcohol consumption and co-consumption). Using an absolute value of less than 3.0 for skewness and less than 10.0 for kurtosis (Kline, 1998), none of these key variables met the criteria for non-normal distribution. Across these variables, 59.7% of participants (46 out of 77) had missing data in at least one variable (see Appendix A, Table A3). On average, participants were missing less than 8% of these variables. As a result, participants with missing data were still included in the analyses. Because only cravings variables were computed using multiple items, it was not possible to (1) conduct Little's missing completely at random (MCAR) analyses on most variables and (2) impute data using expected maximization method. Consequently, missing data were imputed using linear regression imputations.

Using linear regression imputations, multiple imputations (5 total imputations) were calculated for each variable. Across these five imputations, mean values across the thirty one variables remained relatively unchanged (see Appendices F and G for examples). Using repeated measures analysis of covariance (ANCOVA), there were variations in F-statistic and p-values with regard to the effects of odor cues on food and

alcohol attentional bias, cravings, and sympathetic arousal; however, despite these variations, the pattern of mean differences across experimental trials did not appear to differ between imputed and non-imputed data sets (see Appendix I, Tables I1 and I2, for an example). Similarly, there were also variations in coefficients and p-values with regard to the effects of alcohol odor cue elicited food and alcohol attention bias, cravings, and sympathetic arousal on post-lab food and alcohol consumption (see Appendix G for an example); at the same time, pattern of associations remained relatively similar between imputed and non-imputed data sets. Because the imputations appeared to bias the data, the results presented below are from analyses of non-imputed data.

Participant Characteristics and Preliminary Analyses

Participants (n = 77; 51.9% female, 83.1% Caucasian, 2.6% Hispanic, 1.3% Asian, 10.4% African American, and 2.6% comprising other races) had a mean age of 30.84 (SD = 9.46, Range: 18-54). The average participants typically participated in the study in the afternoon (M = 1393.49, SD = 289.11), and at the beginning and end of the week (27.3% Monday, 23.4% Tuesday, 7.8% Wednesday, 11.7% Thursday, and 29.9% Friday). Participants reported being semi-hungry to hungry at the beginning of the lab portion of the study (M = 4.64, SD = 2.66). Most participants were non-college students (60 out of 77; 77.9%), with a minority being college students (17 out of 77; 22.1%). A majority of participants reported consuming alcohol in general (76 out of 77) and beer specifically (74 out of 77) in the past two weeks. Similarly, most participants reported co-consuming food with alcohol (73 out of 77) and with beer (70 out of 77) in the past two weeks. The average participant reported: (1) general alcohol consumption in 5-6 days of

the past two weeks (*M* = 5.63, *SD* = 2.66); (2) beer consumption in 3-4 days of the past two weeks (*M* = 3.93, *SD* = 2.71); (3) co-consuming food with alcohol in 3-4 days of the past two weeks (*M* = 3.67, *SD* = 2.19); and (4) co-consuming food with beer in 2-3 days of the past two weeks (*M* = 2.71, *SD* = 1.99). Participants had a mean of 8.56 (*SD* = 5.11) for problematic alcohol use, which represents a low level of problematic drinking and which suggests a need for simple advice focused on reducing problematic drinking (Babor et al., 1992). Participants had a mean of 2.27 (*SD* = 0.45) for problematic eating, which indicates a low level of disordered eating patterns (de Lauzon et al., 2004) (see Appendix A, Table A1).

Most participants indicated consuming food following the lab portion of the study (93.59%); however, less than half of participants indicated consuming alcohol (46.15%) or co-consuming both food and alcohol (42.37%) after the lab portion of the study. Furthermore, when asked about how full they felt after eating, the average participant indicated being slightly more full than usual (*M* = 66.01, *SD* = 21.44). Relatedly, the average participant indicated consuming the typical amount of food following the lab portion of the study (*M* = 54.86, *SD* = 19.55). Additionally, the average participant indicated consuming between one to two drinks following the lab portion of the study (*M* = 2.49, *SD* = 2.03). Finally, the average participant indicated consuming less alcohol than usual following the lab portion of the study (*M* = 39.65, *SD* = 23.97) (see Appendix A, Table A1).

Demographic Differences

One-way analysis of variance (ANOVA), independent samples t-tests, and correlational analyses were used to examine whether key study variables differed by gender, race, and age (see Appendix A, Table A4). First, age was associated with lower levels of: (1) alcohol cravings following alcohol odors paired with alcohol pictures and food pictures; (2) food cravings following alcohol odors paired with alcohol pictures; (3) food direction attentional bias following alcohol odors; and (4) food duration attentional bias following alcohol and neutral odors (*rs* = -0.24 to -0.34, *ps*= 0.004 to 0.04). In addition to these negative associations, age was also associated with greater alcohol cognitive attentional bias following neutral odors ($r = 0.35$, $p = 0.003$). Second, men had higher alcohol sympathetic arousal following neutral odors ($M = 2.62$, $SD = 6.68$) compared to women ($M = -0.31$, $SD = 5.38$); $t(69) = 2.06$, $p = 0.04$. Finally, a number of variables differed across race: (1) food cognitive attentional bias following alcohol odors; (2) baseline food and alcohol cravings; (3) alcohol cravings following alcohol odors and alcohol pictures; (4) food cravings following neutral odors and alcohol pictures; (5) food cravings following alcohol odors, with both alcohol and food pictures; (6) alcohol duration attentional bias following alcohol odors; (7) food direction attentional bias following alcohol odors; (8) food duration attentional bias following alcohol odors; and (9) food sympathetic arousal following alcohol odors (*Fs*= 2.49 to 4.34, *ps*= 0.004 to 0.047).

Cravings

Repeated measures analyses of covariance (ANCOVAs) were conducted controlling for the effects of age, gender, race, state hunger, time of day, and day of the week. The effect of odor cues on alcohol cravings was significant, $F(4, 240) = 2.72$, $p = 0.03$ (see Appendix C, Figure C1, left panel). Baseline alcohol cravings ($M = 18.28$, $SD = 8.10$) were lower compared to alcohol cravings following (1) alcohol odors and alcohol pictures ($M = 24.18$, $SD = 12.12$, $p < 0.001$), (2) neutral odors and alcohol pictures ($M = 21.93$, $SD = 10.84$, $p = 0.001$), (3) alcohol odors and food pictures ($M = 23.88$, $SD = 12.16$, $p < 0.001$), and (4) neutral odors and food pictures ($M = 21.40$, $SD = 10.10$, $p = 0.001$). Moreover, alcohol cravings following alcohol odors and alcohol pictures were higher compared to alcohol cravings following neutral odors and alcohol pictures ($p = 0.01$). Similarly, alcohol cravings following alcohol odors and food pictures were higher compared to alcohol cravings following neutral odors and food pictures ($p = 0.02$).

After controlling for the effects of the covariates, the effect of odor cues on food cravings was non-significant, $F(4, 244) = 0.99$, $p = 0.42$ (see Appendix C, Figure C1, right panel). However, comparison of mean values across trials revealed two significant differences. Baseline food cravings ($M = 33.57$, $SD = 8.72$) were lower compared to food cravings following (1) alcohol odors and food pictures ($M = 36.94$, $SD = 10.25$, $p = 0.002$) and (2) neutral odors and food pictures ($M = 35.93$, $SD = 9.74$, $p = 0.01$); however, baseline food cravings did not significantly differ from alcohol cravings following (1) alcohol odors and alcohol pictures ($M = 34.82$, $SD = 10.86$, $p = 0.78$) and (2) neutral odors and alcohol pictures ($M = 35.29$, $SD = 10.08$, $p = 0.72$). Interestingly, regardless of the odorant presented, baseline food cravings were lower compared to food cravings

following food pictures. Furthermore, food cravings following alcohol odors and alcohol pictures did not significantly differ from food cravings following neutral odors and alcohol pictures ($p = 0.99$). Finally, food cravings following alcohol odors and food pictures also did not significantly differ from food cravings following neutral odors and food pictures ($p = 0.77$).

Cognitive Attentional Bias

Cognitive attentional bias was calculated by subtracting average reaction time to control pictures (in milliseconds) from average reaction time to alcohol or food pictures (in milliseconds), with greater positive values representing greater food and alcohol cognitive attentional bias. After controlling for the effects of the covariates, repeated measures ANCOVA revealed that the effect of odor cues on alcohol cognitive attentional bias was non-significant, $F(1, 53) = 0.06$, $p = 0.81$ (see Appendix C, Figure C2, left panel). Alcohol cognitive attentional bias did not significantly differ between exposure to alcohol odors ($M = 4.22$, $SD = 52.66$) compared to exposure to neutral odors ($M = 2.85$, $SD = 44.20$, $p = 0.87$). Similarly, the effect of odor cues on food cognitive attentional bias was non-significant, $F(1, 56) = 1.70$, $p = 0.20$ (see Appendix C, Figure C2, right panel). Food cognitive attentional bias did not significantly differ between exposure to alcohol odors ($M = -0.29$, $SD = 46.41$) and exposure to neutral odors ($M = -3.26$, $SD = 55.06$, $p = 0.71$).

Direction Attentional Bias

Direction attentional bias was calculated by dividing total number of fixations on alcohol or food pictures by total number of fixations on all pictures and by multiplying the product of this division by 100. Greater percentage values represented greater food and alcohol duration attentional bias. After controlling for the effects of covariates, repeated measures ANCOVA revealed that the effect of odor cues on alcohol direction attentional bias was non-significant, $F(1, 52) = 0.02$, $p = 0.89$ (see Appendix C, Figure C3, left panel). However, alcohol direction attentional bias following alcohol odors ($M = 55.70$, $SD = 8.22$) was significantly higher compared to alcohol direction attentional bias following neutral odors ($M = 52.37$, $SD = 8.26$, $p = 0.01$). Similarly, the effect of odor cues on food direction attentional bias was non-significant, $F(1, 51) = 0.09$, $p = 0.76$ (see Appendix C, Figure C3, right panel). Despite this non-significance, food direction attentional bias following alcohol odors ($M = 56.21$, $SD = 8.23$) was significantly higher compared to food direction attentional bias following neutral odors ($M = 53.27$, $SD = 7.24$, $p = 0.02$).

Duration Attentional Bias

Duration attentional bias was calculated by subtracting average gaze duration on control pictures (in milliseconds) from average gaze duration on alcohol or food pictures (in milliseconds), with greater positive values representing greater food and alcohol duration attentional bias. After controlling for the effects of covariates, repeated measures ANCOVA revealed that the effect of odor cues on alcohol duration attentional bias was non-significant, $F(1, 53) = 0.45$, $p = 0.51$ (see Appendix C, Figure C4, left panel). At the

same time, alcohol duration attentional bias following alcohol odors (M = 0.17, SD = 0.25) was significantly higher compared to alcohol duration attentional bias following neutral odors (M = 0.10, SD = 0.21, p = 0.02). Similarly, the effect of odor cues on food duration attentional bias was non-significant $F(1, 51) = 0.59$, p = 0.45 (see Appendix C, Figure C4, right panel). However, food duration attentional bias following alcohol odors (M = 0.20, SD = 0.24) was higher compared to food duration attentional bias following neutral odors (M = 0.07, SD = 0.16, $p < 0.001$).

Sympathetic Arousal

Sympathetic arousal was calculated by subtracting pupil diameter in response to control pictures (in millimeters) from pupil diameter in response to food or alcohol pictures (in millimeters), with greater positive values representing greater food and alcohol sympathetic arousal. After controlling for covariates, repeated measures ANCOVA revealed that the effect of odor cues on alcohol sympathetic arousal was non-significant, $F(1, 51) = 0.003$, p = 0.96 (see Appendix C, Figure C5, left panel). Alcohol sympathetic arousal did not significantly differ between exposure to alcohol odors (M = 1.62, SD = 5.24) and exposure to neutral odors (M = 1.11, SD = 5.63, p = 0.56). Similarly, the effect of odor cues on food sympathetic arousal was non-significant $F(1, 53) = 0.03$, p = 0.85 (see Appendix C, Figure C5, right panel). However, food sympathetic arousal was higher following exposure to alcohol odors (M = 1.31, SD = 4.98) compared to exposure to neutral odors (M = -0.30, SD = 3.97), with the difference falling short of significance (p = 0.06).

Post-Lab Food and Alcohol Consumption

Multiple regression and logistic regression analyses were conducted correcting for the effects of age, gender, race, state hunger, time of day, and day of the week. I examined whether alcohol and food cravings, attentional bias, and sympathetic arousal elicited by alcohol odor cues were associated with five post-lab consumption variables: (1) post-lab food satiety, (2) post-lab food typicality, (3) post-lab alcohol use, (4) post-lab alcohol typicality, and (5) the likelihood of post-lab concurrent consumption. There were four significant effects on post-lab alcohol use (see Appendix D, Tables D1 and D2): (1) food cravings paired with alcohol pictures during the visual probe task were associated with greater post-lab alcohol use ($\beta = 0.48$, $p = 0.04$); (2) alcohol cognitive attentional bias was associated with greater post-lab alcohol use ($\beta = 0.30$, $p = 0.03$); (3) food direction attentional bias was associated with lower post-lab alcohol use ($\beta = -0.25$, $p = 0.04$); and (4) food sympathetic arousal was associated with lower post-lab alcohol use ($\beta = -0.28$, $p = 0.02$). In addition to these significant associations, the negative association of food duration attentional bias with post-lab alcohol use fell short of significance ($\beta = -0.24$, $p = 0.06$; see Figure 9). There were no other significant associations with post-lab consumption variables ($\beta s = -0.17 – 0.35$, $ps = 0.10 – 0.99$), including the likelihood of post-lab concurrent consumption (Exp(B)s = 0.49 – 1.83, $ps = 0.33 – 0.91$; see Appendix A, Table A5).

DISCUSSION

General Discussion

The present study aimed to elucidate the role of classical conditioning processes in co-occurring food and alcohol consumption. Using a sample of social drinkers who occasionally co-consume food and alcohol, this study examined whether alcohol odor cues can serve as conditioned stimuli that elicit conditioned food-related and alcohol-related responses (e.g. attentional bias, cravings, and sympathetic arousal), and whether those conditioned responses can influence subsequent food and alcohol consumption. Controlling for covariates, repeated measures analysis of covariance tests revealed that odor cues had: (1) non-significant effects on alcohol and food sympathetic arousal and attentional bias; and (2) a significant effect on alcohol cravings, but a non-significant effect on food cravings. However, when the effects of covariates are not controlled, main effects comparisons indicated that alcohol odor cues elicit increased alcohol and food direction and duration attentional bias, as well as increased alcohol cravings and food sympathetic arousal. Moreover, after controlling for covariates, regression analyses revealed that: (1) cued food cravings and alcohol attentional bias were associated with increased alcohol consumption; (2) cued food attentional bias and sympathetic arousal were associated with decreased alcohol consumption; and (3) cued alcohol and food attentional bias, cravings, and sympathetic arousal were not associated with food consumption or the likelihood of concurrent consumption.

The significant findings of the present study suggest that alcohol odor cues can serve as conditioned stimuli that elicit both conditioned food-related and alcohol-related responses among food and alcohol co-consumers. In addition, these cued conditioned responses might differentially motivate later alcohol consumption, but might not necessarily motivate later food consumption or later concurrent consumption (Martinovic et al., 2014). These findings have important implications, especially in context of previous findings demonstrating that alcohol and food consumption often co-occur (Caton et al., 2004; Caton et al., 2005; Caton et al., 2007). First, these findings suggest that concurrent food and alcohol consumption can potentially lead to either increased or decreased alcohol consumption through classical conditioning mechanisms. Second, these findings suggest that interventions targeting classical conditioning mechanisms can potentially mitigate alcohol consumption, especially among co-consumers. Naturally, future clinical and experimental studies are needed to confirm the robustness of these findings, and to better characterize the implications and clinical relevance of these findings.

The null effects of cued conditioned responses on later food and concurrent consumption, and multiple null effects on later alcohol consumption also raise multiple possibilities that should be explored in future studies. First, conditioned responses elicited by alcohol odor cues might only influence later alcohol consumption, suggesting that alcohol and food odor cues might have differential effects on conditioned responses and consequent consumption among food and alcohol co-consumers. Second, cued conditioned responses might not persist long enough to consistently motivate later consumption, suggesting that alcohol odor cue elicited conditioned responses have

differential effects on immediate versus later consumption among food and alcohol co-consumers. Finally, the role of classical conditioning in concurrent consumption might be indirect: (1) concurrent consumption leads to alcohol odor cues becoming conditioned stimuli that elicit conditioned responses, which might in turn either decrease or increase alcohol consumption; (2) increased alcohol consumption disinhibits food consumption through its pharmacological effects; and (3) decreased alcohol consumption reduces the likelihood of increased food consumption. However, because these explanations are outside the scope of the study, further clinical and experimental investigations are warranted.

Overall, findings of the present study serve as a first step in clarifying the role of classical conditioning mechanisms in concurrent consumption. In particular, study findings suggest that alcohol odor cues can serve as conditioned stimuli that elicit conditioned food-related and alcohol-related responses, both of which can differentially motivate later alcohol consumption. However, in interpreting the aforementioned study findings, there are factors that should be taken into consideration. First, omnibus ANCOVA effects and regression effects were generally non-significant, possibly and partially due to the inclusion of covariates in these analyses. In particular, inclusion of covariates in these analyses (e.g. day of the week, time of day, state hunger, and demographics) might confound the effects of alcohol odor cues on food and alcohol responses and consumption. Moreover, both significant and non-significant effects found in the present study could also be attributed to a number of other factors—including study analyses being underpowered to accurately detect effects due to a small sample size, the possibility of a Type I error due to multiple statistical analyses being performed, and

measurement and methodological limitations (e.g. using self-report questions rather than ad lib measures to assess post-lab consumption). With these factors in mind, study findings should be interpreted cautiously. Future studies are needed to address these aforementioned factors in order to better characterize the implications and clinical relevance of the present study's findings.

Cued Attentional Bias and Consumption

Interpreting Alcohol Cue Elicited Attentional Bias

Findings of the present study serve as a first step in clarifying the role of classical conditioning mechanisms in alcohol odor cue elicited food and alcohol attentional bias. Inconsistent with my hypotheses, omnibus tests revealed that odor cues did not significantly influence alcohol and food direction, duration, and cognitive attentional bias. However, consistent with my hypotheses, unadjusted main effects comparisons revealed that alcohol odor cues elicit increased alcohol and food duration and direction attentional bias, but not cognitive attentional bias. These unadjusted findings suggest that co-consumers who are exposed to alcohol odor cues might demonstrate longer gaze duration and immediate fixation on predictive alcohol and food pictorial cues over non-predictive pictorial cues (direction and duration attentional bias), but might not necessarily demonstrate faster reaction time toward predictive over non-predictive pictorial cues (cognitive attentional bias). In particular, these unadjusted findings raise the possibility that: (1) alcohol odor cues can become imbued with enhanced reinforcing properties after being repeatedly paired with the pleasant effects of alcohol consumption

and food consumption (Dobson & Dozois, 2004); and (2) alcohol odor cues can consequently become conditioned stimuli that elicit the conditioned tendency to more selectively attend to cues (e.g. alcohol and food pictorial cues) that are predictive of alcohol and food consumption and their pleasant effects (Castellanos et al., 2009; Dobson & Dozois, 2004; Field et al., 2007; Yokum et al., 2011).

It should be noted that separate forms of attentional bias (e.g. direction, duration, and cognitive attentional bias) have different characteristics. In particular, duration attentional bias reflects a bias in maintained selective attention, while direction attentional bias reflects a bias in automatic selective attention (Castellanos et al., 2009; Ceballos, Komogotsev, & Turner, 2009; Field & Cox, 2008). Relatedly, cognitive attentional bias reflects either: (1) a bias in maintained selective attention when pictorial cues are presented for 1000ms or longer; or (2) a bias in automatic selective attention when pictorial cues are presented for 200ms or less (Field & Cox, 2008; Schoenmakers, Wiers, & Field, 2008). Because the current study presented pictorial cues for 1000ms, reaction time to visual probes in the present study reflects a bias in maintained selective attention. Collectively, in assessing cognitive and duration attentional bias, individuals are able to shift attention freely between predictive food or alcohol pictorial cues and non-predictive pictorial cues, with longer attention paid to and faster reaction times toward predictive pictorial cues indicating a bias in maintained selective attention. In contrast, direction attentional bias assesses immediate initial orientation toward either predictive pictorial cues or non-predictive pictorial cues, with more frequent immediate initial orientation toward predictive pictorial cues reflecting a bias in automatic selective attention. With those aforementioned features in mind, study findings suggest that food

and alcohol co-consumers might demonstrate alcohol odor cue elicited biases in maintained selective attention (duration but not cognitive attentional bias) and automatic selective attention (direction attentional bias) toward predictive food and alcohol pictorial cues over non-predictive pictorial cues.

These characteristics extend on previous findings demonstrating that: (1) alcohol cue exposure and food cue exposure can increase alcohol attentional bias and food attentional bias, respectively (Cox et al., 2003; Duka & Townshend, 2004; Johansson, Ghaderi, & Andersson, 2004; Papies, Stroebe, & Aarts, 2008; Roberts, Fillmore, & Milich, 2012); and (2) problematic drinkers and eaters have stronger and more consistent attentional bias compared to non-problematic consumers (Castellanos et al., 2009; Field & Cox, 2008). Study results suggests that co-consumers have a distinct pattern of alcohol odor cue elicited food and alcohol attentional bias that might differentiate them from food and alcohol consumers who do not engage in co-consumption (Castellanos et al., 2009; Field & Cox, 2008); however, because the present study did not recruit individuals who did not co-consume food and alcohol, this should be confirmed by future studies. Furthermore, considering that the present study utilized social drinkers who only occasionally co-consume food with alcohol, study results suggest that these attentional bias characteristics might be present among individuals who do not consume problematic levels. However, interpretations of study findings are tentative at best, especially in context of the non-significant omnibus effects of odor cues. Considering that covariates were included in omnibus analyses, future studies should better characterize the role of these covariates. Those future studies will permit firmer conclusions and inferences about alcohol odor cue elicited food and alcohol attentional bias.

Divergence in Attentional Bias Results

It should be noted that: (1) alcohol odor cue elicited bias in maintained attention was only present with the eye-tracking measure (duration attentional bias) and not with the reaction time measure (cognitive attentional bias); and (2) both alcohol and neutral odor cues appear to elicit somewhat faster reaction times toward neutral pictures as opposed to food pictures, with the difference between alcohol and neutral odor cue elicited cognitive food attentional bias being non-significant. This pattern of findings can be partially attributed to differences in measurements, particularly quality of measurements, such that eye-tracking measures might be more sensitive for measuring a bias in maintained selective attention compared to reaction time measures (Field & Cox, 2008). In this way, duration attentional bias might be a more accurate representation of a cued bias in maintained selective attention compared to cognitive attentional bias. Reaction time measures might only be providing a snapshot view of maintained selective attention that is susceptible to multiple factors: (1) task-related strategic influences, such as one's tendency to shift attention from pictorial cues to the central position in anticipation of the visual probe onset; (2) individual differences, including one's ability to cognitively process pictorial cues before responding to the visual probes; and (3) averaging, which might average out actual differences in maintained selective attention toward predictive over non-predictive pictorial cues (Field & Cox, 2008; Tiffany, 1990). In contrast, eye movement measures sample attention continuously and are likely to be a more sensitive index of maintained selective attention (Castellanos et al., 2009; Ceballos et al., 2009; Field et al., 2004).

The Quad model also suggests that there are four distinct processes that might contribute to overt responses in the visual probe task (Conrey, Sherman, Gawronski, & Hugenberg, 2005): (1) automatic activation of an association (association activation), which might represent pure attentional bias toward alcohol or food pictorial cues over matched control pictorial cues; (2) the likelihood of determining a correct response (discriminability), which represents the likelihood of a correct response to visual probes; (3) success at overcoming automatically activated associations (overcoming bias), which represents the ability to inhibit pure attentional bias in producing a correct response to visual probes that appear on the side of the matched control pictorial cues; and (4) the influence of a general response bias on a correct response (guessing), which might include the tendency to respond using the right hand during the visual probe task. Collectively, this suggests that reaction time measures: (1) might represent factors unrelated to attentional bias; and (2) provide a snapshot reflection of attentional bias that is susceptible to strategic influences, individual differences, and averaging. These factors might account for the null effects of alcohol odor cues on food and alcohol cognitive attentional bias. This also suggests that eye-tracking measures might more accurately represent alcohol odor cue elicited bias in maintained selective attention compared to reaction time measures.

Research Directions

To summarize, alcohol odor cues can serve as conditioned stimuli that elicit conditioned food and alcohol direction and duration attentional bias, but not conditioned food and alcohol cognitive attentional bias; however, these significant findings were only

present when covariates (e.g. demographics, time of day, day of the week, and state hunger) were not included. These study findings have important implications for future research. Specifically, future attentional bias studies should take into consideration: (1) the type of measurement used to assess attentional bias; (2) the aspects of attentional bias being measured; (3) sample size; (4) type of odorants used; and (5) the inclusion of covariates. In particular, study results demonstrate that the eye-tracking measurements rather than reaction time measurements can assess alcohol odor cue elicited food and alcohol attentional bias in a small sample of food and alcohol co-consumers. These findings suggest that researchers should utilize eye tracking measurements rather than reaction time measurements for measuring alcohol odor cue elicited attentional bias, especially if they are trying to measure a cued bias in maintained attention and if they only have a small sample size to work with. Furthermore, although both eye-tracking and reaction time measures can tap into multiple aspects of attentional bias (Field & Cox, 2008), the present study only assessed multiple aspects of attentional bias using the eye-tracking measure. To better characterize alcohol odor cue elicited attentional bias, including biases in both maintained and automatic selective attention, future studies need to assess separate aspects of attentional bias using both eye-tracking and reaction time measurements. Additionally, because the study sample consisted of social beer drinkers, beer odor cues utilized in the study may have already been imbibed with reinforcing properties that facilitate attentional bias. Future work should replicate this effect using unconditioned alcohol-related stimuli, particularly novel and unfamiliar alcohol odor cues, to better establish the role of classical conditioning in alcohol odor cue elicited attentional bias (see Kareken et al. 2012 for an example). Finally, considering the null

omnibus effects on attentional bias, there is the possibility that covariates might confound the effects of alcohol odor cues on food and alcohol attentional bias. Future studies should examine how these covariates might influence alcohol odor cue elicited food and alcohol attentional bias.

Interpreting the Attentional Bias-Consumption Relationship

Findings of the present study serve as a first step in clarifying the role of classical conditioning mechanisms in the attentional bias-consumption relationship. More specifically, study results suggest that alcohol odor cue elicited alcohol attentional bias and food attentional bias can serve as conditioned responses that motivate later alcohol consumption (Castellanos et al., 2009; Dobson & Dozois, 2004; Field et al., 2007; Yokum et al., 2011). However, the present study failed to find effects of cued alcohol and food attentional bias on food consumption and concurrent consumption. In particular, partially consistent with my hypotheses, only cued alcohol cognitive attentional bias might lead food and alcohol co-consumers to consume a greater number of alcoholic drinks. Inconsistent with my hypotheses, all forms of cued food attentional bias might not lead to greater food consumption among these food and alcohol co-consumers. Finally, although unhypothesized, cued food direction and duration attentional bias might lead food and alcohol co-consumers to consume a lower number of alcoholic drinks.

There are some possible explanations for these findings. First, cued food direction and duration attentional bias predicted lower alcohol consumption and was unrelated to subsequent food consumption. This suggests that food and alcohol co-consumers: (1) might not consume food to greater levels because they are able to resist the effects of

attention grabbing food pictorial cues; and (2) might consume alcohol at lower levels because attention grabbing food pictorial cues might not necessarily be predictive of alcohol consumption. Second, cued alcohol cognitive attentional bias rather than direction and duration attentional bias predicted higher alcohol consumption, but not food consumption. This suggests that food and alcohol co-consumers: (1) might consume alcohol at greater levels only when they maintain longer attention on and react faster in response to predictive alcohol pictorial cues, both of which incentivize alcohol seeking behaviors; and (2) might not consume alcohol at greater levels when their attention on predictive alcohol cues is automatic, which might be easier to resist and might consequently be less likely to incentivize alcohol use; and (3) might not consume food to greater levels because attention grabbing alcohol pictorial cues are not predictive of food consumption.

These study results extend on previous findings. First, previous studies demonstrated that alcohol and food attentional bias influence alcohol consumption and food consumption, respectively (Ceballos et al., 2009; Yokum et al., 2011). Study results clarify that alcohol odor cue elicited conditioned food and alcohol attentional bias might only influence later alcohol consumption—thus raising the possibility that conditioned responses elicited by food and alcohol odor cues might have differential effects on food and alcohol consumption. Alternately the aforementioned attentional bias-consumption associations might only be present among individuals who engage in problematic eating and alcohol use behaviors (Ceballos et al., 2009; Field, Mogg, Mann, Bennett, & Bradley, 2013; Newman, O'Connor, & Conner, 2008; Yokum et al., 2011). In the present study, co-consumers do not engage in these problematic behaviors, which might explain the

multitude of null effects of attentional bias on consumption. Second, previous studies have demonstrated that food consumption and alcohol consumption often co-occur (Caton et al., 2004; Caton et al., 2005; Caton et al., 2007). Study results clarify that such co-occurrence can lead to alcohol odor cues becoming conditioned stimuli that elicit conditioned food and alcohol attentional bias, which might in turn differentially influence alcohol consumption. In this way, concurrent consumption can lead to either decreased or increased alcohol consumption through cued food and alcohol attentional bias.

There are a number of issues that should be taken into consideration in interpreting the aforementioned findings. In particular, there are many factors that could contribute to these findings. First, the present study utilized indirect self-report questions for measuring post-lab consumption and assessed post-lab consumption following the lab portion of the study. This approach might not accurately tap into food and alcohol consumption elicited by cued attentional bias for a number of reasons: (1) post-lab food and alcohol consumption might be due to external factors unaccounted for by the study (e.g. being invited out for drinks and foods on the day of participation); (2) indirect self-report measurements do not tap into multiple aspects of consumption (e.g. amount consumed in grams and approximate caloric intake) and can subsequently be considered less comprehensive measurements of later consumption; and (3) alcohol odor cue elicited food and alcohol attentional bias might actually more strongly and robustly influence immediate consumption (e.g. ad lib consumption in the lab) as opposed to later consumption (e.g. consumption a few hours after study session). These limitations associated with indirect post-lab consumption measures might account for generally null effects on most consumption outcomes. Second, both significant effects and null effects

of the present study might be due to other factors—including reduced power to accurately detect effects due of the small sample size and the increased likelihood of a Type I error because multiple statistical analyses were performed. Due to these factors, inferences and conclusions drawn from study findings are tentative at best and should be made with caution. Naturally, future studies are needed to better characterize study results.

Research Implications

In summary, alcohol odor cue elicited food and alcohol attentional bias can serve as conditioned responses that differentially influence alcohol consumption. Although these findings should be interpreted in context of methodological and measurement limitations (e.g. limitations associated with post-lab consumption measurement), they can serve as a stepping stone for future studies. First, the present study is the first to demonstrate that alcohol odor cue elicited food and alcohol attentional bias can differentially influence subsequent alcohol consumption. These findings can serve as a stepping stone for future studies, which should focus on replicating and better characterizing these aforementioned effects. Second, cued alcohol cognitive attentional bias rather than cued alcohol duration attentional bias predicted increased alcohol consumption, suggesting that a combination of maintained attention and action stemming from maintained attention toward predictive alcohol pictorial cues are needed to motivate alcohol consumption. Researchers should better characterize the mechanisms through which different aspects of alcohol odor cue elicited bias in maintained attention influence alcohol consumption. Third, cued alcohol and food attentional bias differentially influenced post-lab alcohol consumption, suggesting that differences in aspects and

measurements of attentional bias (e.g. food vs. alcohol, cognitive vs. duration and direction) might contribute to differential effects on alcohol consumption. Researchers should better characterize attentional bias measurements and aspects when examining the cued attentional bias-alcohol consumption associations. Fourth, future studies should consider the possibility that cued food attentional bias might facilitate decreased alcohol consumption. In particular, these findings suggest that future studies should: (1) characterize the mechanisms through which cued food attentional bias lead to decreased alcohol use; (2) examine the effects of food consumption and attention paid to food cues during food consumption on alcohol consumption; and (3) differentiate these aforementioned effects from the disinhibiting effect of alcohol use on food intake (Caton et al., 2005). Fifth, although the present study assessed multiple consumption outcomes, cued attentional bias was associated only with the post-lab alcohol use variable. As mentioned above, considering the limitations of indirect self-report assessment of post-lab consumption, researchers should examine: (1) whether the cued attentional bias-consumption associations would differ in context of immediate (e.g. ad lib consumption) versus later consumption (e.g. consumption a few hours later); and (2) whether more comprehensive assessment of later consumption (e.g. diary measures) would yield different results. Finally, considering that the small sample size of the present study might reduce power for detecting effects, future studies should replicate and confirm study findings using a larger sample of participants. Collectively, these future studies can elucidate the role of classical conditioning mechanisms in concurrent consumption and can clarify the clinical utility of targeting cued conditioned attentional bias for concurrent consumption.

Cued Cravings and Consumption

Interpreting Alcohol Cue Elicited Cravings

Findings of the present study serve as a first step in clarifying the role of classical conditioning mechanisms in food and alcohol cravings. Consistent with my hypotheses, odor cues significantly influenced alcohol cravings, with alcohol odor cues significantly increasing alcohol cravings relative to neutral odor cues and relative to baseline. Inconsistent with my hypotheses, odor cues did not significantly influence food cravings, with alcohol odor cues inconsistently increasing food cravings relative to baseline and not increasing food cravings relative to neutral odor cue exposure. These findings collectively suggest that alcohol odor cues are generally more effective in increasing alcohol cravings, but not food cravings, among social drinkers who occasionally co-consume food and alcohol. These cued cravings characteristics of food and alcohol co-consumers build upon previous findings demonstrating that: (1) food cue exposure and alcohol cue exposure elicit increased food cravings and alcohol cravings, respectively (Fedoroff et al., 2003; Harvey, Kemps, & Tiggemann, 2005; Nederkoorn et al., 2000; Ryan, Kreiner, Chapman, & Stark-Wroblewski, 2010; Smith-Hoerter, Stasiewicz, & Bradizza, 2004); and (2) non-food cues have no effect or negative effects on food cravings (Kemps &Tiggemann, 2007; Kemps &Tiggemann, 2013; Kemps, Tiggemann, & Bettany, 2012). In particular, these findings clarify that alcohol odor cues only increase alcohol cravings and can be considered non-food cues that do not consistently increase food cravings among food and alcohol co-consumers.

These characteristics clarify the role of classical conditioning in the experience of cued food and alcohol cravings. Study results suggest that alcohol odor cues become conditioned stimuli that elicit conditioned alcohol cravings, but not conditioned food cravings, among food and alcohol co-consumers. For these co-consumers, alcohol odor cues might become repeatedly paired with alcohol withdrawal responses (Ludwig & Wikler, 1974; Wikler, 1973) and/or the physiological effects of alcohol (Cox & Klinger, 1988; Wardle, 1990). Consequently, these alcohol odor cues serve as conditioned alcohol-related stimuli that elicit (1) conditioned alcohol withdrawal responses and/or (2) conditioned compensatory responses, both of which can be subjectively perceived as alcohol cravings (Drummond et al., 1990; Jansen, 1998; O'Brien et al., 1990; Powley, 1977; Siegel, 1983). However, for co-consumers, alcohol odor cues might not necessarily become associated with food withdrawal or hunger (Wardle, 1990) and/or the physiological effects of food consumption (Jansen, 1998). Consequently, these alcohol odor cues might not become conditioned food-related stimuli that elicit conditioned food withdrawal responses (Sahakian et al., 1981; Wooley & Wooley, 1981) and/or conditioned food compensatory responses (Jansen, 1998; Wardle, 1990), both of which can be subjectively identified as food cravings. Alternately, food and alcohol cues might differentially influence both food and alcohol cravings, such that food cues might more effectively influence food cravings and alcohol cues might more effectively influence alcohol cravings. Indeed, study results suggest that baseline food cravings were lower compared to food cravings following food pictorial cues during visual probe tasks and regardless of the odorants presented during those tasks; however, it should be noted that the omnibus effects of odor cues on food cravings were non-significant.

Interpreting Associations of Cued Cravings with Consumption

Findings of the present study also serve as a first step in elucidating the role of classical conditioning in the cravings-consumption relationships. Inconsistent with my hypotheses, cued alcohol cravings and food cravings did not influence alcohol and food consumption, respectively. However, although unhypothesized, food cravings following alcohol odor cues and alcohol pictorial cues were associated with an increased number of alcoholic drinks. There are multiple explanations for these findings. First, conditioned alcohol and food cravings elicited by alcohol odor cues were not sufficient in increasing alcohol and food consumption, respectively. Food and alcohol co-consumers of the present study might be less likely to consume alcohol and food because they are better able to resist these cued conditioned cravings (Caton et al., 2004; Hetherington et al., 2001). Instead, study findings suggest that co-consumers might experience increased food cravings and consequently consume more alcohol when they are exposed to alcohol cues (e.g. conditioned stimuli) that engage multiple sensory modalities (e.g. alcohol odor and pictorial cues). Among food and alcohol co-consumers, conditioned food cravings elicited by multiple alcohol cues might increase alcohol use based on expectations and previous learning experiences, wherein alcohol consumption provides caloric intake that can sate food cravings (Gruchow, Sobocinski, Barboriak, & Scheller, 1985).

These results extend on previous findings in multiple ways. First, study findings are inconsistent with previous findings demonstrating food cravings-food consumption and alcohol cravings-alcohol consumption associations (Fedoroff et al., 2003; Gordon et al., 2006; Sobik, Hutchison, & Craighead, 2005; Yoon, Kim, Thurasa, Grant, & Westermeyer, 2006). Instead, study results suggest that such cravings-consumption

associations might not be present among social drinkers who occasionally co-consume alcohol with food, potentially because the aforementioned cravings-consumption associations are present only at more problematic levels of consumption (see Field, Mogg, & Bradley, 2005; Gordon et al., 2006; Litt, Cooney, & Morse, 2000; Polivy, Coleman, & Herman, 2005; Rodin, Mancuso, Granger, & Nelbach, 1991; Meule, Westenhofer, & Kubler, 2011). Second, extending on previous work demonstrating concurrent food and alcohol consumption (Caton et al., 2004; Hetherington et al., 2001), study results clarify that concurrent consumption can lead to alcohol cues (e.g. alcohol odors and pictures) becoming conditioned stimuli that elicit conditioned food cravings and subsequent alcohol consumption. In this way, concurrent consumption can potentially lead to increased alcohol consumption through alcohol cue elicited conditioned food cravings.

Findings of the present study should be interpreted with caution, especially in context of the aforementioned methodological and measurement limitations. In particular, indirect self-report post-lab consumption measures of the present study might not accurately tap into consumption driven by cued cravings and might explain the findings of the present study. Specifically, both null and significant effects of cued cravings on post-lab consumption might be due to: (1) post-lab consumption being due to external factors that cannot be controlled by the study (e.g. being invited out for drinks on the day of participation); and (2) indirect self-report measures not tapping into aspects of consumption influenced by cued cravings (e.g. amount consumed and approximate caloric intake). Of note, there is a possibility that cued alcohol and food cravings serve as conditioned responses that more robustly influenced immediate food and alcohol

consumption (Field et al., 2005; Litt et al., 2000; Martin, O'Neil, Tollefson, Greenway, & White, 2008) rather than later consumption (e.g. ad lib consumption in the lab vs. consumption a few hours after the lab portion of the study). Finally, both null and significant effects in the present study might be due to (1) reduced power to detect effects because of the small sample size and (2) increased likelihood of a Type I error because multiple statistical analyses were performed.

Research Implications

In summary, alcohol odor cues can serve as conditioned stimuli that elicit conditioned alcohol cravings, but not conditioned food cravings; however, only food cravings elicited by a combination of alcohol pictures and odors serve as a conditioned response associated with increased alcohol consumption. As mentioned above, these findings should be cautiously interpreted in context of a number of considerations—including (1) limitations associated with post-lab consumption measures, (2) the non-significant omnibus effects of odor cues on food cravings, (3) the small sample size of the present study, and (4) the number of statistical analyses performed. Despite these considerations, findings of the present study can provide multiple directions for future research studies. First, future studies should work on elucidating whether concurrent consumption can lead to increased alcohol consumption through conditioned food cravings. Such studies can further clarify the clinical utility of targeting food cravings elicited by multiple alcohol cues for reducing alcohol use, especially among food and alcohol co-consumers. Second, considering the limitations of indirect self-report assessment of post-lab consumption, researchers should consider other approaches for

measuring food and alcohol consumption—including ad lib consumption measurements and more comprehensive measurements of later consumption (e.g. diary measures). Third, study findings suggest that: (1) alcohol odor cues more consistently increased alcohol rather than food cravings; (2) food cravings appear to be more strongly influenced by food pictorial cues rather than alcohol odor cues; and (3) only food cravings elicited by alcohol odors and pictures were associated with increased post-lab alcohol consumption. Based on these findings, future studies should examine whether food and alcohol cues that engage multiple sensory modalities (e.g. sight, taste, and smell) would differentially influence cued food and alcohol cravings and subsequent consumption. Finally, considering that the small sample size of the present study might reduce power for detecting effects, future studies should replicate and confirm study findings using a larger sample of participants. Collectively, these studies can further clarify (1) whether classical conditioning can contribute to concurrent food and alcohol consumption through increased food and alcohol cravings, and (2) the clinical utility of targeting cued cravings for reducing concurrent consumption.

Exploratory Analyses on Sympathetic Arousal and Consumption

In exploratory analyses, the present study examined the effects of alcohol odor cues on food and alcohol sympathetic arousal and subsequent consumption. Although omnibus effects of odor cues on food and alcohol sympathetic arousal were non-significant, main effects comparisons suggest that alcohol odor cues increased pupil diameter toward food pictures relative to non-food pictures (food sympathetic arousal), but did not increase pupil diameter toward alcohol pictures relative to non-alcohol

pictures (alcohol sympathetic arousal). Moreover, study findings also suggest that: (1) cued alcohol and food sympathetic arousal did not predict alcohol and food consumption, respectively; and (2) cued food sympathetic arousal was associated with decreased number of alcoholic drinks. Increased pupil size, particularly pupil dilation, has been considered an index of arousal and interest (Blaha, 1977; Metalis, Hess, & Beaver, 1982); in this way, increased pupillary dilation in response to alcohol or food pictures would be suggestive of increased interest and arousal in response to those pictures. With this in mind, study findings suggest that social drinkers who occasionally co-consume food with alcohol: (1) demonstrate increased arousal and interest in food pictures, but not alcohol pictures, after being exposed to alcohol odorants; (2) do not consume alcohol and food to greater levels regardless of their level of cued arousal and interest in alcohol and food pictures, potentially because they are able to resist their arousal and interest in these pictures; and (3) consume less alcohol when they have increased cued arousal and interest in food pictures, potentially because food pictures are not necessarily predictive of alcohol consumption.

These characteristics of food and alcohol co-consumers are difficult to reconcile with previous findings, which have generally been mixed in nature. First, although consistent with findings that demonstrate no effect of alcohol taste cues on pupillary dilation (Adams, Brown, Haegerstrom-Portnoy, Flom, & Jones, 1978), study results are not consistent with previous work demonstrating increased pupillary dilation following alcohol odor cue exposure (Kennedy, 1971). Second, study results are consistent with work demonstrating that pupil diameter toward alcohol cues do not predict alcohol consumption (Ceballos et al., 2009), but inconsistent with findings demonstrating that

increased alcohol cue elicited pupil dilation is associated with a higher likelihood of alcohol relapse (Kennedy, 1971). Third, extending on findings demonstrating that food odors elicit increased pupillary dilation (Metalis et al., 1982), study results clarify that alcohol odors also increase pupillary size in response to food pictures. Finally, although inconsistent with findings demonstrating a positive relationship between pupillary dilation in response to food cues and food consumption (Graham, Hoover, Ceballos, & Komogortsev, 2011; Metalis et al., 1982), study results clarify that alcohol odor cue elicited pupil diameter changes in response to food pictures likely do not affect food consumption and might have the potential to reduce alcohol consumption.

Again, all study findings should be interpreted in context of the issues mentioned above—including (1) small sample size reducing power for detecting effects, (2) the number of statistical analyses increasing Type I error, (3) issues with indirect self-report post-lab consumption measures, and (4) the non-significant omnibus effects of odor cues on food and alcohol sympathetic arousal. In addition to those issues, it should be noted that inconsistencies with some previous findings can also be partially attributed to differences pupil diameter measurements. In particular, previous studies did not examine: (1) the effects of alcohol odor cues on pupillary responses to alcohol and food pictures relative to matched control pictures (cued alcohol and food sympathetic arousal); and (2) the effects of cued alcohol and food sympathetic arousal on alcohol and food consumption, respectively. Those studies only examined the effects of alcohol and food cue exposure on pupil diameter size and subsequent consumption, and did not present participants with pictorial cues (e.g. side-by-side food or alcohol versus neutral pictures). Separate measures of pupillary responses might be affected differently by alcohol odor

cues and might differentially predict consumption (see Schmitz, Krummenauer, Henn, & Dick, 2003; Yoon, Schmidt, Lietman, & McLeod, 2007). Finally, present study results could also be attributed to limitations associated with pupil diameter measurements. In particular, results of pupil diameter measurements can be affected by numerous factors: (1) distance between eye tracking apparatus and participants, (2) time between exposure to odor cues and assessments of pupil diameter, (3) emotional and cognitive processing of pictorial cues, and (4) length of presentation of pictorial cues (Bradley et al., 2008; Field & Eastwood, 2005; Field et al., 2007; Schmitz et al., 2003; Stormark et al., 2000).

Alternative Explanations

There are a number of alternative explanations for study findings. First, level of alcohol and food consumption might have contributed to the inconsistent effects of alcohol odor cues on food and alcohol attentional bias, cravings, sympathetic arousal, and consumption. In particular, previous studies have suggested that the aforementioned food-related and alcohol-related responses might be greater among problematic eaters (Calitri et al., 2010; Castellanos et al., 2009; Graham et al., 2011; Metalis et al., 1982; Meule et al., 2011; Polivy et al., 2005) and problematic drinkers (Field et al., 2007; Field et al., 2005; Gordon et al., 2006; Kennedy, 1971; Rubin, Gottheil, Roberts, Alterman, & Holstine, 1978; Townshend & Duka, 2011). Compared to social drinkers who occasionally co-consume food with alcohol, problematic drinkers and eaters might demonstrate more consistent increases in alcohol odor cue elicited conditioned food-related and alcohol-related responses, both of which might in turn more consistently increase food and alcohol consumption. Relatedly, considering that the level of food and

alcohol co-consumption is low among study participants, higher levels of co-consumption of food and alcohol (e.g. more regular pairing of alcohol odor cues with food and alcohol consumption effects) might be needed before alcohol odor cues can consistently elicit increased conditioned food-related and alcohol-related response, as well as increased food and alcohol consumption.

Second, a related possibility is that food and alcohol consumption might predict alcohol odor cue elicited conditioned food-related responses (Calitri et al., 2010; Castellanos et al., 2009; Graham et al., 2011; Metalis et al., 1982; Meule et al., 2011; Polivy et al., 2005) and conditioned alcohol-related responses (Field 2007; Field et al., 2005; Gordon et al., 2006; Kennedy, 1971; Rubin et al., 1978; Townshend & Duka, 2011). In this way, cravings, attentional bias, and sympathetic arousal might serve as correlates of level of consumption rather than predictors of consumption—such that more problematic levels of consumption might lead to greater levels of the these conditioned responses following alcohol odor cue exposure. Indeed, exploratory correlational analyses of study data provided partial support for this: (1) problematic eating was associated with greater alcohol odor cue elicited food cravings, food duration and direction attentional bias, and food sympathetic arousal; and (2) problematic alcohol use was associated with greater alcohol odor cue elicited alcohol cravings and food cravings, but only when food cravings were elicited by both alcohol odor and pictorial cues[1]. These

[1] Problematic alcohol use (AUDIT) was associated with: (1) alcohol cravings following alcohol odor and pictorial cues, (2) alcohol cravings following alcohol odor and food pictorial cues, and (3) food cravings following alcohol odor and pictorial cues (rs= 0.25 to 0.30, ps = 0.01 to 0.04). Problematic eating (TFEQ-18) was associated with: (1) food cravings following alcohol odor and pictorial cues, (2) food cravings following alcohol odor and food pictorial cues, and (3) food duration and direction attentional bias following alcohol odors (rs= 0.24 to 0.32, ps = 0.004 to 0.04). The association of problematic eating with food sympathetic arousal following alcohol odors fell short of significance (r = 0.22, p = 0.07).

exploratory analyses suggest that alcohol and food consumption might serve as predictors rather than outcomes of the aforementioned conditioned food-related and alcohol-related responses.

Third, study findings clarify that: (1) odor cues have non-significant omnibus effects on alcohol and food attentional bias and sympathetic arousal, as well as food cravings, and significant effects on alcohol cravings; and (2) all alcohol odor cue elicited conditioned food-related responses have no effect on food consumption. One possibility is that food odor cues might be more effective in eliciting these aforementioned food-related responses. Indeed, previous studies have provided support for this (Fedoroff et al., 2003; Harvey et al., 2005; Johansson et al., 2004; Metalis et al., 1982; Papies et al., 2008). In further support of this, study results also indicate that food cravings were increased following exposure to food pictures during the visual probe task and regardless of the type of odors presented (water vs. alcohol odors); however, it should be noted that the omnibus effects of odor cues on food cravings were non-significant. Another possibility is that food odor cue elicited food-related responses rather than alcohol odor cue elicited food-related responses might more consistently predict increased food consumption. Indeed, some findings have also provided some support for this (Fedoroff et al., 2003; Graham et al., 2001; Papies et al., 2008; Sobik et al., 2005). Overall, these findings emphasize the possible differential roles of alcohol odor cues and food odor cues in eliciting conditioned food-related responses and consequent food consumption.

Fourth, the effects of alcohol odor cues on food and alcohol sympathetic arousal, cravings, attentional bias, and consumption might be dependent on the type of foods and alcoholic beverages presented. Beer odor cues, particularly Bud Light odor cues, might

trigger conditioned cravings, attentional bias, and increased pupil diameter for specific types of foods and alcoholic beverages—all of which might trigger increased consumption of specific types of foods and alcoholic beverages. Indeed, previous studies have suggested that conditioned responses and subsequent consumption might be food and alcohol specific (Fedoroff et al., 2003; Field et al., 2005; Graham et al., 2011; Kemps &Tiggemann, 2009; Martin et al., 2008; Weafer & Fillmore, 2012; Willner, Field, Pitts, & Reeve, 1998). However, the present study utilized: (1) general rather than specific measures of food and alcohol cravings and consumption; and (2) non-specific food and alcohol pictorial cues during the visual probe task. The use of food and alcohol specific cravings and consumption measures, as well as the use of more specific pictorial cues, might result in beer odor cues being more consistent in eliciting increased conditioned food-related and alcohol-related responses, as well as increased food and alcohol consumption.

Fifth, study results broadly indicate that alcohol odor cues can serve as conditioned stimuli that elicit conditioned food-related and alcohol-related responses, both of which persist long enough to influence only alcohol consumption. This suggests that classical conditioning mechanisms might play a stronger role in later alcohol consumption compared to later food consumption or later concurrent consumption. At the same time, an alternative explanation is that the role of classical conditioning in concurrent consumption might be indirect. For instance, through frequent food and alcohol co-consumption (Caton et al., 2004; Caton et al., 2005; Caton t al., 2007), alcohol odor cues become associated with alcohol and food consumption effects. Consequently, these alcohol odor cues become conditioned stimuli that elicit conditioned food-related

and alcohol-related responses, some of which might increase later alcohol consumption. In turn, increased alcohol consumption disinhibits food consumption through its pharmacological effects. Confirmation of this pathway would elucidate the role of classical conditioning in concurrent food and alcohol consumption.

Sixth, the present study utilized five self-report questions to measure post-lab food and alcohol consumption. These questions assessed later consumption rather than immediate consumption (e.g. a few hours after the lab portion of the study) and provided only a snapshot view of later consumption (e.g. only consumption after the lab portion of the study). Alcohol odor cue elicited conditioned responses might more consistently and robustly influence other aspects of food and alcohol consumption. One alternative approach would be to assess ad libitum food and alcohol consumption during the lab portion of the study. Using this approach, participants would be given food and alcohol to consume, with food and alcohol consumption being measured as the weight difference in food and alcohol before and after consumption (Marlatt, Demming, & Reid, 1973; Weafer & Fillmore, 2008; Yip, Wiessing, Budgett, & Poppitt, 2013). Another alternative approach would be to utilize daily diary measures to get more comprehensive measurements of later consumption. With this approach, participants will be instructed to report on multiple aspects of food and alcohol consumption (e.g. approximate amount consumed and approximate amount of caloric intake) each day for a period of time (Carney, Armeli, Tennen, Affleck, & O'Neil, 2000; Conner, 1999; Subar et al., 2012).

Seventh, Pavlovian-to-instrumental transfer (PIT) might explain the generally null effects of cued food and alcohol attentional bias, cravings, and sympathetic arousal on post-lab food and alcohol consumption. In particular, PIT refers to a behavioral

phenomenon of increased instrumental responding for reinfocers (e.g. alcohol or food) when in the presence of conditioned cues (e.g. alcohol odor cues) that have been paired separately with those reinforcers (Cartoni, Puglisi-Allegra, & Baldassarre, 2013; Garbussow et al., in press; Garbusow et al., 2014; Hogarth, Field, & Rose, 2013; Martinovic et al., 2014). Through PIT, exposure to conditioned alcohol odor cues associated with food and alcohol consumption might evoke expectations of consumption effects (e.g. sated cravings and euphoria), which might in turn elicit food and alcohol seeking behaviors (Martinovic et al., 2014). In this way, exposure to conditioned alcohol odor cues might play a vital role in subsequent alcohol and food consumption. However, conditioned alcohol odor cues were present only in the lab portion of the present study and might not necessarily be present outside of the lab. The absence of those conditioned cues might explain the multitude of null effects of cued food and alcohol responses on consumption. Alcohol odor cue elicited food and alcohol responses might more robustly influence immediate (ad lib) consumption in the lab due to there being less delay among alcohol cue exposure, cued conditioned responses, and subsequent consumption.

Finally, there might be external factors that influence the effects of alcohol odor cues on food and alcohol cravings, attentional bias, sympathetic arousal, and consumption. The absence of these factors in the present study might have contributed to the inconsistent effects of alcohol odor cues on food and alcohol responses and consumption. In particular, the effects of alcohol odor cues on conditioned responses and subsequent consumption might be influenced by a number of factors—including dieting status and habits (Harvey et al., 2005; Papies et al., 2008), negative and positive emotions (Cooney et al., 1997; Hepworth et al., 2010; Newman et al., 2008; Waters, Hill, &

Waller, 2001; Willner et al., 1998), trait eating and drinking style (Fedoroff et al., 2003), complexity of pictorial cues (Miller & Fillmore, 2010), trait reactivity to food and alcohol cues (Field et al., 2005; Johansson et al., 2004), and exposure to familiar environmental and social cues associated with drinking and eating (Litt et al., 2000; Ryan et al., 2010). These factors might mediate or moderate the effects of alcohol odor cues on conditioned food-related and alcohol-related responses, as well as the effects of those conditioned responses on food and alcohol consumption.

Collectively, future research studies should examine whether the effects of alcohol odor cues on food and alcohol responses and consumption: (1) differ between problematic and non-problematic food and alcohol consumers; (2) are dependent on participants' level of concurrent food and alcohol consumption; (3) differ compared to the effects of food odor cues; (4) are dependent on specific types of food and alcoholic beverages; and (5) are moderated and/or mediated by multiple factors. Relatedly, future studies should also elucidate whether alcohol odor cue elicited cravings, attentional bias, and sympathetic arousal serve as predictors and/or outcomes of consumption. Furthermore, future studies should also examine indirect pathways through which classical conditioning mechanisms might influence concurrent consumption. Such studies would further clarify (1) the importance of classical conditioning mechanisms in concurrent food and alcohol consumption, and (2) the clinical utility of targeting classically conditioned responses for reducing concurrent consumption.

Limitations

There are limitations that might limit the generalization of study findings (Kazdin, 2002). First, the present study recruited participants who co-consumed food with alcohol at non-problematic levels; in particular, most participants reported co-consuming alcohol and food in only 3-4 days out of the past two weeks and most participants did not co-consume food with alcohol following the lab portion of the study. Relationships among food and alcohol attentional bias, cravings, sympathetic arousal, and consumption might not necessarily apply to populations who (1) more regularly co-consume food with alcohol and/or (2) have comorbid eating and alcohol use problems. Second, the present study did not recruit food and alcohol consumers who do not co-consume food and alcohol. As such, direct comparisons between individuals who co-consume and individuals who do not co-consume food and alcohol were not possible in the present study. Third, because the present study only recruited beer consumers, relationships of the present study might not generalize to individuals who prefer to concurrently consume food with other alcoholic beverages. Fourth, because the present study utilized Bud Light as the beer odorant and water as the control odorant, the present study results might not necessarily apply to the effects of other alcohol odor cues (e.g. unconditioned novel alcohol odor cues and other light beer odor cues) and appetitive odor cues (e.g. hot chocolate and grape juice) on food and alcohol cravings, attentional bias, sympathetic arousal, and consumption. Relatedly, because non-appetitive water odorant was utilized as the control odorant, it is unclear whether the effects of alcohol odor cues represent a specific response to an alcohol odorant or a general response to an appetitive odorant. Sixth, the present study had to exclude data from three participants; at the same time,

such exclusion did not affect the pattern of study results. Seventh, although there were many significant main effects comparisons, most omnibus effects of odor cues were non-significant. This suggests that the inclusion of covariates might be confounding the effects of odor cues. As such, study results might not generalize to non-adjusted effects of odor cues on food-related and alcohol-related responses. Eighth, sample size of the present study might reduce power for accurately detecting effects; consequently, study results might not be comparable to results from studies with larger sample sizes. Finally, although present study examined multiple effects (60 total effects), no corrections were made (1) to reduce the likelihood of type II error, and (2) because such corrections might render potentially important effects null and consequently not interpretable. However, because no corrections were made, there is a possibility that some significant effects are due to type I error. As such, replication is needed to increase confidence in study results.

Furthermore, eye-tracking measures of the present study might generate some limitations. First, participants might not have eye movement data for the entire duration of their participation. Because the eye-tracker is sensitive to participant movement, pupil recognition can be lost during administration of the visual probe task. Loss of pupil recognition might lead to missing eye movement data until the eye-tracker is able to capture the participant's corneal reflection again. Loss of pupil recognition might happen when participants start to squint or move outside of the optimal recognition range (24 inches from the eye-tracker). At the same time, the present study minimized this limitation by (1) utilizing a forehead rest and a chinrest to minimize participant movement and facilitate eye calibration, (2) positioning the forehead rest and chinrest in the same position (24 inches from the monitor) for all participants, and (3) requiring eye

recalibration prior to each of the four trials. Second, although the present study assessed a bias in initial orientation of attention (direction attentional bias) by dividing fixations on alcohol or food pictures by total fixations on all pictures, this particular form of attentional bias can be assessed in other ways. For instance, a bias in initial orientation can be assessed by (1) determining the number of initial fixations on alcohol or food versus control pictures, and (2) subtracting the number of initial fixations on control pictures by the number of first fixations on alcohol or food pictures. This approach to calculating a bias in initial orientation of attention might yield different results. Finally, measurements of direction attentional bias, duration attentional bias, and sympathetic arousal might be affected by multiple factors—including (1) distance between eye tracking apparatus and participants, (2) time between exposure to odor cues and assessments of eye movement, (3) emotional and cognitive processing of pictorial cues, and (4) length of presentation of pictorial cues (Bradley et al., 2008; Field & Eastwood, 2005; Field et al., 2007; Schmitz et al., 2003; Stormark et al., 2000).

Similarly, other measures used in the present study can generate some limitations. For instance, cravings were measured using self-report questionnaires. Other measurements of reactivity food or alcohol odor exposure might be more objective, and might consequently yield different or more accurate results. To address this, the present study examined whether alcohol odor cues would influence food and alcohol sympathetic arousal, as well as subsequent food and alcohol consumption, in exploratory analyses. Furthermore, alcohol and food consumption were measured using self-report questionnaires, which provided only a snapshot view of later consumption. In particular, food and alcohol consumption were measured in terms of (1) level of satiety following

later food consumption, (2) amount of food consumed relative to usual consumption, (3) number of drinks consumed, (4) amount of alcohol consumed relative to usual consumption, and (5) whether participants consumed alcohol with food. Study results might differ with direct ad libitum measurements of consumption and with more comprehensive measurements of later consumption.

Finally, study procedures might generate some limitations. For instance, food and alcohol cravings were measured following exposure to different odorants and following engagement in the visual probe tasks. Exposure to food or alcohol pictures during the visual probe tasks might influence cravings and might consequently confound the effect of odor exposure on cravings. However, there are some considerations that should be noted: (1) prior studies have indicated that odor cue exposure might be more powerful in eliciting alcohol cravings (Litt & Cooney, 1990) and food cravings (Drobes et al., 2001; Hawk et al., 2004) compared to pictorial cue exposure; and (2) study results indicate that the effect of odor cue exposure generally remain consistent, regardless of the type of pictorial cues presented. Furthermore, participants were asked to report on their food and alcohol consumption following the lab portion of the study. Their experiences following the lab portion of the study cannot be controlled and might exert a confounding effect on study results. In particular, post-lab food and alcohol consumption might be due to external factors (i.e., being invited out for drinks, usual day for drinking, etc.) unaccounted for by the study.

Conclusions

The present study is the first to demonstrate that alcohol odor cues can serve as conditioned stimuli that elicit conditioned food-related and alcohol-related responses, both of which might contribute differentially to alcohol consumption. As mentioned above, these findings should be interpreted in context of multiple study issues—including (1) small sample size reducing power for detecting effects, (2) multiple statistical analyses increasing Type I error, (3) measurement and methodological limitations (e.g. indirect self-report assessment of post-lab consumption and reaction time measurements limitations), and (4) multiple non-significant omnibus effects of odor cues. In lieu of these issues, the present study can be more accurately described as a pilot study, whose results can guide future studies. In particular, study findings suggest that the role of classical conditioning mechanisms in food and alcohol responses and consumption warrants further investigation. For instance, future experimental and longitudinal studies should consider: (1) utilizing more diverse measurements of conditioned responses; (2) utilizing more proximal measurements of consumption and/or more comprehensive measurements of later consumption; (3) utilizing more specific food and alcohol pictorial cues; (4) examining the effects of food odor cues and unconditioned alcohol odor cues; and (5) recruiting a larger sample of participants with higher levels of food, alcohol, and concurrent consumption. In addition, findings of the present study also suggest that interventions targeting alcohol odor cue elicited conditioned food-related and alcohol-related responses have the potential for reducing alcohol consumption. Naturally, clinical studies are needed to examine whether these approaches are effective. Overall, the present study serves as a first step in elucidating the role of classical conditioning

mechanisms in conditioned food-related and alcohol-related responses, and in subsequent food and alcohol consumption. Building upon the findings of the present study, future studies can further elucidate these classical conditioning mechanisms, as well as clarify whether these classical conditioning mechanisms might have clinical relevance for addressing concurrent food and alcohol consumption.

REFERENCES

REFERENCES

Adams, A. J., Brown, B., Haegerstrom-Portnoy, G., Flom, M. C., & Jones, R. T. (1978). Marijuana, alcohol, and combined drug effects on the time course of glare recovery. *Psychopharmacology, 56,* 81-86.

Agras, W. S., & Telch, C. F. (1998). The effects of caloric deprivation and negative affect on binge eating in obese binge-eating disordered women. *Behavior Therapy, 29,* 491-503.

Babor, T. F., de la Fuente, J. R., Saunders, J., & Grant, M. (1992). *The alcohol use disorders identification test: Guidelines for use in primary health care*. Geneva, Switzerland: World Health Organization.

Barrow, O. (2014). Top 20 best selling beers of 2013. *Dayton Business Journal.* Retrieved on June 25, 2015, from http://www.bizjournals.com/dayton/news/2014/01/27/top-20-best-selling-beers-of-2013.html.

Baumeister, R. F., Heatherton, T. F., & Tice, D. M. (1994). *Losing control: How and why people fail at self-regulation.* San Diego, CA: Academic Press.

Blaha, S. J. M. (1977). *Measurements of pupil size in response to pictures of food with changes in the percentage of blood glucose in underweight, normal weight, overweight, and obese women.* Unpublished master's thesis, Oregon State University, Corvallis, Oregon.

Bohn, M. J., Krahn, D. D., & Staehler, B. A. (1995). Development and initial validation of a measure of drinking urges in abstinent alcoholics. *Alcoholism: Clinical and Experimental Research*, *19*, 600-606.

Bradley, B. P., Mogg, K., Wright, T., & Field, M. (2003). Attentional bias in drug dependence: Vigilance for cigarette-related cues in smokers. *Psychology of Addictive Behaviors, 17,* 66-72.

Bradley, M. M., Miccoli, L., Escrig, M. A., & Lang, P. J. (2008). The pupil as a measure of emotional arousal and autonomic activation. *Psychophysiology, 45,* 602-607.

Bragulat, V., Dzemidzic, M., Talavage, T., Davidson, D., O'Connor, S. J., & Kareken, D. A. (2008). Alcohol sensitizes cerebral responses to the odors of alcoholic drinks: An fMRI study. *Alcoholism: Clinical and Experimental Research, 32,* 1124-1134.

Braun, D. L., Sunday, S. R., & Halmi, K. A. (1994). Psychiatric comorbidity in patients with eating disorders. *Psychological Medicine, 24,* 859-867.

Buchner, A., Erdfelder, E., Faul, F., & Lang, A. G. (2009). G* Power version 3.1.7. *Kiel: University of Kiel*.

Burton, S. M., & Tiffany, S. T. (1997). The effect of alcohol consumption on craving to smoke. *Addiction, 92,* 15-26.

Calitri, R., Pothos, E. M., Tapper, K., Brunstrom, J. M., & Rogers, P. J. (2010). Cognitive biases to healthy and unhealthy food words predict change in BMI. *Obesity*, *18*, 2282-2287.

Cardinal, R. N., & Aitken, M. R. F. (2006). *ANOVA for the behavioural sciences researcher.* London: Lawrence Erlbaum Associates.

Carney, M. A., Armeli, S., Tennen, H., Affleck, G., & O'Neil, T. P. (2000). Positive and negative daily events, perceived stress, and alcohol use: A diary study. *Journal of Consulting and Clinical Psychology, 68,* 788-798.

Cartoni, E., Puglisi-Allegra, S., & Baldassarre, G. (2013). The three principles of action: A Pavlovian-instrumental transfer hypothesis. *Frontiers in Behavioral Neuroscience, 7,* 1-11.

Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R. L. (2009). Obese adults have visual attentional bias for food images: Evidence for altered reward system function. *International Journal of Obesity, 33,* 1063-1073.

Caton, S. J., Ball, M., Ahern, A., & Hetherington, M. M. (2004). Dose-dependent effects of alcohol on appetite and food intake. *Physiology and Behavior, 81,* 51-58.

Caton, S. J., Bate, L., & Hetherington, M. M. (2007). Acute effects of an alcoholic drink on food intake: aperitif versus co-ingestion. *Physiology & behavior*, *90*, 368-375.

Caton, S. J., Marks, J. E., & Hetherington, M. M. (2005). Pleasure and alcohol: manipulating pleasantness and the acute effects of alcohol on food intake. *Physiology and behavior*, *84*, 371-377.

Ceballos, N. A., Komogotsev, O. V., & Turner, G. M. (2009). Ocular imaging of attentional bias among college students: Automatic and controlled processing of alcohol-related scenes. *Journal of Studies on Alcohol and Drugs, 70,* 652-659.

Chambers, R., Lo, B. C. Y., & Allen, N. B. (2008). The impact of intensive mindfulness training on attentional control, cognitive style, and affect. *Cognitive Therapy and Research*, *32*, 303-322.

Childress, A. R., Ehrman, R., Rohsenow, D. J., Robbins, S. J., & O'Brien, C. (1992). Classically conditioned factors in drug dependence. In J. Lowinson, P. Ruiz, R. Millman, & J. Langrod (Eds.), *Substance abuse: A comprehensive textbook* (56-69). Baltimore: Williams and Wilkins.

Collins, R. L., Parks, G. A., & Marlatt, G. A. (1985). Social determinants of alcohol consumption: The effects of social interaction and model status on the self-administration of alcohol. *Journal of Consulting and Clinical Psychology, 53,* 189-200.

Conklin, C. A., & Tiffany, S. T. (2002). Applying extinction research and theory to cue-exposure addiction treatments. *Addiction*, *97*, 155-167.

Conner, M. (1999). Stress and snacking: A diary study of daily hassles and between-meal snacking. *Psychology and Health, 14,* 51-63.

Conrey, F. R., Sherman, J. W., Gawronski, B., & Hugenberg, K. (2005). Separating multiple processes in implicit social cognition: The Quad Model of implicit task performance. *Journal of Personality and Social Psychology, 89,* 469-487.

Cooney, N. L., Litt, M. D., Morse, P. A., Bauer, L. O., & Gaupp, L. (1997). Alcohol cue reactivity, negative-mood reactivity, and relapse in treated alcoholic men. *Journal of Abnormal Psychology, 106,* 243-250.

Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: A motivational model of alcohol use. *Journal of Personality and Social Psychology, 69,* 990-1005.

Cooper, S. J., & Turkish, S. (1989). Effects of naltrexone on food preference and concurrent behavioral responses in food-deprived rats. *Pharmacology Biochemistry and Behavior, 33,* 17-20.

Coskunpinar, A., Dir, A. L., Karyadi, K. A., Koo, C., & Cyders, M. A. (2013). Mechanisms underlying the relationship between negative affectivity and problematic alcohol use. *Journal of Experimental Psychopathology*, *4*, 263-278.

Cox, W. M., & Klinger, E. (1988). A motivational model of alcohol use. *Journal of Abnormal Psychology*, *97*, 168-180.

Cox, W. M., Brown, M. A., & Rowlands, L. J. (2003). The effects of alcohol cue exposure on non-dependent drinkers' attentional bias for alcohol-related stimuli. *Alcohol and Alcoholism*, *38,* 45-49.

Cox, W. M., Hogan, L. M., Kristian, M. R., & Race, J. H. (2002). Alcohol attentional bias as a predictor of alcohol abusers' treatment outcome. *Drug and Alcohol Dependence, 68,* 237-243.

Cravens, R. W., & Renner, K. E. (1969). Conditioned hunger. *Journal of Experimental Psychology*, *81*, 312-316.

de Lauzon, B., Romon, M., Deschamps, V., Lafay, L., Borys, J. M., Karlsson, J., ... & Charles, M. A. (2004). The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in a general population. *The Journal of Nutrition*, *134*, 2372-2380.

Dobson, K. S., & Dozois, D. J. (2004). Attentional biases in eating disorders: A meta-analytic review of Stroop performance. *Clinical Psychology Review*, *23*, 1001-1022.

Drobes, D. J. (2002). Concurrent alcohol and tobacco dependence: mechanisms and treatment. *Alcohol Research and Health*, *26*, 136-142.

Drobes, D. J., Miller, E. J., Hillman, C. H., Bradley, M. M., Cuthbert, B. N., & Lang, P. J. (2001). Food deprivation and emotional reactions to food cues: Implications for eating disorders. *Biological Psychology, 57,* 153-177.

Drummond, D. C., & Glautier, S. (1994). A controlled trial of cue exposure treatment in alcohol dependence. *Journal of Consulting and Clinical Psychology*, *62*, 809-817.

Drummond, D. C., & Phillips, T. S. (2002). Alcohol urges in alcohol-dependent drinkers: Further validation of the Alcohol Urge Questionnaire in an untreated community clinical population. *Addiction, 97,* 1465-1472.

Drummond, D. C., Cooper, T., & Glautier, S. P. (1990). Conditioned learning in alcohol dependence: Implications for cue exposure treatment. *British Journal of Addiction*, *85*, 725-743.

Duka, T., & Townshend, J. M. (2004). The priming effect of alcohol pre-load on attentional bias to alcohol-related stimuli. *Psychopharmacology*, *176*, 353-361.

Dunn, E. C., Larimer, M. E., & Neighbors, C. (2002). Alcohol and drug-related negative consequences in college students with bulimia nervosa and binge eating disorder. *International Journal of Eating Disorders*, *32*, 171-178.

Edwards, G. (1990). Withdrawal symptoms and alcohol dependence: Fruitful mysteries. *British Journal of Addictions, 85,* 447-461.

Eiler II, W. J., Dzemidzic, M., Case, K. R., Considine, R. V., & Kareken, D. A. (2012). Correlation between ventromedial prefrontal cortex activation to food aromas and cue-driven eating: an fMRI study. *Chemosensory Perception*, *5*, 27-36.

Fedoroff, I., Polivy, J., & Herman, C. P. (2003). The specificity of restrained versus unrestrained eaters' responses to food cues: General desire to eat, or craving for the cued food? *Appetite*, *41*, 7-13.

Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug and Alcohol Dependence, 97,* 1-20.

Field, M., & Eastwood, B. (2005). Experimental manipulation of attentional bias increases the motivation to drink alcohol. *Psychopharmacology*, *183*, 350-357..

Field, M., Duka, T., Eastwood, B., Child, R., Santarcangelo, M., & Gayton, M. (2007). Experimental manipulation of attentional biases in heavy drinkers: do the effects generalize? *Psychopharmacology*, *192*(4), 593-608.

Field, M., Mogg, K., & Bradley, B. P. (2005). Craving and cognitive biases for alcohol cues in social drinkers. *Alcohol and Alcoholism, 6,* 504-510.

Field, M., Mogg, K., Mann, B., Bennett, G. A., & Bradley, B. P. (2013). Attentional biases in abstinent alcoholics and their association with cravings. *Psychology of Addictive Behaviors, 27,* 71-80.

Field, M., Mogg, K., Zetteler, J., & Bradley, B. P. (2004). Attentional biases for alcohol cues in heavy and light social drinkers: The roles of initial orienting and maintained attention. *Psychopharmacology, 176,* 88-93.

Field, M., Munafò, M. R., & Franken, I. H. (2009). A meta-analytic investigation of the relationship between attentional bias and subjective craving in substance abuse. *Psychological bulletin*, *135*(4), 589.

Fischer, S., Settles, R., Collins, B., Gunn, R., & Smith, G. T. (2012). The role of negative urgency and expectancies in problem drinking and disordered eating: Testing a model of comorbidity in pathological and at-risk samples. *Psychology of Addictive Behaviors, 26*, 112-123.

Fischer, S., Smith, G. T., Annus, A., & Hendricks, M. (2007). The relationship of neuroticism and urgency to negative consequences of alcohol use in women with bulimic symptoms. *Personality and Individual Differences, 43,* 1199-1209.

Garbusow, M., Schad, D. J., Sebold, M., Friedel, E., Bernhardt, N., Koch, S. P.,…& Heinz, A. (in press). Pavlovian-to-instrumental transfer effects in the nucleus accumbens relate to relapse in alcohol dependence. *Addiction Biology*. Advance online publication.

Garbusow, M., Schad, D. J., Sommer, C., Junger, E., Sebold, M., Friedel, E.,…& Rapp, M. A. (2014). Pavlovian-to-instrumental transfer in alcohol dependence: A pilot study. *Neuropsychobiology, 70,* 111-121.

Glautier, S., Clements, K., White, J. A. W., Taylor, C., & Stolerman, I. P. (1996). Alcohol and the reward value of cigarette smoking. *Behavioural Pharmacology*, *7*, 144-154.

Glinder, J. G., Beckjord, E., Kaise, C. R., & Compas, B. E. (2007). Psychological adjustment to breast cancer: Automatic and controlled responses to stress. *Psychology and Health, 22,* 337-359.

Gordon, S. M., Sterling, R., Slatkowski, C., Raively, K., Weinstein, S., & Hill, P. C. (2006). Inpatient desire to drink as a predictor of relapse to alcohol use following treatment. *The American Journal of Addictions, 15,* 242-245.

Graham, R., Hoover, A., Ceballos, N. A., & Komogortsev, O. (2011). Body mass index moderates gaze orienting biases and pupil diameter to high and low calorie food images. *Appetite, 56,* 577-586.

Grant, V. V., Stewart, S. H., O'Connor, R. M., Blackwell, E., & Conrod, P. J. (2007). Psychometric evaluation of the five-factor Modified Drinking Motives Questionnaire—Revised in undergraduates. *Addictive Behaviors*, *32*, 2611-2632.

Grilo, C. M., White, M. A., & Masheb, R. M. (2009). DSM-IV psychiatric disorder comorbidity and its correlates in binge eating disorder. *International Journal of Eating Disorders*, *42*, 228-234.

Gruchow, H. W., Sobocinski, K. A., Barboriak, J. J., & Scheller, J. G. (1985). Alcohol consumption, nutrient intake and relative body weight among US adults. *American Journal of Clinical Nutrition, 42,* 289-295.

Guerrieri, R., Nederkoorn, C., & Jansen, A. (2007). The interaction between impulsivity and varied food environment: Its influence on food intake and overweight. *International Journal of Obesity, 32*, 708-714.

Hardman, C. A., Rogers, P. J., Etchells, K. A., Houstoun, K. V., & Munafò, M. R. (2013). The effects of food-related attentional bias training on appetite and food intake. *Appetite*, *71*, 295-300.

Harvey, K., Kemps, E., & Tiggemann, M. (2005). The nature of imagery processes underlying food cravings. *British Journal of Health Psychology, 10,* 49-56.

Hawk Jr, L. W., Baschnagel, J. S., Ashare, R. L., & Epstein, L. H. (2004). Craving and startle modification during in vivo exposure to food cues. *Appetite*, *43*, 285-294.

Heatherton, T. F., Polivy, J., Herman, P., & Baumeister, R. F. (1991). Self-awareness, task failure, and disinhibition: How attentional focus affects eating. *Journal of Personality, 61*, 49-61.

Hepworth, R., Mogg, K., Brignell, C., & Bradley, B. P. (2010). Negative mood increases selective attention to food cues and subjective appetite. *Appetite, 54,* 134-142.

Hetherington, M. M., Cameron, F., Wallis, D. J., & Pirie, L. M. (2001). Stimulation of appetite by alcohol. *Physiology and Behavior, 74,* 283-289.

Hingson, R. W., Heeren, T., Winter, M., & Wechsler, H. (2005). Magnitude of alcohol-related mortality and morbidity among US college students ages 18-24: Changes from 1998 to 2001. *Annual Review of Public Health, 26,* 259-279.

Hoffmann, W., & Friese, M. (2008). Impulses got the better of me: Alcohol moderates the influence of implicit attitudes toward food cues on eating behavior. *Journal of Abnormal Psychology, 117*, 420-427.

Hohlstein, L. A., Smith, G. T., & Atlas, J. G. (1998). An application of the expectancy theory to eating disorders: Development and validation of measures of eating and dieting expectancies. *Psychological Assessment, 10,* 49-58.

Holt, S. H. A., Miller, J. C. B., Petocz, P., & Farmakalidis, E. (1995). A satiety index of common foods. *European Journal of Clinical Nutrition, 49,* 675-690.

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey replication. *Biological Psychiatry, 61,* 348-358.

Hull, J. G., & Bond, C. J. (1986). Social behavioral consequences of alcohol consumption and expectancy: A meta-analysis. *Psychological Bulletin, 99*, 374-360.

Jansen, A. (1998). A learning model of binge eating: Cue reactivity and cue exposure. *Behavior Therapy and Research, 36,* 257-272.

Jasinska, A. J., Yasuda, M., Burant, C. F., Gregor, N., Khatri, S., Sweet, M., & Falk, E. B. (2012). Impulsivity and inhibitory control deficits are associated with unhealthy eating in young adults. *Appetite, 59*, 738-747.

Johansson, L., Ghaderi, A., & Andersson, G. (2004). The role of sensitivity to external food cues in attentional allocation to food words on dot probe and Stroop tasks. *Eating Behaviors*, *5*, 261-271.

Kareken, D. A., Bragulat, V., Dzemidzic, M., Cox, C., Talavage, T., Davidson, D., & O'Connor, S. J. (2010). Family history of alcoholism mediates the frontal response to alcoholic drink odors and alcohol in at-risk drinkers. *Neuroimage, 50,* 267-276.

Kareken, D. A., Claus, E. D., Sari, M., Dzemidzic, M., Kosobud, A. E. K., Radnovich, A. J., Hector, D., Ramchandani, V. A., O'Connor, S. J., Lowe, M., & Li, T. (2004). Alcohol-related olfactory cues activate the nucleus accumbens and ventral tegmental area in high-risk drinkers: Preliminary findings. *Alcoholism: Clinical and Experimental Research, 28,* 550-557.

Kareken, D. A., Grahame, N., Dzemidzic, M., Walker, M. J., Lehigh, C. A., & O'Connor, S. J. (2012). fMRI of the brain's response to stimuli experimentally paired with alcohol intoxication. *Psychopharmacology, 220,* 787-797.

Karlsson, J., Persson, L. O., Sjöström, L., & Sullivan, M. (2000). Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *International journal of obesity and related metabolic disorders: Journal of the International Association for the Study of Obesity*, *24*, 1715-1725.

Karyadi, K. A., & Cyders, M. A. (2014). *The effects of food and alcohol odors on attentional bias, cravings, and consumption.* Unpublished manuscript, Department of Psychology, Indiana University-Purdue University in Indianapolis, Indianapolis, United States.

Karyadi, K. A., Coskunpinar, A., Entezari, A., Long, C., & Cyders, M. A. (2013). Mechanisms underlying the co-prevalence between problematic eating and drinking. In S. B. Harris (Ed.), *Binge Eating and Binge Drinking: Psychological, Social, and Medical Implications* (pp. 97-126). New York: Nova Science Publisher, Inc.

Kazdin, A. E. (2002). Research design in clinical psychology. Boston, MA: Allyn & Bacon.

Kemps, E., & Tiggemann, M. (2007). Modality-specific imagery reduces cravings for food: An application of the elaborated intrusion theory of desire to food craving. *Journal of Experimental Psychology: Applied*, *13*, 95-104.

Kemps, E., & Tiggemann, M. (2009). Attentional bias for craving-related (chocolate) food cues. *Experimental and Clinical Psychopharmacology, 17,* 425-433.

Kemps, E., & Tiggemann, M. (2013). Olfactory stimulation curbs food cravings. *Addictive Behaviors, 38,* 1550-1554.

Kemps, E., Tiggemann, M., & Bettany, S. (2012). Non-food odorants reduce chocolate cravings. *Appetite*, *58*, 1087-1090.

Kennedy, D. (1971). *Pupilometrics as an aid in the assessment of motivation, impact of treatment, and prognosis of chronic alcoholics* (Unpublished doctoral dissertation). University of Utah, Salt Lake City, Utah.

Kennedy, S. H., Katz, R., Neitzert, C. S., Ralevsky, E., & Mendlowitz, S. (1995). Exposure with response prevention treatment of anorexia nervosa–bulimic subtype and bulimia nervosa. *Behavior Research and Therapy, 33,* 685–689.

Kivlahan, D. R., Marlatt, G. A., Fromme, K., Coppel, D. B., & Williams, E. (1990). Secondary prevention of alcohol-related problems in young adults at risk. In K. D. Craig & S. M. Weiss (Eds.), *Health enhancement, disease prevention, and early intervention.* New York: Springer.

Kline, R. B. (1998). *Principles and practice of structured equation modeling.* New York: Guilford Press.

Laberg, J. C. (1990). What is presented, and what prevented, in cue exposure and response prevention with alcohol dependent subjects? *Addictive Behaviors*, *15*, 367-386.

Laibson, D. (2001). A cue-theory of consumption. *The Quarterly Journal of Economics*, *116*, 81-119.

Litt, M. D., & Cooney, N. L. (1999). Inducing craving for alcohol in the laboratory. *Alcohol Research and Health*, *23*, 174-178.

Litt, M. D., Cooney, N. L., & Morse, P. (2000). Reactivity to alcohol-related stimuli in the laboratory and in the field: Predictors of craving in treated alcoholics. *Addiction, 95,* 889-900.

Litt, M. D., Cooney, N. L., Kadden, R. M., & Gaupp, L. (1990). Reactivity to alcohol cues and induced moods in alcoholics. *Addictive Behaviors*, *15*, 137-146.

Ludwig, A. M., Wikler, A. (1974). Craving and relapse to drink. *Quarterly Journal of Studies on Alcohol, 35,* 108-130.

Lundholm, J. K. (1989). Alcohol use among university females: Relationship to eating disordered behavior. *Addictive Behaviors, 14,* 181-185.

Marlatt, A. G., Demming, B., & Reid, J. B. (1973). Loss of control drinking in alcoholics: An experimental analogue. *Journal of Abnormal Psychology, 81,* 233-241.

Martin, C. K., O'Neil, P. M., Tollefson, G., Greenway, F. L., & White, M. A. (2008). The association between food cravings and consumption of specific foods in a laboratory test. *Appetite, 51,* 324-326.

Martinovic, J., Jones, A., Christiansen, P., Rose, A. K., Hogarth, L., & Field M. (2014). Electrophysiological responses to alcohol cues are not associated with Pavlovian-to-Instrumental transfer in social drinkers. *PLOS One, 9,* 1-10.

McCusker, C. G., & Brown, K. (1990). Alcohol-predictive cues enhance tolerance and precipitate "craving" for alcohol in social drinkers. *Journal of Studies on Alcoholism, 51,* 494-499.

Metalis, S. A., Hess, E. H., & Beaver, P. W. (1982). Pupillometric analysis of two theories of obesity. *Perceptual and Motor Skills, 55,* 87-92.

Meule, A., Westenhofer, J., & Kubler, A. (2011). Food cravings mediate the relationship between rigid, but not flexible control of eating behavior and dieting success. *Appetite, 57,* 582-584.

Miller, M. A., & Fillmore, M. T. (2010). The effect of image complexity on attentional bias toward alcohol-related images in adult drinkers. *Addiction, 105,* 883-890.

Mineka, S. (1975). Some new perspectives on conditioned hunger. *Journal of Experimental Psychology: Animal Behavior Processes*, *1*, 134-138.

Mogg, K., Bradley, B. P., Field, M., & De Houwer, J. (2003). Eye movements to smoking-related pictures in smokers: Relationship between attentional biases and implicit and explicit measures of stimulus valence. *Addiction*, *98*, 825-836.

Mogg, K., Holmes, A., Garner, M., & Bradley, B. P. (2008). Effects of threat cues on attentional shifting, disengagement and response slowing in anxious individuals. *Behavior Research and Therapy, 46,* 656-667.

Monti, P. M., Rohsenow, D. J., Abrams, D. B., & Binkoff, J. A. (1988). Social learning approaches to alcohol relapse: Selected illustrations and implications. *NIDA Research Monograph, 84,* 141-160.

Nederkoorn, C., Smulders, F. T. Y., & Jansen, A. (2000). Cephalic phase responses, craving and food intake in normal subjects. *Appetite, 35,* 45-55.

Newlin, D. B. (1985). Human conditioned compensatory response to alcohol cues: Initial evidence. *Alcohol, 2,* 507-509.

Newlin, D. B. (1986). Conditioned compensatory response to alcohol placebo in humans. *Psychopharmacology, 88,* 247-251.

Newman, E., O'Connor, D. B., & Conner, M. (2008). Attentional biases for food stimuli in external eaters: Possible mechanism for stress-induced eating? *Appetite, 51,* 339-342.

O'Brien, C. P., Childress, A. R., McLellan, A. T., & Ehrman, R. (1992). Classical conditioning in drug-dependent humans. *Annals of the New York Academy of Sciences*, *654*, 400-415.

O'Brien, C. P., Childress, A. R., McLellan, T., & Ehrman, R. (1990). Integrating systematic cue exposure with standard treatment in recovering drug dependent patients. *Addictive Behaviors*, *15*, 355-365.

Papies, E. K., Stroebe, W., & Aarts, H. (2008). The allure of forbidden food: On the role of attention in self-regulation. *Journal of Experimental Social Psychology*, *44*, 1283-1292.

Pavlov, I. P. (1927). *Conditioned reflexes: An investigation of the physiological activity of the cerebral cortex.* London: Oxford University Press.

Pearce, J. M. (1987). A model for stimulus generalization in Pavlovian conditioning. *Psychological Review, 94,* 61-73.

Polivy, J., & Herman, C. P. (1976). The effects of alcohol on eating behavior: Disinhibition or Sedation? *Addictive Behaviors, 1*, 121-125.

Polivy, J., Coleman, J., & Herman, C. P. (2005). The effect of deprivation on food cravings and eating behavior in restrained and unrestrained eaters. *International Journal of Eating Disorders, 38,* 301-309.

Poppitt, S. D., & Prentice, A. M. (1996). Energy density and its role in the control of food intake: evidence from metabolic and community studies. *Appetite*, *26*, 153-174.

Powley, T. L. (1977). The ventromedial hypothalamic syndrome, satiety and cephalic phase hypothesis. *Psychological Review, 84,* 89-126.

Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 25*, 7-13.

Reed, S. C., Levin, F. R., & Evans, S. M. (2012). Alcohol increases impulsivity and abuse liability in heavy drinking women. *Experimental and Clinical Psychopharmacology, 20*, 454-465.

Reinert, D. F., & Allen, J. P. (2002). The alcohol use disorders identification test (AUDIT): A review of recent research. *Alcoholism: Clinical and Experimental Research*, *26*, 272-279.

Roberts, W., Fillmore, M. T., & Milich, R. (2012). Drinking to distraction: Does alcohol increase attentional bias in adults with ADHD? *Experimental and Clinical Psychopharmacology, 20,* 107-117.

Rodin, J., Mancuso, J., Granger, J., & Nelbach, E. (1991). Food cravings in relation to body mass index, restraint and estradiol levels: A repeated measures study in healthy women. *Appetite, 17,* 177-185.

Rothman, K. J. (1990). No adjustments are needed for multiple comparisons. *Epidemiology, 1,* 43-46.

Rubin, L. S., Gottheil, E., Roberts, A., Alterman, A. I., & Holstine, J. (1978). Autonomic nervous system concomitants of short-term abstinence in alcoholics. *Journal of Studies on Alcohol, 39,* 1895-1907.

Rush, C. C., Becker, S. J., & Curry, J. F. (2009). Personality factors and styles among college students who binge eat and drink. *Psychology of Addictive Behaviors, 23,* 140-145.

Ryan, J. J., Kreiner, D. S., Chapman, M. D., & Stark-Wroblewski, K. (2010). Virtual reality cues for binge drinking in college students. *Cyberpsychology, Behavior, and Social Networking, 13,* 159-162.

Sahakian, B. J., Lean, M. E. J., Robbins, T. W., & James, W. P. T. (1981). Salivation and insulin secretion in response to food in non-obese men and women. *Appetite*, *2*, 209-216.

Sayette, M. A., Martin, C. S., Wertz, J. M., Perrott, M. A., & Peters, A. R. (2005). The effects of alcohol on cigarette craving in heavy smokers and tobacco chippers. *Psychology of Addictive Behaviors*, *19*, 263-270.

Schmitz, S., Krummenauer, F., Henn, S., & Dick, H. B. (2003). Comparison of three different technologies for pupil diameter measurement. *Graefe's Archive of Clinical and Experimental Ophthalmology, 241,* 472-477.

Schoenmakers, T. M., de Bruin, M., Lux, I. F., Goertz, A. G., Van Kerkhof, D. H., & Wiers, R. W. (2010). Clinical effectiveness of attentional bias modification training in abstinent alcoholic patients. *Drug and Alcohol Dependence*, *109*, 30-36.

Schoenmakers, T., Wiers, R. W., & Field, M. (2008). Effects of a low dose of alcohol on cognitive biases and craving in heavy drinkers. *Psychopharmacology*, *197*, 169-178.

Shafran, R., Lee, M., Cooper, Z., Palmer, R. L., & Fairburn, C. G. (2007). Attentional bias in eating disorders. *International Journal of Eating disorders, 40,* 369-380.

Siegel, S. (1983). Classical conditioning, drug tolerance and drug dependence. *Research Advances in Alcohol and Drug Problems, 7,* 207–246.

Sinha, R., & O'Malley, S. S. (2000). Alcohol and eating disorders: Implications for alcohol treatment and health services research. *Alcoholism: Clinical and Experimental Research, 24,* 1312-1319.

Smeets, E., Roefs, A., van Furth, E., & Jansen, A. (2008). Attentional bias for body and food in eating disorders: Increased distraction, speeded detection, or both? *Behavior Research and Therapy*, *46*(2), 229-238.

Smith-Hoerter, K., Stasiewicz, P. R., & Bradizza, C. M. (2004). Subjective reactions to alcohol cue exposure: A qualitative analysis of patients' self-reports. *Psychology of Addictive Behaviors, 18,* 402-406.

Sobik, L., Hutchison, K., & Craighead, L. (2005). Cue-elicited craving for food: A fresh approach to the study of binge eating. *Appetite, 44,* 253-261.

Staiger, P. K., & White, J. M. (1988). Conditioned alcohol-like and alcohol-opposite responses in humans. *Psychopharmacology, 95,* 87-91.

Stewart, S. B., Brown, C. G., Devoulyte, K., Theakston, J., & Larsen, S. E. (2006). Why do women with alcohol problems binge eat? Exploring connections between binge eating and heavy drinking in women receiving treatment for alcohol problems. *Journal of Health Psychology, 31,* 409-425.

Stormark, K. M., Laberg, J. C., Nordby, H., & Hugdahl, K. (2000). Alcoholics' Selective Attention to Alcohol Stimuli: Automated Processing? *Journal of Studies on Alcohol and Drugs*, *61*, 18-23.

Subar, A. F., Kirkpatrick, S. I., Mittl, B., Zimmerman, T. P., Thompson, F. E., Bingley, C., ... & Potischman, N. (2012). The Automated Self-Administered 24-hour dietary recall (ASA24): A resource for researchers, clinicians, and educators from the National Cancer Institute. *Journal of the Academy of Nutrition and Dietetics*, *112*, 1134-1137.

Svaldi, J., Brand, M., & Tuschen-Caffier, B. (2010). Decision-making impairments in women with binge eating disorder. *Appetite, 54*, 84-92.

Taylor, A. V., Peveler, R. C., Hibbert, G. A., & Fairburn, C. G. (1993). Eating disorders among women receiving treatment for an alcohol problem. *International Journal of Eating Disorders, 14,* 147-151.

Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and non-automatic processes. *Psychological Review, 97,* 147-168.

Till, B. D., & Priluck, R. L. (2000). Stimulus generalization in classical conditioning: An initial investigation and extension. *Psychology and Marketing*, *17*, 55-72.

Toro, J., Cervera, M., Feliu, M. H., Garriga, N., Jou, M., Martinez, E., & Toro, E. (2003). Cue exposure in the treatment of resistant bulimia nervosa. *International Journal of Eating Disorders*, *34*, 227-234.

Townshend, J., & Duka, T. (2001). Attentional bias associated with alcohol cues: differences between heavy and occasional social drinkers. *Psychopharmacology*, *157*, 67-74.

Ward, A. & Mann, T. (2000). Don't mind if I do: Disinhibited eating under cognitive load. *Journal of Personality and Social Psychology, 78*, 753-763.

Wardle, J. (1990). Conditioning processes and cue exposure in the modification of excessive eating. *Addictive Behaviors, 15,* 387–393.

Waters, A., Hill, A., & Waller, G. (2001). Bulimics' response to food cravings: IS binge-eating a product of hunger or emotional state. *Behavior Research and Therapy, 39,* 877-886.

Weafer, J. & Fillmore, M. T. (2012). Comparison of alcohol impairment of behavioral and attentional inhibition. *Drug and Alcohol Dependence, 126*, 176-182.

Weafer, J., & Fillmore, M. T. (2008). Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. *Psychopharmacology, 2013,* 315-324.

Werthmann, J., Field, M., Roefs, A., Nederkoorn, C., & Jansen, A. (2014). Attention bias for chocolate increases chocolate consumption–An attention bias modification study. *Journal of Behavior Therapy and Experimental Psychiatry*, *45*, 136-143.

Wikler, A. (1973). Conditioning of successive adaptive responses to the initial effects of drugs. *Conditioned Reflex, 8,* 193–210.

Willner, P., Field, M., Pitts, K., & Reeve, G. (1998). Mood, cue and gender influences on motivation, craving and liking for alcohol in recreational drinkers. *Behavioural Pharmacology, 9,* 631-642.

Wolfe, W. L., & Maisto, S. A. (2000). The relationship between eating disorders and substance use: Moving beyond co-prevalence research. *Clinical Psychology Review, 20,* 617-631.

Wooley, O. W., & Wooley, S. C. (1981). Relationship of salivation in humans to deprivation, inhibition and the encephalization of hunger. *Appetite*, *2*, 331-350.

Yeomans, M. R. (2010). Short term effects of alcohol on appetite in humans. Effects of context and restrained eating. *Appetite*, *55*, 565-573.

Yeomans, M. R., Caton, S., & Hetherington, M. M. (2003). Alcohol and food intake. *Current Opinion* in *Clinical Nutrition & Metabolic Care, 6, 639-644.*

Yeomans, M. R., Hails, N. J., & Nesic, J. S. (1999). Alcohol and the appetizer effect. *Behavioural Pharmacology*, *10*, 151-161.

Yip, W., Wiessing, K. R., Budgett, S., & Poppitt, S. D. (2013). Using a smaller dining plate does not suppress food intake from a buffet lunch meal in overweight, unrestrained women. *Appetite, 69,* 102-107.

Yokum, S., Ng, J., & Stice, E. (2011). Attentional bias to food images associated with elevated weight and future weight gain: an fMRI study. *Obesity*, *19*, 1775-1783.

Yoon, G., Kim, S. W., Thuras, P., Grant, J. E., & Westermeyer, J. (2006). Alcohol craving in outpatients with alcohol dependence: Rate and clinical correlates. *Journal of Studies on Alcohol and Drugs, 67,* 770-777.

Yoon, M. K., Schmidt, G., Lietman, T., & McLeod, S. D. (2007). Inter- and intraobserver reliability of pupil diameter measurement during 24 hours using the Colvard pupillometer. *Journal of Refractive Surgery, 23,* 266-271.

APPENDICES

Appendix A: Preliminary Analyses and Regression Effects

Table A1

Covariates and Consumptive Behaviors

Continuous Variables	***M***	***SD***	***Range***	**Categorical Variables**	***Frequency***	***Percentage***
Age	30.84	9.46	18-54	**Gender**		
				Male	37	48.1
Time of Day	1393.49	289.11	900-1900	Female	40	51.9
State Hunger	4.64	1.3	2-7	**Race**		
				Caucasian	64	83.1
Alcohol Use Days	5.63	2.66	1-13	African	8	10.4
				Hispanic	2	2.6
Beer Use Days	3.93	2.71	1-13	Asian	1	1.3
				Other	2	2.6
Alcohol and Food Days	3.67	2.19	1-11			
				Day of the Week		
Beer and Food Days	2.71	1.99	1-11	Monday	21	27.3
				Tuesday	18	23.4
Post-Lab Food Satiety	66.01	21.44	10-100	Wednesday	6	7.8
				Thursday	9	11.7
Post-Lab Food Typicality	54.86	19.55	0-100	Friday	23	29.9
Post-Lab Alcohol use	2.49	2.03	1-9	**Post-Lab Food**		
				Yes	69	93.2
Post-Lab Alcohol Typicality	39.65	23.97	0-100	No	5	6.8
Problematic Alcohol Use	8.56	5.11	2-27	**Post-Lab Alcohol**		
				Yes	35	47.3
Problematic Eating	2.27	0.45	1.30-3.60	No	39	52.7
				Post-Lab Co-Consumption		
				Yes	24	42.1
				No	33	57.9

Table A2

Key Study Variables across Trials

	Baseline		Trial 1		Trial 2		Trial 3		Trial 4	
	M	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**
Alcohol Cravings (L)	18.64	8.09	24.31	12.31	23.85	12.46	22.25	11.01	21.65	10.43
Food Cravings (L)	33.08	9.01	34.33	10.89	36.01	10.54	34.44	10.30	35.35	10.20
Alcohol Cognitive AB (ms)			1.64	54.34			3.39	43.84		
Food Cognitive AB (ms)					-0.32	46.53			-3.61	58.69
Alcohol Duration AB (ms)			0.16	0.24			0.09	0.23		
Food Duration AB (ms)					0.20	0.27			0.09	0.18
Alcohol Direction AB (%)			55.29	8.09			52.13	9.92		
Food Direction AB (%)					56.20	9.95			54.04	7.75
Alcohol Arousal (mm)			1.69	5.09			1.05	6.14		
Food Arousal (mm)					1.63	6.25			0.14	4.78
Odor Intensity (L)			2.87	0.80	2.90	0.83	1.35	0.66	1.38	0.72
Odor Pleasantness (L)			2.94	0.63	2.93	0.60	2.71	0.68	2.77	0.54
Odor Represantiveness (L)			3.16	0.77	3.20	0.65	1.55	0.70	1.85	1.56

Note. Parentheses: L-Likert; ms-millisecond; %-percent; mm-millimeter. Trials: Trial 1-alcohol odors and alcohol pictures; Trial 2-alcohol odors and food pictures; Trial 3-neutral odors and alcohol pictures; Trial 4-neutral odors and food pictures. AB-attentional bias. Arousal-sympathetic arousal.

Table A3

Percentage of Missing Data.

Missing Data Breakdown

Overall (total n = 77)
59.7% were missing at least one variable
(46 out of 77 participants)

Alcohol Cravings
Baseline: 5.19% (4 out of 77)
Trial 1: 7.79% (6 out of 77)
Trial 2: 2.60% (2 out of 77)
Trial 3: 0.00% (0 out of 77)
Trial 4: 0.00% (0 out of 77)

Food Cravings
Baseline: 1.30% (1 out of 77)
Trial 1: 6.49% (5 out of 77)
Trial 2: 1.30% (1 out of 77)
Trial 3: 0.00% (0 out of 77)
Trial 4: 0.00% (0 out of 77)

Alcohol Cognitive AB
Trial 1: 16.88% (13 out of 77)
Trial 3: 9.10% (7 out of 77)

Food Cognitive AB
Trial 2: 7.79% (6 out of 77)
Trial 4: 10.39% (8 out of 77)

Alcohol Duration AB
Trial 1: 15.58% (12 out of 77)
Trial 3: 7.79% (6 out of 77)

Food Duration AB
Trial 2: 10.39% (8 out of 77)
Trial 4: 10.39% (8 out of 77)

Alcohol Direction AB
Trial 1: 15.58% (12 out of 77)
Trial 3: 7.79% (6 out of 77)

Food Direction AB
Trial 2: 10.39% (8 out of 77)
Trial 4: 10.39% (8 out of 77)

Alcohol Sympathetic Arousal
Trial 1: 15.58% (12 out of 77)
Trial 3: 7.79% (6 out of 77)

Food Sympathetic Arousal
Trial 2: 10.39% (8 out of 77)
Trial 4: 10.39% (8 out of 77)

Alcohol Consumption (Post-Lab)
Alcohol Use: 9.09% (7 out of 77)
Alcohol Typicality: 7.79% (6 out of 77)
Concurrent Consumption: 26% (20 out of 77)

Food Consumption (Post-Lab)
Food Satiety: 3.90% (3 out of 77)
Food Typicality: 3.89% (3 out of 77)

Note. AB-attentional bias.

Table A4

Demographic Differences across Key Study Variables

	Age		Gender		Race	
	r	p-value	t-stat	p-value	F-stat	p-value
Alcohol Cravings (L)						
Baseline	-.16	.19	1.41	.16	**2.55**	**.046**
Alcohol Odor, Alcohol Pictures	**-.24**	**.04**	-.01	.99	**3.10**	**.02**
Alcohol Odor, Food Pictures	**-.26**	**.02**	-.16	.87	1.46	.22
Neutral Odor, Alcohol Pictures	-.18	.12	.10	.92	2.10	.09
Neutral Odor, Food pictures	-.08	.47	.15	.88	2.49	.051
Food Cravings (L)						
Baseline	-.14	.23	1.89	.06	**3.13**	**.02**
Alcohol Odor, Alcohol Pictures	**-.27**	**.02**	.68	.50	**3.91**	**.01**
Alcohol Odor, Food Pictures	-.22	.06	.55	.58	**3.72**	**.01**
Neutral Odor, Alcohol Pictures	-.18	.11	1.19	.24	**3.83**	**.01**
Neutral Odor, Food pictures	-.12	.31	.31	.76	2.00	.10
Cognitive Attentional Bias (ms)						
Alcohol Attentional Bias (AOD)	-.04	.75	-1.30	.20	.62	.61
Alcohol Attentional Bias (NOD)	**.35**	**.00**	-1.39	.17	.12	.95
Food Attentional Bias (AOD)	.04	.72	.00	.99	**3.07**	**.03**
Food Attentional Bias (NOD)	.09	.46	-.23	.82	.36	.78
Direction Attentional Bias (%)						
Alcohol Attentional Bias (AOD)	-.20	.12	.68	.50	1.60	.20
Alcohol Attentional Bias (NOD)	-.16	.17	1.86	.07	1.87	.13
Food Attentional Bias (AOD)	**-.34**	**.00**	-.53	.60	**2.89**	**.03**
Food Attentional Bias (NOD)	-.21	.08	.55	.59	1.17	.33
Duration Attentional Bias (ms)						
Alcohol Attentional Bias (AOD)	-.20	.11	1.14	.26	**2.96**	**.03**
Alcohol Attentional Bias (NOD)	-.08	.53	1.51	.13	1.79	.14
Food Attentional Bias (AOD)	**-.32**	**.01**	-.01	.99	**4.35**	**.00**
Food Attentional Bias (NOD)	**-.32**	**.01**	-1.07	.29	.52	.72
Sympathetic Arousal (mm)						
Alcohol Sympathetic Arousal (AOD)	-.14	.28	.87	.39	.32	.87
Alcohol Sympathetic Arousal (NOD)	.06	.62	**2.05**	**.04**	1.17	.33
Food Sympathetic Arousal (AOD)	-.17	.17	-.07	.94	**3.29**	**.02**
Food Sympathetic Arousal (NOD)	-.06	.64	-.46	.64	.46	.76
Post-Lab Consumption (L)						
Post-Lab Food Satiety	-.13	.28	-.72	.47	.90	.47
Post-Lab Food Typicality	-.17	.15	-.12	.91	.09	.98
Post-Lab Alcohol Use	.16	.19	.12	.91	.53	.72
Post-Lab Alcohol Typicality	.19	.11	-.02	.98	1.14	.34
Post-Lab Concurrent Consumption	.12	.37	.33	.74	1.16	.33

Note. Bolded values are significant. AOD-alcohol odorants. NOD-neutral odorants. L-Likert. ms-millisecond. %-percent. mm-millimeter.

Table A5

Effects of Key Study Variables on Post-Lab Consumption

	Food Satiety		Food Typicality		Alcohol Use		Alcohol Typicality		Co-consumption	
	β	p-value	*β*	p-value	*β*	p-value	*β*	p-value	Exp(b)	p-value
Alcohol Cravings (Alcohol)	.001	.99	.02	.95	.35	.29	.14	.67	1.01	.85
Alcohol Cravings (Food)	.07	.84	.10	.78	-.08	.81	-.09	.79	.95	.49
Food Cravings (Alcohol)	-.002	.99	-.01	.98	**.48**	**.04**	.15	.53	.95	.33
Food Cravings (Food)	.20	.45	.004	.99	-.15	.50	-.14	.56	1.02	.80
Alcohol Cognitive AB (ms)	.06	.67	.02	.88	**.30**	**.03**	-.06	.70	1.00	.84
Food Cognitive AB (ms)	-.09	.50	-.15	.26	-.07	.57	-.15	.23	1.01	.49
Alcohol Direction AB (%)	.26	.46	.25	.44	.03	.37	-.01	.97	.98	.59
Food Direction AB (%)	.22	.11	.08	.57	**-.25**	**.04**	-.12	.36	1.00	.91
Alcohol Duration AB (ms)	.01	.96	.11	.44	.14	.31	.05	.70	1.18	.90
Food Duration AB (ms)	.02	.88	.14	.32	-.24	.06	-.16	.22	1.83	.62
Alcohol Sympathetic Arousal (mm)	.02	.86	.23	.10	.06	.64	-.07	.59	.95	.49
Food Sympathetic Arousal (mm)	-.01	.92	-.04	.78	**-.28**	**.02**	-.17	.19	1.04	.43

Note. Bolded coefficients are significant. Parentheses: Alcohol-alcohol pictures; Food-food pictures; ms-millisecond; %-percent; mm-millimeter. AB-attentional bias. Likert scale used for cravings.

Appendix B: Methodology Figures

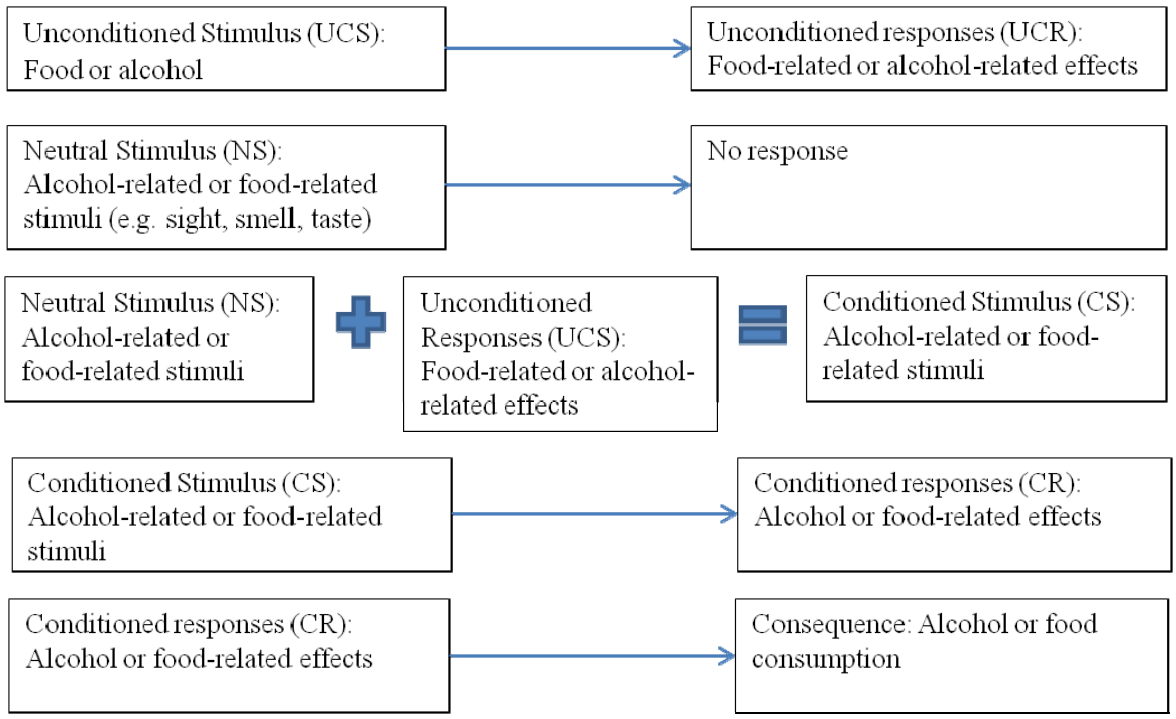


Figure B1

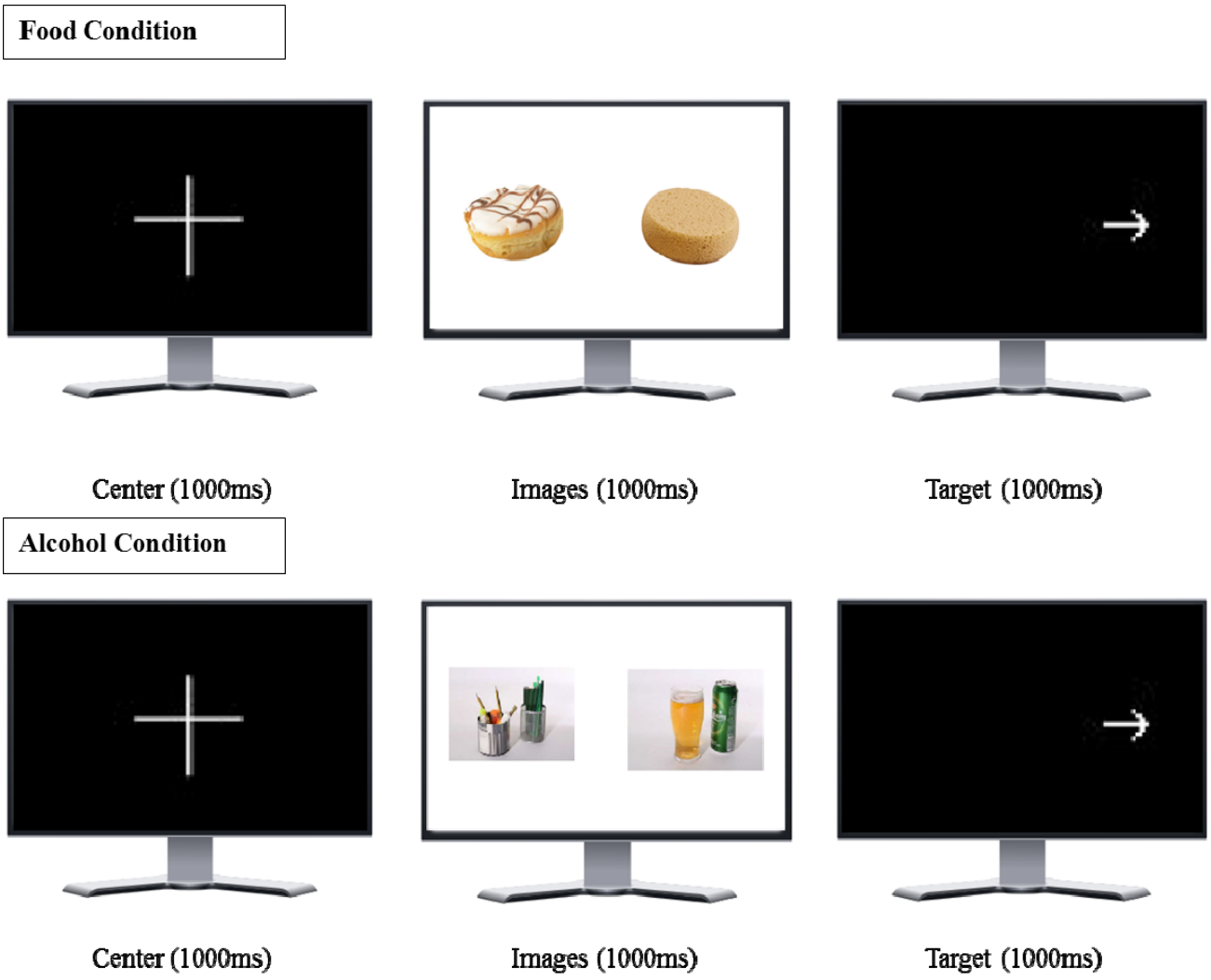
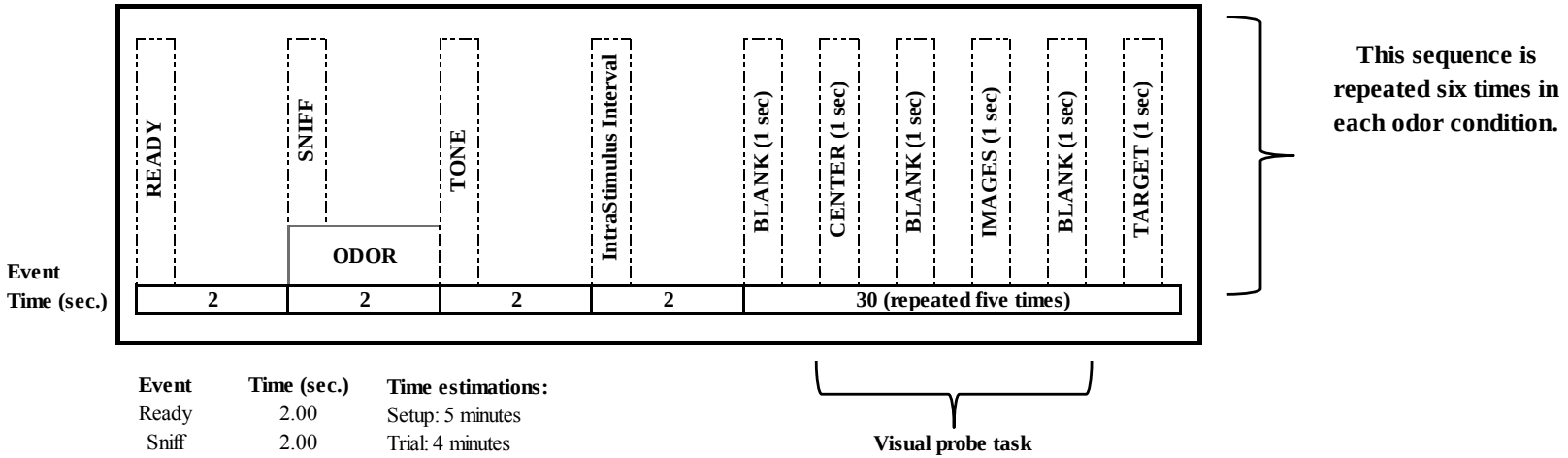

Food Condition
Center (1000ms)
Images (1000ms)
Target (1000ms)
Alcohol Condition
Center (1000ms)
Images (1000ms)
Target (1000ms)

Figure B2


Participants' Computer
Olfactometer Laptop
Eye-Tracker Interface Computer

Figure B3



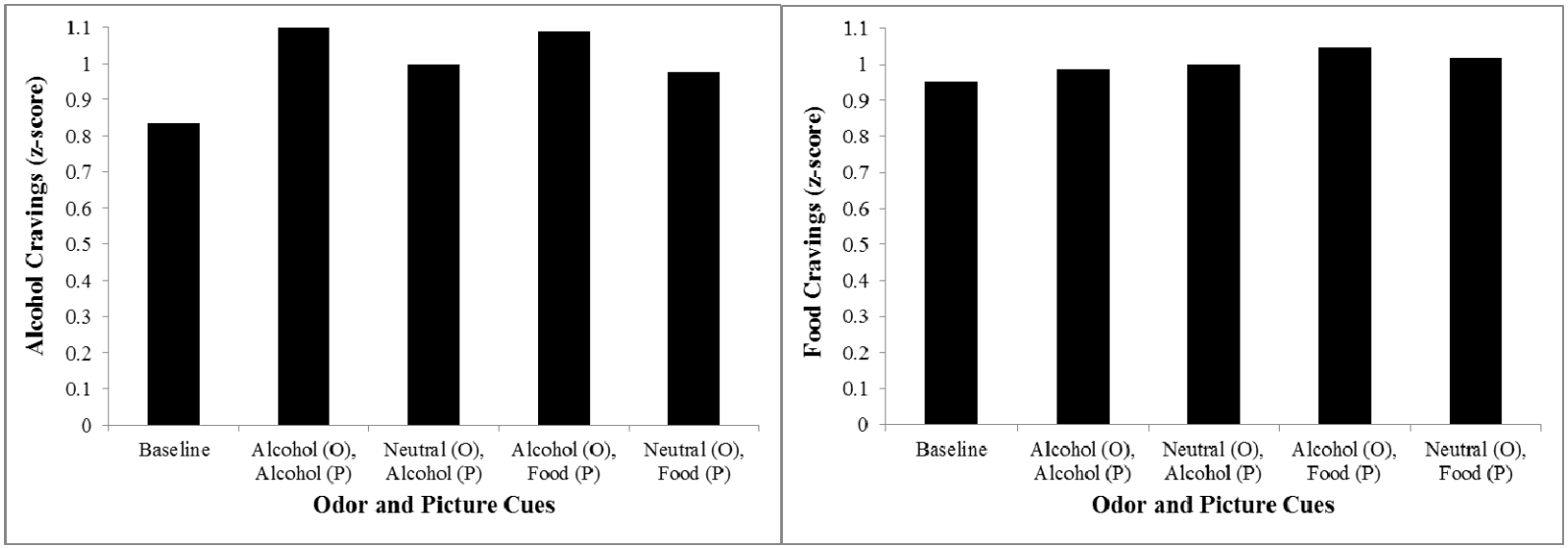
Event	Time (sec.)
Ready	2.00
Sniff	2.00
Tone	2.00
ISI	2.00
Visual Probe	30.00

Time estimations:
Setup: 5 minutes
Trial: 4 minutes
Total: 9 minutes/trial

Figure B4

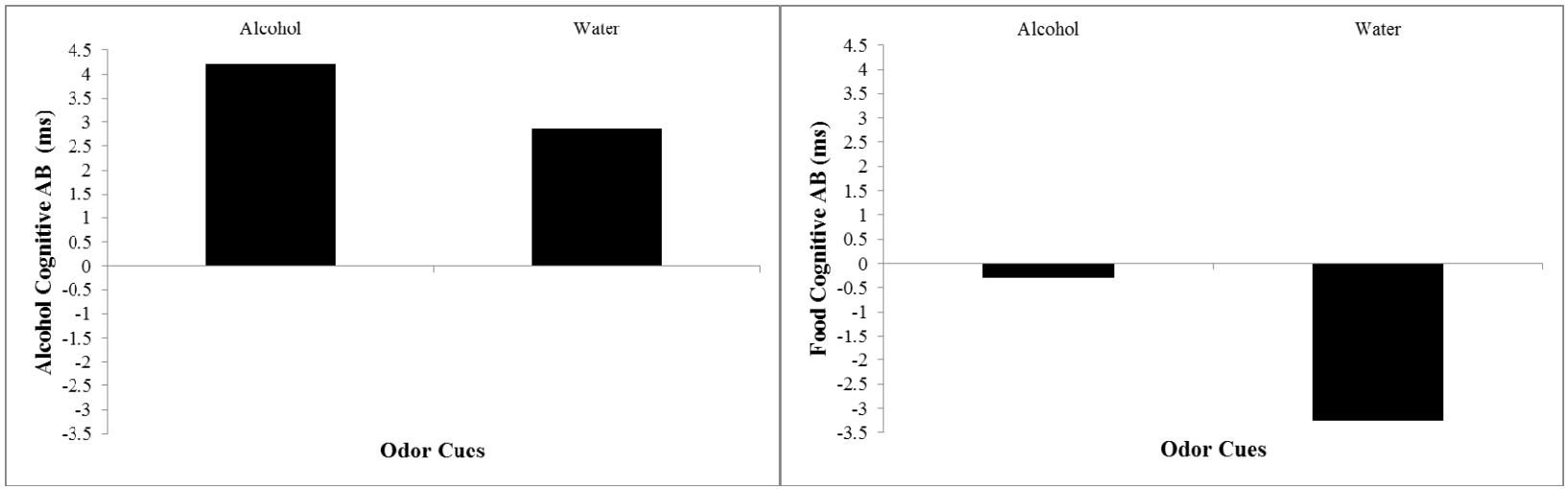
Figure B5

Appendix C: Alcohol Cue Elicited Responses



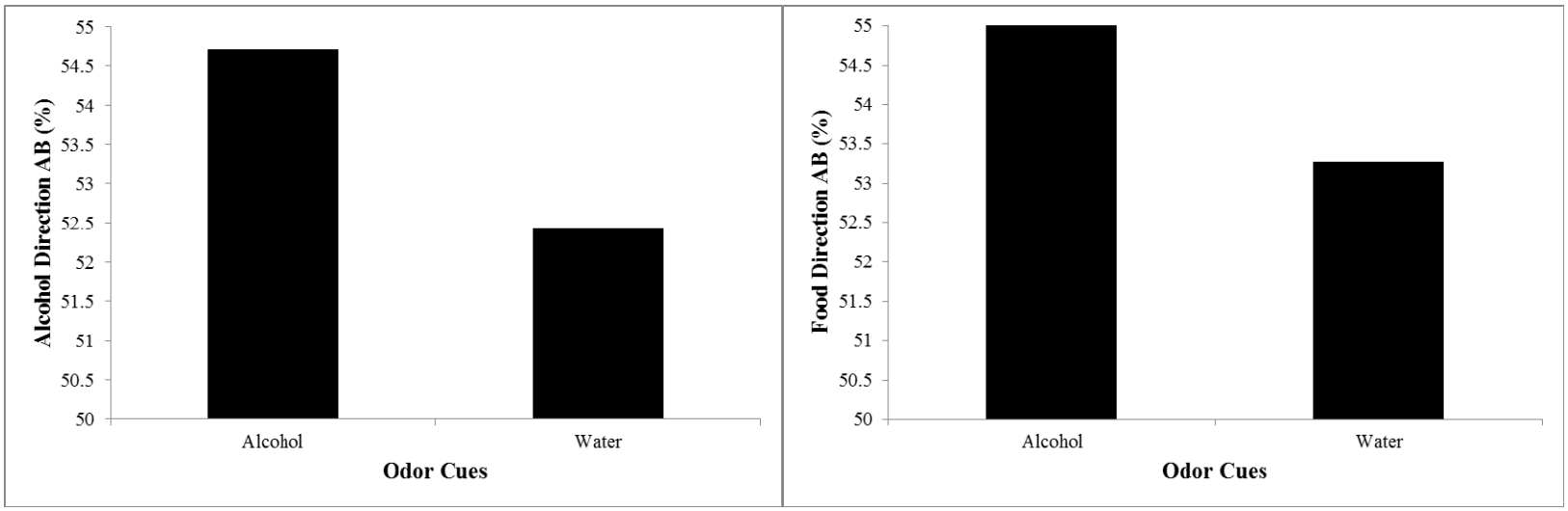
Note. Likert scale used to assess food and alcohol cravings. Overall cravings were calculated by averaging mean cravings across the four trials. Z-scores were calculated by comparing mean cravings in each condition to overall cravings. Odor and Pictorial Cues: O-odorants; P-pictures.

Figure C1



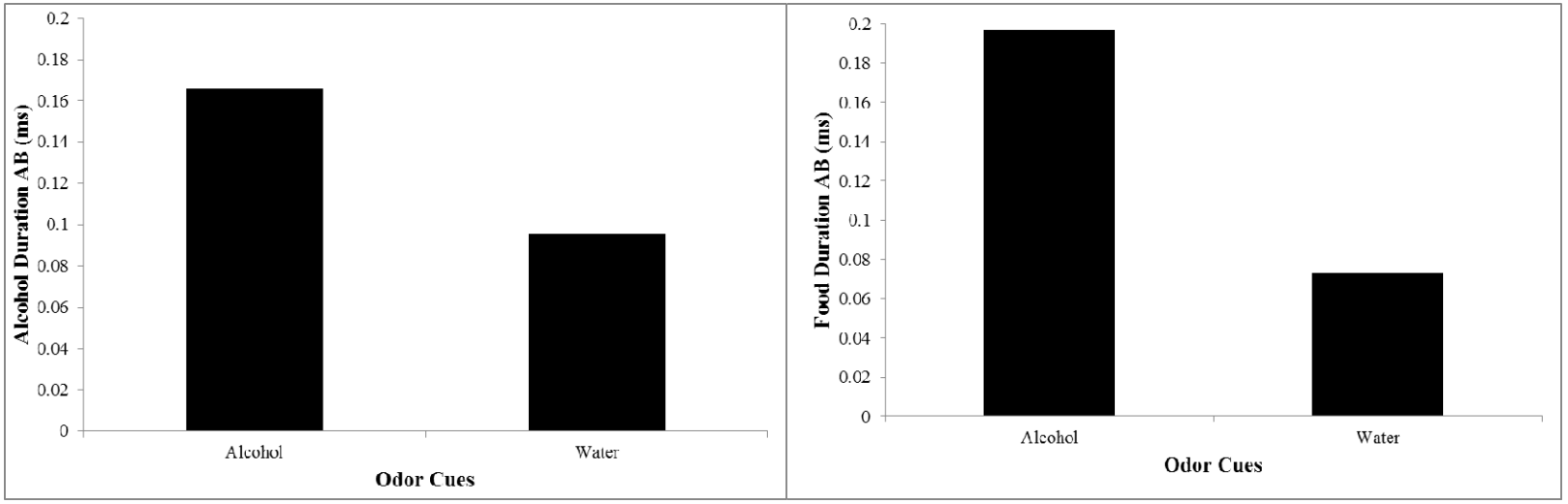
Note. AB-attentional bias. ms-millisecond.

Figure C2



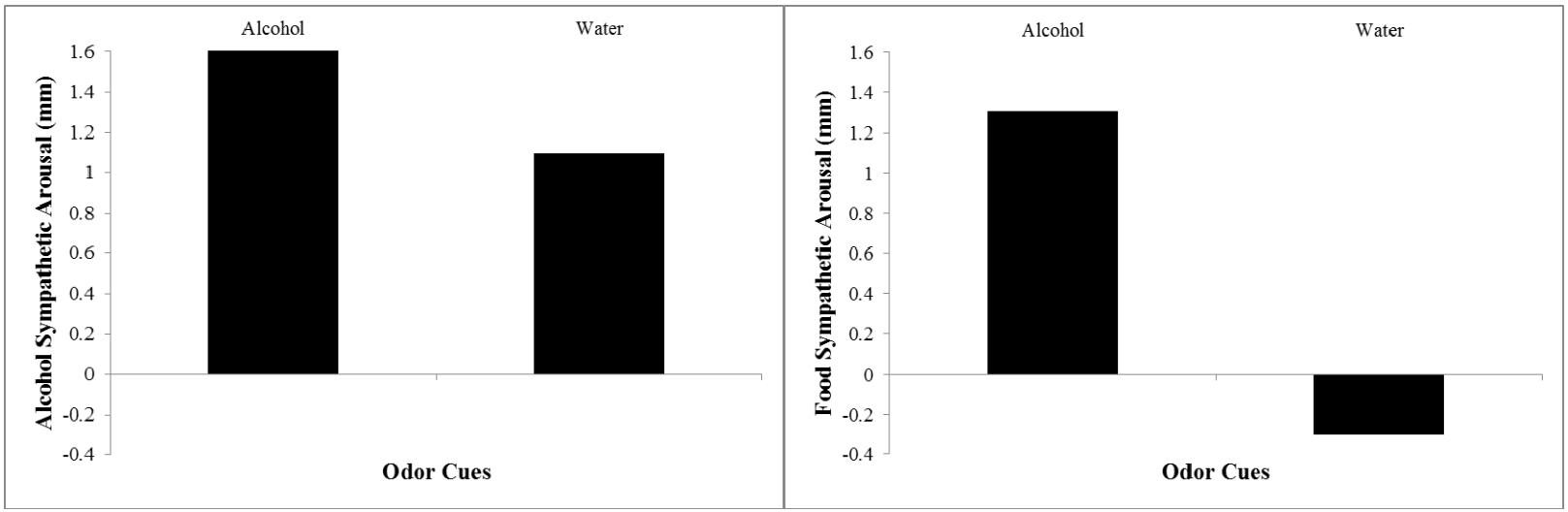
Note. AB-attentional bias. %-percent.

Figure C3



Note. AB-attentional bias. ms-millisecond.

Figure C4



Note. AB-attentional bias. mm-millimeter.

Figure C5

Appendix D: Effects on Post-Lab Consumption

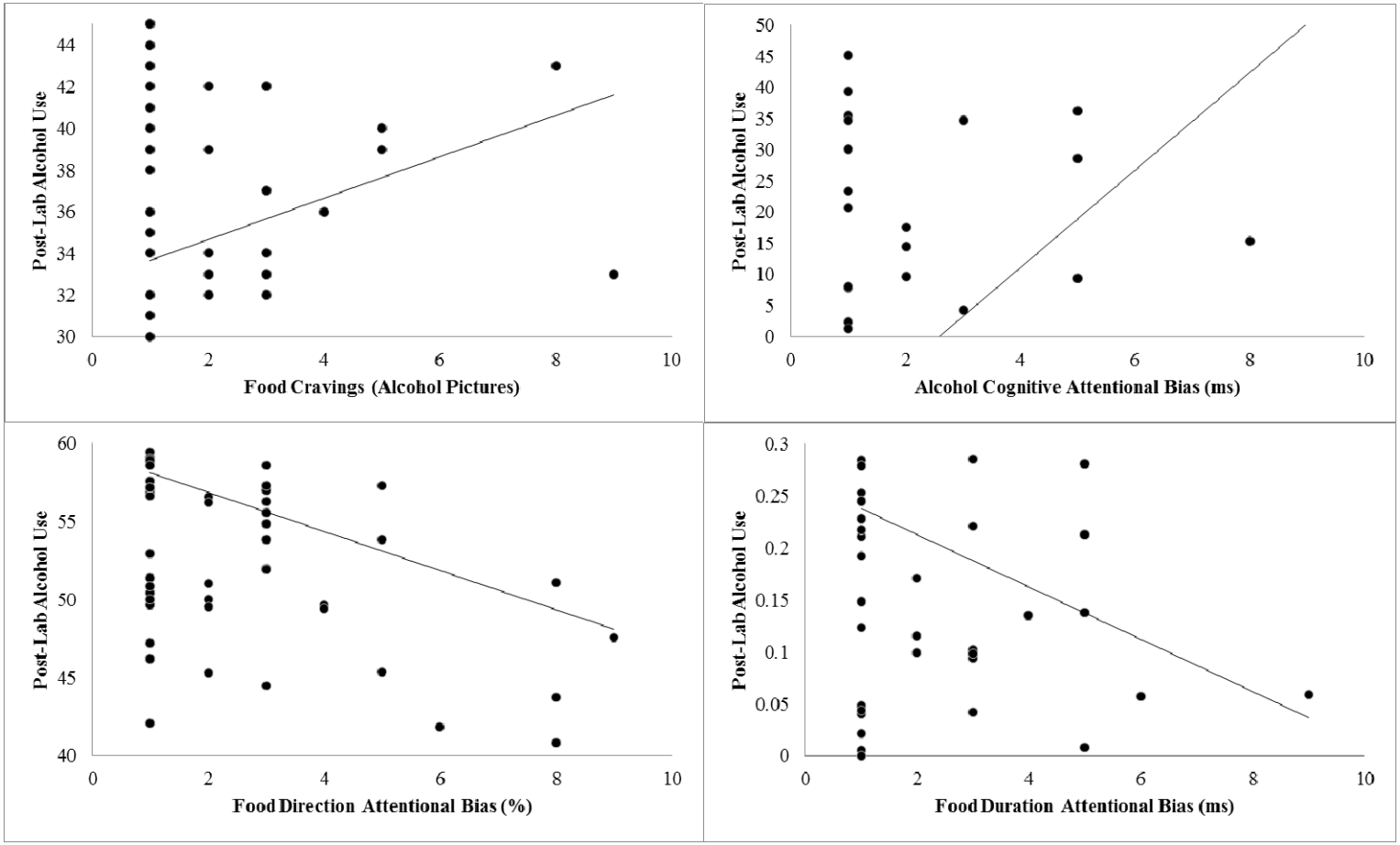
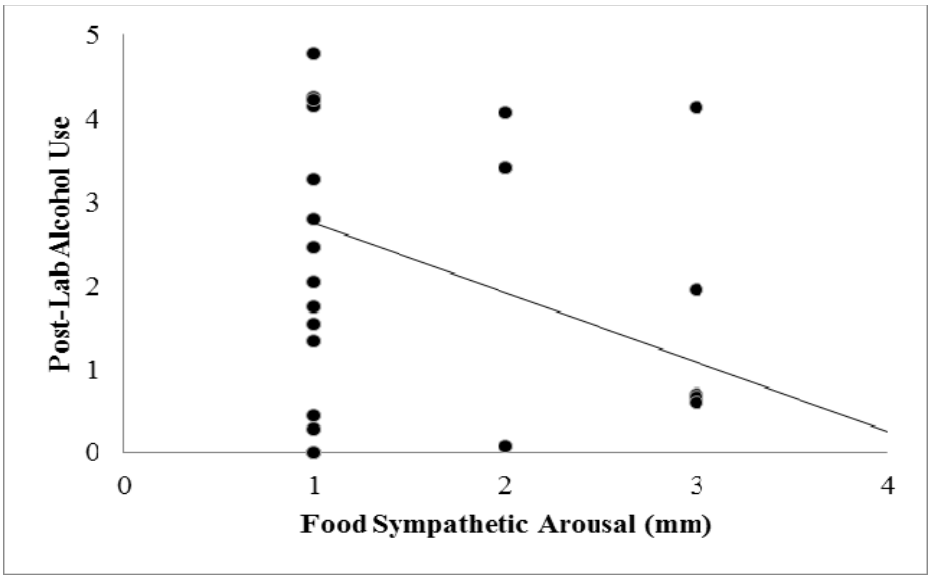


Figure D1



Note. Effect of food duration attentional bias fell short of significance ($p = 0.06$).

Figure D2

Appendix E: Pilot Study Summary

METHOD

Design

The pilot study examined the effects of: (1) odor cues (beer, pizza, and water odorants) on food and alcohol cravings and attentional bias (cognitive, direction, and duration); and (2) food and alcohol cravings and attentional bias elicited by food and alcohol odor cues on post-lab food and alcohol consumption. Participants were assigned to one of two conditions: (1) food condition, wherein participants saw food pictures matched with control pictures, and had their food cravings and attentional bias assessed; and (2) alcohol condition, wherein participants saw alcohol pictures matched with control pictures, and had their alcohol cravings and attentional bias assessed. On the day after the lab portion of the study, all participants completed a short online survey, which contained two questions on post-lab food and alcohol consumption.

Participants

Participant in the pilot study consisted solely of college students enrolled in introductory psychology courses. In order to eligible for the study, students must indicate that they are (1) at least 18 years of age, (2) speak English fluently, (3) consume alcohol at least once a week, (4) consume at least one drink every drinking session, and (5) occasionally consume food and alcohol concurrently. Data were collected from 20 participants. Data from one participant were excluded from the analyses due to an

olfactometer malfunction (e.g. olfactometer was not administering odorants during the trials). Of the remaining 19 participants, 10 had been assigned to the alcohol condition and 9 had been assigned to the food condition. These participants had a mean age of 29.21 (*SD* = 12.35); 57.9% were female and 42.1% were male; and 78.9% of the participants were Caucasian, with 10.5% reporting being Hispanic American and 10.5% reporting being African American. Most participants (17 out of 19) reported consuming beer in the past two weeks, and less than half of the participants (8 out of 19) reported consuming pizza with beer in the past two weeks. Participants reported beer consumption in 3-4 days out of the past two weeks (*M* = 3.53, *SD* = 2.55), and those who concurrently consumed beer and pizza reported doing so at least once every two weeks (*M* = 1.38, *SD* = 0.52).

PILOT RESULTS

Missing Data

All participants (19 participants) completed measures of post-lab food and alcohol consumption. Participants in the food condition (9 participants) completed measurements of food cravings and attentional bias, while participants in the alcohol condition (10 participants) completed measurements of alcohol cravings and attentional bias. For each participant, there should be a total of 15 separate variables of interest: 4 cravings variables (baseline, neutral odors, alcohol odors, and food odors), 9 attentional bias variables (neutral odors, alcohol odors, and food odors for duration, direction, and cognitive attentional bias), and 2 post-lab consumption variables (food and alcohol

consumption). In the alcohol condition, 70% of participants (7 out of 10 participants) had missing data in at least one of these fifteen variables. In the food condition, 56% of participants (5 out of 9 participants) had missing data in at least one of these fifteen variables. On average, participants do not appear to be missing more than 14.5% of these variables. As a result, participants with missing data were still included in the analyses.

Aforementioned variables with missing data were imputed using linear regression imputations. Using this approach, multiple imputations (5 total imputations) were calculated for each variable. Although F-statistics and p-values vary greatly across separate imputations, pooled mean values for attentional bias, cravings, and post-lab consumption across separate imputations did not substantially differ from the original mean values. Relatedly, the pattern of mean differences across odorant trials does not seem to differ between analyses of imputed and non-imputed data sets. Due to the similarities in mean values and differences across trials, and because F-statistics and p-values cannot be pooled across separate imputations in SPSS, the results presented below are from analyses of non-imputed data.

Cravings

A repeated measures analysis of variance (ANOVA) indicated that the main effect of odor trials (baseline, neutral odors, beer odors, pizza odors exposure) on alcohol cravings was not significant, F (3, 24) = 1.08, p = 0.38. Despite this non-significant effect, which would be expected given the small sample size, examination of mean alcohol cravings across the four trials indicates a pattern of meaningful trends. In particular, mean alcohol cravings was higher following alcohol odorant trials (M = 24.00,

SD = 15.69) and food odorant trials (*M* = 22.22, *SD* = 12.30) relative to neutral odorant trials (*M* = 20.67, *SD* = 11.74) and baseline (*M* = 20.44, *SD* = 12.24). In general, the pattern of results indicates that exposure to food and alcohol odorants might be producing higher alcohol cravings compared to exposure to neutral odorants and compared to baseline alcohol cravings.

The main effect of trials on food cravings was not significant, F (3, 21) = 0.45, p = 0.45. Again, despite the non-significant effect, food cravings across the four trials appear to be meaningfully different. More specifically, mean food cravings were higher following alcohol odorant trials (*M* = 42.75, *SD* = 10.14) relative to neutral odorant trials (*M* = 39.50, *SD* = 9.61) and baseline (*M* = 37.25, *SD* = 5.70). However, mean food cravings following food odorant trials (*M* = 37.75, *SD* = 10.32) and baseline food cravings (*M* = 37.25, *SD* = 5.70) might not be meaningfully different. Moreover, mean food cravings were lower following food odorant trials (*M* = 37.75, *SD* = 10.32) relative to neutral odorant trials (*M* = 39.50, *SD* = 9.61). In general, the pattern of results indicate that exposure to alcohol odorants, but not food odorants, might be producing higher food cravings compared to neutral odorants and compared to baseline.

Attentional Bias

A repeated measures ANOVA indicated that the main effect of trials (neutral odors, beer odors, pizza odors exposure) on cognitive alcohol attentional bias was not significant, F (2, 12) = 0.08, p = 0.71. Examination of cognitive alcohol attentional bias across the three trials indicates that there might be meaningful differences in a larger sample. In particular, alcohol attentional bias was higher following alcohol odorant trials

(M = 10.72, SD = 35.53) and food odorant trials (M = 10.43, SD = 32.05) relative to neutral odorant trials (M = 4.47, SD = 29.73). In general, pattern of results indicate that exposure to both food odorants and alcohol odorants might be producing higher cognitive alcohol attentional bias compared to exposure to neutral odorants.

The main effect of trials on cognitive food attentional bias was not significant, F (2, 12) = 0.35, p = 0.72. Similarly, examination of cognitive food attentional bias suggests that there might be meaningful differences across the three trials. Food attentional bias was higher following alcohol odorant trials (M = 7.53, SD = 37.35) and food odorant trials (M = 13.69, SD = 27.71) relative to neutral odorant trials (M = -2.10, SD = 27.49). Pattern of results indicate that exposure to food odorants and alcohol odorants might be producing higher cognitive food attentional bias compared to exposure to neutral odorants.

Using a repeated measures ANOVA indicated, the main effect of trials (neutral, beer, pizza odors exposure) on duration alcohol attentional bias was significant, F (2, 12) = 6.99, p = 0.01. Using post-hoc LSD (least significant difference) test, duration alcohol attentional bias was higher following alcohol odorant trials (M = 0.15, SD = 0.24) relative to neutral odorant trials (M = 0.05, SD = 0.29), with the difference falling short of significance (p = 0.06). In contrast, duration alcohol attentional bias appeared to be lower following food odorant trials (M = 0.00, SD = 0.29) relative to neutral odorant trials (M = 0.05, SD = 0.29), with the difference also falling short of significance (p = 0.07). These findings suggest that alcohol odorants might be increasing duration alcohol attentional bias relative to neutral odorants, and that food odorants might be decreasing duration alcohol attentional bias relative to neutral odorants.

The main effect of trials on duration food attentional bias was not significant, F (2, 8) = 0.92, p = 0.44. However, examination of duration food attentional bias indicates possible meaningful differences across the three trials. In particular, duration food attentional bias was higher following alcohol odorant trials (M = 0.23, SD = 0.31) relative to neutral odorant trials (M = 0.11, SD = 0.30). In contrast, duration food attentional bias was lower following food odorant trials (M = 0.06, SD = 0.10) relative to neutral odorant trials (M = 0.11, SD = 0.30). This pattern of results suggest that exposure to alcohol odorants might be increasing duration food attentional bias relative to exposure to neutral odorants, and that exposure to food odorants might be decreasing duration food attentional bias relative to exposure to neutral odorants.

A repeated measures ANOVA indicated that the main effect of trials (neutral odors, beer odors, pizza odors) on direction alcohol attentional bias was not significant, F (2, 12) = 2.72, p = 0.11. Examination of direction alcohol attentional bias across the three trials indicates non-meaningful differences. More specifically, direction alcohol attentional bias did not seem meaningfully different following alcohol odorant trials (M = 56.04%, SD = 12.13%, Range = 40% - 78%) and food odorant trials (M = 52.60%, SD = 10.01%, Range = 35% - 70%) relative to neutral odorant trials (M = 50.47%, SD = 13.41%, Range = 27% - 69%). The main effect of odor exposure on direction food attentional bias was also not significant, F (2, 8) = 3.18, p = 0.10. Similarly, direction food attentional bias across the three trials indicates non-meaningful differences. In particular, direction food attentional bias following alcohol odorant trials (M = 60.13%, SD = 11.21%, Range = 47% - 75%) and food odorant trials (M = 49.74%, SD = 3.56%, Range = 44% - 55%) did not seem meaningfully different compared to direction food

attentional bias following neutral odorant trials (M = 57.18%, SD = 12.22%, Range = 45% - 78%, p = 0.19). In summary, exposure to food and alcohol odorants does not seem to be increasing direction food and alcohol attentional bias relative to exposure to neutral odorants.

Post-Lab Consumption

All participants completed the post-lab survey (19 out of 19). For post-lab alcohol consumption, participants had a mean of 2.26 (SD = 2.64). This indicates that, on average, participants consumed two to three drinks following their participation in the lab portion of the study. For post-lab food consumption, participants had a mean of 2.00 (SD = 0.45). This suggests that the average participants ate an average amount of food following their participation in the lab portion of the study, such that they are neither full nor hungry following their food consumption. Correlational analyses were conducted to examine the relationships of cravings and attentional bias with post-lab food and alcohol consumption.

Alcohol cravings and attentional bias (cognitive, duration, and direction) following food and alcohol odorant trials were not significantly associated with post-lab alcohol consumption ($p > 0.05$). However, findings suggest that there might still be meaningful associations. For instance, cognitive alcohol attentional bias following alcohol odorant trials might be associated with higher levels of post-lab alcohol consumption (r = 0.40); however, cognitive alcohol attentional bias following food odorant trials might not be meaningfully associated with post-lab alcohol consumption (r = -0.02). Furthermore, alcohol cravings following food odorant trials might be associated

with somewhat higher levels of post-lab alcohol consumption ($r = 0.25$); in contrast, alcohol cravings following alcohol odorant trials might be associated with somewhat lower levels of post-lab alcohol consumption ($r = -0.16$). In addition, duration alcohol attentional bias following food odorant trials ($r = 0.07$) and alcohol odorant trials ($r = 0.01$) might not be meaningfully associated with post-lab alcohol consumption. Finally, direction alcohol attentional bias following food odorant trials might be associated with somewhat higher levels of post-lab alcohol consumption ($r = 0.15$), while direction alcohol attentional bias following alcohol odorant trials might not be meaningfully associated with post-lab alcohol consumption ($r = 0.04$). In summary, increased cognitive attentional bias following alcohol odorant trials might be associated with higher post-lab alcohol consumption; however, other types of alcohol attentional bias (duration and direction) and alcohol cravings following alcohol odorant trials might not be associated with increased post-lab alcohol consumption. In contrast, alcohol cravings and direction alcohol attentional bias following food odorant trials might be associated with increased post-lab alcohol consumption; however, cognitive and duration alcohol attentional bias elicited by food odorant trials might not be associated with increased post-lab alcohol consumption.

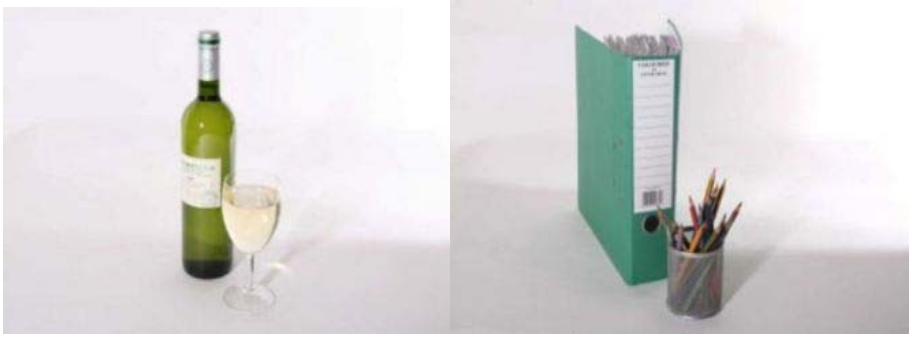
For post-lab food consumption, duration food attentional bias following alcohol odorant trials ($p = 0.001$) and food odorant trials ($p = 0.046$) were significantly associated with post-lab food consumption. In particular, duration food attentional bias following alcohol odorant trials ($r = -0.99$) and food odorant trials ($r =- 0.82$) were associated with lower levels of post-lab food consumption. Although food cravings, cognitive food attentional bias, and direction food attentional bias were not significantly associated with

post-lab food consumption ($p > 0.05$), findings suggest there might be meaningful associations. For instance, food cravings following alcohol odorant trials ($r = -0.38$) and food odorant trials ($r = -0.41$) might be associated with lower levels of post-lab food consumption. Similarly, cognitive food attentional bias following alcohol odorant trials ($r = -0.40$) and food odorant trials ($r = -0.62$) might also be associated with lower levels of post-lab food consumption. Finally, direction food attentional bias following alcohol odorant trials ($r = 0.03$) and food odorant trials ($r = 0.08$) might not be meaningfully associated with post-lab food consumption. In general, food cravings, duration food attentional bias, and cognitive food attentional bias, but not direction food attentional bias, might be decreasing post-lab food consumption.

Appendix F: Food and Matched Control Pictures

Appendix G: Alcohol and Matched Control Pictures







BELL'S
BELL'S
BELL'S

Appendix H: Results without Covariates

Table H1

Effects of Odor Cues on Key Study Variables (Repeated Measures ANOVA)

	Baseline		Trial 1		Trial 2		Trial 3		Trial 4		ANOVA	
	M	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**	**F-Stat**	**p-value**
Alcohol Cravings (L)	18.21	8.06	24.01	12.11	23.68	12.19	21.79	10.81	21.28	10.08	17.06	.00
Food Cravings (L)	33.55	8.72	34.44	10.93	36.65	10.37	34.92	10.17	35.75	9.97	6.36	.00
Alcohol Cognitive AB (ms)			1.84	54.75			2.27	43.62			.00	.96
Food Cognitive AB (ms)					-.18	45.93			-3.32	59.08	.15	.70
Alcohol Duration AB (ms)			.16	.24			.09	.21			6.20	.02
Food Duration AB (ms)					.19	.24			.08	.16	17.40	.00
Alcohol Direction AB (%)			55.42	8.16			51.87	9.29			1.55	.20
Food Direction AB (%)					55.50	8.62			53.62	7.35	2.39	.13
Alcohol Arousal (mm)			1.76	5.15			.90	5.66			.91	.34
Food Arousal (mm)					.98	4.96			-.14	4.19	1.94	.17

Note. AB-attentional bias. Arousal-sympathetic arousal. Parentheses: L-Likert scale; ms-millisecond; %-percent; mm-millimeter.

Table H2

Correlations of Key Study Variables with Post-Lab Consumption

	Alcohol Cravings (Alcohol)	Alcohol Cravings (Food)	Food Cravings (Alcohol)	Food Cravings Food)	Alcohol Cognitive AB (ms)	Food Cognitive AB (ms)	Alcohol Direction AB (%)	Food Direction AB (%)	Alcohol Duration AB (ms)	Food Duration AB (ms)	Alcohol Arousal (mm)	Food Arousal (mm)	*M (SD)*
Post-Lab Food Satiety	.07	.09	.19	.23	.06	-.04	.13	.26*	.02	.08	.00	.01	66.01 (21.44)
Post-Lab Food Typicality	.16	.20	.06	.11	.04	-.17	.17	.17	.15	.20	.21	.03	54.86 (19.55)
Post-Lab Alcohol Use	.21	.19	.18	.02	.29*	-.09	.10	-.26*	.12	-.19	.05	-.27*	2.49 (2.03)
Post-Lab Alcohol Typicality	.01	.01	-.12	-.13	.04	-.16	-.01	-.13	.04	-.16	-.06	-.17	39.65 (23.97)
Post-Lab Concurrent Consumption	-.18	-.16	-.18	-.14	-.09	.07	-.10	-.05	-.05	-.06	-.11	.10	1.58 (0.50)

Note. * indicates $p < 0.05$. Cravings: Alcohol-alcohol pictures; Food-food pictures. AB-attentional bias. Parentheses: L-Likert scale; ms-millisecond; %-percent; mm-millimeter. Likert scale used for cravings and post-lab consumption questions.

Appendix I: Imputations

Table I1

Effects of Cues on Alcohol Cravings across Imputations

	Baseline		Trial 1		Trial 2		Trial 3		Trial 4		ANOVA	
	M	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**	**M**	**SD**	**F-Stat**	**p-value**
Alcohol Cravings (Original)	18.28	8.10	24.18	12.12	23.88	12.16	21.93	10.84	21.40	10.10	2.72	0.03
Alcohol Cravings (1)	20.81	19.96	27.22	20.57	25.32	14.30	22.50	11.29	21.75	10.65	1.38	0.24
Alcohol Cravings (2)	17.70	11.52	24.81	13.73	23.70	13.69	22.50	11.29	21.75	10.65	0.41	0.80
Alcohol Cravings (3)	17.19	14.88	20.46	40.57	20.89	29.96	22.50	11.29	21.75	10.65	1.11	0.35
Alcohol Cravings (4)	18.89	8.33	25.86	15.15	25.31	14.15	22.50	11.29	21.75	10.65	1.50	0.20
Alcohol Cravings (5)	22.48	33.37	25.98	14.74	24.42	12.54	22.50	11.29	21.75	10.65	1.45	0.22

Note. Likert scale used for cravings questions.

Table I2

Effects of Food Cravings on Post-Lab Consumption across Imputations

	Food Satiety		Food Typicality		Alcohol Use		Alcohol Typicality		Co-consumption			
	β	p-value	*β*	p-value	*β*	p-value	*β*	p-value	Exp(b)	p-value	Mean	SD
Food Cravings (Original)	.00	.99	-.01	.98	.48	.04	.15	.53	.95	.33	34.33	10.89
Food Cravings (1)	-.02	.89	-.22	.20	.20	.23	-.01	.95	.95	.22	36.03	29.81
Food Cravings (2)	.08	.61	.16	.33	-.22	.16	-.01	.96	.99	.72	31.90	15.62
Food Cravings (3)	-.14	.58	.04	.88	.14	.54	.19	.39	1.00	.93	36.14	34.86
Food Cravings (4)	-.09	.63	-.30	.11	.17	.34	.03	.87	.97	.43	34.31	12.17
Food Cravings (5)	.06	.72	-.14	.39	-.10	.53	.03	.84	.96	.24	33.82	17.39

Note. Likert scale used for cravings and post-lab consumption questions.

VITA

VITA

Kenny A. Karyadi

Indiana University-Purdue University Indianapolis

Department of Psychology

Email: kkaryadi@iupui.edu

EDUCATION

2016 **Ph.D. in Clinical Psychology**

Indiana University-Purdue University Indianapolis

Dissertation title (defended June 2015): *The effects of alcohol odorants on food and alcohol attentional bias, cravings, and consumption.*

2013 **M.S. in Clinical Psychology**

Indiana University-Purdue University Indianapolis

Thesis title (defended February 2013): *The interactive effects of alcohol cravings, cue reactivity, and urgency on college student problematic drinking.*

2010 **B.S. in Psychology**, with Distinction

University of Washington

Honors thesis title (submitted June 2010): *The moderating role of negative emotions on urgency and alcohol engagement.*

GRANTS AND FELLOWSHIPS

2013-Present **Pre-doctoral Fellowship (T32 AA07462)**

National Institute on Alcohol Abuse and Alcoholism

Food and Alcohol Consumption Project

Principal Investigators: Melissa A. Cyders, Ph.D.; Cristine Czachowski, Ph.D.

Role: Pre-doctoral trainee

HONORS AND AWARDS

2012-2014 **RSA Student Merit Meeting Award,** Research Society on Alcoholism

2012-2013 **Travel Award,** Indiana University-Purdue University Indianapolis

2010 **Guthrie Prize for Best Empirical Paper,** University of Washington

2009-2010 **Psychology Honors Program,** University of Washington

2009-2010 **Psi Chi,** National Honors Society in Psychology

2007-2010 **Dean's List**, University of Washington

PROFESSIONAL MEMBERSHIPS

2014-Present **Indiana Psychological Association,** Student Member

2014-Present **National Academy of Neuropsychology,** Student Member

2012- Present **Research Society on Alcoholism,** Student Member

CLINICAL INTERESTS

Neuropsychological and psychological assessments of individuals diagnosed with neuropathological, neuropsychological, psychological, and medical disorders.

CLINICAL EXPERIENCES

2014 **Practicum Student,** *Community South Bariatric Center,* Indianapolis, IN.

Supervisor: Theresa Rader, Psy.D.

Duration and Hours: 220 hours over 4 months

- Administered psychodiagnostic assessments to morbidly obese patients (27 total) who were seeking bariatric procedure.
- Conducted psychodiagnostic interviews with morbidly obese patients who were seeking bariatric procedure.
- Scored and interpreted the results of psychological assessments.
- Wrote integrated reports (18 total) based on assessment results, psychodiagnostic interview, and behavioral observation.

2014 **Practicum Student,** *Midtown Base Clinic,* Indianapolis, IN.

Supervisor: Joan Farrell, Ph.D.

Duration and Hours: 303 hours over 5 months

- Co-led group schema therapy sessions with clients (25 total) diagnosed with borderline personality disorder.
- Conducted individual schema therapy sessions with a client diagnosed with borderline personality disorder.

- Conducted psychodiagnostic assessments with clients (5 total) diagnosed with borderline personality disorder.

2013 **Practicum Student,** *Larue D. Carter Memorial Hospital,* Indianapolis, IN.

Supervisor: Jennifer L. Vohs, Ph.D.

Duration and Hours: 206 hours over 4 months

- Conducted psychoeducational group therapy with patients (24 total) diagnosed with severe mental illness (e.g. schizophrenia, schizoaffective disorder, personality disorders).
- Conducted individual psychotherapy with patients (4 total) diagnosed with severe mental illness (e.g. schizophrenia, schizoaffective disorder, personality disorders).
- Engaged in milieu therapy with patients (24 total) diagnosed with severe mental illness (e.g. schizophrenia, schizoaffective disorder, personality disorders).

2012-2013 **Practicum Student,** *IU Health Neuroscience Center,* Indianapolis, IN.

Supervisor: Daniel Rexroth, Psy.D.

Duration and Hours: 434 hours over 12 months

- Neuropsychological and psychodiagnostic assessments of patients (50 total) presenting with neurological problems and/or psychiatric problems.
- Scored neuropsychological and psychodiagnostic assessments.
- Wrote multiple integrated reports (15 total) based on information from

assessment results, clinical interview, and behavioral observation.

2007-2011 **Patient Care Facilitator**, *Schick Shadel Hospital,* Seattle, WA.

Program Director: Jerome F. Walters, M. Div, CDP.

Supervisor: Paula Fisher, MBA, CDP.

- Conducted structured interviews with substance dependent patients who were undergoing conscious sedation.
- Conducted psychodiagnostic interviews with substance dependent patients who were undergoing detoxification.
- Conducted orientation interviews with patients diagnosed with substance dependence.
- Conducted phone interviews with patients in recovery from substance dependence.
- Prepared materials for clinical workshops and interviews.

SUPERVISION EXPERIENCES

2014 Provided clinical supervision to an early stage graduate-level student during her practicum placement at a neuropsychology clinic.

2014 Provided clinical supervision to an early stage graduate-level student during her practicum placement at an inpatient psychiatric hospital.

SELECTED CLINICAL AND RESEARCH WORKSHOPS AND TRAINING

2014 *Neuropsychophysiology* (Online Course)

Gerald Kozlowski, Ph.D., Teaching Faculty, Saybrook University

2014 *Biofeedback Workshop*

Eric Scott, Ph.D., Assistant Professor, Indiana University

2014 *Mediation, Moderation, and Conditional Process Analysis Workshop*

Andrew F. Hayes, Ph.D., Professor, Ohio State University

2013 *Introduction to Meta-analysis Workshop*

Noel Card, Ph.D., Associate Professor, University of Arizona

2013 *Self-Hypnosis for Chronic Pain Management Workshop*

Mark P. Jensen, Ph.D., Associate Professor, University of Washington

2013 *Writing from the Reader's Perspective*

George D. Gopen, PhD., Professor Emeritus, Duke University

2013 *Grant Writers' Seminars and Workshops*

David C. Morrison, Ph.D., Professor, University of Missouri Kansas City

2012-2014 *Meta-supervision*

Attended monthly supervision meetings with students enrolled in practicum and a licensed clinical psychologist. Received supervision on clinical work and reported intervention progress. Received feedback on a transcribed therapy session. Received didactics and supervision on peer supervision.

2012-Present *Workshops at Annual Research Society on Alcoholism meetings.*
Relevant topics include: intervention and prevention approaches for problematic alcohol use, mechanisms underlying problematic alcohol use behaviors, grant writing, research writing, and professional development.

2011-Present *Proseminar on Professional Issues in Clinical Psychology*
Weekly professional development course covering advanced clinical topics, such as case conference/case conceptualization and clinical practice issues. Relevant topics include: supervision, consulting, diversity, ethics, professionalism, teaching, research methods, licensure, grant writing, and research writing.

SELECTED TESTS ADMINISTERED

Neuropsychological Assessments	**Personality and Other Assessments**
Boston Naming Test	BPD Severity Index
Category Fluency Test	Minnesota Multiphasic Personality
Controlled Oral Word Association Test	Inventory-II and -RF
Grip Strength Test	Rorschach Test
Grooved Pegboard Test	Thematic Apperception Test
Hooper Visual Organization Test	Addiction Severity Index
Judgment of Lines	EASY Diagnostic Survey
Mini-mental State Examination	Millon Clinical Multiaxial Inventory-III
National Alzheimer's Coordinating Center's FTLD Module	Semi-Structured Assessment for the Genetics of Alcoholism

Rey Auditory Verbal Learning Test

Rey-Osterrieth Complex Figure

Stroop Color and Word Test

Test of Memory Malingering

Trail Making Test

Wechsler Adult Intelligence Scale-III & -IV

Wechsler Intelligence Scale for Children-IV

Wechsler Memory Scale-Revised

Wechsler Test of Adult Reading

Wisconsin Card Sorting Test

Structured Clinical Interview for DSM-IV

Weight and Lifestyle Inventory

RESEARCH INTERESTS

Dispositional (e.g. mindfulness and impulsivity), neurocognitive (e.g. cue reactivity and attentional bias), and neurobiological (e.g. brain functioning) mechanisms underlying substance use behaviors.

PEER-REVIEWED MANUSCRIPTS

1. **Karyadi, K. A.,** & Cyders, M. A. (*in press).* Elucidating the association between trait mindfulness and alcohol use behaviors among college students. *Mindfulness.* doi:10.1007/s12671-015-0386-7

2. Cyders, M. A., Dzemidzic, M., Eiler, W. J., Coskunpinar, A., **Karyadi, K. A.**, & Kareken, D. A. (*in press*). Negative urgency mediates the relationship between amygdala and orbitofrontal cortex activation to negative emotional stimuli and general risk-taking. *Cerebral Cortex.* doi:10.1093/cercor/bhu123
3. **Karyadi, K. A.,** Vanderveen, J. D., & Cyders, M. A. (2014). A meta-analysis of the relationship between mindfulness and substance use behaviors. *Drug and Alcohol Dependence, 143,* 1-10. doi:10.1016/j.drugalcdep.2014.07.014
4. Cyders, M. A., Littlefield, A. K., Coffey, S., & **Karyadi, K. A.** (2014). Examination of a short version of the UPPS-P Impulsive Behavior Scale. *Addictive Behaviors, 39,* 1372-1376. doi:10.1016/j.addbeh.2014.02.013
5. Cyders, M. A., Dzemidzic, M., Eiler, W. J., Coskunpinar, A., **Karyadi, K. A.**, & Kareken, D. A. (2013). Negative urgency and ventromedial prefrontal cortex responses to alcohol cues: fMRI evidence of emotion-based impulsivity. *Alcoholism: Clinical and Experimental Research, 38,* 409-417. doi:10.1111/acer.12266
6. Coskunpinar, A., Dir, A. L., **Karyadi, K. A**., Koo, C., & Cyders, M. A. (2013). Mechanisms underlying the relationship between negative affectivity and problematic alcohol use. *Journal of Experimental Psychology, 4,* 263-278. doi:10.5127/jep.029612
7. Dir, A., **Karyadi, K. A.,** & Cyders, M. A. (2013). The uniqueness of negative urgency as a common risk factor for self-harm behaviors, alcohol consumption, and eating problems. *Addictive Behaviors, 38,* 2158-2162. doi:10.1016/j.addbeh.2013.01.025.

8. **Karyadi, K. A.,** Coskunpinar, A., Dir, A., & Cyders, M. A. (2013). The interactive effects of affect lability, urgency, and sensation seeking on young adult problematic drinking. *Journal of Addiction, 2013,* 1-7. doi:10.1155/2013/636854
9. Luk, J. W., Emery, R. L., **Karyadi, K. A.,** Patock-Peckham, J. A., & King, K. M. (2012). Religiosity and substance use among Asian American college students: Moderated effects of race and acculturation. *Drug and Alcohol Dependence, 130,* 142-149. doi:10.1016/j.drugalcdep.2012.10.023
10. King, K. M., **Karyadi, K. A.**, Luk, J. W., & Patock-Peckham, J. A. (2011). Dispositions to rash action moderate the association between concurrent drinking, depressive symptoms, and alcohol problems during emerging adulthood. *Psychology of Addictive Behaviors, 25,* 446-454. doi:10.1037/a0023777
11. **Karyadi, K. A.**, & King, K. M. (2011). Urgency and negative emotions: Evidence for moderation on negative alcohol consequences. *Personality and Individual Differences, 51,* 635-640. doi: 10.1016/j.paid.2011.05.030

PUBLICATIONS UNDER REVIEW

1. Fossati, A., Somma, A., **Karyadi, K. A.**, Cyders, M. A., & Bortolla, R. (under review). Reliability and validity of the Italian translation of the UPPS-P Impulsive Behavior Scale in a sample of consecutively admitted psychotherapy patients.
2. Hershberger, A. R., **Karyadi, K. A.**, Vanderveen, J. D., & Cyders, M. A. (under review). Assessing positive comparative expectancies of e-cigarettes to cigarettes using CECE-Q.

3. Hershberger, A. R., **Karyadi, K. A.**, Vanderveen, J. D., & Cyders, M. A. (under review). Direct comparisons of e-cigarettes and cigarettes.
4. Hershberger, A. R., **Karyadi, K. A.**, Vanderveen, J. D., & Cyders, M. A. (under review). U.S. smoke-free policies that do not include e-cigarettes are not effective in reducing e-cigarette use and alcohol consumption.

CHAPTERS

1. **Karyadi, K. A.,** Coskunpinar, A., Entezari, A., Long, C., & Cyders, M. A. (2013). Mechanisms underlying the co-prevalence between problematic eating and drinking. In S. B. Harris (Ed.), *Binge Eating and Binge Drinking: Psychological, Social, and Medical Implications* (pp. 97-126). New York: Nova Science Publisher, Inc.
2. **Karyadi, K. A.,** Coskunpinar, A., & Cyders, M. A. (2012). Neurobiological underpinnings of dispositions to rash action. In M. A. Cyders (Ed.), *Psychology of Impulsivity* (pp. 95-110). New York: Nova Science Publisher, Inc.

PRESENTATIONS

1. Entezari, A. B., **Karyadi, K. A.**, & Cyders, M. A. (2015). *Role of positive and negative urgency and social context on problematic alcohol use behaviors.* Poster presented at IUPUI Research Day, Indianapolis, IN.
2. Hershberger, A., **Karyadi, K. A.**, & Cyders, M. A. (2015). *E-Cigarettes: A novel measure for the expectancies of e-cigarette use as directly compared to cigarette use.* Poster presented at IUPUI Research Day, Indianapolis, IN.

3. **Karyadi, K. A.,** Vanderveen, J. D., & Cyders, M. A. (2014). *A meta-analysis of the relationship between mindfulness and substance use behaviors.* Poster presented at the 37th Annual Scientific Meeting of the Research Society on Alcoholism, Seattle, WA.
4. **Karyadi, K. A.,** & Cyders, M. A. (2014). *The effects of food and alcohol odors on attentional biases and cravings: A pilot study.* Poster presented at the 37th Annual Scientific Meeting of the Research Society on Alcoholism, Seattle, WA.
5. **Karyadi, K. A.,** & Cyders, M. A. (2014). *The interactive effects of alcohol cravings, cue reactivity, and urgency on college student problematic drinking.* Poster presented at IUPUI Research Day, Indianapolis, IN.
6. **Karyadi, K. A.,** & Cyders, M. A. (2013). *A meta-analysis of the relationship between mindfulness and substance use behaviors.* Poster presented at the 2013 Indiana Psychology Association conference, Indianapolis, IN.
7. **Karyadi, K. A.,** & Cyders, M. A. (2013). *The interactive effects of alcohol cravings, cue reactivity, and urgency on college student problematic drinking.* Poster presented at the 36th Annual Scientific Meeting of the Research Society on Alcoholism, Orlando, FL.
8. Dir, A. L., **Karyadi, K. A.**, Cyders, M. A. (2013). *The uniqueness of negative urgency as a common risk factor for alcohol consumption, self-harm behaviors, and eating problems in college students.* Poster presented at IUPUI Research Day, Indianapolis, IN.

9. Coskunpinar, A., Dir, A. L., **Karyadi, K. A.**, Koo, C., & Cyders, M. A. (2013). *Mechanisms underlying the relationship between negative affectivity and problematic alcohol use.* Poster presented at IUPUI Research Day, Indianapolis, IN.
10. **Karyadi, K. A.,** Dir, A., & Cyders, M. A. (2013). *The interaction between sensation seeking and negative affect lability on alcohol involvement.* Poster presented at IUPUI Research Day, Indianapolis, IN.
11. **Karyadi, K. A.,** Dir, A., & Cyders, M. A. (2012). *The interaction between sensation seeking and negative affect lability on alcohol involvement.* Poster presented at the 35th Annual Scientific Meeting of the Research Society on Alcoholism, San Francisco, CA.
12. **Karyadi, K. A.,** Coskunpinar, A., & Cyders, M. A. (2011). *The neural correlates of emotion regulation and urgency.* Poster presented at IUPUI Research Day, Indianapolis, IN.
13. **Karyadi, K. A.,** Coskunpinar, A., & Cyders, M. A. (2011). *The neural correlates of emotion regulation and urgency.* Poster presented at the Indianapolis Society for Neuroscience Annual meeting, Indianapolis, IN.
14. Luk, J. W., Emery, R. L., **Karyadi, K. A.**, Patock-Peckham, J. A., & King, K. M. (2011). *Does acculturation moderate the effects of religiosity on substance use and problems in Asian American college students?* Poster presented at the 34th Annual Scientific Meeting of the Research Society on Alcoholism, Atlanta, GA.

15. Elkins, R. L., Hobbs, S. H., Edwards, G. L., Carl, G. F., & **Karyadi, K. A.** (2011). *Ethanol induced expression differences in heat shock protein 27 in ventral striatum of taste aversion-prone and taste aversion-resistant rats.* Poster presented at the 34th Annual Scientific Meeting of the Research Society on Alcoholism, Atlanta, GA.
16. Elkins, R. L., Dandala, K. R., Oksendahl, J. E., **Karyadi, K. A.**, & Whitford, J. L. (2011). *Age related enrollment and abstinence outcomes of inpatient counter conditioning combined with other alcohol use disorder treatments.* Poster presented at the 34th Annual Scientific Meeting of the Research Society on Alcoholism, Atlanta, GA.
17. King, K. M., **Karyadi, K. A.**, Luk, J. W., Patock-Peckham, J. A. (2010). *Depressive and anxiety symptoms moderate the impact of motivational and cognitive impulsiveness on alcohol use and problems.* Poster presented at the 33th Annual Scientific Meeting of the Research Society on Alcoholism, San Antonio, TX.
18. **Karyadi, K. A.**, & King, K. M. (2010). *The interaction between urgency and negative emotions on alcohol engagement.* Poster presented at the University of Washington's Undergraduate Research Symposium, Seattle, WA.
19. **Karyadi, K. A**., & King, K. M. (2010). *The interaction between urgency and negative emotions on alcohol engagement.* Poster presented at the at the University of Washington's Psychology Honors Poster Session, Seattle, WA.

RESEARCH EXPERIENCE

2013-Present **Dissertation Project,** *Food and Alcohol Consumption Project,* IUPUI, Indianapolis, IN.

Graduate Investigator: Kenny A. Karyadi, M.S.

Graduate Investigator's Grant Information: T32 AA07462

Supervisor: Melissa A. Cyders, Ph.D.

- Developed and implemented an original research project that examined the effects of alcohol odorants on food and alcohol cravings, attentional bias, and consumption.
- Submitted a human subjects review application.
- Conducted an extensive review of the literature on food and alcohol attentional bias and cravings.
- Conducted an online survey study to determine the most frequently consumed alcoholic beverage and food.
- Prepared E-prime and DASYLab syntaxes for visual probe task and odor administration.
- Prepared eye tracking instruments for collecting attentional bias data.
- Set up olfactometer instruments for administering alcohol odorants.
- Prepared online surveys to collect lab data and post-lab self-report data.
- Interacted with human participants for data collection.

2013-2014 **Preliminary Examination,** *Mindfulness and Substance Use,* IUPUI, Indianapolis, IN.

Graduate Investigator: Kenny A. Karyadi, M.S.

Supervisor: Melissa A. Cyders, Ph.D.

- Completed a meta-analytic review of the relationship between trait mindfulness and substance use behaviors.
- Created a codebook for coding relevant research articles.
- Conducted an extensive review of the literature on the relationship between trait mindfulness and substance use behaviors.
- Conducted literature searches of multiple electronic databases for relevant research articles.
- Coded approximately 40 research articles for the meta-analytic analyses.
- Conducted meta-analytic analyses of coded research articles using SPSS and macros.

2012-2014 **Master's Thesis**, *College Student Health and Personality Survey*, IUPUI, Indianapolis, IN.

Supervisor: Melissa A. Cyders, Ph.D.

Graduate Investigator: Kenny A. Karyadi, M.S.

- Developed and implemented an original research project that examined the interactive effects of alcohol cravings, cue reactivity, and urgency on problematic alcohol use.
- Conducted an extensive review of the literature on cue reactivity,

alcohol cravings, urgency, and problematic alcohol use.

- Submitted a human subjects review application.
- Interacted with human subjects during data collection.
- Utilized SPSS for the management and analyses of a large dataset.

2012-2013 **Graduate Research Assistant,** *Brain Functioning with External Stimuli,* IUPUI, Indianapolis, IN.

Principal Investigator: Melissa A. Cyders, Ph.D.

PI's Grant Information: K01 AA020102

- Conducted phone and in-person interviews for the purpose of recruiting subjects.
- Aided in running neuroimaging sessions.
- Compiled neuroimaging-related data sets.

2011-2012 **Graduate Research Assistant,** *Computer Performance and Behavior,* IUPUI, Indianapolis, IN.

Principal Investigator: Melissa A. Cyders, Ph.D.

PI's Grant Information: HRSA-10-175; D76HP20905

- Interacted with human subjects during data collection.
- Assisted in setting up olfactometer instruments for alcohol odorant administration.
- Assisted in setting up the eye tracker for collecting attentional bias data.
- Completed E-prime syntaxes for mood manipulations, visual probe task, and odor administration.

- Utilized SPSS for the management and analyses of a large dataset.

2010-2011 **Research Assistant,** *Counter Conditioning Research,* Schick Shadel Hospital, Seattle, WA.

Principal Investigator: Ralph Elkins, Ph.D.

- Assisted in revising a research compendium.
- Assisted in applying the CONSORT model to a manuscript.
- Wrote a treatment efficacy summary.
- Assisted in writing a research protocol, a research proposal, and a human subjects review application for a neuroimaging study.
- Collected and compiled longitudinal data on counter conditioning patients.

2010-2011 **Undergraduate Research Assistant,** *Asian Student Health Survey,* University of Washington, Seattle, WA.

Graduate Investigator: Jeremy W. Luk, B.S., B.A.

Supervisor: Kevin M. King, Ph.D.

- Assisted in creating a codebook of measures.
- Provided technical support for online data collection.
- Utilized SPSS for the management and analyses of a large dataset.

2009-2010 **Undergraduate Honors Thesis**, *College Student Alcohol Use Survey,* University of Washington, Seattle, WA.

Undergraduate Investigator: Kenny A. Karyadi

Supervisor: Kevin M. King, Ph.D.

- Developed and implemented an original research project that examined the interaction between negative emotions and urgency on problematic alcohol use.
- Conducted an extensive literature review on negative emotions, urgency, and alcohol use behaviors.
- Submitted a human subjects review application.
- Utilized an online survey for data collection.
- Utilized SPSS for the management and analyses of a large dataset.

2009-2010 **Honors Research Assistant**, *College Student Health and Behaviors Survey*, University of Washington, Seattle, WA

Supervisor: Kevin M. King, Ph.D.

- Created codebooks of measures.
- Gained fluency in conducting extensive literature reviews.
- Utilized SPSS for the management and analyses of several large datasets.
- Aided in data collection and interacted with human subjects during data collection.
- Assisted with the submission of human subjects review applications.
- Played a key role in setting up and launching research projects.
- Played a key role in laboratory management.

TEACHING INTERESTS

Clinical psychology, neuropsychology, cognitive psychology, substance use and abuse, and research methods in psychology.

TEACHING EXPERIENCES

2013 **Graduate Teaching Assistant,** *Psychology B370: Social Psychology.*

Instructor: Ayca Coskunpinar, M.S.

- Graded lab assignments and final paper.
- Prepared class-related materials.

2011-2013 **Graduate Teaching Assistant,** *Psychology B311: Research Methods in Psychology*

Instructors: Robert Stewart, Ph.D.; Milena Petrovic, Ph.D.

- Taught two laboratory sections per week (two and a half hours per week; each section with 15-25 students).
- Held flexible office hours.
- Prepared teaching materials for laboratory sections.
- Graded lab assignments and research papers.

2012 **Graduate Teaching Assistant,** *Psychology B110: Introduction to Psychology.*

Instructors: Melina Petrovic, Ph.D.; Shenan Kroupa, Ph.D.; Sarah Altman, M.A.

- Prepared teaching materials for class and laboratory sections.
- Graded lab assignments and final class paper.

REFERENCES

Melissa A. Cyders, Ph.D., HSPP
Assistant Professor
Department of Psychology, IUPUI
Telephone: (317) 274-6752
Email: mcyders@iupui.edu

Theresa Rader, Psy.D., HSPP, LCAC
Bariatric Psychologist
Community South Bariatric Center
Telephone: (317) 460-9455
Email: TRaderWilson@ecommunity.com

Daniel Rexroth, Psy.D.
Assistant Professor
Indiana University School of Medicine
Telephone: (317) 963-7301
Email: drexroth@iupui.edu

Joan Farrell, Ph.D.
Research and Training Director
Midtown Community Mental Health Center
Telephone: (317) 283-3623
Email: ijinindy@sbcglobal.net